Heart rate variability during propofol anaesthesia

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SUMMARY

We have examined the effect of i.v. propofol anaesthesia on beat-to-beat heart rate variability. The power in all frequency bands (0.02-0.08, 0.08-0.15, 0.15-0.45 Hz) decreased, although the reduction in lower power was significantly less than that for mid (P = 0.001) and high (P = 0.04) power. (Br. J. Anaesth. 1994; 72: 219-220)

KEY WORDS

Anaesthetics, i.v., propofol Heart: heart rate variability.

Beat-to-beat heart rate variability (HRV) is caused by the fluctuating balance of sympathetic and parasympathetic tone at the sino-atrial node. These autonomic inputs are driven by the central nervous system in response to the interaction of ventilation, baroreflexes and fluctuations in vasomotor tone. Three periodicities are present in normal HRV: a low frequency component at 0.05 Hz, a mid frequency component at 0.1 Hz and a higher frequency component in phase with ventilation. Low and mid frequency components are mediated by the sympathetic and parasympathetic nervous systems, while ventilatory fluctuations (> 0.15 Hz) are solely parasympathetic [1]. Components of HRV are affected variably by inhalation and i.v. anaesthetics [2, 3], nitrous oxide [4] and opioids [3]. In this present study we have examined the effect of total i.v. anaesthesia with propofol on HRV.

METHODS AND RESULTS

After local Ethics Committee approval and patient consent were obtained, we studied 18 ASA I patients (13 male) undergoing general anaesthesia for day-case surgery. Mean age was 29.1 yr (range 21-38 yr) and all were fasted and unpremedicated. Patients were monitored non-invasively using pulse oximetry, intermittent non-invasive arterial pressure (Dinamap) and continuous ECG (CM5). Anaesthesia was induced with propofol 2.0 mg kg⁻¹ injected over 30 s, followed by an infusion of 0.17 mg kg⁻¹ min⁻¹, increased if necessary to maintain loss of eyelash reflex and spontaneous ventilation (15 min after induction, the mean infusion rate was 0.19 mg kg⁻¹ min⁻¹). Patients breathed room air unless the oxygen saturation decreased to 95% or less. In two patients, ventilation was assisted for 15 and 30 s with 100% oxygen.

The ECG output was interfaced to a computer system which determined and stored the value of consecutive R–R intervals. As described previously [4], 5-min segments were processed using Fourier analysis to provide the power spectrum of the heart rate time series. These segments were taken from the 5 min immediately before induction and from the 10–15 min after induction. Total spectral power was calculated by integrating the power spectra between 0.02 and 0.45 Hz. Low, mid and high frequency bands were calculated by integrating the frequency ranges 0.02–0.08 Hz, 0.08–0.15 Hz and 0.15–0.45 Hz. Comparison of measurements obtained before and after induction were made using a paired Student’s t test for R–R interval, sn of R–R interval and mean arterial pressure. Spectral data were analysed using the Wilcoxon sign rank test. P < 0.05 was considered significant.

There was a small, non-significant increase in heart rate, a significant reduction in sn of heart rate and a significant decrease in mean arterial pressure (table I). Total spectral power decreased, while individual component periodicities were affected differently. The power in all frequency bands decreased, although the decrease in low power was significantly less than that for mid (P = 0.001) and high (P = 0.04) power. Reductions in mid and high frequency powers were not significantly different (P = 0.5). Thus the proportion of spectral power in the low frequency band increased while that in the mid frequency range decreased. The proportion of high frequency power was decreased, but not significantly.

COMMENT

We have observed previously that nitrous oxide–isoflurane anaesthesia was associated with marked depression of total power (59%) and component frequencies (low 61%; mid 74%; high 53%) of the HRV power spectrum [2]. Using propofol as the sole anaesthetic agent in the present study, we observed a relative preservation of low frequencies, in contrast with the more marked reduction of mid and high frequency components. A recent study [3] observed a lesser reduction of power in the range < 0.125 Hz compared with higher frequencies. Our own study separated the frequency bands 0.02–0.08 Hz and 0.08–0.15 Hz, observing a shift to, or preservation
of, the lowest frequencies. This is similar to our previous observation of slow heart rate fluctuations with a mean frequency of 0.056 Hz (range 0.049–0.059 Hz) after induction of anaesthesia with propofol. Therefore, our observations suggest that propofol, unlike isoflurane–nitrous oxide, may be associated with relative preservation of low frequency (< 0.08 Hz) components of HRV.

It is believed that the two spectral components at frequencies less than 0.15 Hz are mediated, in part, via cardiac sympathetic efferents, as they are diminished by beta adrenergic block, are absent in quadriplegic humans and are exaggerated by agents and manoeuvres which demand a sympathetic response — for example vasodilators, haemorrhage and standing [6]. The low frequency component centred between 0.04 and 0.08 Hz may reflect thermoregulatory fluctuations in peripheral vasomotor tone or a vasomotor fluctuation in peripheral vascular beds matching blood flow to demand. Low frequency fluctuations are enhanced by ACE inhibitors and alpha adrenergic antagonists. Although a direct myocardial depressant action has been reported for propofol, many studies have indicated that propofol produces significant reductions in systemic vascular resistance or venous capacitance. It is possible that the low frequency oscillations which appear to be relatively maintained with propofol are related to this peripheral vascular action. This might indicate, for example, the response of the cardiovascular system to altered capacitance and may suggest that propofol preserves, at least in part, those mechanisms which regulate peripheral vascular beds or maintain thermoregulatory control.

In many studies, the ratio of spectral power < 0.15 Hz to that > 0.15 Hz is used as a measure of sympathetic/parasympathetic balance. Our own study and that of Latson and O’Flaherty [5] show a shift towards lower frequencies in the HRV spectrum, suggesting sympathetic dominance. Our present observations suggest that the two frequency ranges less than 0.15 Hz may be affected differently and do not reflect identical physiological processes, and thus may be changed by mechanisms other than sympathetic activation. It is likely, therefore, that there is not a simple relationship between power < 0.125 Hz and sympathetic activation.

In conclusion, we have observed that spectral analysis of heart rate variability during propofol infusion showed relative preservation of low frequency “vasomotor” fluctuations (< 0.08 Hz) with markedly attenuated mid (0.08–0.15 Hz) and high (> 0.15 Hz) frequency ventilatory oscillations. These observations differ from those changes we have made previously during isoflurane–nitrous oxide anaesthesia, when there was depression of all frequency components of HRV. These effects may relate to an action of propofol on vascular control.

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REFERENCES