Studies of Hemodynamic Changes in Humans Following Induction of Low and High Spinal Anesthesia


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After a lapse of some years, surgeons and anesthesiologists alike are re-exploring the use of high spinal anesthesia, which is designed to produce a “useful” hypotension coupled with maximal relaxation. In the present study the hemodynamic changes induced by low and high spinal anesthesia, the latter arbitrarily defined as ablation of sympathetic, sensory and somatic nerves fibers above the fourth dermatomic segment, have been investigated in a series of waking patients not undergoing surgery.

Since the introduction of spinal anesthesia 54 years ago, no method of anesthesia has had a more controversial course. Some investigators have found it applicable to surgery on any part of the body, while others place strict limitations upon the level of anesthesia for which it is safe, and also restrict its use to carefully selected patients. Surgeons and anesthesiologists alike have objected to the hypotension which often accompanies this type of anesthesia, especially when high levels of anesthesia are reached. In recent years, however, this very property of high spinal anesthesia has been offered as an advantage in that it provides the surgeon with excellent relaxation of the patients and permits an operation with virtually no blood loss. Since it seemed apparent that this technic would gain in popularity, it was thought advisable to evaluate the cardiovascular effects of hypotension induced by its employment in patients undergoing surgery and in others not undergoing surgical intervention.

This report deals with the hemodynamic and blood oxygen content changes in 10 patients, five of whom were given low and five high spinal anesthesia, and in whom no surgical procedure was performed.

Material and Methods

Ten hospitalized patients were studied, six males and four females. The average age was 51.4 years. At first patients were excluded who showed evidence of cardiovascular disease on the basis of history, physical examination and electrocardiography. Subsequently, some subjects were included who showed evidence of mild heart disease or of healed cerebrovascular accidents. The majority of patients had varying degrees of anemia and pulmonary emphysema. The lowest hemoglobin level encountered was 8.8 Gm. per 100 cc. None had azotemia and the majority had normal bromsulfalein retention.

The conventional 12 lead electrocardiogram was recorded pre-operatively, during the spinal anesthetic, on the same afternoon as the investigation, and daily thereafter for seven days, when possible.
in all patients. Preocardial leads were not obtained during the period of anesthesia. All patients had a regular sinus rhythm.

On the evening before the tests, the patients received 1.0 Gm. of chloral hydrate orally. On the morning of the examination, 0.1 to 0.2 Gm. of sodium pentobarbital was administered orally. The patients were then transferred to the cardiovascular laboratory which was maintained at a temperature of 23 to 24 C.

The right side of the heart was catheterized with the standard Courand type single lumen 7F catheter. The latter was introduced and kept in the pulmonary artery for the duration of the studies. The “zero” point for reference was taken to be 10 cm. from the posterior surface of the thorax. The peripheral arterial pressure was recorded through a number 19 indwelling intra-arterial needle introduced into the brachial artery of the same arm used for the cardiac catheterization. Pressures were transduced through Statham strain gauges and recorded by the Brush multichannel oscillograph.45

The baseline cardiac outputs were determined in duplicate according to the direct Fick principle, after a rest period of 30 minutes following introduction of the cardiac catheter and the intra-arterial needle. The volumes of expired air were measured through a gas test meter. Five minute samples were collected in Douglas bags. The oxygen content of room and expired air was measured on the Pauling oxygen analyzer after passage through a drier. All values were corrected to 0 C. and 760 mm. of Hg. The hemato-crit levels of simultaneously drawn and heparinized specimens of brachial and pulmonary arterial blood were determined by the Wintrobe method.46 Blood oxygen contents were determined by means of a previously standardized Beckman DU spectrophotometer.46 The blood samples were obtained simultaneously from the pulmonary and brachial arteries. The specimens were collected by means of heparinized Luer-Lock syringes air-sealed with Aquaresin,* and immediately placed on ice. All determinations were done in duplicate. As an added control, direct arteriovenous oxygen differences were determined for simultaneously drawn samples of blood.

Mean auricular, pulmonary and brachial arterial pressures were calculated by the planimetric integration of the area under the recorded pressure curves over a period of two successive respiratory cycles. The total peripheral resistance was calculated according to the standard dyne formula:

\[
T.P.R. = \frac{\text{Mean systemic blood pressure}}{\text{Cardiac Output}} \times 1332,
\]

where pressure is expressed in millimeters of Hg, cardiac output in cubic centimeters per second, and

\[
1332 \text{ is the conversion factor to reduce T.P.R. to dynes per second per cm.}^{-2}, \text{ in terms of absolute units.}
\]

Left ventricular work was calculated according to the formula:

\[
L.V.W. = 0.0135 \times \text{cardiac output} \times \text{mean systemic blood pressure},
\]

wherein the cardiac output is expressed in liters per minute, the mean arterial blood pressure in millimeters of Hg, and left ventricular work in kilogram meters per minute. The existence of basal cardiac outputs within the normal range and the directional changes in the brachial arterial pressures and in the cardiac outputs to lower levels, validates this estimation in the face of the inability to account for the kinetic factor in the formula:

\[
L.V.W. = (0.0135 \times C.O. \times B.P.) + \frac{(mV)^2}{2}.
\]

Peripheral blood flows and pulse volumes were measured by means of the Grass oscillographic plethysmograph,* employing air transmission through Grass and Statham strain gauges. The finger oncometer was placed on the index finger of the noncatheterized extremity, and simultaneous pulse volumes were determined on the second toe of either foot. The oncometers were sealed with Kal-Kord to the digits. When blood flows were estimated, an occlusion cuff was placed at the base of the digit and venous occlusion obtained by inflating the cuff from a pressure tank. The blood flows were expressed in cubic centimeters per 10 cc. of digit per minute and the pulse volumes in cubic centimeters per 10 cc. of digit.

After the control data had been obtained, the effects of spinal anesthesia were studied. Low spinal anesthesia, arbitrarily determined to be below the level of T-4, was induced by injecting 75 to 175 mg. of procaine slowly into the subarachnoid space at the level of the third or fourth lumbar interspace. High spinal anesthesia, determined to be above the level of T-4, was attained by injection of 150 to 175 mg. of procaine into the subarachnoid space, rapidly and with barbotage. In all cases of high spinal anesthesia there was a release of vasomotor tone in the finger, as against a vasoconstriction in low spinal anesthesia. In all instances a period of 15 minutes was arbitrarily taken as the time necessary to ensure proper fixation of the anesthetic. Vasoconstrictor drugs were not used with either low or high spinal anesthesia, despite the development of hypotension.

The cardiac output and the pressures in the brachial and pulmonary arteries were recorded 30 and 60 minutes after the administration of the spinal anesthetic. Right auricular pressures were recorded

* Glyco Products Co., Inc., 26 Court St., Brooklyn, N. Y.

again at the end of the 60 minute period when the
cardiac catheter was withdrawn.

The results to be reported below will be confined
to the general hemodynamic and blood oxygen
changes developing during low and high spinal
anesthesia. The results of individual organ studies
will be presented in later communications.

**Results**

1. *Mean Brachial Arterial Pressure.* The
mean brachial arterial pressure fell significantly
in both groups of patients. The low spinal
group showed an average reduction of 21
per cent from the basal level, and the high
spinal group a reduction of 44 per cent. The
systolic and diastolic pressures dropped
proportionately, and when the former decreased
to circa 90 mm of Hg in normotensive patients,
the pulse pressure was definitely diminished.

2. *Cardiac Output and Index.* These
decreased in a manner similar to the
blood pressure. In the low spinal group the maximal
average reduction in the cardiac output was
16.2 per cent, and in the high spinal group it
was 31 per cent from the basal level. The
cardiac indices followed closely. Although the
reduction in the blood pressure and the cardiac
index showed a gross relationship, the trends
of the two were not always parallel (fig 1).
In one patient (G. T.) who was given a low
spinal anesthetic, there was no significant
change in the cardiac output. This was the
only patient who showed a minimal rise in
blood pressure. In one patient who was given
a high spinal anesthetic (F. D.) there was a
transient fall and then a terminal rise in the
cardiac output in spite of a profound decrease
in arterial pressure. This remains unexplained.

3. *Heart Rate and Stroke Volume.* The heart
rate decreased in all patients with the exception
of one who experienced a small and transient
rise (P. P.). There was an average maximal
decrease of 8 beats in the low, and 10.8 beats
in the high spinal group. The heart rate
uniformly falls in the basal conscious patient
under spinal anesthesia, following stabilization,
in the absence of physical or psychic stimula-
tion, and when vasoconstrictor drugs are not
employed. The changes in the stroke volume
reflected those of the cardiac output. In the
low spinal group the stroke volume showed a
small average decrease from 73.1 cc. to 68.0 cc.,

![Fig. 1. Low and high spinal anesthesia. Average changes in cardiac index, brachial arterial mean pressure, pulmonary arterial mean pressure and right auricular mean pressure. Values for control, 30 and 60 minute determinations.](http://circ.ahajournals.org/)

whereas in the high spinal group the reduction
was more marked (57.0 to 43.5 cc.).
<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Anesthetic &amp; Level</th>
<th>Brachial Arterial Pressure (mm. of Hg)</th>
<th>Right Auricular Pressure (mm. of Hg)</th>
<th>Pulmonary Arterial Pressure (mm. of Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Control 30 min. 60 min.</td>
<td>Control 60 min.</td>
<td>Control 30 min. 60 min.</td>
</tr>
<tr>
<td>L. S.</td>
<td>46</td>
<td>M</td>
<td>Diabetes</td>
<td>Procaaine 100 T-4</td>
<td>121/67  98.5/51 98.8/48</td>
<td>6.4/2.3  4.5/0</td>
<td>25.1/8.7  21.4/7.2</td>
</tr>
<tr>
<td>N. W.</td>
<td>56</td>
<td>F</td>
<td>Coronary H.D. hyperthyroidism</td>
<td>Procaaine 100 T-6</td>
<td>153/60  138/52 155/70</td>
<td>4.55  3.42</td>
<td>17.3  14.9</td>
</tr>
<tr>
<td>G. T.</td>
<td>46</td>
<td>M</td>
<td>Hepatitis</td>
<td>Procaaine 75 T-11</td>
<td>120/66  126/72 132/78</td>
<td>19.0  12.3</td>
<td>3.7/1.1  19.6/8.2</td>
</tr>
<tr>
<td>P. P.</td>
<td>45</td>
<td>M</td>
<td>Chronic alcoholism</td>
<td>Procaaine 175 T-4</td>
<td>136/84  97/64 101/64</td>
<td>1.94  1.08</td>
<td>13.9  12.4</td>
</tr>
<tr>
<td>T. D.</td>
<td>75</td>
<td>M</td>
<td>Bronchogenic carcinoma</td>
<td>Procaaine 100 T-4 and 5</td>
<td>177/90  90/52.2 80.4/46</td>
<td>6.2/0.5  1.7/1.0</td>
<td>24.8/11.3  20.6/8.7</td>
</tr>
</tbody>
</table>

Averages...... 53.6
Percentile Change. .... -21.0  -17.6  -36.3  -17.4  -8.7

<table>
<thead>
<tr>
<th>Finger Blood Flow (cc./10 cc. part/min.)</th>
<th>Finger Pulse Volume (cc./10 cc. part)</th>
<th>Toe Pulse Volume (cc./10 cc. part)</th>
<th>Cardiac Output (cc./min.)</th>
<th>Stroke Volume (cc.)</th>
<th>Pulse Rate (cc.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
</tr>
<tr>
<td>0.63 0.24 0.21</td>
<td>.0054 0.0020 0.0025</td>
<td>.0007 0.0026 0.0018</td>
<td>7040 6213 6090</td>
<td>83.7 75.7 78.0</td>
<td>83 82 78</td>
</tr>
<tr>
<td>2.17 1.25 0.22</td>
<td>.0140 0.0086 0.0033</td>
<td>.0003 0.0017 0.0002</td>
<td>4627 4911 4603</td>
<td>60.8 72.1 65.7</td>
<td>76 68 70</td>
</tr>
<tr>
<td>0.15 0.10 0.12</td>
<td>.0018 0.0010 0.0013</td>
<td>.0003 0.0030 0.0012</td>
<td>6308 6353 5833</td>
<td>76.0 77.4 71.1</td>
<td>83 82 82</td>
</tr>
<tr>
<td>2.16 1.62 1.44</td>
<td>.0083 0.0060 0.0055</td>
<td>.0006 0.0068 0.0066</td>
<td>5773 4456 3679</td>
<td>83.0 50.4 55.0</td>
<td>67 74 64</td>
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<tr>
<td>1.28 0.80 0.50</td>
<td>.0065 0.0039 0.0025</td>
<td>.0018 0.0052 0.0052</td>
<td>5669 5136 4746</td>
<td>70.0 71.1 68.0</td>
<td>77 72 69</td>
</tr>
<tr>
<td>0.63 0.24 0.21</td>
<td>.0054 0.0020 0.0025</td>
<td>.0007 0.0026 0.0018</td>
<td>7040 6213 6090</td>
<td>83.7 75.7 78.0</td>
<td>83 82 78</td>
</tr>
</tbody>
</table>

Oxygen Consumption (cc./min.)

<table>
<thead>
<tr>
<th>Arterial Oxygen Capacity (vol. %)</th>
<th>Brachial Arterial Oxygen Content (vol. %)</th>
<th>Pulmonary Arterial Oxygen Content (vol. %)</th>
<th>Arteriovenous Oxygen Difference (vol. %)</th>
<th>Arterial Oxygen Saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
</tr>
<tr>
<td>229 223 224</td>
<td>13.00 12.85 13.00</td>
<td>11.85 11.75 11.81</td>
<td>8.21 8.24 7.97</td>
<td>3.63 3.51 3.84</td>
</tr>
<tr>
<td>189 169 160</td>
<td>16.35 16.70 16.75</td>
<td>15.07 15.56 15.09</td>
<td>10.96 11.05 10.55</td>
<td>4.11 4.52 4.54</td>
</tr>
<tr>
<td>224 192 188</td>
<td>12.91 12.54 12.50</td>
<td>11.14 10.24 10.43</td>
<td>7.26 5.94 5.32</td>
<td>3.88 4.30 5.11</td>
</tr>
</tbody>
</table>

Pressures are recorded as systolic, diastolic and mean pressures. Average pressures are mean pressures.
4. Right Auricular Pressure. The pressure in the right auricle fell in every instance in both groups of patients. The reduction of the right auricular mean pressure from the average resting level was 36.3 per cent in the low, and 53.1 per cent in the high spinal group. It could be presumed that had it been feasible to record right auricular pressures in the latter group at the 30 minute period, even lower levels might have been found. The basal right auricular mean pressures were within the normal range in all except one patient (N. W.) in whom it was 19.0 mm. of Hg.

5. Pulmonary Arterial Pressure. The pulmonary arterial pressure decreased in all subjects. The maximal reduction from the average mean resting level was 17.4 per cent in the low, and 34.2 per cent in the high spinal group. While decreases of as much as 5 mm. of Hg may be demonstrable in the pulmonary arterial pressure of resting patients when serial pressures are recorded, the uniform reductions recorded in all subjects and the 100 per cent difference between the low and high spinal groups lend significance to the values.

It will be noted that the central and peripheral blood pressures and the cardiac index undergo essentially similar reductions with the induction of spinal anesthesia (fig. 1).

6. Oxygen Consumption. This showed a reduction in 8 of 10 patients, with no change in one (L. S.) and a definite rise in another (F. D.) patient. Slightly greater reductions occurred in the high spinal group than in the low spinal group.

7. Brachial Arterial Oxygen Content. The oxygen content of brachial arterial blood fell slightly in all patients with the exception of patients L. S. and M. G., in whom slight increases were noted. The average changes were from 13.06 to 12.92 in the low, and from 13.14 to 12.54 volumes per cent in the high spinal group. These slight changes are accounted for solely by the slight but constant reduction in the arterial oxygen capacity (see below).

8. Pulmonary Arterial Oxygen Content. This was ultimately reduced in all patients studied. The average decrease in the low spinal group was from 9.25 to 8.64, and in the high spinal group from 9.23 to 7.49 volumes per cent.

9. Arteriovenous Oxygen Difference. In the low spinal group a rise from the average basal level of 3.81 to 4.29 volumes per cent was noted, whereas in the high spinal group there was an average increase from 3.91 to 5.05 volumes per cent.

10. Brachial Arterial Oxygen Capacity. In the low spinal group this changed inconstantly and insignificantly, but in subjects to whom high spinal anesthesia was given there was a definite trend toward a decrease in capacity.

11. Arterial Oxygen Saturation. No significant alteration was noted in any subject with the exception of patient T. D., in whom there was a decrease from 86.2 to 81.0 and 83.4 per cent at the 30 and 60 minute periods respectively. This patient was 75 years of age and had significant pulmonary disease. Anesthesia to the level of T-4 to T-5, superimposing a decreased ventilation upon a probably reduced alveolar-pulmonary capillary oxygen diffusion, most likely accounted for the reduction which was observed.

12. Hematocrit Level. Although the individual values are not tabulated, there was an average decrease of 1 per cent in the low, and 2 per cent in the high spinal group. Constant decreases occurred in all instances, equal in specimens withdrawn simultaneously from the brachial and pulmonary arteries.

In both low and high spinal anesthesia, the reduction in brachial arterial oxygen capacity, hematocrit level and brachial arterial oxygen content can be explained partly by hemodilution from the catheter drip system and the withdrawal of numerous blood specimens. There is some evidence, however, that spinal anesthesia per se may produce such reductions, possibly by the displacement of red cells in the capillary bed. The causes of these changes were not assessed.

13. Peripheral Blood Flows and Pulse Volumes. These underwent divergent responses in the fingers and toes in the low spinal group. In the finger the blood flow was reduced from 1.28 to 0.5 cc. per 10 cc. of part per minute, and the pulse volume fell from 0.0065 cc. to 0.0025 cc. per 10 cc. of part. On the other hand
### Table 2.—The Hemodynamic and Blood Oxygen Changes Induced by High Spinal Anesthesia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Anesthetic &amp; Level mg.</th>
<th>Brachial Arterial Pressure (mm. of Hg)</th>
<th>Right Auricular Pressure (mm. of Hg)</th>
<th>Pulmonary Arterial Pressure (mm. of Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Control 30 min. 60 min.</td>
<td>Control 60 min.</td>
<td>Control 30 min. 60 min.</td>
<td>Control 60 min.</td>
</tr>
<tr>
<td>J. K.</td>
<td>56</td>
<td>M</td>
<td>Senility</td>
<td>Procaine 150 T-3</td>
<td>132/78 84/58 102/60</td>
<td>6.1/3.0 5.9/2.0</td>
<td>28.7/9.5 22.0/8.5 25.0/10</td>
</tr>
<tr>
<td>F. B.</td>
<td>43</td>
<td>F</td>
<td>Obesity</td>
<td>Procaine 175 T-2</td>
<td>138/83 68/37.5</td>
<td>8.0/-2.5 2.0/-1.0</td>
<td>21.5/11</td>
</tr>
<tr>
<td>M. G.</td>
<td>41</td>
<td>F</td>
<td>Abdominal mass</td>
<td>Procaine 150 T-1</td>
<td>13/69 67/45 87/60</td>
<td>3.0/1.5 -2.5/-2.0</td>
<td>16.5/6.5 9.5/2.5 10.0/2.5</td>
</tr>
<tr>
<td>F. D.</td>
<td>46</td>
<td>F</td>
<td>Paroxysmal hypertension</td>
<td>Procaine 150 T-1 2</td>
<td>144/66</td>
<td>64/36</td>
<td>66.42</td>
</tr>
<tr>
<td>T. G.</td>
<td>60</td>
<td>M</td>
<td>Drug reaction</td>
<td>Procaine 175 T-1 2</td>
<td>145/71 63/36 75/41</td>
<td>10.5/1.8 4.7/1.2</td>
<td>17.5/9.7 12.5/4.7 13.5/6.0</td>
</tr>
</tbody>
</table>

Averages ........ 49.2

Percentile change ........

- Finger Blood Flow (cc./10 cc. part/min.)
  - Control 30 min. 60 min.
  - Finger Pulse Volume (cc./10 cc. part)
  - Toe Pulse Volume (cc./10 cc. part)
  - Cardiac Output (cc./min.)
  - Stroke Volume (cc.)
  - Pulse Rate

<table>
<thead>
<tr>
<th>Oxygen Consumption (cc./min.)</th>
<th>Arterial Oxygen Capacity (vol. %)</th>
<th>Brachial Arterial Oxygen Content (vol. %)</th>
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<th>Arteriovenous Oxygen Difference (vol. %)</th>
<th>Arterial Oxygen Saturation (%)</th>
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<td>Control 30 min. 60 min.</td>
<td>Control 30 min. 60 min.</td>
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<tr>
<td>218</td>
<td>195 203</td>
<td>13.78 13.58</td>
<td>12.36 11.58</td>
<td>8.21 6.55</td>
<td>4.15 5.03</td>
</tr>
<tr>
<td>195</td>
<td>172</td>
<td>15.85 15.80</td>
<td>14.38 14.28</td>
<td>10.97 8.65</td>
<td>3.41 5.62</td>
</tr>
<tr>
<td>134</td>
<td>122 127</td>
<td>15.63 15.63</td>
<td>14.30 14.65</td>
<td>10.38 9.40</td>
<td>3.91 5.89</td>
</tr>
<tr>
<td>153</td>
<td>155 176</td>
<td>15.09 15.09</td>
<td>13.80 13.68</td>
<td>9.85 9.26</td>
<td>3.95 4.42</td>
</tr>
<tr>
<td>219</td>
<td>184 198</td>
<td>11.80 11.41</td>
<td>10.84 10.26</td>
<td>6.72 4.71</td>
<td>4.12 5.55</td>
</tr>
<tr>
<td>184</td>
<td>164 175</td>
<td>14.43 13.82</td>
<td>13.14 12.54</td>
<td>9.23 7.49</td>
<td>3.91 5.05</td>
</tr>
</tbody>
</table>

Pressures are recorded as systolic, diastolic and mean pressures. Average pressures are mean pressures.
the pulse volume in the toe rose from a basal average of 0.0018 to 0.0052 cc. per 10 cc. of part. These divergent responses between fingers and toes were a constant feature in all the studies with low spinal anesthesia. The vasocostriction in the finger persisted and in most instances progressed for the duration of the investigation.

On the other hand the induction of high spinal anesthesia was followed by increased rates of circulation in both the fingers and toes.

In high spinal anesthesia. In this group the peripheral circulation tended to increase further in most cases at the 60 minute period when the blood pressure was rising.

14. Estimated Total Peripheral Resistance (T.P.R.). Total peripheral resistance showed a maximal reduction of 13.5 per cent in the low, and 18.8 per cent in the high spinal group. This fell to a greater extent in those patients who showed the greater decreases in blood pressure, but the validity of the formula is open to ques-

### Table 3.—The Changes in Cardiac Index, Left Ventricular Work and Estimated Total Peripheral Resistance Induced by Low and High Spinal Anesthesia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Cardiac Index (L/min.)</th>
<th>Left Ventricular Work (Kg. meters/min.)</th>
<th>Total Peripheral Resistance (Absolute units)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>30 min.</td>
<td>60 min.</td>
</tr>
<tr>
<td>L. S.</td>
<td>4.24</td>
<td>3.70</td>
<td>3.64</td>
</tr>
<tr>
<td>N. W.</td>
<td>3.23</td>
<td>3.43</td>
<td>3.21</td>
</tr>
<tr>
<td>G. T.</td>
<td>3.68</td>
<td>3.71</td>
<td>3.41</td>
</tr>
<tr>
<td>P. P.</td>
<td>2.43</td>
<td>1.97</td>
<td>1.87</td>
</tr>
<tr>
<td>T. D.</td>
<td>3.30</td>
<td>2.50</td>
<td>2.07</td>
</tr>
<tr>
<td>Averages</td>
<td>3.37</td>
<td>3.06</td>
<td>2.84</td>
</tr>
<tr>
<td>Percentile change</td>
<td>—</td>
<td>—9.2</td>
<td>—15.7</td>
</tr>
</tbody>
</table>

High Spinal Anesthesia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Cardiac Index (L/min.)</th>
<th>Left Ventricular Work (Kg. meters/min.)</th>
<th>Total Peripheral Resistance (Absolute units)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>30 min.</td>
<td>60 min.</td>
</tr>
<tr>
<td>J. K.</td>
<td>2.79</td>
<td>2.08</td>
<td>2.34</td>
</tr>
<tr>
<td>F. B.</td>
<td>3.00</td>
<td>—</td>
<td>1.62</td>
</tr>
<tr>
<td>M. G.</td>
<td>2.46</td>
<td>1.69</td>
<td>1.94</td>
</tr>
<tr>
<td>F. D.</td>
<td>2.76</td>
<td>2.45</td>
<td>3.02</td>
</tr>
<tr>
<td>T. G.</td>
<td>2.90</td>
<td>1.80</td>
<td>2.43</td>
</tr>
<tr>
<td>Averages</td>
<td>2.78</td>
<td>2.00</td>
<td>2.27</td>
</tr>
<tr>
<td>Percentile change</td>
<td>—</td>
<td>—28.1</td>
<td>—18.4</td>
</tr>
</tbody>
</table>

In this group the finger blood flow rose from a resting average level of 0.32 cc. to 1.11 cc. per 10 cc. of part per minute, and the finger pulse volume from an average of 0.0021 cc. to 0.0126 cc. per 10 cc. of part; the pulse volume in the toe increased from 0.0018 cc. to 0.0082 cc. per 10 cc. of part. The circulatory responses in the fingers are in contrast to those developing after low spinal anesthesia, and are the result of vasomotor paralysis of the preganglionic sympathetic outflow to the upper extremities. When treating arteriovenous pressure differences of less than 80 mm. of Hg.15

15. Calculated Left Ventricular Work (L.V.W.). This decreased in both groups. The low spinal group showed an average maximal reduction of 30 per cent from the basal level, and the high spinal group a maximal reduction of 62.4 per cent. The one patient (G. T.) (low spinal), who showed an increase in left ventricular work, failed to develop the usual decrease in blood pressure, and the cardiac out-
put was maintained. It is interesting that this one patient was exceptional in that the level of anesthesia was as low as T-11 and the blood pressure rose slightly.

16. Clinical Observations. The clinical events which accompany low spinal anesthesia are well known, but those which occur during high spinal anesthesia are of some interest. The authors have employed the procedure in 25 conscious patients, 20 of whom will be discussed in other reports concerned with studies in blood flow in surgical patients and in individual organs. As the anesthetic agent rises in the subarachnoid space, and particularly when levels above the fourth thoracic dermatome are rendered analgesic, the patients often yawn repeatedly. Following this event there may be a period of transient and slight nausea which usually subsides quickly and spontaneously or is alleviated by the inhalation of 100 per cent oxygen for a few minutes. In two instances severe nausea and vomiting occurred. This was the case in patient F. B. in whom it was not possible to record values at the 30 minute period since she was maintained on 100 per cent oxygen until that time because of nausea and retching associated with almost total body analgesia. Hyperventilation and considerable apprehension on the part of the patient may occur. In one instance the patient became so emotionally upset that it was thought best to terminate the procedure. In a third subject a 15 minute period of Cheyne-Stokes respiration occurred.

Although it has been implied that these occurrences, and particularly vomiting, are related to the high level of the anesthesia, our impression is that they are more likely associated with the rapidity with which the anesthetic agent rises in the subarachnoid space. Individual variations in the response to spinal anesthesia also play a role. In both instances mentioned, the patients became anesthetic to the clavicles in approximately two or three minutes. On the other hand, three patients (M. D., F. D., and T. G.) who developed sensory anesthesia to the first thoracic level within an optimum period of 8 to 10 minutes, experienced no untoward reactions.

When profound hypotension develops, however, nausea, vomiting and other symptoms become apparent regardless of the rapidity with which anesthesia is induced.

As has been noted by others, the peripheral arterial pressure usually reaches a maximal reduction within 15 minutes. This is maintained for about an hour, and then a gradual rise commences. The blood pressure returns to near basal levels within two or two and one-half hours, and this can be accelerated by the parenteral administration of a vasopressor agent such as Neosynephrine, which is free to act directly on the decentralized blood vessels. The administration of such an agent is seldom indicated.

Discussion

The hemodynamic changes produced by low and high spinal anesthesia are similar, with the exception of the effects on the peripheral circulation.

1. Blood Pressure. When vasoconstrictor drugs are not used, a constant accompaniment of both low and high spinal anesthesia is a reduction of the blood pressure. If the anesthetic agent is confined to the lower thoracic segments, the reduction in blood pressure is usually slight. If the anesthetic agent is allowed to reach the upper thoracic segments, marked lowering of the blood pressure usually occurs.

The cause of the hypotension in spinal anesthesia is not settled. Hematogenous intoxication, direct action on the medullary centers and reduced secretion of adrenaline by paralysis of the adrenal nerves are hypotheses which may be mentioned as of historical interest. The hypothesis that paralysis of the skeletal muscles with resultant loss of tone leads to a diminished venous return to the heart has been found invalid. The role of respiratory depression, although of little importance in low spinal anesthesia, merits consideration as a cause of hypotension in high spinal anesthesia. The view that paralysis of the sympathetic vasoconstrictor fibers to the arterioles, capillaries and veins occurs at the preganglionic level is the one best explaining the hemodynamic changes occurring in spinal anesthesia, whether high or low.
The argument that systemic hypotension is due primarily to respiratory depression or paralysis may now be examined. In high spinal anesthesia complete intercostal depression or even paralysis usually develops, and the respiratory pattern becomes diaphragmatic. There is no doubt that paralysis of the abdominal and thoracic muscles interferes with respiratory function,\textsuperscript{14, 21} causing a reduction in the vital capacity,\textsuperscript{21} but that this so impairs the venous return to the heart as to explain the hypotension has been questioned.\textsuperscript{7, 29, 38, 39}

Our observations suggest that respiratory depression is not the cause of the systemic hypotension which develops during high spinal anesthesia, since this occurs even when respiratory function is maintained efficiently by intermittent positive pressure breathing.\textsuperscript{26} One recent investigation\textsuperscript{38} has shown rather conclusively by the use of differential spinal block, that extreme hypotension can exist with little or no intercostal paralysis. Should paralysis of the phrenic nerves occur with high spinal anesthesia, cerebral anoxia and secondary vasomotor collapse would result, were artificial respiration not initiated. The fact that extreme hypotension can exist in the absence of phrenic or intercostal nerve paralysis,\textsuperscript{38} and that such hypotension cannot be corrected by means of artificial respiration, supports the view that respiratory paralysis is not a major factor in the hypotension. Our observations indicate that respiratory exchange is sufficient to saturate pulmonary blood, since the oxygen saturation of the arterial blood remained virtually unaltered even in the face of marked degrees of hypotension. Contrariwise, spinal anesthesia may be attended by a significant and dangerous decrease in arterial oxygen saturation in the patient with impaired respiratory function, as occurred in one patient (T. D.). This does not occur if efficient aided or artificial respiration is maintained.\textsuperscript{26}

Experimental and clinical evidence has accumulated supporting the role of sympathetic vasoconstrictor paralysis in the production of the hypotension during spinal anesthesia. It is manifestly impractical to consider arteriolar paralysis and paralysis of the postarteriolar beds separately,\textsuperscript{29, 32, 41} as it is extremely unlikely that spinal anesthesia could effect one and not the other. The demonstration that serial destruction of the spinal cord in dogs\textsuperscript{19, 36} produces profound hypotension only when the fourth thoracic segment is destroyed suggests that up to a certain level the body can compensate for the loss of sympathetic vasoconstrictor elements and maintain a fairly normal blood pressure. These findings have been substantiated by spinal procaine block in dogs\textsuperscript{4, 10, 42} in which the blood pressure is well maintained as long as the upper thoracic segments are not paralyzed. The results of this study would support this contention, since the fall in blood pressure in the low spinal group was not usually severe. The demonstration that spinal anesthesia produces no appreciable drop in blood pressure after total sympathectomy\textsuperscript{5} is additional proof of the role of that system in the production of the hypotension of spinal anesthesia.

It is therefore suggested that the hypotension so constant in spinal anesthesia is the result of paralysis of vasomotor tone, proportional to the degree of sympathetic paralysis. The result is a decreased peripheral resistance and an increased capacity of the arteriolar and postarteriolar vascular beds in the anesthetized regions, factors which cause a reduction in the blood pressure. If vasomotor paralysis were the only factor, it should be possible to demonstrate an increased blood flow in all the anesthetized regions due to the release of arteriolar tone; there should be a decreased arteriovenous oxygen difference in the blood from those areas; and there should be an increase in the venous and right heart pressures secondary to the increased venous return. Our data do not support wholly these suppositions, but, in the light of data obtained during the course of observations on individual organ flows,\textsuperscript{17, 30} indicate regional variations in the rate of blood flow and oxygen extraction.

2. Peripheral Blood Flow. The data in this report substantiate the observations of others,\textsuperscript{5, 11, 29, 31, 38, 40} that the rate of blood flow increases in the extremities after spinal block, and that in the case of low spinal
anesthesia there is a compensatory vasoconstriction in the upper extremities. The increased blood flow in the anesthetized fingers and toes is maintained only so long as the mean blood pressure remains above the normal arteriolar pressure. When the mean arterial blood pressure falls below circa 30 mm. of Hg, the peripheral blood flow falls below the normal resting level. In most cases of high spinal anesthesia such low levels of blood pressure do not occur.

3. Oxygen Consumption. This was found to be reduced by both low and high spinal anesthesia. This reduction was minimal in the subjects who had low spinal anesthesia and somewhat greater in the subjects who had high spinal anesthesia. Were one to consider only the average group changes, it would be difficult to ascribe significance to the reductions encountered which were maximally no greater than 10 per cent. However, a reduction was noted in four of five patients in each group, and the greater average reduction occurred in subjects who were given high spinal anesthesia. Hypotensive states in themselves may be associated with a drop in the basal metabolic rate. An additional factor in spinal anesthesia is the existence of a flaccid paralysis of the striated muscles in the anesthetized areas, and a reduction of the oxygen utilization would be expected, proportional to the mass of paralyzed muscle.

4. Arteriovenous Oxygen Difference. In most cases of low and in all cases of high spinal anesthesia the arteriovenous oxygen difference is increased. If one were to assume that the increased blood flows noted in the fingers and toes are representative of similar changes occurring elsewhere in the entire vascular bed, it would be difficult to reconcile such increases with a rise in the arteriovenous oxygen difference and a fall in the cardiac output. The greatly increased arteriovenous oxygen differences noted particularly in high spinal anesthesia, are believed to be due to a combination of two factors: (a) a marked slowing of the rate of blood flow in areas other than the extremities, and, (b) the possible opening of prevenous channels that are normally non-functioning in the basal state. Investigations now in progress in this laboratory indicate that there is a definite reduction in the hepatic and coronary flows, with a greatly exaggerated relative extraction of oxygen from the splanchnic blood, and a decreased extraction from the blood in the extremities. Kety and his associates have shown that under differential spinal block in hypertensive patients, the hypotension so produced is associated with a reduction in the rate of cerebral blood flow. Data have been reported suggesting that a state of stagnation of the peripheral circulation exists in the anesthetized regions. That the circulation time increases has also been demonstrated. It would follow from such observations that the arterial-venous oxygen differences in the blood from the involved areas would increase. The data presented in this report demonstrate an increased arteriovenous oxygen difference between the brachial and pulmonary arterial bloods, but this is attributed in part not to stagnation in the extremities, but rather to a decrease in the rate of flow and a predominant increase in the oxygen extraction in the splanchnic bed.

5. Total Peripheral Resistance. This falls, provided there is a decrease in the blood pressure. Total peripheral resistance, however, does not afford a measure of regional variations in resistance. Increases in resistance in the renal and splanchnic vasculature have been demonstrated following the administration of spinal anesthesia, but they are slight, and the evidence ascribing such changes to selective vasoconstriction is lacking of conclusive proof.

6. Right Heart Pressures. Our data show a decrease in the pressures within the right auricle and the pulmonary artery, as has been found by others. It has been shown that the peripheral venous pressures fall with the arterial pressure during spinal anesthesia. That the drop in right heart pressures may be due to the release of vasomotor tone in the pulmonary arterioles is worthy of consideration, but it is probably not a factor of major importance. The reductions in right heart pressures noted herein are consistent with the finding of a decreased cardiac output, and with the view that there is an over-all reduction in the rate of blood flow in the viscera. The
evidence for this is the increased difference between the oxygen content of the brachial and pulmonary arterial bloods, associated with a decreased blood flow through the splanchnic coronary and renal beds, proportionately greater than the increased flow in the extremities.

7. Bradycardia. In general the heart rate slows after spinal anesthesia has become established. The bradycardia under high spinal anesthesia may be due to paralysis of the cardiac accelerator fibers. This explanation would not apply to low spinal anesthesia where the innervation of the heart is intact, and the heart rate similarly slows. In these cases it is difficult to explain the failure of the hydrostatic carotid sinus reflex to induce tachycardia. All the patients had a decrease in right heart pressure, due to a decrease in venous return to the heart. The tendency of this to cause bradycardia (Bainbridge reflex), may have overcome any activity on the part of the carotid sinus.

8. Cardiac Output. The cardiac output dropped in all patients after spinal anesthesia became established. In one patient the drop was minimal (F. D.) despite the fact that there was a profound drop in the blood pressure, and at the end of 60 minutes there was an actual increase in the output, a sequence of events which remains unexplained; in another (G. T.) the cardiac output did not change significantly, but neither did the blood pressure with an anesthetic level of only T-11. From the data already discussed it would appear that the decrease in the cardiac output is the result of a decreased venous return to the heart, and in general this decrease is greater after high than after low spinal anesthesia. A similar relationship exists between the right heart pressures under the two types of anesthesia.

9. Left Ventricular Work. With the exception of one subject (G. T.) who did not have a reduction of blood pressure following spinal anesthesia, all patients showed a significant decrease in left ventricular work. The greater reductions were observed in the patients who were given high spinal anesthesia, mainly a reflection of the diminished arterial pressure. Unpublished observations reveal a marked decrease in coronary flow, with a stable or slightly reduced oxygen extraction. Under the circumstances it is possible that the reduced coronary circulation is sufficient to meet the lessened work demand of the myocardium.

It is believed that the cause of the cardiovascular changes observed in spinal anesthesia is the paralysis of the preganglionic sympathetic nerve fibers of the spinal cord, and these changes become more marked as higher cord levels are blocked. The vasomotor paralysis which follows leads to local alterations in hemodynamics. Although in the skin there is a demonstrable increase in the rate of blood flow in the anesthetized regions, it is possible that this increase may represent a combination of decreased resistance in the arteriolar bed and the opening of channels (metarterioles) not functioning in the basal state. It is reasonable to assume that there is an increased rate of blood flow in the muscles as has been found to occur in general anesthesia. Our data show that there is a decreased venous return to the heart with an increased circulation in the extremities. This must result from a substantially greater reduction of venous return with increased oxygen extraction from other parts of the body, presumably, in the main, the splanchnic bed. This view is supported by the known decrease in renal flow and by unpublished observations demonstrating a decrease in splanchic and coronary blood flows. The increase in the arteriovenous oxygen difference is in itself indicative only of a greater relative extraction of oxygen by the body tissues, whether this be due to more prolonged contact between the tissues and the red cells (reduced rate of blood flow as in stagnant anoxia), to a greater surface contact (as a result of the opening of previously nonfunctioning vascular compartments), or both.

We have observed no significant ill effects in this group of patients consequent to the administration of high spinal anesthesia, provided that adequate oxygenation of the arterial blood is maintained. In no instance have pathologic reflexes appeared, and there has been no evidence of delayed neurologic de-
kidney function as demonstrated by urinary concentration tests. No electrocardiographic changes indicative of myocardial anoxia have been demonstrated during or after the administration of anesthesia.

**SUMMARY AND CONCLUSIONS**

1. The hemodynamic changes induced by spinal anesthesia, low and high, were studied in 10 patients, without the use of vasoconstrictor drugs.

2. Certain effects upon hemodynamics were induced by both low and high spinal anesthesia, as follows:
   (a) The cardiac output, the cardiac index and the stroke volume of the heart decreased.
   (b) The right auricular, the pulmonary arterial and the brachial arterial blood pressures fell.
   (c) The total peripheral resistance decreased.
   (d) The heart rate decreased in four out of five patients who had low, and in all subjects who had high spinal anesthesia.
   (e) The rate of oxygen consumption decreased.
   (f) The calculated work of the left ventricle decreased in all except one case in whom the systemic blood pressure did not fall.
   (g) The pulmonary arterial oxygen content dropped, and, with relatively little decrease in the brachial arterial oxygen content and no decrease in arterial oxygen saturation, the arteriovenous oxygen difference increased.

3. The effects upon the blood flow in the extremities differed between low and high spinal anesthesia, as follows:
   (a) The peripheral blood flow under low spinal anesthesia increased in the toes, but diminished in the fingers.
   (b) The peripheral blood flow and pulse volume increased in both fingers and toes under high spinal anesthesia.

4. The differences in the hemodynamic effects of low and high spinal anesthesia are only quantitative, and are more marked with high spinal anesthesia.

5. The hemodynamic changes induced by spinal anesthesia are attributed to vasomotor paralysis.

6. Acute sequential studies of the circulation, which may shed light on the immediate adjustments of the circulation to spinal anesthesia, are needed.

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