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


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Low and high spinal anesthesia were administered to a group of 10 waking patients without the use of vasopressor drugs. Preliminary evidence does not support the view that the induced hypotension is accompanied by significant splanchnic vasoconstriction or by homeostatic diversion of blood from the splanchnic bed to maintain the circulation in “more vital” areas.

In previous communications we have presented the data on the general hemodynamic and blood oxygen changes induced in humans by the administration of low and high spinal anesthesia.11,18 This paper deals with the changes observed in the splanchnic vascular bed in two groups of patients receiving low and high spinal anesthesia, but in whom no surgical procedure was performed. The general technic and methods have been previously described15 and mention will be made of the technic only as it applies to the problem at hand.

Material and Methods

Ten patients, nine men and one woman, with various medical diseases, were selected at random from the medical wards. Their ages ranged from 31 to 77 years; the average age was 52.4 years. Although none had active infections or temperature elevations at the time of the study, it must be emphasized that only one of these patients was entirely free of disease, and that most of them could not be considered normal control subjects. In three there was a history of chronic alcoholism, one had a fatty liver which was thought to be healed, and another

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was found to have mild congestive failure, conditions which are all known to derange the normal liver function and splanchnic blood flow. For the purpose of this study, however, the selection of completely normal individuals was not deemed necessary.

After an overnight fast, 0.1 to 0.2 Gm. of pentobarbital sodium was administered and the patients were transported to the cardiovascular laboratory where the air temperature was maintained at 24 C., and the humidity was kept fairly constant. The right hepatic vein was uniformly catheterized employing a 6 to 8F Goodale birdseye catheter, introduced into a medial arm vein, and the position of the tip checked repeatedly by fluoroscopic observation. An indwelling round tip no. 16 venous needle was introduced into a lateral arm vein for delivery of the bromsulfalein drip. After a priming dose of 150 mg., the chemical was administered at a rate of 3.0 mg. per square meter of body surface per minute. Constancy of flow was insured by the use of a tunnel clamp.*

The estimated hepatic blood flow was calculated according to the method of Bradley and associates,2 and is expressed in cubic centimeters per square meter of body surface per minute (EHBFM). In all instances minor variations in the peripheral concentration of bromsulfalein were adjusted by computing the estimated hepatic blood flow according to the correction factors suggested by the same authors. In this instance, total plasma volume was calculated from the tables of Gibson and Evans.3 Cases in which the peripheral concentration of bromsulfalein (dP) exceeded .0005 mg. per minute, were discarded.

The calculated splanchnic oxygen consumption (CSOC) was obtained by using the formula:

\[ CSOC = \frac{EHBFM^2 \times A - V \text{ Difference}}{100}, \]

wherein A-V difference represents subtraction of the oxygen content of hepatic vein blood from that of brachial arterial blood in volumes per cent, and CSOC is expressed in cc. of oxygen per minute per square meter of body surface.\(^6\)

The brachial arterial and the hepatic venous pressure were measured respectively through a no. 19 intra-arterial needle and the hepatic catheter. Pressures were transduced via Statham strain gauges and recorded on the Brush multichannel oscillograph.

The splanchnic vascular resistance (SVR) was calculated according to the formula:

\[ SVR = \frac{\text{Mean arterial blood pressure}}{\text{Hepatic blood flow in cubic centimeters per second per square meter body surface}}, \]

and expressed as mm. Hg per cubic centimeter per second per square meter body surface.\(^4\)

Bromsulphalein levels were determined on the Beckman DU spectrophotometer, previously calibrated to known amounts and dilutions of the dye, employing a wave length setting of 680 m\(\nu\). Blood oxygen contents were determined spectrophotometrically according to the method of Hickam and Frayser.\(^8\)

Following a stabilization period which did not exceed 60 minutes, the basal values were obtained. Low (five patients) or high (five patients) spinal anesthesia was then induced according to the previously described technic,\(^1\) wherein the former produces a sensory anesthetic level lower than T-4 and is accompanied by diminished blood flow in the finger, and the latter leads to anesthesia above T-4 with a release of vasomotor tone in the finger. In all instances these criteria were met. Finger blood flows and volumes and toe pulse volumes were measured through onometer air transmission, transduced via Grass and Statham strain gauges, and recorded on the Grass oscillographic plethysmograph.

All determinations were repeated 30 and 60 minutes after the induction of spinal anesthesia. Specimens for the determination of estimated hepatic blood flow were drawn in groups of three, five minutes apart, and the averages recorded. All other determinations were carried out in duplicate. Vasopressor drugs were not employed at any time.

As a control all determinations were carried out in an additional group of five patients over a two or three hour period. Spinal anesthesia was not administered to these patients, but all other maneuvers repeated as in the previous group.

**RESULTS**

The data obtained in patients undergoing low and high spinal anesthesia are presented in tables 1 and 2, respectively.

1. **Mean Brachial Arterial Pressure.** This dropped an average maximal of 22.4 per cent in the “low” and 27 per cent in the “high” spinal group. These changes compare favorably with those previously reported,\(^1\) particularly in the low spinal group. In the high spinal group the drop in blood pressure was not as pronounced as we might have expected. This was probably due to the fortuitous circumstance that the low spinal group was, on the average, 10 years older than the high spinal group, and the latter had an average resting mean pressure of 89.4 mm. Hg, as compared with the former where it was 110 mm. Hg.

2. **Estimated Hepatic Blood Flow (EHBFM\(^2\)).** There was an average maximal decrease of 24 per cent in the “low,” and 33 per cent in the “high” spinal group. The estimated blood flow (EHBFM\(^2\)) decreased in all instances with one exception (patient A. C.) in whom an insignificant rise and fall occurred at the 30 and 60 minute periods, respectively, following low spinal anesthesia. This was the only patient with an elevated right atrial pressure (5.7 mm. Hg).

In three subjects (J. K., J. N., J. C.) the control flows exceeded the expected upper range of normal\(^1\); in another (P. P.) it was slightly lower.

3. **Brachial Arterial-Hepatic Venous Oxygen Difference.** There was an average maximal increase of 16.7 per cent in the “low” and 35.5 per cent in the “high” spinal group. Here again, patient A. C. showed a transient decrease of 1.13 volumes per cent of oxygen at the 30 minute period, and an increase of only 0.57 volumes per cent at the 60 minute period.

4. **Hepatic Vein Oxygen Content.** The oxygen content of hepatic venous blood decreased a maximal average of 16.2 per cent in the “low,” and 30.3 per cent in the “high” spinal group. This decrease occurred in all instances except in patient C. V., in the low spinal group, who demonstrated an insignificant rise and fall at the 30 and 60 minute periods respectively. In patient A. C. there was an insignificant
### Table 1. The Hemodynamic and Oxygen Changes Consequent to Low Spinal Anesthesia. Determinations Recorded in the Resting State, and 30 and 60 Minutes Following the Administration of the Anesthetic. Arterial Pressure Expressed as Systolic, Diastolic and Mean

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Anesthetic and Level</th>
<th>Brachial Arterial Pressure (mm. of Hg)</th>
<th>Estimated Hepatic Blood Flow (cc./Ml./min.)</th>
<th>Brachial Arterial-Hepatic Venous Oxygen Difference (vol. %)</th>
<th>Hepatic Venous Oxygen Content (vol. %)</th>
<th>Splanchnic Oxygen Consumption (cc./Ml./min.)</th>
<th>Splanchnic Vascular Resistance (mm. of Hg/cc./sec./Ml.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. K.</td>
<td>58</td>
<td>Cerebral thrombosis</td>
<td>Procaine 100 mg. T-10</td>
<td>Control 149/90 117.6 135/77 105.6</td>
<td>127/79 99.6</td>
<td>1413 996 1161</td>
<td>5.33 7.06 7.19</td>
<td>8.27 5.96 6.44</td>
<td>75 80 83</td>
</tr>
<tr>
<td>J. N.</td>
<td>41</td>
<td>Fatty liver (healed)</td>
<td>Procaine 100 mg. T-8</td>
<td>Control 137/80 106.0 102/63 90.4</td>
<td>105/69 82.2</td>
<td>1315 1256 945</td>
<td>4.40 4.57 4.99</td>
<td>8.23 6.87 6.73</td>
<td>58 57 48</td>
</tr>
<tr>
<td>A. J.</td>
<td>77</td>
<td>Hypertrophy of prostate</td>
<td>Procaine 100 mg. T-9</td>
<td>Control 139/72 100.5 93/53 68.1</td>
<td>99.0</td>
<td>928 613 572</td>
<td>3.81 5.12 5.11</td>
<td>3.89 2.34 2.43</td>
<td>35 31 29</td>
</tr>
<tr>
<td>A. C.*</td>
<td>66</td>
<td>Resolved pneumonia</td>
<td>Procaine 100 mg. T-8</td>
<td>Control 154/81 111.6 116/60 79.0</td>
<td>85.8</td>
<td>578 583 521</td>
<td>5.29 4.06 5.86</td>
<td>7.08 6.86 6.20</td>
<td>31 24 31</td>
</tr>
<tr>
<td>C. V.</td>
<td>44</td>
<td>Chronic alcoholism</td>
<td>Procaine 100 mg. T-7</td>
<td>Control 136/90 114.0 117/77 94.4</td>
<td>90.0</td>
<td>662 544 510</td>
<td>5.10 5.28 5.36</td>
<td>8.13 8.16 8.00</td>
<td>34 29 27</td>
</tr>
<tr>
<td>Averages</td>
<td>57</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<td>—</td>
</tr>
<tr>
<td>Percentile change</td>
<td>57</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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</tr>
</tbody>
</table>

* Patient A.C. in minimal congestive failure (right atrial mean pressure 5.7 mm. of Hg).
Table 2.—The Hemodynamic and Oxygen Changes Consequent to High Spinal Anesthesia. Determinations Recorded in the Resting State, and 30 and 60 Minutes Following the Administration of the Anesthetic. Arterial Pressure Expressed as Systolic, Diastolic and Mean

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Anesthetic and Level</th>
<th>Brachial Arterial Pressure (mm. Hg)</th>
<th>Estimated Hepatic Blood Flow (cc./M.2/min.)</th>
<th>Brachial Arterial-Hepatic Venous Oxygen Difference (vol. %)</th>
<th>Hepatic Venous Oxygen Content (vol. %)</th>
<th>Splanchnic Oxygen Consumption (cc./M.2/min.)</th>
<th>Splanchnic Vascular Resistance (mm. of Hg/cc./sec./M.2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></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>
<td>Control 30 min. 60 min.</td>
</tr>
<tr>
<td>H. W.</td>
<td>39</td>
<td>Korsakow psychosis</td>
<td>Proxime 160 mg T-3</td>
<td>150/88 114.6 61.5 85/56 67.7</td>
<td>1085 910 919</td>
<td>10.18 6.13 7.02</td>
<td>60 76 66</td>
<td>6.4 4.1 4.4</td>
<td></td>
</tr>
<tr>
<td>W. R.</td>
<td>57</td>
<td>No disease</td>
<td>Proxime 160 mg T-2</td>
<td>94/65 80.5 60.0 86/55 73.8</td>
<td>744 511 656</td>
<td>4.80 7.22 6.10</td>
<td>10.54 7.43 8.60</td>
<td>36 37 40</td>
<td>6.5 7.1 6.8</td>
</tr>
<tr>
<td>J. C.</td>
<td>31</td>
<td>Peptic ulcer</td>
<td>Proxime 125 mg T-2</td>
<td>107/68 98.8 72.6 79/54 67.8</td>
<td>1292 934 647</td>
<td>4.59 6.41 7.23</td>
<td>13.58 11.17 10.34</td>
<td>59 60 47</td>
<td>4.1 4.7 6.3</td>
</tr>
<tr>
<td>P. P.</td>
<td>45</td>
<td>Alcoholic neuropathy</td>
<td>Proxime 200 mg T-7</td>
<td>105/61 81.0 63.6 59/42 46.8</td>
<td>493 303 205</td>
<td>5.10 7.50 8.63</td>
<td>11.49 9.18 7.98</td>
<td>25 23 25</td>
<td>9.9 12.5 9.5</td>
</tr>
<tr>
<td>C. P.</td>
<td>50</td>
<td>Psychoneurosis</td>
<td>Proxime 175 mg T-2</td>
<td>98/66 82.2 55.4 66/50 60.6</td>
<td>910 504 432</td>
<td>5.46 9.98 9.22</td>
<td>9.48 5.52</td>
<td>44 50 40</td>
<td>6.1 7.0 8.4</td>
</tr>
<tr>
<td>Averages</td>
<td>46</td>
<td></td>
<td></td>
<td>80.4 83.2 63.3 883 822 500</td>
<td>5.03 7.90 7.67</td>
<td>11.05 7.70 7.83</td>
<td>45 49 44</td>
<td>6.5 7.1 7.1</td>
<td></td>
</tr>
<tr>
<td>Percentile change</td>
<td>46</td>
<td></td>
<td></td>
<td>80.4 83.2 63.3 883 822 500</td>
<td>5.03 7.90 7.67</td>
<td>11.05 7.70 7.83</td>
<td>45 49 44</td>
<td>6.5 7.1 7.1</td>
<td></td>
</tr>
</tbody>
</table>


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drop at the 30 minute period, as would be expected from the values already noted in his instance.

The data so far presented are significant when compared with the results in the five control patients. Because of the individual variability in the data, the changes in the estimated hepatic blood flow and in the arteriovenous oxygen difference at the 30 minute period in the low spinal group are not statistically significant. The general trend of change here, as in the remainder of the study, however, is in a downward and upward direction respectively.

5. Calculated Splanchnic Oxygen Consumption (CSOC). In the “low” spinal group there were average decreases of 4.7 and 6.8 per cent at the 30 and 60 minute periods respectively. In the “high” spinal group there was an average rise of 8.8 per cent and a fall of 2.2 per cent at these same periods.

In four subjects (J. K., J. N., H. W., J. C.) the calculated splanchnic oxygen consumption (CSOC) exceeded the known upper range of normal (a reflection of the high estimated hepatic blood flows.)

6. Splanchnic Vascular Resistance (SVR). This showed no significant change in the patients undergoing “low” spinal anesthesia, and a rise of only 7 per cent in the patients undergoing “high” spinal anesthesia. When compared with the average decreases of 8.5 and 11 per cent in the control group, these changes are not sufficiently clear cut to permit conclusive evaluation.

7. Hepatic Vein Mean Pressure. The extreme variability which occurred in individual patients, and the lack of data in regard to extrascular pressures, preclude assessment of these values.

DISCUSSION

As has been previously shown the higher the level of anesthesia, the greater is the drop in the brachial arterial pressure and in the cardiac output. A close but not strict correlation was found in the decrease of the arterial pressure and the cardiac output, and the increasing level of anesthesia.

The splanchnic bed participates in the decreased rate of circulating blood during spinal anesthesia. The estimated hepatic blood flow decreases, the hepatic venous oxygen content decreases, and the brachial arterial-hepatic venous oxygen difference increases. These changes are intensified as higher cord levels are anesthetized. A close but not absolute relation may be noted between these changes and the level of anesthesia (fig. 1). The increased extraction of oxygen from the splanchnic blood is readily explained by the probable existence of a more prolonged contact between blood circulating in this bed and cells available for extraction. The cause of the retardation of flow which has been shown to exist is more difficult to explain. It is of considerable interest that Habif and his associates have shown that cyclopropane, ether and Thiopental anesthetics cause similar changes in flow in the absence of a decrease in the peripheral arterial pressure, and their findings indicate that renal and splanchnic vasoconstriction causes an increased
resistance in the vascular beds of these organs. This would support the belief that the renal and splanchnic vascular beds sacrifice their recipient blood as part of a homeostatic mechanism designed to maintain an elevation of the arterial pressure and the diversion of blood to more “vital” areas, in the same fashion as is stated to occur in oligemic shock, congestive heart failure and periods of violent exercise.³,¹⁰,¹²

It is not likely that this same phenomenon is operative during spinal anesthesia, at least in regard to the splanchnic bed, at levels of blood pressure decreases in the order of those herein presented. Both in “high” and in “low” spinal anesthesia the changes in splanchnic vascular resistance are not significant and are variable from patient to patient. The slight average increase in splanchnic resistance noted only in “high” spinal anesthesia is so slight as to defy any definite conclusion.

Added evidence against participation of the splanchnic bed in such a diversionary homeostasis during spinal anesthesia is afforded by a combined study in patient W. R. (table 3). By placing cardiac catheters into the pulmonary artery and the hepatic vein, simultaneous determinations of the cardiac output, the estimated hepatic blood flow and blood oxygen contents could be obtained. Reduction of the saline catheter drip systems to 10 drops a minute and doubling of the amount of heparin employed prevented the development of hypervolemia. It will be noted that the percentile contribution of the splanchnic flow to the total cardiac output remained remarkably constant. Further work to test these findings is in progress. The very close relation of changes between the cardiac index, the brachial arterial pressure, the estimated hepatic blood flow and the oxygen contents of the hepatic venous and the pulmonary arterial bloods in this patient is readily seen. Papper and his associates¹⁴ have likewise found a relation between the estimated hepatic blood flow and the arterial pressure in patients undergoing surgery under high spinal anesthesia.

As is the case in general anesthesia,⁴ spinal anesthesia is followed by little or no alteration in splanchnic oxygen consumption. Contrary to the mechanism which is said to be involved in general anesthesia, however, the decrease in estimated hepatic blood flow does not appear to be due to splanchnic vasoconstriction, but to a reduced rate of inflow of blood into the splanchnic bed.

Investigation now in progress indicates that “high” spinal anesthesia leads to a marked reduction of the coronary blood flow.⁷ The

| Table 3.—The Data in Patient W. R. Catheters Introduced in Hepatic Vein and Pulmonary Artery with Simultaneous Determinations of Cardiac Output and Hepatic Flow |
|-------------------------------------------------|---|---|---|
| Heart rate                                     | Control | 30 min. | 60 min. |
| Brachial arterial pressure                      | 94/65   | 74.5/46  | 86/55.5 |
| Brachial arterial mean pressure                 | 80.4    | 60.0     | 73.8    |
| Brachial arterial oxygen content                | 15.34   | 14.65    | 14.70   |
| Pulmonary arterial oxygen content               | 11.10   | 9.14     | 10.15   |
| Brach. art.—pulm. art. oxygen difference       | 4.24    | 5.51     | 4.55    |
| Brach. art.—hep. ven. oxygen difference        | 4.80    | 7.22     | 6.10    |
| Cardiac index                                   | 2.80    | 2.02     | 2.70    |
| EHBFM²                                         | 0.74    | 0.51     | 0.65    |
| EHBFM² % contribution to total cardiac index   | 26      | 25       | 24      |

* Pressures in mm. of Hg, oxygen contents and differences in volumes per cent, cardiac index and EHBFM² in liters per minute.

marked reduction and, in some instances, total temporary abolition of urinary flow during “high” spinal anesthesia, is indirect evidence that the renal bed likewise is subject to a putative decrease in blood flow. Cerebral blood flow does not appear to show any appreciable change in normotensive subjects.⁹ Only in the skin, and probably in the muscles, has an increase in blood flow been demonstrated, associated with an increase in femoral vein oxygen content and a decrease in brachial arterial-femoral venous oxygen difference.¹⁶

There is a proportionately greater increase in the brachial arterial-hepatic venous than in the brachial arterial-pulmonary arterial oxygen difference. The conclusion is tentatively
reached that the increase in arteriovenous oxygen difference between arterial and mixed venous blood which follows spinal anesthesia is in part due to an increased extraction of oxygen in the splanchnic bed preponderant over the total changes which occur in the remaining vascular beds of the body. The important role of the renal vasculature, normally the recipient of 25 per cent of the resting cardiac output, has not as yet been assessed.

**Summary and Conclusions**

1. The brachial arterial pressure, the hepatic venous blood flow, the oxygen contents of hepatic, pulmonary (one case) and brachial arterial bloods, the splanchnic oxygen consumption and the splanchnic vascular resistance were determined in 10 patients with various medical diseases, to whom a spinal anesthetic was administered. The level of anesthesia was "low" in five patients and "high" in five patients. Determinations were made basally, 30 and 60 minutes following the administration of the anesthetic. Surgery was not performed in these patients. Vasoconstrictor drugs were not employed despite the development of hypotension. Control observations were made in five patients not receiving a spinal anesthetic.

2. Spinal anesthesia produces the following alterations: (a) a decrease in the peripheral arterial pressure; (b) a decrease in the estimated hepatic blood flow; (c) an increase in the brachial arterial–hepatic venous oxygen difference, and a decrease in the hepatic venous oxygen content.

3. Neither "low" nor "high" spinal anesthesia produce significant changes in splanchnic oxygen consumption and resistance. The absence of grossly calculable splanchnic vasoconstriction, coupled with preliminary observations that the percentile contribution of the hepatic blood flow to the total cardiac output remains unchanged during spinal anesthesia productive of hypotension of the order observed, indicates that blood is not diverted from the splanchnic bed during such hypotension as part of a homeostatic mechanism designed to maintain circulation in "vital" areas.

4. Spinal anesthesia has previously been shown to increase the brachial arterial–pulmonary arterial oxygen difference. Partial evidence is presented which indicates that a relative increase in oxygen extraction in the splanchnic bed, predominating over the extraction changes occurring in the remaining beds, is in part responsible for this change.

5. The data corroborate previous tentative conclusions which correlate the degree of hemodynamic change consequent to spinal anesthesia to the dermatonic level of the anesthetic.

**References**


Unpublished observations.

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