Background: The changes in sympathovagal balance induced by spinal anesthesia remain controversial. The spontaneous baroreflex method allows the continuous assessment of the spontaneous engagement of the cardiac baroreflex, giving an index of sympathovagal balance. The purpose of this study was to follow the effects of spinal anesthesia on spontaneous baroreflex sensitivity.

Methods: Continuous electrocardiogram and noninvasive blood pressure were recorded in 24 patients scheduled for elective inguinal hernia repair and randomly assigned to three groups: (1) no volume loading, (2) volume loading of 15 ml/kg lactated Ringer's solution, and (3) continuous infusion of etilefrine (an epinephrine-like drug). Each patient was studied before, during, and after bupivacaine-induced spinal anesthesia (mean sensory block: T4). Spontaneous baroreflex sensitivity and parameters of time-domain analysis of heart rate variability were calculated from 30 min of recording of each period.

Results: No significant change in spontaneous baroreflex slope or parameters of time-domain analysis were observed after regional anesthesia in any group. However, three patients experienced episodes of bradycardia and hypotension in the absence of a high block; these three patients showed an increase in spontaneous baroreflex sensitivity and time-domain parameters.

Conclusions: Using a noninvasive, continuous technique to estimate cardiac sympathovagal balance, no significant variation in autonomic balance induced by spinal anesthesia was observed. However, untoward episodes of bradycardia and hypotension occurred in three patients, who could not be prospectively identified by the parameters studied. (Key words: Anesthesia technique; spinal. Anesthetic, local; bupivacaine. Autonomic nervous system: sympathovagal balance. Measurement techniques: electrocardiography; heart rate.)

* Staff Anesthesiologist.
† Professor of Physiology.

Received from the Department of Anesthesia, Hôpital E. Herriot, and Laboratory of Physiology, School of Medicine, Lyon, France. Submitted for publication October 31, 1996. Accepted for publication August 7, 1997. Supported by a grant from the Ministère de l’Éducation Nationale, de l’Enseignement Supérieur et de la Recherche (EA 1896) CNRS 5578.

Address reprint requests to Dr. Quintin: Laboratoire de Physiologie, Faculté de Médecine, 8, avenue Rockefeller, 69373 Lyon Cedex 08, France. Address electronic mail to: quintin@cimsun.univ-lyon1.fr

Anesthesiology, V 87, No 6, Dec 1997

Sinus bradycardia associated with hypotension may occur during spinal anesthesia in some patients in the absence of an inordinately high level of block. Rarely, this bradycardia has culminated in cardiac arrest.1-3 An imbalance between sympathetic and parasympathetic control of the heart rate has been suggested among the possible causes responsible for this untoward event. According to this hypothesis, local anesthetic blockade inhibits sympathetic outflow, whereas vagal activity is preserved or enhanced, resulting in an alteration in sympathovagal balance during spinal anesthesia. However, this hypothesis has not been clearly confirmed, with some studies reporting unchanged cardiac sympathovagal balance during spinal anesthesia4-5 and others demonstrating either increased vagal activity6,7 or even a shift toward a sympathetic predominance.8 These contradictory findings could result either from the complexity of the functioning of the autonomic nervous system or from the various methods used to estimate cardiac sympathovagal balance during spinal anesthesia.

Recently, a noninvasive technique was proposed that can assess continuously the relation between high-pressure baroreceptor activity and heart rate. This relation is defined as the spontaneous baroreflex (SBR), the sensitivity of which gives an index of sympathovagal balance.9 Based on simultaneous beat-by-beat analysis of heart rate and blood pressure, this method yields a mean slope of spontaneous engagement of the cardiac baroreflex over a given period of time, defining the SBR sensitivity at any given time.10,11 We recently showed that the SBR slope is closely related to the one calculated after drug-induced changes in pressure and heart rate.12

The objective of this study was to determine whether changes occur in cardiac sympathovagal balance during spinal anesthesia using the SBR method and also a traditional analysis of time-domain heart rate variability. The effects of prophylactic volume loading and vasoconstriction administration on SBR slope were also studied.
Materials and Methods

Patients
The study was approved by the Ethics Committee of the Hospices Civils de Lyon. After giving informed written consent, 24 patients classified as American Society of Anesthesiologists physical status I who were scheduled for elective inguinal hernia repair were studied. All patients were free of cardiovascular, neurologic, or metabolic disease, as assessed by results of a medical history, physical examination, 12-lead electrocardiogram, and chest radiograph. At the time of the study, no patient was receiving concurrent medication.

Regional Anesthesia
Patients were orally premedicated with 70 µg/kg midazolam 1 h before administration of the anesthetic. An intravenous catheter was inserted. Spinal anesthesia was performed with patients placed in a lateral decubitus position. Lumbar puncture was performed with a 25-gauge needle at the L3-L4 or L4-L5 level, followed by injection of 18-20 mg hyperbaric 0.5% bupivacaine in 8% dextrose (Marclain Rachianesthesia; Astra, Nantes, France) into the subarachnoid space. The dose, chosen by the attending physician, depended on the age and height of the patient. Thereafter, the patient was immediately turned to the supine position. No further position change was imposed throughout the surgical procedure. The level of sensory blockade was evaluated by pinprick at 5-min intervals until stability was achieved. Measurements were always performed twice by the same investigator to ensure consistency of the assessment. The upper stable level of spinal blockade, reached at least 20 min after the lumbar puncture, was reported as dermatomal level of loss of painful sensation to pinprick.

Study Protocol
Three 30-min measurement periods were performed in each patient: (1) while premedicated and supine before venous catheter insertion and anesthesia, (2) during spinal anesthesia after spinal blockade achieved a stable level (approximately 20 min after bupivacaine injection), and (3) after complete resolution of the spinal blockade as assessed by the recovery of lower limb mobility and sensitivity. On arrival in the recovery room, before the third measurement period, each patient received a subcutaneous injection of morphine (150 µg/kg). These three measurement periods were those taken for data analysis. Furthermore, for each patient, heart rate and blood pressure were recorded continuously throughout the study.

Each patient was randomly assigned to one of three groups. The first group (n = 8) did not receive any volume loading before anesthesia; the second group (n = 8) received 15 ml/kg lactated Ringer’s solution intravenously over 20 min after the control period recording. Finally, in a third group (n = 8) of patients, a continuous infusion of etilefrine (4 µg·kg⁻¹·min⁻¹) (Effortil, Boehringer Ingelheim, Mainz, Germany) was initiated after the onset of regional anesthesia and continued until resolution of spinal blockade. Etilefrine is a sympathomimetic drug with mixed α- and β-agonist activity similar to ephedrine.¹⁵

Measurements
Continuous signals of electrocardiogram (lead II) and blood pressure were recorded for off-line analysis using an FM cassette recorder (TEAC R61, Tokyo, Japan). An electrocardiogram was obtained using an oscillographic monitor (VSM 1, Physiocontrol, Redmond, WA). Blood pressure was measured continuously using the volume-clamp method and a noninvasive blood pressure monitor (Finapres 2300; Ohmeda, Englewood, CO). This monitor has been shown to provide a reliable beat-by-beat measurement of systolic blood pressure during various autonomic testing conditions when compared with intra-arterial measures.¹⁴ The servo-reset mode of the Finapres monitor was turned off during the recordings and was reset between recordings. As a confirmation, upper limb blood pressure was measured by cuff on the opposite arm to the Finapres, using a Dinamap vital signs monitor (Critikon, Tampa, FL). Recordings were digitized at a sampling rate of 1,000 Hz (DAS-16G, Metrabyte, Taunton, MA) and transferred to a computer for off-line analysis. Electrocardiograph signals were passed through a window-discriminator circuit set to detect R wave peaks. The recordings were observed on an oscilloscope during transfer for elimination of nonsinus beats or artifactual signals caused if the patient moved.

Data Analysis
Spontaneous Cardiac Baroreflex. The distance between all R-wave peaks of the electrocardiogram recording (RR intervals) were paired with the systolic pressure value of the preceding beat (fig. 1A). During each 30-min period of recording, computer software selected all sequences of three or more successive heart beats in which there were simultaneous increases or
CARDIAC BAROREFLEX AND SPINAL ANESTHESIA

**Time-domain Analysis.** In addition to these measures, two parameters related to the analysis of heart rate in the time domain were calculated. The first was the percentage of absolute differences between successive normal RR intervals that exceeded 50 ms (pNN50). The second was the root mean squared successive difference (rMMSD) calculated as follows

\[
r_{\text{MMSD}} = \sqrt{\frac{1}{n-1} \sum_{i=1}^{n-1} (x_i - x_{i+1})^2}
\]

where \( n \) is the number of RR intervals and \( x_i \) is the duration of the \( i \)th interval. These two time-domain parameters are the most commonly used to quantify measures derived from interval differences between consecutive RR intervals, and they estimate short-term variations of RR intervals that are related primarily to parasympathetic control of the heart rate. Any increase in parasympathetic activity leads to an enhanced variation of RR intervals and thus to an increased value of pNN50 and rMMSD.

**Statistical Analysis**

Values are presented as mean ± SD unless otherwise stated. Patient demographic data were compared using one-way analysis of variance. The changes in mean values of RR intervals, systolic and mean blood pressures, SBR sensitivity, and time-domain indices were analyzed by two-way analysis of variance, with repeated measurement on one factor (study period). The analysis of SBR sensitivity and time-domain indices were performed on log transformed data to account for their non-normal distribution. When analysis of variance showed significance, comparison of means were performed by a Scheffé’s test. Because of post hoc selection, no comparison was attempted between patients experiencing an episode of hypotension and bradycardia and the other patients.

**Results**

The three groups of patients were similar with respect to age and weight (40 ± 8 yr, 41 ± 6 yr, 45 ± 13 yr; and 73 ± 9 kg, 73 ± 9 kg, 77 ± 6 kg, respectively) and circulatory data for the three study periods (table 1). Surgery was performed without narcotic or sedative supplementation. The mean doses of hyperbaric bupivacaine were not different among the three groups (table 1). Sensory blockade was achieved rapidly to a mean dermatomal level of T4.
Table 1. Bupivacaine Administered Dosage, Level of Sensory Block, Heart Rate, Systolic Blood Pressure, and Mean Blood Pressure Changes Induced by Spinal Anesthesia for the Three Groups

<table>
<thead>
<tr>
<th></th>
<th>Bupivacaine Dosage (mg)</th>
<th>Sensory Blockade Level (range)</th>
<th>Heart Rate (beats/min)</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Mean Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untreated group</td>
<td>Baseline</td>
<td></td>
<td>65 ± 8</td>
<td>119 ± 13</td>
<td>86 ± 10</td>
</tr>
<tr>
<td></td>
<td>Spinal anesthesia</td>
<td>19 ± 2</td>
<td>63 ± 6</td>
<td>104 ± 16</td>
<td>79 ± 13</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td></td>
<td>68 ± 10</td>
<td>115 ± 16</td>
<td>83 ± 10</td>
</tr>
<tr>
<td>Volume loaded group</td>
<td>Baseline</td>
<td></td>
<td>66 ± 8</td>
<td>114 ± 12</td>
<td>81 ± 10</td>
</tr>
<tr>
<td></td>
<td>Spinal anesthesia</td>
<td>19 ± 2</td>
<td>63 ± 7</td>
<td>115 ± 20</td>
<td>87 ± 17</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td></td>
<td>70 ± 12</td>
<td>113 ± 21</td>
<td>83 ± 15</td>
</tr>
<tr>
<td>Etilefrine group</td>
<td>Baseline</td>
<td></td>
<td>65 ± 11</td>
<td>112 ± 14</td>
<td>82 ± 12</td>
</tr>
<tr>
<td></td>
<td>Spinal anesthesia</td>
<td>20 ± 1</td>
<td>66 ± 9</td>
<td>126 ± 20</td>
<td>91 ± 17</td>
</tr>
<tr>
<td></td>
<td>Recovery</td>
<td></td>
<td>68 ± 15</td>
<td>125 ± 16</td>
<td>90 ± 11</td>
</tr>
</tbody>
</table>

Values are mean ± SD for each set of 30-min measurements of the three study periods. Each set was acquired during stable periods, excluding the hypotension-bradycardia episodes occurring in three patients.

within 20 min after spinal bupivacaine injection, with no difference between groups.

The mean heart rate, and systolic and mean arterial pressures remained fairly stable throughout the study periods (table 1). In patients receiving continuous infusions of etilefrine, systolic blood pressure was slightly but nonsignificantly increased compared with other groups. No significant change in SBR sensitivity was observed after spinal blockade in any group (fig. 2A). Similarly, there was no significant change in the number of baroreflex sequences per 1,000 cardiac beats among the study periods (fig. 2B). The values of pNN50 and rMMSD were not significantly modified by spinal anesthesia in any group (figs. 2C, 2D), although there was a tendency toward lower values in the etilefrine group.

Three patients experienced bradycardia and hypotension during spinal anesthesia that occurred at the 45th, 56th, and 110th min after the time of injection, with final block levels of T5, T4, and T6, respectively. Figure 3 shows the time course of blood pressure and heart rate of these three patients. All three patients remained conscious throughout the bradycardia-hypotension episodes. Ephedrine (5 mg) was administered to one patient. Two patients belonged to the volume-loaded group and the other to the etilefrine group. Table 2 shows individual circulatory data, SBR slope, and the time-domain index for these three patients. Figure 4 is an illustration of the change in RR interval, systolic blood pressure, and SBR sensitivity occurring before and during one of these episodes.

Discussion

The present study examined the effect of spinal anesthesia on sympathovagal balance assessed by the SBR. In patients undergoing spinal anesthesia with a mean block level of T4, the slope of the SBR remains unaltered. However, three patients experienced unexpected episodes of bradycardia and hypotension during the course of spinal anesthesia. These episodes were accompanied by an increase in mean baroreflex slope only during the actual hypotension, indicating greater sensitivity of the cardiac baroreflex.

The baroreflex sensitivity is defined by the ratio of change in heart rate to change in systolic blood pressure. It has been firmly established that these variations in heart rate are brought about by changes in parasympathetic and sympathetic efferent influences on the heart. Thus the baroreflex sensitivity could be viewed as the result of the balance between the two components of the autonomic nervous system. However, the relative role of the two efferent pathways that control the heart rate are not strictly simultaneous and reciprocal. In particular, using autonomic blocking drugs, it was shown that in supine resting conditions, the parasympathetic pathway plays the major role in heart rate control, whereas the sympathetic system provides a more minor modifying influence. Thus, in our study, the baroreflex sensitivity was likely to be influenced primarily by the parasympathetic drive. This same drive is known to influence the time domain indices derived from interval differences between cardiac cycles. They reflected mainly the respiratory sinus arrhythmia, which is under parasympathetic cardiac control.
CARDIAC BAROREFLEX AND SPINAL ANESTHESIA

Fig. 2. Time course of spontaneous cardiac baroreflex and time-domain indices during spinal anesthesia. (A) Spontaneous baroreflex sensitivity (ms/mmHg); (B) number of spontaneous baroreflex sequences per 1,000 heart beats (N beat/1,000 beats); (C) pNN50 = percentage of RR intervals with absolute differences between adjacent RR intervals greater than 50 ms, computed for the 30 min of electrocardiographic recording; (D) rMMSD = square root of the mean of the squared differences between adjacent normal RR intervals computed over the 30-min recording before (baseline), during (block), and after recovery (recovery) of bupivacaaine spinal anesthesia. Values (means ± SD) are reported for the three groups of randomly assigned patients: the untreated group (n = 8); the volume-load group (lactated Ringer’s solution, 15 ml/kg of body weight; n = 8); and the etilefrine group receiving continuous intravenous injection of etilefrine (4 μg·kg⁻¹·min⁻¹ of body weight; n = 8). All values were recorded during stable periods, as stated in the Materials and Methods section, excluding hypotension and bradycardia episodes occurring in three patients. No significant change was observed for the three groups of patients during and after spinal blockade within and between the three groups of patients.

Fig. 3. Time course of systolic blood pressure (closed circles), diastolic blood pressure (open circles), and heart rate (open triangles) of the three patients experiencing an episode of bradycardia and hypotension. Data are shown in relation to the bradycardia–hypotension episode (time 0). Values are mean ± SD.

Our failure to observe any difference in the baroreflex sensitivity induced by spinal anesthesia may have been due to the statistical power of our study.²² Given our sample size, the observed variability derived from the analysis of variance, a 0.05 type I error, and a 0.20 type II error, the smallest difference we could anticipate detecting was 30% of the preanesthetic value. Thus a smaller change in SBR sensitivity could not be ruled out, though this would not likely be of clinical significance. The observed absence of change in SBR sensitivity after induction of spinal anesthesia contrasts with the findings of a previous study. Using vasopressor injections, Baron et al.⁷ reported a 40% increase in baroreflex sensitivity induced by low-level epidural anesthesia ranging from T8–T12. The proposed explanation for this variation was a decreased venous return that lowered the activity of cardiopulmonary receptors, which have a tonic inhibitory action on the parasympathetic system. According to this hypothesis, we should have observed a change in baroreflex slope induced by spinal anesthesia with a T4 level of anesthesia. This level is likely to induce a more pronounced change in venous return due to an increase in splanchic capacitance than is an T8–T12 anesthesia level.²³ Further, we might have expected that the three groups of patients would have behaved differently, because volume loading or etilefrine infusion should modify venous return by increasing the volume of blood of the splanchic area, or by decreasing splanchic capacitance. Another explanation of the discrepancy between the study of Baron et
al. and the present one could be ascribed to the way the baroreflex slopes were acquired. The mean SBR slope represents baroreflex sensitivity at a point close to the resting point of blood pressure, whereas the drug-induced baroreflex slope assesses the baroreflex sensitivity over an extreme range of induced pressure variation. However, in a validation study, the baroreflex sensitivity by both the SBR and the drug-induced methods were found to change in a parallel manner on autonomic blockade. Thus the discrepancy between Baron et al.’s study and ours may relate to the time dependency of the two techniques. The SBR method continuously measures cardiac baroreflex sensitivity in the normal physiologic range of blood pressure over a period of time, whereas the drug-induced baroreflex method measures extreme induced blood pressure perturbations during a brief period. Indeed, our results acquired in three patients experiencing bradycardia and hypotension clearly showed that parasympathetic activity may change over time in one single patient.

The stable sympathovagal profile under spinal anesthesia is in keeping with several previous studies examining indices of heart rate variability to assess autonomic nervous system activity. These studies reported unchanged sympathovagal balance during regional anesthesia in adult patients and in infants. Only two studies mentioned a relatively increased parasympathetic activity, whereas another suggested a shift toward sympathetic predominance. In the current study, the observation of unchanged sympathovagal balance under spinal anesthesia is further reinforced by the lack of change in the time-domain indices, pNN50 and rMMSD. This stable autonomic balance could be the result of a constant level of parasympathetic activity associated with unchanged sympathetic outflow. However, the mean level of sensory block was T1 and thus the sympathetic block was two or more dermatomes higher, which would be expected to decrease sympathetic outflow originating from T1 to T4. Nevertheless, it is possible that sympathetic outflow could remain unchanged even in the presence of a high spinal anesthesia if the sympathetic tonic activity begins at a lower level before spinal blockade. This could be the case in our patients because the vagal outflow has been shown to be largely predominant over a low sympathetic activity in supine resting conditions. On the other hand, unchanged sympathovagal balance during regional blockade could be obtained by a simultaneously decreased activity of both components of the autonomic nervous system, which has been suggested by previous work using heart rate analysis in the frequency domain. It has been hypothesized that the reduction of the parasympathetic activity is a reflex response triggered by a reduction of the sympathetic activity, due to the existence of a reciprocal relation between these two components. However, during high spinal anes-

---

Table 2. Individual Circulatory Data, Spontaneous Baroreflex (SBR) Slopes, and Time Domain Indices Data for the Three Patients Experiencing Bradycardia–Hypotension Episodes

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Block Level</th>
<th>Heart Rate (beats/min)</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Mean Blood Pressure (mmHg)</th>
<th>SBR Slope (ms · mmHg⁻¹)</th>
<th>Sequence No. (/1,000 beats)</th>
<th>pNN50 (%)</th>
<th>RMMSD (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Baseline</td>
<td>T₅</td>
<td>73 ± 3</td>
<td>116 ± 6</td>
<td>84 ± 4</td>
<td>8.5</td>
<td>95</td>
<td>0</td>
<td>11.3</td>
</tr>
<tr>
<td>Spinal anesthesia</td>
<td>62 ± 7</td>
<td>110 ± 13</td>
<td>87 ± 10</td>
<td>14.1</td>
<td>78</td>
<td>3</td>
<td>23.3</td>
<td></td>
</tr>
<tr>
<td>Bradycardia–hypotension</td>
<td>58 ± 3</td>
<td>77 ± 6</td>
<td>61 ± 6</td>
<td>18.7</td>
<td>66</td>
<td>9</td>
<td>80.2</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>81 ± 3</td>
<td>113 ± 6</td>
<td>80 ± 4</td>
<td>7.44</td>
<td>222</td>
<td>1</td>
<td>23.2</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>64 ± 6</td>
<td>135 ± 5</td>
<td>92 ± 4</td>
<td>20.8</td>
<td>101</td>
<td>56</td>
<td>105.9</td>
<td></td>
</tr>
<tr>
<td>Spinal anesthesia</td>
<td>54 ± 17</td>
<td>112 ± 12</td>
<td>87 ± 9</td>
<td>32.3</td>
<td>70</td>
<td>63</td>
<td>192.4</td>
<td></td>
</tr>
<tr>
<td>Bradycardia–hypotension</td>
<td>45 ± 1</td>
<td>62 ± 6</td>
<td>46 ± 5</td>
<td>37.8</td>
<td>57</td>
<td>73</td>
<td>196.2</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>57 ± 6</td>
<td>109 ± 8</td>
<td>80 ± 6</td>
<td>20.5</td>
<td>92</td>
<td>56</td>
<td>116.3</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>51 ± 2</td>
<td>137 ± 5</td>
<td>106 ± 3</td>
<td>15.9</td>
<td>42</td>
<td>6</td>
<td>26.7</td>
<td></td>
</tr>
<tr>
<td>Spinal anesthesia</td>
<td>56 ± 3</td>
<td>153 ± 9</td>
<td>115 ± 7</td>
<td>22.2</td>
<td>49</td>
<td>18</td>
<td>38.7</td>
<td></td>
</tr>
<tr>
<td>Bradycardia–hypotension</td>
<td>40 ± 5</td>
<td>97 ± 9</td>
<td>75 ± 10</td>
<td>33.01</td>
<td>59</td>
<td>37</td>
<td>70.3</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>47 ± 2</td>
<td>128 ± 5</td>
<td>97 ± 4</td>
<td>9.4</td>
<td>57</td>
<td>10</td>
<td>31.2</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD for each set of 30-min measurements of the three study periods and for the bradycardia–hypotension events. Spinal anesthesia values were recorded out of bradycardia–hypotension episode.
Fig. 4. Circulatory variables and spontaneous baroreflex sequences during bradycardia-hypotension in one patient who received 5 mg ephedrine to restore blood pressure (arrow). Baroreflex sequences were searched before (time interval and line 1), during (time interval and line 2), and after the bradycardia–hypotension episode (time interval and line 3); the sequences detected during the third interval (recovery) are omitted for clarity. The mean slope of each time interval is shown (heavy tracing). Note (1) the abrupt drop in blood pressure; (2) the large oscillations in RR intervals during the second period (600 and 2,000 ms); i.e., instantaneous heart rate was oscillating 30–90 beats/min on a beat-by-beat basis; (3) the large increase in baroreflex slope that occurred simultaneously with bradycardia and hypotension and returned toward baseline value after restoration of pressure.

Anesthesiology, V 87, No 6, Dec 1997

Caplan et al., these events occurred suddenly and unpredictably, with hemodynamics apparently stable minutes before. Although the current study was not designed specifically to address this issue, it suggests that patients who experienced bradycardia and hypotension episodes did not have a high parasympathetic activity at baseline (i.e., before spinal anesthesia).

The association of hypotension with bradycardia can only be explained by a significant alteration in the balance between the two limbs of the autonomic nervous system: withdrawal of sympathetic activity,
parasympathetic activation, or both. The present observation is in line with several previous published observations: reduced calculated vascular resistance evoked by fear, reduced sympathetic nerve activity observed during nitroprusside challenge, and increased oscillation of heart rate observed immediately after cessation of exercise. However, the cause of the episodes of hypotension and bradycardia is not clear. First, suprabulbar inputs generated by emotion may affect the sympathetic premotoneurons in the vasomotor center leading to reduced sympathetic activity. Suprabulbar inputs may also affect the cardiac vagal motoneurons in the nucleus ambiguus, leading to their activation with resulting bradycardia. In an analogous manner, the stimulation of the ventral periaqueductal gray matter leads to hypotension and bradycardia, the basis of the “playing dead” reaction. Similarly in humans, α-adrenergic agonists induce relative hypotension and bradycardia due to a combination of decreased sympathetic activity and increased parasympathetic activity. Second, ventricular mechanoreceptors may become activated in the presence of low end-diastolic volume. This can be observed during severe hemorrhage or, during spinal anesthesia, an increase in venous capacitance. In turn, the ventricular mechanoreceptors may trigger arterial dilatation and bradycardia. Regardless of the central or peripheral genesis of the hypotension and bradycardia episodes, the striking phenomenon is a resetting toward lower pressure combined with an increase in sensitivity of the cardiac baroreflex (fig. 4). This observation is in line with the suggestion that the baroreflex is intact but “switched off.” There may be a continuum of response for the cardiac baroreflex ranging from the “playing dead” reaction (reduced set point, increased sensitivity) to the adaptation to exercise (increased set point, reduced sensitivity), the so-called fight or flight response.

In conclusion, using a noninvasive, continuous technique to estimate cardiac sympathovagal balance, we failed to observe any significant variation of the autonomic nervous system balance during spinal anesthesia to a T₁ block level. However, three patients suffered episodes of bradycardia and hypotension in the absence of high block, which were associated with evidence of increased parasympathetic activity. These patients could not be identified prospectively.

The authors thank Professor Petit for his support.

References

17. Anonymous: Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the Euro-
CARDIAC BAROREFLEX AND SPINAL ANESTHESIA


37. Inui K, Murase S, Nosaka S: Facilitation of the arterial baroreflex by the ventrolateral part of the midbrain periaqueductal grey matter in rats. J Physiol (Lond) 1994; 477:89-101


