Cardiovascular and Respiratory Effects of Subarachnoid Block in the Presence of Acute Blood Loss

William F. Kennedy, Jr., M.D.,* John J. Bonica, M.D.,† T. Jason Akamatsu, M.D.,*
Richard J. Ward, M.D.,* Wayne E. Martin, M.D.,‡
Alexander Grinstein, M.D.§

Subarachnoid block under normovolemic conditions was compared with subarachnoid block after 10 ml. of whole blood per kilogram of body weight had been removed in 15 unpremedicated, normal human volunteers. Each subject served as his own control. The results of cardiovascular and blood gas measurements revealed significant reductions in mean arterial pressure, cardiac output, stroke volume and central venous pressure after acute blood loss. Mean arterial pressure was about 30 per cent below the control value, cardiac output was reduced 15 per cent, stroke volume had decreased 12 to 22 per cent, and central venous pressure was 66 per cent below the control value. Subarachnoid block should be used with caution in the presence of acute untreated blood loss.

IN MODERN ANESTHESIOLOGIC PRACTICE subarachnoid block is considered contraindicated in hypovolemic patients because the resulting vasomotor blockade impairs cardiovascular performance. We,[1, 2, 3] and others,[4, 5, 6, 7] have studied the effects of subarachnoid block on normovolemic subjects, but to our knowledge no well-designed controlled studies on hypovolemic human subjects have been reported.

* Assistant Professor, Department of Anesthesiology, University of Washington, School of Medicine, Seattle, Washington.
† Professor and Chairman, Department of Anesthesiology, University of Washington, School of Medicine, Seattle, Washington.
‡ Senior Fellow, Cardiovascular Trainnig Program (HTS 5147), Departments of Physiology and Biophysics and Anesthesiology, University of Washington, School of Medicine, Seattle, Washington.
§ The Cobb Medical Center, Seattle, Washington.

This study was initiated to provide such information.

Method

Fifteen healthy male volunteers 22 to 37 years of age had fasted for eight hours. They were unpremedicated. Under local anesthesia, Teflon and vinyl catheters were inserted percutaneously into the brachial artery and the basilic vein and advanced into the subclavian artery and the superior vena cava, respectively. Arterial and venous pressures were measured with Statham strain gauges and recorded continuously on a Gilson GME polygraph; the electrocardiogram was recorded continuously. After the subject had rested for 30 minutes, control measurements were made of mean arterial pressure, cardiac rate and cardiac output. Cardiac output was determined by the indicator-dilution technique with indocyanine green. The area under the dye curve was measured planimetrically.[8] Arterial blood was sampled anaerobically to determine P_{O_2} and pH, with a modified Clark electrode and an Astrup pH electrode, respectively. We calculated Pa_{CO_2} by the tonometric method of Astrup. Stroke volume was determined from cardiac output and cardiac rate. Total peripheral resistance is represented by the following formula:

$$\text{T.P.R. units (dynes per second per cm.}^{-5}) = \frac{\text{Mean arterial blood pressure (mm. Hg)}}{\text{Cardiac output (ml. per second)}} \times 1,332$$

The arterial pressure wave was differentiated electronically to obtain the initial ventricular impulse, which was used to assess changes in left ventricular performance. In this study, alterations in the initial ventricular impulse probably reflected changes in left ven-

Table 1. Results of Cardiovascular and Blood Gas Measurements

Event	Mean Arterial Pressure	Total Peripheral Resistance	Cardiac Output	Cardiac Rate	Stroke Volume	Central Venous Pressure	Initial Ventricular Impulse	P_{aO_2}	P_{aCO_2}	pH*
C1	85.4	1080.1	0.5	70.3	81.2	5.2	388.8	89.0	34.0	7.308
T5	−0.5±1.5	−10.0±2.8	5.8±3.0	3.5±2.5	3.4±2.0	−21.2±10.3	5.1±0.8	4.5±2.2	−3.1±3.0	7.387±0.005
15 min.	−10.2±1.8	−13.2±3.3	5.2±4.1	4.1±3.5	2.2±2.7	−17.0±17.0	3.4±5.5	5.3±2.5	−2.8±3.0	7.405±0.007
30 min.	−8.4±1.7	−3.2±3.0	−3.7±3.6	−5.9±2.0	3.3±3.2	−8.3±11.0	3.1±0.2	4.7±2.0	−3.5±3.2	7.300±0.000
60 min.	−3.6±1.2	0.1±4.2	−0.8±4.4	−8.8±3.0	3.0±2.8	8.3±13.4	−5.0±5.5	7.7±2.5	0.7±2.3	7.304±0.005
90 min.	0.4±1.8	4.2±4.0	−2.0±3.7	−7.5±2.4	7.0±3.0	14.0± 0.4	1.7±0.2	0.4±3.0	1.1±2.1	7.382±0.005
C2	90.0	1115.7	6.0	73.7	90.4	5.1	401.0	94.0	34.0	7.380
II+5 min.	−0.8±2.8	2.0±3.1	−10.5±3.8	0.0±3.0	−10.3±2.4	−00.6± 4.7	17.8±8.3	−1.4±1.1	−4.2±2.4	7.398±0.007
II+30 min.	−9.1±2.3	2.0±4.0	−0.5±3.5	8.4±2.0	−10.8±2.2	−00.1± 3.7	11.9±7.3	2.3±2.2	−5.0±2.6	7.398±0.010
T5	−20.2±4.3	−14.0±4.7	−14.8±6.8	11.0±8.8	−22.1±3.0	−70.6± 0.8	8.2±8.0	−0.3±3.5	−11.1±4.0	7.390±0.013
15 min.	−25.8±3.5	−15.4±4.0	−10.0±5.4	8.2±4.9	−17.2±3.0	−55.5± 5.1	3.2±0.0	−1.0±4.2	−3.7±3.0	7.385±0.010
30 min.	−18.7±2.6	−3.2±5.0	−14.1±3.0	−1.5±3.2	−12.0±3.1	−57.5± 0.1	2.4±0.4	−2.0±3.1	−4.0±3.0	7.370±0.007
60 min.	−0.0±1.7	0.9±3.3	−13.9±3.1	4.0±2.5	−17.3±2.3	−52.0± 8.1	1.0±0.0	−0.4±2.0	−3.3±2.2	7.370±0.006
90 min.	−5.9±3.0	3.5±3.0	−8.0±3.0	10.4±2.0	−17.5±2.7	−45.7± 8.1	3.5±8.7	−0.2±2.1	−0.3±2.3	7.370±0.007

Mean per cent change from control ± standard error of mean.
* Mean change from mean of control ± standard error of mean.

tricular contractility.[9] All measurements were made while the subject was supine.

Following the control measurements (C1), subarachnoid block to a T5 sensory level was performed at L3 or L4 with 40 to 50 mg. of 5 per cent lidocaine hydrochloride (Xylocaine) in 7.5 per cent dextrose solution; the dose related to the size of the subject. Cardiovascular and blood gas measurements were repeated when the analgesic level to pin prick reached T5; at 15 and at 30 minutes; and at subsequent 30-minute intervals until cutaneous analgesia disappeared. The average duration of the subarachnoid block was 2.3 hours (range 2.0 to 2.5 hours). In previous studies of blood flow in the lower extremity during subarachnoid block with a Whitney gauge, we found that flow rates actually returned to normal before the disappearance of cutaneous analgesia.[10]

After the subject had again rested for 30 minutes, new control measurements (C2) were obtained. Then 10 ml. of whole blood per kilogram of body weight were removed over a period of 11 to 35 minutes. Cardiovascular and blood gas measurements were repeated at 5 and 30 minutes and the subarachnoid block was repeated with the same dose. In no instance did the resulting analgesic level differ by more than one dermatome from that produced by the first block. Cardiovascular and blood gas measurements were made as before. When cutaneous analgesia had disappeared, at 1.5 hours, the previously-removed blood was reinfused. No intravenous fluids or vasopressors were administered. The study was completed in approximately seven hours.

Cardiovascular and blood gas measurements were converted to per cent change from control values. The significance of the change was determined by the use of Student's *t* test.

Results

The results are summarized in table 1 and figures 1 to 6. Results of measurements after the disappearance of the first subarachnoid block were not statistically different from the first control (C1) values and served as the new control (C2). Because the second control measurements (C2) were made prior to bleeding and to the second subarachnoid block,
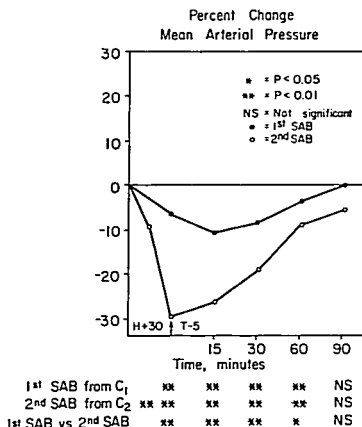

Fig. 1. Per cent change in mean arterial pressure during subarachnoid block (SAB). ●——● = change from C1 under normovolemic conditions; ○——○ = change from C2 under hypovolemic conditions. H + 30 represents the value 30 minutes after removal of blood expressed as per cent change from C2, thus reflecting the effects of hemorrhage. T5 represents the value when the sensory level reached the fifth thoracic dermatome.

changes observed during the second subarachnoid block represented the effects of both hemorrhage and the block.

MEAN ARTERIAL PRESSURE

During the first subarachnoid block the mean arterial pressure decreased a maximum of 10 per cent. Hemorrhage alone produced a 9 per cent fall in the mean arterial pressure from control (C2) ($P < 0.01$). During the second subarachnoid block, mean arterial pressure was almost 30 per cent below control (C2) and remained decreased for 60 minutes. The difference between the pressures before and after hemorrhage was statistically significant ($P < 0.01$ to 0.05) (fig. 1).

TOTAL PERIPHERAL RESISTANCE

Changes in the total peripheral resistance were remarkably similar during both blocks. For the first 15 minutes there was a 10 to 15 per cent decrease in total peripheral resistance ($P < 0.01$), which then returned to
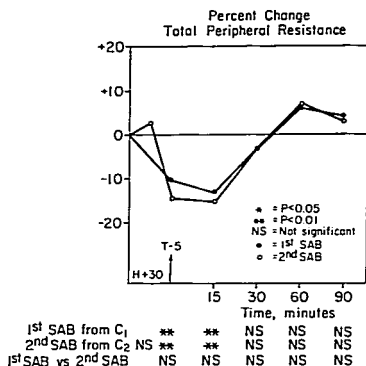
FIG. 2. Per cent change in total peripheral resistance from mean control value. For explanation of symbols, see legend to fig. 1.

the control level. The effect of hemorrhage was minimal, causing an increase of 3 per cent in total peripheral resistance (fig. 2).

CARDIAC OUTPUT

No significant changes in cardiac output were seen during the first block. Hemorrhage reduced the cardiac output by 9 per cent. During the second subarachnoid block there was a significant decrease in the cardiac output for the duration of the anesthetic ($P < 0.01$ to 0.05). During the early phase of the anesthesia, cardiac output was significantly lower after the second subarachnoid block than after the first ($P < 0.01$ to 0.05) (fig. 3).

CARDIAC RATE

During the first subarachnoid block there was an initial insignificant increase in the cardiac rate followed by a significant decrease of 6 to 9 per cent ($P < 0.01$). Hemorrhage produced a 7 per cent increase ($P < 0.05$). During the first 30 minutes of the second subarachnoid block, the changes were similar to those seen during the first block. Cardiac rate then rose above control level as the block began to recede (fig. 4).

STROKE VOLUME

With the first block the increase was significant only at the 90-minute period ($P <$

0.05). Thirty minutes after removal of blood, the stroke volume had decreased 17 per cent ($P < 0.01$). Throughout the second block, it remained significantly below the control value by 12 to 22 per cent ($P < 0.01$). The differences in stroke volume between the normovolemic first anesthetic and the second anesthetic under hypovolemic conditions were highly significant ($P < 0.01$) (fig. 5).

CENTRAL VENOUS PRESSURE

During the first subarachnoid block an initial 20 per cent decrease in central venous pressure was followed by a gradual return to control level and a slight increase as the block disappeared. Thirty minutes after hemorrhage there was a 66 per cent decrease from the control level. During the initial phase of the second subarachnoid block there was a further fall of 4 per cent in central venous pressure, which gradually recovered toward but not to the pre-hemorrhage control level (C2) (fig. 6).

INITIAL VENTRICULAR IMPULSE

Both anesthetic and hemorrhage caused insignificant increases. There were no significant differences between the initial ventricular impulses during the two blocks.
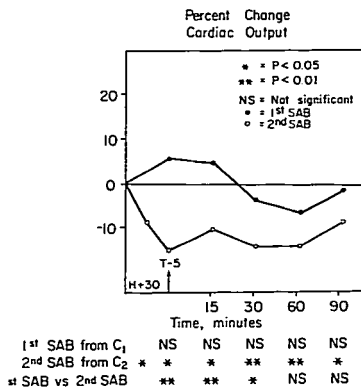

FIG. 3. Per cent change in cardiac output from mean control value. For explanation of symbols, see legend to fig. 1.

Pa_{O_2}

During the initial anesthetic, there was a 4 to 9 per cent increase in Pa_{O_2} ($P < 0.01$). With hemorrhage there was a slight increase. During the second anesthetic Pa_{O_2} did not change significantly from control. At only one point (90 minutes from the onset of the block) was the difference between values recorded during the two blocks significant ($P < 0.05$).

Pa_{CO_2}

There were only minimal changes in the Pa_{CO_2} during the first block. The slight effect of hemorrhage was not significant. During the second block there was a 12 per cent decrease in Pa_{CO_2} when the analgesic level reached T5 ($P < 0.05$). As with the arterial oxygen tension, there was only the one point—90 minutes from the onset of the block—at which there was any statistically significant difference between the values for the two subarachnoid blocks ($P < 0.05$).
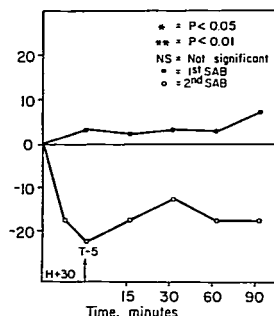
pH

Only minimal changes in the pH occurred during both blocks and following hemorrhage.
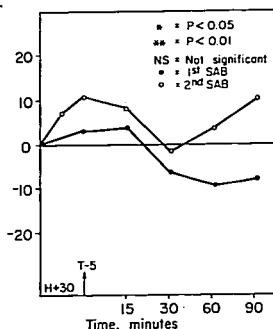
FIG. 4. Per cent change in cardiac rate from mean control value. For explanation of symbols, see legend to fig. 1.

FIG. 5. Per cent change in stroke volume from mean control value. For explanation of symbols, see legend to fig. 1.

Discussion

The significant differences between the effects of subarachnoid block under normovolemic conditions and subarachnoid block in the presence of hypovolemia were found in mean arterial pressure, cardiac output, stroke volume, and central venous pressure. With hemorrhage there was, surprisingly, an increase of only 3 per cent in total peripheral resistance. With both subarachnoid blocks, the decrease in the total peripheral resistance was about the same, indicating that arteriolar constriction was not appreciably greater during hypovolemic block than during normovolemic block.

Although the decrease in central venous pressure was less during the second subarachnoid block than during the first, in both, as the analgesic level receded, the central venous pressure increased to slightly above the control level. This could represent a shift of interstitial fluid to the intravascular compartment. As a reduction in central venous pressure reflects a diminished venous return to the right heart, this decrease in central venous pressure accounted for the decrease in stroke volume. Cardiac rate did not increase suffi-
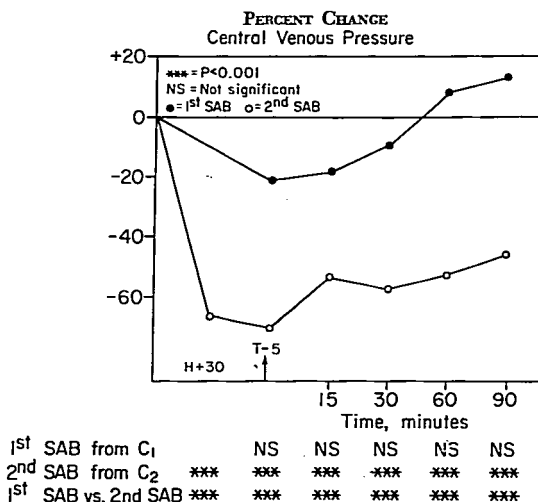
PERCENT CHANGE
Central Venous Pressure

*** = P<0.001
NS = Not significant
● = Iˢᵗ SAB   o = 2ⁿᵈ SAB

	15	30	60	90
Iˢᵗ SAB from C₁	NS . NS	NS	NS	NS	NS

Iˢᵗ SAB from C₁   NS .NS   NS   NS   NS
2ⁿᵈ SAB from C₂ *** *** *** *** *** ***
Iˢᵗ SAB vs. 2ⁿᵈ SAB *** *** *** *** *** ***

ciently to offset the decrease in stroke volume. As a result, cardiac output decreased. Since the total peripheral resistance was unchanged with both anesthetics, the fall in cardiac output accounted for the decrease in the mean arterial pressure.

The initial 17 to 21 per cent reductions in central venous pressure agreed with the 25 per cent decrease observed by Adriani and Rovenstine [14] in their report on the effect of spinal anesthesia on peripheral venous pressure. Sancetta et al.,[6] noted decreases of 53 per cent in right auricular pressure during high spinal anesthesia. Thus, while a decrease in venous pressure is consistently found with high subarachnoid block, there is considerable variation in the literature.

In the presence of hypovolemia, the 12 per cent decrease in Pa_{CO_2} ($P < 0.05$) when the analgesic level reached T5, can be explained by two possible mechanisms. Floyd and Neil [11] have shown that with decreased perfusion of the carotid body there is an increase in chemoreceptor activity providing an increased drive to ventilation. Apprehension is another possible explanation. This, too, has been reported.[3, 12, 13] Whatever the explana-

tion, this again demonstrates that patients with high subarachnoid blocks can still hyperventilate.

Although there was a significant increase in Pa_{O_2} at the 60- and 90-minute periods ($P < 0.01$), there is no apparent explanation for this result as reflected in changes in the Pa_{CO_2} and pH. The mean control value of Pa_{O_2} for the second block (C2) was 4.4 mm. Hg higher than that of the first control, exaggerating the actual difference between values for the two blocks. Had the control values been the same, the differences in Pa_{O_2} would have been less apparent.

The initial ventricular impulse appears to reflect primarily the performance of the left ventricle.[9] Since this parameter was unchanged in both the normovolemic and hypovolemic subjects, left ventricular performance, by this criterion, appears unaffected by subarachnoid block. The stability of this parameter coupled with an unchanged electrocardiogram suggests that significant hypoxia secondary to a fall in coronary perfusion pressure did not occur in these subjects.

It has been demonstrated [15, 16, 17] that high subarachnoid block under normovolemic con-

ditions was accompanied by a significant decrease in splanchnic and coronary, but not cerebral, blood flow. As the reduction in splanchnic and coronary blood flows parallels the decrease in arterial pressure, one can postulate that during subarachnoid block under hypovolemic conditions there could be a further decrease in splanchnic and coronary blood flows. Despite a significant fall in mean arterial pressure from 93 to 63 mm. Hg, Kleinerman [17] observed that the mean cerebral blood flow did not change significantly. A corresponding decrease in cerebral vascular resistance maintained normal flow. Whether this occurs under hypovolemic conditions remains to be studied.

It should be emphasized that subjects used in this investigation were young, healthy unpremedicated volunteers upon whom no surgical procedures had been performed. Patients of lesser physical status might not have compensated as effectively.

Summary

The cardiovascular and respiratory changes which accompanied subarachnoid block under normovolemic and hypovolemic conditions were compared. Marked decreases in the stroke volume accounted for significant reductions in mean arterial pressure and cardiac output after removal of blood. In the presence of acute untreated blood loss, subarachnoid block should be used with discretion.

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