Altitude Distress Influence on Cardiac Function

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Abstract

*To study the influence of altitude exposure on heart rate variability (HRV) and cardiac ventricular function 11 male climbers performed 24 hour ECG and echocardiographic evaluation during an expedition to the Everest. Recordings were done as soon as they reached the altitude (=>5000 m, P1), after at least 15 days of acclimatization (P2) and at sea level (before and after the expedition, B1, B2). HRV indices were computed (in the time and frequency domain) on the RR time series; major cardiac measures were calculated from the best echocardiographic view. Significant changes of variability and cardiac indices were detected after altitude exposition (mean±SD, *p<.05). Altitude distress induces tachycardia and reduces some HRV indices. Respiratory pattern consents to document sleep apnea which greatly interferes on RR interval time series. Altitude distress modifies cardiac function whose interference on heart rate reflex could explain HRV changes.*

1. Introduction

The extreme hypoxemia occurring at high altitude is responsible of impairment in cardiac function (1) and of respiratory disorder during sleep. Periodic breathing with prolonged apnea is a frequent phenomenon in normal subjects at high altitude occurring largely during sleep, but it have also been described during wakefulness. Apnea is an interruption of airflow to the mouth lasting more than ten seconds. Marked arterial hypoxemia occurring during sleep at high altitude is responsible of the lack of respiratory central drive stimulation with periodic breathing appearance. Associated changes in heart rate and rhythm have been described during periodic breathing. The cyclic variations in heart rate (HR) are heavily influenced by respiration (2): reflex arch with parasympathetic efferent on the sinus node may account for this phenomenon and its predominance is considered an index of the vagal tone. Heart rate variability represents an useful tool to follow the autonomic cardiovascular regulation during the daily activity, with lower frequency (LF) oscillations attributed to the sympatho vagal influence and high frequency (HF) band expression of the respiratory component.

Modification of cardiac function at increasing systemic hypoxemia may be related to exaggerated pulmonary artery pressure due to hypossic vasoconstriction or to beta adrenergic stimulation described at altitude. Doppler echocardiography has facilitated the detection of the cardiac involvement during hypoxemia obtained in controlled situation as hypobaric chamber studies simulated altitudes (3) rarely at high level (4). Moreover no studies have described the possible correlation between the HRV indices, signs of adrenergic balance and the parameters of cardiac function.

Aim of our investigation was to detect cardiac ventricular function and cycling ECG changes during and after prolonged high altitude exposure, to find out the possible correlation between cardiac and HRV indices.

2. Methods

Data collection was performed on eleven male climbers who reached an altitude of 5000 m during an expedition to the Satopanth (Indian Himalayan).

All subjects performed echocardiographic evaluation and 24-h Holter monitoring at sea level, before and after the expedition (B1 and B2 respectively), as soon as they reached altitude (≥ 5000 meters, P1) and after at least 15 days of exposition (P2). Between the two observations at altitude the subjects were engaged in climbing activities to reach the 7000 meters level. Unidimensional (M-Mode) echocardiograms were recorded using an Esaote Biomedica SIM 5000 instrument, with 2,5 or 3,5 MHz transducer and pulsed Doppler velocity recording. All studies were recorded on a standard VHS videotape for future analysis. Echocardiograms were recorded in the supine left lateral position. The protocol included the parasternal long and short axis views with Doppler sample volume for systemic flow velocity at the left ventricle outflow tract just below the valve in the apical long-axis view. The following measurements were collected: end-diastolic left ventricular dimension, end-

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systolic left ventricular dimension, aortic root dimension, interventricular septum thickness and left posterior wall thickness, the diameter of the aorta, ejection fraction, left ventricular mass (5), cardiac output.
Respiratory signal was monitored by a polymeric thoracic transducer (PVDF transducer) inserted into a chest belt fed on the second channel of a Remco Cardioline ambulatory recorder (modified LP203 recorder), the first channel being used for the ECG.
The ECG and the respiratory signal were off-line digitized at 250 Hz sampling frequency throughout the 24-h recording period and processed to extract their characteristic features. Respiratory signal was analyzed to obtain information on the respiratory rate.
Time series of RR interval were analyzed by power spectral analysis using an autoregressive method (Levinson Durbin Algorithm, model order 12 and 6 respectively). As regard the frequency bands, three major frequency components were considered: very low frequency (VLF) component (from 0.003 to 0.03 Hz); low frequency (LF) component (from 0.03 to 0.15 Hz); and high frequency(HF) component (from 0.15 to 0.5 Hz). The power spectrum components calculated from segments of 256 R-R interval were averaged per hour and over the 24 hour period and stored for statistical analysis.
Standard deviation of normal 24 hours mean RR intervals (SD) reflecting the overall variance of the signal and the proportion of adjacent RR intervals that differed by more than 50 ms, pNN50, were also obtained. This last index is related to the highest frequency components of the HRV, index of vagal activity.
Statistical analysis was obtained by Person's product-moment correlation coefficient, used to evaluate the association among the measures of heart period variability and echocardiographic variables. Moreover Neumann-Keuls for multiple comparison (NKS) was applied. Data are presented as mean ± SD.
A p value < .05 was considered significant.

3. Results

Four recordings were obtained for each subject.
No major arrhythmias were observed at sea level or at high altitude.
Respiratory signal analysis identifies, in the recordings performed at altitude (Fig.1) the constant presence of periodic breathing with hyperpnoeic phases and periodic apnea at night-time. The corresponding RR series showed alternation of bradycardia synchronous with apnea and tachycardia during the resumption of breathing.
The average 24-hour values of variability indices are shown in Table 1. The mean RR interval value was significantly higher at altitude, still increased in the second registration, but promptly resumed the values recorded before departure upon return to sea level.

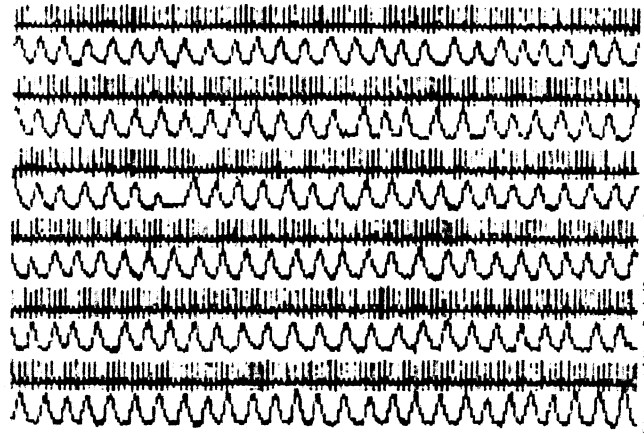
Fig. 1. ECG and respiratory signals

Table 1. Average 24-hour values of variability indices

		B1	P1	P2	B2
RR(msec)	*	971±131	837±109	776±95	978±91
SD (msec)	*	187±36	169±61	147±47	211±43
pNN50 (%)	*	19±11	13±8	6±6	19±11
TP (msec2)	NS	2848±1544	3923±2139	2124±1681	2692±1401
LF/HF	*	4±3	6±5	4±4	2±1
VLF (msec2)	*	1701±1376	675±892	479±418	2178±1318
LF (msec2)	NS	1835±797	2258±1327	1658±1298	1650±774
HF (msec2)	NS	882±795	817±865	315±265	900±705
LF nu (%)	NS	68.1±11.5	65.7±18.0	73.0±22	65.5±12.7
HF nu (%)	*	27.2±12.3	21.6±13.7	14.9±7.2	29.5±10.7

Conversely statistical indices of HRV, SD and pNN50, decreased significantly at high altitude.
Table 2 shows averaged values of major echocardiographic indices.

Table 2. Average 24-hour values of echocardiographic indices

		B1	P1	P2	B2
LVED(mm)	NS	50,3±2,8	50,1±2,6	51±5,4	52,9±2,8
SS (mm)	NS	10±1,4	9,8±1,4	9 ,5±1,4	9±1
PP (mm)	*	10±1,2	8,8±1	8±1,3	9,2±1,9
LVES (mm)	*	35,3±3,6	31,7±3,1	32,4±4,6	33,2±3,5
FE(%)	*	57,5±8,8	65,8±5,4	66,2±7	65,7±7,6
FA (%)	*	30,7±6,4	37,2±5	36,9±4,9	36,7±5,9
Mass (gr)	*	218,8±26,6	193,9±25,8	185,5±19,9	209,9±29,9
CO (l/min)	NS	5,4±1,5	4,6±1,4	5,3±1,9	4,5±1,6
Ao-Vm (m/sec)	*	0,68±0,15	0 ,59±0,08	0,63±0,11	0,6±0,08
Ao-Vmax (m/sec)	*	1,11±0,23	1,02±0,15	1.03±0,18	0,96±0,13

There was an increase of left ventricular function at altitude as documented by significant changes in ejection fraction and shortening fraction, which persisted also after coming back to sea level. Moreover a reduction of left ventricular mass (normal values: male 131-134 g/m2, female 100-120 g/m2) was documented at altitude

with a rapid restoration toward basal values at sea level.
At sea level before expedition significant positive correlation (R > 0.5) was found between heart rate and HRV indices. This correlation was not reproduced at altitude, both acutely and after acclimatization (Fig.2).
Looking at the cardiac analyzed measures, at sea level there was a correlation between interventricular septal thickness and left ventricular mass as expected. No apparently correlation were found between HRV and echocardiographic indices, both at sea level and at altitude. Only a negative correlation between posterior wall thickness and power spectrum in the three bands was found at sea level. These correlation were not reproduced

CORRELATION MATRIX

	RR	SD	TOTAL P	VLF	LF	HF	PP	SIV	FE	MASS
RR	1.0000									
SD	0.4754	1.0000								
TOTAL P	0.6486	0.6531	1.0000							
VLF	0.7143	0.5442	0.7525	1.0000						
LF	0.7060	0.6307	0.9530	0.8193	1.0000					
HF	0.5246	0.6403	0.9488	0.6082	0.8106	1.0000				
PP	-0.4951	-0.1492	-0.7547	-0.7401	-0.7751	-0.6359	1.0000			
SIV	0.1397	-0.1276	0.1937	-0.1558	0.0662	0.3072	-0.2485	1.0000		
FE	0.3090	-0.1919	-0.0405	-0.2645	0.0609	-0.1615	0.0279	0.3557	1.0000	
MASS	-0.0113	0.0369	0.1720	-0.2060	0.0553	0.2744	0.0554	0.6695	0.3632	1.0000

	RR	SD	TOTAL P	VLF	LF	HF	PP	SIV	FE	MASS
RR	1.0000									
SD	0.8021	1.0000								
TOTAL P	0.2805	0.3145	1.0000							
VLF	0.8242	0.7124	0.2431	1.0000						
LF	0.4473	0.4071	0.7599	0.3871	1.0000					
HF	0.2940	0.4016	0.7734	0.2968	0.3997	1.0000				
PP	0.2241	0.1431	-0.0475	0.2314	-0.1175	0.1295	1.0000			
SIV	0.0700	0.1023	-0.0723	-0.0059	-0.0020	0.0056	0.2224	1.0000		
FE	0.0465	0.0577	-0.1039	0.0891	-0.0531	-0.2057	0.1657	0.2893	1.0000	
MASS	0.4615	0.4655	0.0736	0.4126	0.0231	0.2627	0.4093	0.5272	0.1119	1.0000

Fig.2: Correlation Matrix at sea level (upper) and at altitude(below). R >0.5= ** p<.05.

at altitude.

4. Discussion

The aim of this study was to examine cardiac function and HR variability in climbers because they represent an interesting model to study the influence of hypoxemia. At high altitudes the hypoxemia becomes extreme and hyperventilation is the compensatory mechanism. HR was increased acutely and persisted high after acclimatization as the first compensation to hypoxemia. The proportion of adjacent RR intervals reflects the changes from one QRS to another and is most sensitive to the highest frequency components of heart rate variability. Its reduction seems to reflect a reduction of vagal tone, which is detected by the HF component of the power spectrum of the RR series. The respiratory frequency varies during the day and in different populations. The respiratory monitoring, as described (6), permits to observe the exact respiratory frequency and to assess the presence of respiratory apnea during the night.
Correlation present at sea level between heart rate and HRV indices was lost at high level. These changes might reflect a compromised sympatho- vagal balance on ascent to altitude. An enhanced sympathetic activity has been described secondary to hypoxemia (3). Adrenergic stimulation is suggested in our population by the increase, even not significantly, of LF band of power spectrum, as soon after climb. Augmented sympathetic activity appears to be also the basis for the increase in contractility at high level expressed by the persistent increase of ejection fraction. Maintenance but not increase of cardiac output seems indicate a parallel increase in peripheral resistance, still induced by adrenergic stimulation, which might download baroriflex responsivenessFinally a significant reduction in left ventricular mass was observed at altitude, parallel to a reduction of septum thickness; no data are available to explain this result.

5. Conclusions

Altitude distress induces tachycardia and reduces HRV time domain indices.
Respiratory pattern consents to document sleep apnea which interferes on RR interval time series.
Altitude distress modifies HRV indices correlation present at sea level probably enhancing sympathetic stimulation..
Left ventricular function in maintained or somewhat increased at high level.
The hemodynamic interference on heart rate reflex could explain HRV changes. To clarify this phenomena further studies needed.

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