POWER SPECTRAL ANALYSIS OF HEART RATE VARIABILITY
AFTER SPINAL ANAESTHESIA

M. KAWAMOTO, N. TANAKA AND M. TAKASAKI

SUMMARY
Power spectral analysis of heart rate (HR) variability is a useful tool with which to assess cardiac autonomic activity. The low frequency bands have been considered as both sympathetically and parasympathetically mediated components, while the high frequency bands have been assumed to be the parasympathetically mediated respiratory components. It has been anticipated that spinal anaesthesia to the thoracic level may modulate cardiac autonomic activity to reduce HR and arterial pressure by blocking cardiac sympathetic activity. In order to quantify the alterations in cardiac autonomic activity, we have analysed the power spectra of HR variability for 30 min after subarachnoid administration of hyperbaric amethocaine. Using 256-s R-R interval data obtained from continuously recorded ECG, low frequency (Lo: 0.04-0.15 Hz) and high frequency (Hi: 0.15-0.40 Hz) band widths were integrated and their serial alterations were computed by shifting subjected R-R intervals at 60-s intervals. After the subarachnoid injection, arterial pressure, HR and Lo decreased and Hi and the Hi:Lo ratio increased. These changes were observed within 15-20 min. Ventilatory frequency did not change throughout the study. These findings suggest that the decrease in HR and arterial pressure after subarachnoid administration of hyperbaric amethocaine reflect decreased sympathetic activity and increased parasympathetic activity in the cardiac autonomic nervous system. (Br. J. Anaesth. 1993; 71: 523-527)

KEY WORDS

Bradyarrhythmia associated with spinal anaesthesia occurs commonly when a high level of block is produced. Both unopposed parasympathetic activity and reduced sympathetic tone are thought to be the causes [1]; of the two neurogenic mechanisms, increased parasympathetic activity is considered more important [1, 2]. However, the respective sympathetic and parasympathetic contributions have not been quantified clearly, because no effective monitor is available during anaesthesia.

The cardiac autonomic nervous system plays a vital role in neural control for maintaining cardiovascular stability. To elucidate its contribution to cardiac sinus rhythm, we have used power spectral analysis of heart rate (HR) variability to study the physiological and pathophysiological properties of the system [3-5]. Two spectral components of HR variability have been observed in dogs and humans: a low frequency component, considered to be of vasomotor origin mediated by the parasympathetic and sympathetic systems, and a high frequency component, centered at the frequency of ventilation mediated primarily by the parasympathetic system. A decreased high frequency component during isoflurane anaesthesia has been reported [6] and shown to be dose related [7]. However, no study has been carried out during regional block including spinal anaesthesia.

The purpose of this study was to examine the effect of hyperbaric spinal anaesthesia on the cardiac autonomic nervous system using successive power spectral analyses of HR variability, calculated from the digitized R-R intervals from taped ECG.

PATIENTS AND METHODS
We studied 26 ASA class I and II patients having elective lower abdominal or limb surgery under spinal anaesthesia. After approval by the local Research Review Board, informed consent was obtained from all participants. Patients with a history of cardiovascular disease, arrhythmia or cardiac autonomic nervous system diseases such as diabetes mellitus were excluded from the study. No patient was receiving concurrent medication.

Each patient received midazolam 0.1 mg kg^-1 1 h before induction of anaesthesia. Atropine was not given. On arrival in the operating room, patients were placed in the supine position and routine monitors including pulse oximetry, ECG (lead II), impedance respiratory signals and automated sphygmomanometry were started (BSM-8500, Nihon Kohden, Tokyo, Japan). An i.v. cannula was inserted and 6% hydroxyethylated starch in saline admin
istered at a rate of 10 ml kg⁻¹ h⁻¹ for 30 min, followed by Hartmann’s solution 10 ml kg⁻¹ h⁻¹. With the patient in the left lateral position, the extradural space was located at L1-2 with a 17-gauge needle and a catheter was placed cephalad. A 25-gauge spinal needle was inserted at L2-3 and a small dose of hyperbaric 0.5% plain amethocaine in 10% glucose was injected at a rate of 0.2 ml s⁻¹ without barbotage. No injection was made through the catheter at this time.

Timing commenced from the start of the spinal injection. The patient was then returned to the supine position. Baseline variables were measured before spinal injection in the supine position and at 5, 10, 15, 20, 25 and 30 min after spinal injection. Consecutive recordings of impedance respiratory signals and ECG were made onto magnetic tape for 256 s at baseline and for 30 min during the study. The dermal level of anaesthesia was determined by absence of sensation to pinprick in the mid axillary line bilaterally at 5, 10, 15, 20, 25 and 30 min. Surgery proceeded when an adequate block was achieved more than 30 min after spinal injection. Routine management of the patient was not affected by their inclusion in the study; patients who required ephedrine 4-8 mg to treat hypotension (≤ 80 mm Hg systolic) or atropine 0.25-0.5 mg for bradycardia (≤ 50 beat min⁻¹) were excluded from the study, as these vasoactive agents modify the power spectrum. No patient was placed in the Trendelenburg position. All were kept awake and supine during the study. If spinal anaesthesia was thought insufficient at 30 min, extradural anaesthesia was commenced.

### Data analysis

Mean arterial pressure (MAP) was calculated as:

$$\text{MAP} = \frac{[(\text{systolic pressure}) + 2 \times (\text{diastolic pressure})]}{3}$$

The monitor was equipped with a high-cut filter above 5 Hz for impedance respiratory signals and a drift-free filter for the ECG channel. Ventilatory frequency (f) was determined from impedance respiratory signals.

The ECG channel was recorded onto magnetic tape (RD-111T, TEAC, Tokyo, Japan) and digitized at 500 Hz for off-line analysis. ECG was recorded with care to avoid artefacts such as muscle contractions and electrical noise. The computer program processed the digitized data using a 14-bit A-D converter-equipped desk-top computer system (PC98, NEC, Tokyo, Japan) and detected QRS complexes and R waves. The program measured the time difference between two R waves to obtain an R–R interval tachogram (fig. 1A, a) [8, 9]. Instantaneous HR data from 256-s R–R interval segments were converted to 1/R–R interval length by sampling at 4 Hz and the 256-s segment of R–R intervals was subjected to off-line spectral analysis because the raw R–R data were inappropriate for fast Fourier transform analysis. For fast Fourier transform-based power spectral analysis, a rectangular local window periodogram method was used as a low-pass anti-aliasing digital filter beyond the Nyquist sampling rate (2 Hz), which enabled computation of reliable spectral estimates between 0 and 1 Hz [8, 9]. The power spectra at frequencies less than 0.5 Hz were standardized as the square of the mean of HR (Hz⁻¹) (fig. 1C) [8, 9].

At every 60-s interval, the analysis was repeated on successive records of R–R interval data; by overlapping the R–R interval data of 196 s duration, the power spectral analysis was carried repeatedly using the next incoming 60-s segment of R–R interval data (fig. 1B). Spectral accuracy was confirmed by testing a sequence of simulated R–R intervals generated by an integral pulse frequency modulation model [9]. The spectra were quantified by determining the areas of the spectrum in two components: low frequency (Lo: 0.04–0.15 Hz) and high frequency (Hi: 0.15–0.40 Hz) band areas. The peak areas of the power spectral densities were integrated and the Hi:Lo ratio computed [3, 10]. Log power of these peak areas and the Hi:Lo ratio were calculated by taking their common logarithms (base 10) from confirmed normal distributions [5, 8, 11, 12].

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**FIG. 1.** Method of power spectrum analysis from stored R–R interval tachograms obtained from continuously recorded ECG. A: Recorded ECG. B: R–R interval tachogram. C: Each 256-s R–R interval is transformed into 1024 instantaneous heart rate for power spectral analysis based on fast Fourier transform. D: A power spectrum from 256-s interval data.
Data of normal distribution are presented as mean (SD). From segments of R–R interval tachograms at each sampling time, simple statistics (mean and variance) were calculated. HR was calculated from the reciprocal of R–R interval. Variations in ventilatory frequency, MAP, R–R interval and R–R variance were compared using one-way analysis of variance (ANOVA) for repeated measurements, followed by Scheffe’s test. Variations in log power and HR calculated from R–R interval tachograms were compared repeatedly using ANOVA, followed by Fisher’s protected least significant difference test. The thoracic dermal level of anaesthesia was recorded as median (range) and compared using Wilcoxon matched pairs signed rank test for paired comparisons (right vs left side), as a normal distribution could not be assumed. Comparisons between the median level of anaesthesia and mean log Hi:Lo used linear regression analysis and Kendall’s rank correlation. \( P < 0.05 \) was considered statistically significant.

**RESULTS**

No serious complications including hypoxaemia were observed in any of the 26 participants. However, six patients were excluded from the study as they received ephedrine \((n = 3)\), atropine \((n = 1)\) and both ephedrine and atropine \((n = 2)\) within 30 min of the spinal injection. Subjects for the study thus consisted of 20 patients \((13 \text{ female})\) aged 21–56 yr \((\text{mean} 36 \text{ yr})\). The mean dose of hyperbaric amethocaine solution was 1.80 \((0.14)\) ml \((\text{range} 1.6–2.0 \text{ ml})\). No patient required extradural analgesia at 30 min. No serious complications including hypoxaemia were observed in any of the 26 participants. How- ever, six patients were excluded from the study as they received ephedrine \((n = 3)\), atropine \((n = 1)\) and both ephedrine and atropine \((n = 2)\) within 30 min of the spinal injection. Subjects for the study thus consisted of 20 patients \((13 \text{ female})\) aged 21–56 yr \((\text{mean} 36 \text{ yr})\). 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the range < 0.5 Hz. The former is considered to reflect the sympathetically and parasympathetically mediated outflows and the latter (respiratory sinus arrhythmia) to reflect parasympathetic vagal efferent activity [3, 5, 13]. Lo reflects both sympathetic and parasympathetic input to the heart, because pharmacological blocking studies suggest that parasympathetic reflexes are involved [12]. Hi primarily represents the variation caused by respiratory sinus arrhythmia. The Hi:Lo ratio is thus considered a convenient index of the interaction of the two neural systems [3, 8, 14].

The bandwidth for integration varies between reports, as it has been analysed by different methods [3, 5, 8, 10, 14, 15]. The oscillations about a frequency of 0.1–0.25 Hz comprise the main component of information on neural modulation of the sinoatrial node [12]. Oscillations with a frequency < 0.03 Hz cannot be described accurately, hence those components between 0 and 0.03 Hz are considered to be d.c. noise [3, 12]. The clearest sympathetic component is that which centres around 0.1 Hz; consequently, we analysed our data with the window for Lo set at 0.04–0.15 Hz. Some reports subdivide Lo into two components: very low frequency (0.04–0.10 Hz) and a mid-frequency band (0.10–0.15 Hz) [16]. The latter component may be related to the frequency response of the baroreceptor reflex, which was not investigated in this study. The high frequency component (Hi: 0.15–0.4 Hz) is reported as being proportional to tidal volume and inversely proportional to ventilatory frequency [13, 17]. As Hi disappears after atropine, it may also represent a projec- tion of information on neural modulation of the sinoatrial node [12]. Oscillations with a frequency of 0.1–0.25 Hz are considered to reflect the sympathetically and parasympathetically mediated outflows of the central nervous system and the cardiac autonomic nervous system may be involved in this mechanism [6, 7]. However, the results observed in this study may reflect a different mechanism. Time-dependent sympathetic cephalad block might mainly reduce sympathetic activity, as the preganglionic sympathetic cardiac accelerator fibres leaving the spinal cord at T1–4 are easily blocked at higher levels of spinal anaesthesia. Cardiac autonomic nervous system activity is known to be depressed by spinal analgesia, as sympathetic block may extend two to six dural segments higher than sensory loss [1, 18]. Our dural level of sympathetic block may have been sufficiently higher than T1–2 to block cardiac accelerator fibres.

It was not clear why spinal analgesia increased parasympathetic activity as shown in this study. We presumed two major reasons: ventilation-mediated enhancement of parasympathetic activity and parasympathetic reflex mechanisms in the heart. Ventilation mediates parasympathetic activity via the vasomotor centre and modulates cardiac sinus node activity: it can influence both Lo and Hi [17]. As shown in our study, ventilatory frequency appears not to play an important role in this mechanism, although tidal volume was not measured.

Within 20 min after the spinal injection, Lo and Hi seemed to have reached their maximal upper dermal level. Simultaneous alterations in HR and MAP were also observed with changes in Lo and Hi. These findings suggest that spinal anaesthesia affects both sympathetic and parasympathetic activity to make the cardiac autonomic nervous system vagotonic.

REFERENCES


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