Effect of Controlled Breathing on Short-term Cardiovascular Variability: An Investigation in Chronic Heart Failure Patients

GD Pinna [1], R Maestri [1], E Robbi [2], M Gnemmi [2], MT La Rovere [2]

[1] Biomedical Engineering Unit, [2] Division of Cardiology,
S. Maugeri Foundation, IRCCS, Scientific Institute of Montescano, Italy

Abstract

Voluntary control of breathing (CB) has been proposed as a means to standardize short-term investigations on cardiovascular variability (CVV). In this study we assessed the changes induced by CB on ventilatory and cardiovascular parameters in 17 chronic heart failure (CHF) patients. We recorded instantaneous lung volume (ILV), RR interval, systolic arterial pressure (SAP) and end-tidal CO_2 ($ETCO_2$, 10 patients) during 8' of spontaneous breathing (SB) and 8' of CB at 0.22 Hz. CB caused a – 14% reduction in Respiratory Rate, a + 57% and +27% increase respectively in Tidal Volume and Minute Ventilation and a –7% decrease in $ETCO_2$. A negligible change was observed in mean RR and SAP. No significant change was observed in LF power of RR and SAP and in baroreflex sensitivity, whereas HF power increased markedly. Hence, CB in CHF patients causes a mild hyperventilation but does not seem to influence autonomic cardiovascular control. Only respiratory-related oscillations of CVV signals are affected by CB.

1. Introduction

It has long been known that respiration exerts a marked influence on spontaneous fluctuations of RR interval and arterial blood pressure and that this influence is dependent on the rate, depth and pattern of breathing activity [1]. These characteristics of breathing, in turn, may change within- and between subjects, even though experimental conditions are kept strictly homogeneous. As a consequence, voluntary control of respiration, either through simple paced breathing [2] or including control of tidal volume [1], has been advocated as a means to standardize short-term (< 10 min) laboratory investigations on cardiovascular variability (CVV) [1]. Moreover, in some occasions, CVV may be fully entrained by respiratory activity, e.g. when respiratory rate spontaneously slows down to the frequency of the Mayer rhythm (≈ 0.1 Hz) or during periodic breathing [3]. In these instances, control of breathing may also be used as a means to avoid confounding [4].

Consensus on the use of controlled breathing in CVV analysis, however, has not been reached yet. Some investigators have argued that paced breathing in the range 0.2-0.3 Hz shifts the sympathovagal balance in favor of the vagal component [5], while others have maintained the opposite, namely that the act of voluntary control of breathing causes a mild mental stress leading to a reduced parasympathetic influence to the heart [6]. In between the two, a third group of investigators have recently suggested that changes in breathing frequency, with or without tidal volume control, do not alter the absolute level of cardiac-vagal nerve activity [2]. Of note, all these studies have been carried out on healthy individuals.

In the light of these highly contradictory results it is hard for an investigator to soundly decide as to whether using or not controlled breathing during laboratory recordings, this choice being particularly difficult when measurements are performed on pathological subjects.

On the grounds of these considerations, we carried out this study on a population of stable CHF subjects with the aim of assessing the changes induced by controlled breathing on ventilatory and CVV parameters as well as on indirect measurements of vagal and sympathetic activity, namely heart rate and arterial blood pressure. Leave one line space above and below all headings from now on.

2. Methods

We considered for the study moderate to severe CHF patients (NYHA class: II-III, left ventricular ejection fraction: 26±7%) consecutively admitted to our laboratory for the assessment of the autonomic function. Inclusion criteria were: i) stable clinical conditions, ii) sinus rhythm, iii) no previous history of pulmonary or neurological disease, iv) ectopy rate < 5%. The experimental protocol comprised: 1) instrumentation and signal stabilization (15 min), 2) 8 min supine resting recording of ECG, calibrated instantaneous lung volume (ILV) by inductive plethysmography (Respitrace Plus),

0276-6547/01 $17.00 © 2001 IEEE

Computers in Cardiology 2001;28:217-219.

noninvasive arterial blood pressure at the finger (Finapres 2300) and arterial oxygen saturation at the ear (SaO_2, Biox 3740), 3) 8 min supine resting recording during paced breathing at 0.25 Hz. Pacing was induced using a digitally recorded human voice signaling to breath "in" and "out" with a ratio 2:3 between inspiration and expiration. A few minutes trial was allowed to let the patient become familiar with the experiment. To the purpose of improving patient's comfort, upon request, the breathing frequency was reduced up to a minimum value of 0.2 Hz. In a subset of patients breath-by-breath CO2 (Beckman LB-12) was also monitored during both spontaneous and paced breathing.

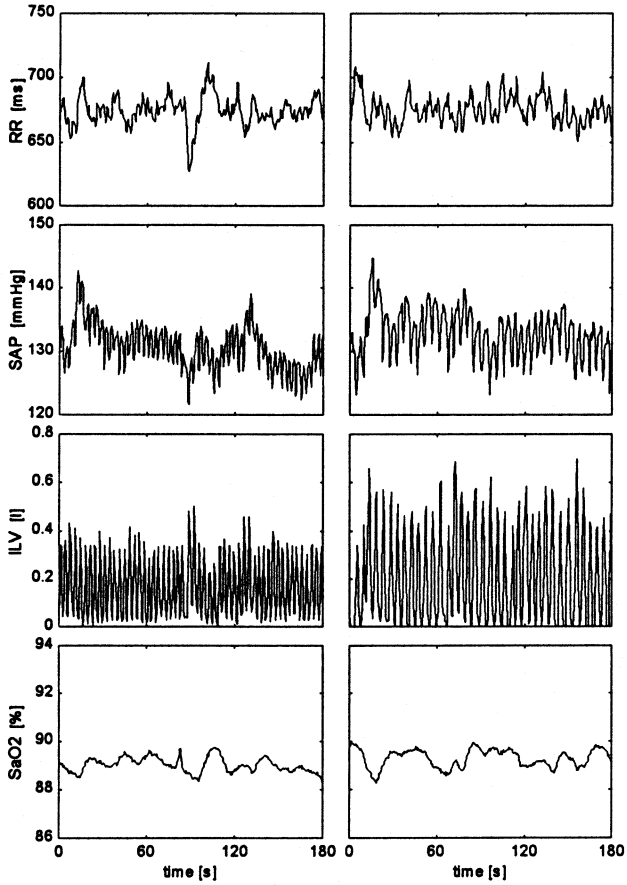

Figure 1. A representative example of recorded signals during spontaneous (left panels) and controlled (right panels) breathing. From top to bottom: RR time series, systolic arterial pressure (SAP), istantaneous lung volume (ILV) and oxygen saturation (SaO2)

RR and systolic arterial pressure (SAP) time series were obtained from row signals. Ectopic beats were linearly interpolated. From the ILV we derived the instantaneous tidal volume (ITV) and the instantaneous minute ventilation (IMV) signal [3], while, from the CO_2 signal, end-tidal CO_2 (ET_CO_2) was obtained. All signals were visually inspected and a 5 min sub-record with all signals free from artifacts, large transients or marked changes in the fluctuating behavior of the signals was interactively selected [7]. After linear detrending univariate spectral analysis was performed on ILV, RR and SAP signals using the autoregressive approach with spectral decomposition [7]. The power in the low frequency (LF) band (0.04÷0.15 Hz) and high frequency (HF) or respiratory band (0.15÷0.45 Hz) was then computed adding up spectral components > 10% of the total power in the band. From bivariate spectral analysis between SAP and RR, the transfer function between them was estimated.

Patients showing a periodic breathing pattern (either apneic or non-apneic) or having more than 20% of the ILV power outside the HF band were discarded from analysis. In the remaining (N=17), measurements were performed of: i) respiratory rate, ii) mean values of RR interval, SAP, ITV, IMV, SaO_2, ET_CO_2, iii) LF and HF power of RR, SAP DAP; iii) Baroreflex sensitivity was estimated as average transfer function between SAP and RR in the LF band (TF_BRS). ET_CO2 was measured in 10 subjects.

Results are expressed as mean (SD). Due to their markedly skewed distribution, power measurements were log transformed. Paired comparisons between spontaneous and paced breathing were performed by the t-test and verified by the Wilcoxon matched pairs test. The significance level was 0.05.

3. Results

A representative example of recorded signals during spontaneous and controlled breathing is given in fig. 1. Results of the study are given in table 1.

4. Discussion

We have shown in a sample of supine CHF patients during short-term (< 10 min) laboratory recordings that paced breathing at 0.2÷0.25 Hz modestly reduces respiratory rate compared to spontaneous breathing (-14%) and markedly increases tidal volume (+57% on average). As a consequence, a moderate increment in minute ventilation (+27%) was observed. These ventilatory changes were accompanied by an increase in O_2 saturation from, on average, 93.3% to 94.2% and by a slight (-7%), although statistically significant, decrease in end-tidal CO_2. These findings suggest that controlled breathing in moderate-to-severe CHF patients is associated with a mild hyperventilation. We also observed a negligible increase in mean RR (+2%) and mean SAP (+2%, ns) and no systematic change in baroreflex sensitivity (-1.5%, ns). Hence, voluntary

control of breathing frequency does not seem to affect autonomic activity in a physiologically meaningful way as a result of mental effort and mild hyperventilation. As regards cardiovascular variability parameters, LF power of RR intervals and SAP did not show any directional change during paced breathing, whereas HF power significantly increased in both signals, with a dramatic rise in SAP. Augment of respiratory fluctuations of RR interval likely reflects the increased central respiratory drive and increased afferent traffic from lung stretch receptors and cardiopulmonary receptors brought about by increased tidal volume and negative intrathoracic pressure, as well as augmented stimulation of arterial baroreceptors by enlarged respiratory oscillations of blood pressure. The latter, in turn, simply reflects increased mechanical coupling between respiration and circulation. Interestingly, changes in HF power of RR interval during controlled breathing significantly decreased the LF/HF ratio. However, due to the substantial constancy of heart rate, this reduction cannot be explained as a shift of the sympathovagal balance.

Table 1. Comparison of ventilatory and cardiovascular variability parameters during spontaneous and paced breathing. N=17 (*N=10).

	Spontaneous Breathing	Paced Breathing	P
Respiratory rate (Hz)	0.26±0.05	0.22±0.01	0.004
Tidal volume (l)	0.35±0.1	0.53±0.14	<0.001
Minute Ventilation (l/min)	0.092±0.02	0.116±0.03	<0.001
O_2 saturation (%)	93.3±1.7	94.2±1.6	0.002
End-tidal CO_2 (%) *	4.9±0.5	4.5±0.5	0.006
RR interval (ms)	957±134	979±141	0.01
SAP (mmHg)	104±13	106±16	0.21
LF Power of RR ($log(ms^2)$)	4.5±1.6	4.4±1.6	0.87
HF Power of RR ($log(ms^2)$)	4.2±1.0	4.6±1.0	0.005
LF / HF Power	2.2±1.7	1.4±1.8	0.02
LF Power of SAP ($log(mmHg^2)$)	0.72±0.9	0.60±1.1	0.38
HF Power of SAP ($log(mmHg^2)$)	0.06±0.9	0.46±1.0	0.004
BRS (ms/mmHg)	4.2±2.0	4.2±2.9	0.99

References

[1] Brown Troy E, Beightol LA, Koh J, Eckberg Dwain L. Important influence of respiration on human R-R interval power spectra is largely ignored. J.Appl.Physiol 1993, vol 75 (5), 2310-2317

[2] Cooke WH, Cox JF, Diedrich AM, Taylor JA, Beightol LA, Ames JE 4th, Hoag JB, Seidel H, Eckberg DL Controlled breathing protocols probe human autonomic cardiovascular rhythms Am J Physiol 1998, vol 274, H709-H718

[3] Pinna GD, Maestri R, Mortara A, La Rovere MT. Cardiorespiratory interactions during periodic breathing in awake chronic heart failure patients, Am J Physiol Heart Circ Physiol 2000, Vol 278(3), H932-941

[4] Mortara A, Sleight P, Pinna GD, Maestri R, Prpa A, La Rovere MT, Cobelli F, Tavazzi L. Abnormal awake respiratory patterns are common in chronic heart failure and may prevent the evaluation of autonomic tone by measures of heart rate variability. Circulation 1997, vol 96, 246-252

[5] Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. Circulation 1991 vol 84, 482-492

[6] Patwardhan AR, Evans JM, Bruce EN, Eckberg DL, Knapp CF. Voluntary control of breathing does not alter vagal modulation of heart rate. J.Appl.Physiol 1995, vol 78 (6), 2087-2094

[7] Maestri R, Pinna GD. POLYAN: A computer program for polyparametric analysis of cardio-respiratory variability signals. Comput Meth Prog Bio 1998, vol 56, 37-48

Address for correspondence:

Ing. Gian Domenico Pinna,
Fondazione S. Maugeri, Centro Medico di Montescano,
I-27040, Montescano (PV), ITALY
gdpinna@fsm.it