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A STUDY OF HEART RATE AND HEART RATE VARIABILITY DURING RAPID DECOMPRESSION TO 50,000 FT

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13. ABSTRACT *(Maximum 200 words)*

A previous study of the heart rate (HR) response to positive pressure breathing (PPB), anxiety and hypoxia during rapid decompression (RD) revealed a consistent pattern in the beat-to-beat or interbeat interval variability of HR, known in the literature as heart rate variability (HRV). One method of analyzing HRV is a noninvasive measure of the respiratory-cardiac neural reflex known as vagal tone monitoring. This method is based on the amplitude of respiratory sinus arrhythmia (RSA) as a manifestation of vagal cardioinhibitory influence on the heart. The RD/HR data were interpreted by two HRV analysis methods, vagal tone monitoring (VTM) and a method developed in-house based on maximum entropy method (MEM) (spectral analysis), as a means of studying the heart rate regulating mechanisms during RD. A significant drop in vagal tone in both HRV measures was shown during the PPB and hypoxia profiles. Conflicting results regarding the anxiety profile during RD leave room for questions as to the use of these HRV measures as a tool for assessing anxiety or emotional stress. However, these measures only examined a subset of the HRV frequency spectrum (the respiratory frequency range) which provides information only on the parasympathetic autonomic contributions. Investigation of other frequency bands may provide more complete data on the autonomic regulation of the cardiovascular system.

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INTRODUCTION

A previous study of the heart rate response during rapid decompression (RD) (4) revealed a consistent pattern in the beat-to-beat or interbeat interval of heart rate (known in the literature as heart rate variability (HRV)). We observed a marked reduction in HRV with the increase in heart rate after RD. This observation led to further interest in the HRV response under the stresses resulting from RD.

An excellent review article on HRV has been published by Saul (32). A more detailed survey of the literature has shown that spectral analysis of HRV is a powerful noninvasive tool for quantifying estimates of autonomic nervous system control of the cardiovascular system (1,28,33). Experiments (1,27,28,33) have been performed using drugs which act upon the autonomic control of the heart to define the frequency ranges involved in the power spectrum of heart rate fluctuations. These studies have concluded that sympathetic and parasympathetic activity make frequency-specific contributions to the heart rate power spectrum; the former contributes to the lower frequencies (>0.12 Hz) and the latter to both the lower and higher frequencies (>0.2 Hz)(1,27,28). The HRV frequency spectrum has also been correlated with specific physiologic activity: The higher frequencies (centered about 0.35 Hz) are mediated by respiration; the middle frequencies (centered about 0.1 Hz) are associated with the baroreceptor reflex and vasomotor activity; and lowest frequencies (centered about 0.04 Hz) are related to cyclic fluctuations in peripheral vasomotor tone associated with thermoregulation (1,33) and changing plasma levels of neurohormones (31).

Methods for computing the HRV frequency spectrum vary widely depending on application and historical origin (2,5,22,29,39). One patented method for analyzing the HRV spectrum, called "vagal tone monitoring (VTM)" by its inventor (Porges-Bohrer), uses the HRV caused by the respiratory-cardiac neural reflex (29). VTM focuses on the contribution to HRV by respiratory sinus arrhythmia (RSA), the rhythmic oscillation in HR which occurs with breathing, described as a decrease in the interbeat interval with inspiration and an increase with expiration. Essentially, VTM measures the magnitude of the HRV power spectrum within the range of respiratory frequencies. On the basis the RSA amplitude is mediated by vagal tone, the nonneural and direct sympathetic influences can be separated from the HRV using spectral analysis. In VTM, the HRV component

associated with normal respiration, quantified by summing the variances within the respiratory frequency band, is defined as vagal tone (V) (7,24,42).

Because the VTM is proprietary, an alternate method of computing the HRV spectrum which can be used to extract a measure of HRV similar to the vagal tone methods has been developed. This method is based on the maximum entropy method (spectral analysis) (MEM); it has the flexibility to look at other frequency ranges in the spectrum besides the respiratory frequency range. See the Appendix for detailed explanation to the method of computation. An interclass correlation between V and the scores computed by the MEM has been accomplished. One hundred and sixty, 1-min data files from the RD study were scored by both methods. The HRV measures from MEM correlate well with those from VTM; the interclass correlation value of 0.86 has limits of 0.85 to 0.87. We have employed both VTM and MEM to evaluate data that were collected during a series of high and low altitude rapid decompressions in a hypobaric chamber during which the subjects were exposed to stresses from positive breathing (PPB), anxiety, and hypoxia. This study simulated the most physiologically stressful altitude situation which can occur in flight. This type of analysis supports the study of the heart rate regulating mechanisms; it elucidates the autonomic nervous system activity of the stresses resulting from rapid decompression.

MATERIALS AND METHODS

Experimental Design

This paper reports one part of a large study to evaluate the impact of reduced oxygen breathing gas mixtures during RD. The focus of this report is on the cardiovascular response. However, the experiment was designed to examine other physiologic responses as well. The results from the pulmonary and task performance data are reported elsewhere. Thus, the description of the experimental methods includes all aspects of the study.

The subject pool from the USAFSAM Altitude Research Panel consisted of 17 active duty USAF male volunteers, 20 to 42 years of age. All had passed a Class III flying physical examination and had received physiological and PPB training. During all experiments the subjects wore standard USAF flight equipment, used standard USAF breathing equipment, and were seated in a chair in a hypobaric chamber (15). Three chest electrodes were fitted to acquire the electrocardiogram (ECG) throughout the experiment. These data were stored on magnetic tape for later analysis along with the other measured parameters which included:

Inspiratory flow, mask cavity pressure, chamber pressure, and respiratory gas composition. At the time of the RD each subject breathed one of two gas mixtures (either aviator's breathing oxygen (ABO) or 93% O_2) using two regulator conditions: dilution (i.e., supply gas diluted with ambient air according to cabin altitude) or nondilution (99.5% O_2 for ABO or 93% O_2). In the dilution mode, oxygen concentration was about half that of the nondilution mode.

The chamber altitude profiles are shown in Figure 1. Each procedure began with 1 h of preoxygenation breathing ABO at ground level (GL) during which time two psychomotor and memory tasks were carried out (26). The prebreathe was followed by an ear and sinus check at 1,524 m (5,000 ft) and return to GL. For the high altitude profile, an ascent to 8,229 (27,000 ft) for abdominal gas elimination was also performed. The chamber altitude was then changed to and held at 6,096 m (20,000 ft), where the subject carried out the second set of performance tasks. Before the second set was started the breathing regulator supply was switched from ABO to the test gas mixture and selecting either the dilution or the nondilution regulator mode. The subject breathed the new gas mixture for 3-5 min to achieve a stabilized pulmonary and circulatory state. Upon completion of these tasks, the subject was warned of the impending RD and advised to breathe normally. About 20 s after the warning, the chamber was decompressed from 6,096 to 15,239 m (20,000 to 50,000 ft), 34.5 kPa (5 psi) differential, in approximately 1 s. On decompression, the regulator automatically delivered undiluted gas (ABO or 93% O_2) at a positive breathing pressure of 3.9 kPa (30 mm Hg). During the 10 s following RD, equipment function and mask seal were checked, and the third performance task was started. The chamber altitude remained at 15,239 m (50,000 ft) for 1 min before it was decreased to 12,191 m (40,000 ft) in 15 s where it was held until the third task was finished. Pressure breathing at this altitude was approximately 1.1 kPa (8 mm Hg). Descent to 6,096 m (20,000 ft) at 1,524 m/min (5,000 ft/min) to complete another set of performance tasks was followed by a final descent to GL.

Some of the subjects participated in two additional experimental profiles. The first profile was designed to eliminate the influence of hypoxia by repeating the decompression profile pattern but at a lower altitude (Figure 1); the subject breathed nondiluted ABO. In this case, the RD was from 2,438 to 7,010 m (8,000 to 23,000 ft), also a 34.5 kPa (5 psi) differential; initial descent after 1 min was to 5,489 m (18,000 ft), instead of to 12,191 m (40,000 ft), and then to 2,438 m (8,000 ft) instead of to 6,096 m (20,000 ft). The performance tasks were completed in the same pattern as they had been in the original altitude profile. Positive pressure breathing of 3.9 kPa (30 mm Hg) was delivered upon RD for the 1 min at peak

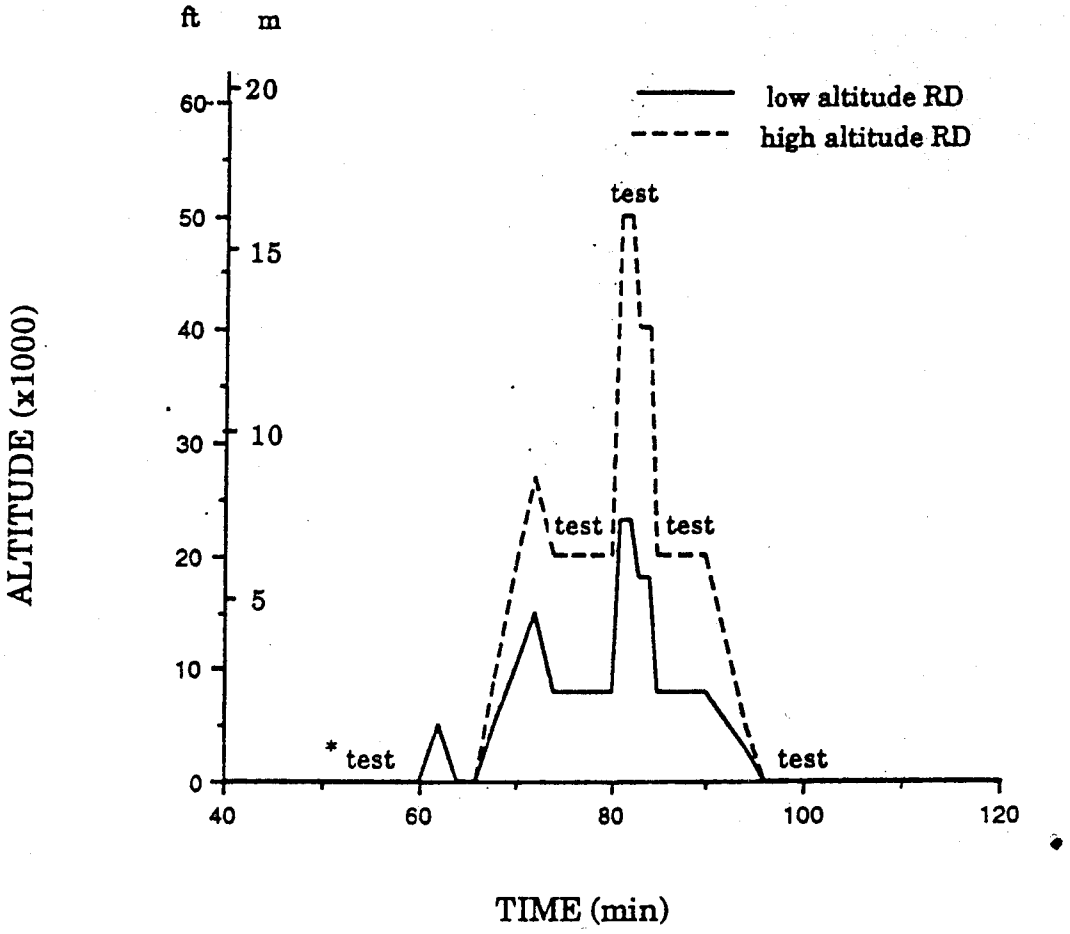

Figure 1. Chamber altitude profiles [*test indicates a set of psychomotor performance].

altitude. The subjects were not informed that the altitudes would be lower. This test condition eliminated the hypoxia effects, but left the effects of anxiety and PPB. The next profile was designed to isolate the effect of PPB on HR by eliminating both effects of hypoxia and the additional anxiety normally associated with RD. In this case, the complete profile was performed at ground level. The subjects wore the same flight equipment, used the same breathing system, and were seated inside the hypobaric chamber at GL breathing nondiluted ABO. The subjects did not experience hypoxia, and they were also aware that no RD would occur. Positive pressure breathing at 3.9 kPa (30 mm Hg) was, however, delivered for 1 min as with the full RD profiles. The subjects undertook the same performance tasks as they had during the RD profiles.

To extract the R-R intervals (also known as interbeat intervals or heart period) from the analog magnetic tape, the ECG signals were replayed through a specially designed peak detector and automatic R-wave Tracker (ART) (38). This system measured the interbeat intervals of the ECG to the nearest millisecond. The data were channeled to a Gould strip chart recorder for monitoring in real time. A Zenith model 248 microcomputer was used to score the final output. For comparative analysis over time, and in order to coordinate times of the RD period, the raw interbeat intervals were interpolated to generate data points at equal time intervals (0.5 s) using cubic splines. This method produced a virtually identical time history as the raw interbeat interval data, but with a uniform (0.5 s) sampling interval. These data were further analyzed by the following heart rate variability and statistical analysis methods.

Vagal Tone Analysis

The interbeat interval data with uniform sampling intervals were input into Dr. Porges software (MXEDIT) on an IBM PC XT286 and analyzed as follows. Each file was automatically at converted to a binary data file, plotted on the monitor, divided into 60-s segments, and plotted on a graphics plotter. The data points within each segment were examined to ensure they fell within reasonable physiological limits for interbeat interval or heart period (HP) data (300 to 1200 msec) and edited to remove artifacts and poor data segments. The edited segments were replotted and stored with the raw data plots to document the editing. John Dellinger who did editing, estimated that less than 20% of the data points needed editing. After editing, the Porges-Bohrer patented method (PB Filter Command in MXEDIT) for computing an estimate of vagal tone was used to calculate the dependent variables at epochs of 10 s. This method uses a 21-point cubic polynomial as a band pass

filter to cutoff to achieve the specified normal respiratory frequency range of 0.12 to 0.45 Hz. We set the respiratory range by using the known respiratory data for these subjects across all experimental profiles.

Every 10 s, the PB Filter yielded estimated heart rate (bpm), mean heart period (HP) (ms), heart period variability (HPV (in ms^2), heart period range (ms), estimates of vagal tone (V) (in ms^2). These data were stored on paper and magnetic computer diskettes (ASCII files) for subsequent statistical analysis and data summaries.

Maximum Entropy Method (Spectral Analysis)

The MEM program begins with time and interbeat intervals or heart period from the data files. It then computes the first difference of HP by successively subtracting the previous HP from the current one and stores the result in a second field corresponding to the time of the current value, i.e., x_t is replaced by $\Delta x_t = x_t - x_{t-1}$. Next, use an interpolating polynomial (cubic spline) to interpolate the Δx_t series at 0.5 s intervals so that the resulting time series can be transformed by the MEM routines to yield the power spectral density of the HRV (also called heart period variability by Porges-Bohrer). The user can specify how many samples (or total time) are used for estimation of the spectrum. The more samples one has in the time domain, the better the frequency domain resolution of the spectral peaks; however, the spectrum represents the "average" HPV power over the time interval. This is the dilemma of spectral analysis: good resolution in the frequency domain implies poor resolution in the time domain. In general, we have found that a minimum of sixty samples (30 s) are required to produce an acceptable estimate of the HPV spectrum. Thus, a new HPV spectrum can be computed for each 30-s period of heart period data. We have chosen to update the computation after each 5-s period. At each 5-s interval, we compute a new HPV spectrum over the last 30 s of HP data. After that computation, the 30-s window is advanced 5 s in time and the HPV spectrum is recomputed; the new score is assigned to the most recent 5-s period. Consequently, the 5-s scores represent a "moving average" of the 30-s HPV spectrum and they are, therefore, serially correlated. Scores at 30-s intervals are completely independent (computationally, but probably not physiologically) from the previous 30-s period. To differentiate our method of computation of an estimate of "vagal tone" from that of VTM, our score for an epoch will be labeled V'. We computed the V' scores from the MEM spectrum as the natural logarithm of the mean square of the

magnitude of the power spectral ordinates between 0.12 and 0.45 Hz; we used the same frequency range to compute vagal tone by VTM.

Statistical Analysis

We performed the statistical analysis on the results from the group of data analyzed by both methods from six of the subjects who completed the additional two experimental profiles. The three test conditions compared were the high altitude RD using 93% oxygen in the dilution mode (a profile in which the breathing gas produced the greatest degree of hypoxia), the low altitude RD, and the GL/PPB profiles. We evaluated the means of data resulting from the VTM and MEM analysis (mean heart period-HP, heart period variability-HPV, vagal tone-V and V') by a 3-way repeated measures factorial analysis of variance (ANOVA); subject was a random factor. The fixed factors of the statistical model were condition (altitude profile) and time interval. We applied tests to determine the significance of the condition effect, the time effect, and the interaction of condition by time for each variable (HP, HPV, V, V') at a significant level of 0.05. Where appropriate, we performed subsequent t-tests at each 20 s epoch to aid in interpreting significant interactions.

RESULTS

Figure 2 shows the typical HR response for this experiment. Time zero identifies the RD; the greatest change in HR occurred during 0-1 min following RD. Typically, an increase in HR occurred immediately following the RD and reached a plateau after several seconds. The HR decreased after the chamber had descended to 12,191 m (40,000 ft). HR variability was markedly decreased at high altitude; this response, too, was typical.

We demonstrated that the individual effects of PPB, anxiety, and hypoxia on HR could be statistically isolated (4). Figure 3 shows the HR response to the three separate experimental profiles, as well as the hypothesized environmental/physiologic cause of the HR response (shaded areas).

This HR, calculated from the interbeat interval or HP data, represented an inversion of the HR data. Figure 4 displays the same HP data used to calculate HR in Figure 3, but in a larger window around the RD and processed by the Porges-Bohrer software and statistical analysis (ANOVA). However, the same trend is apparent in both graphs, where an

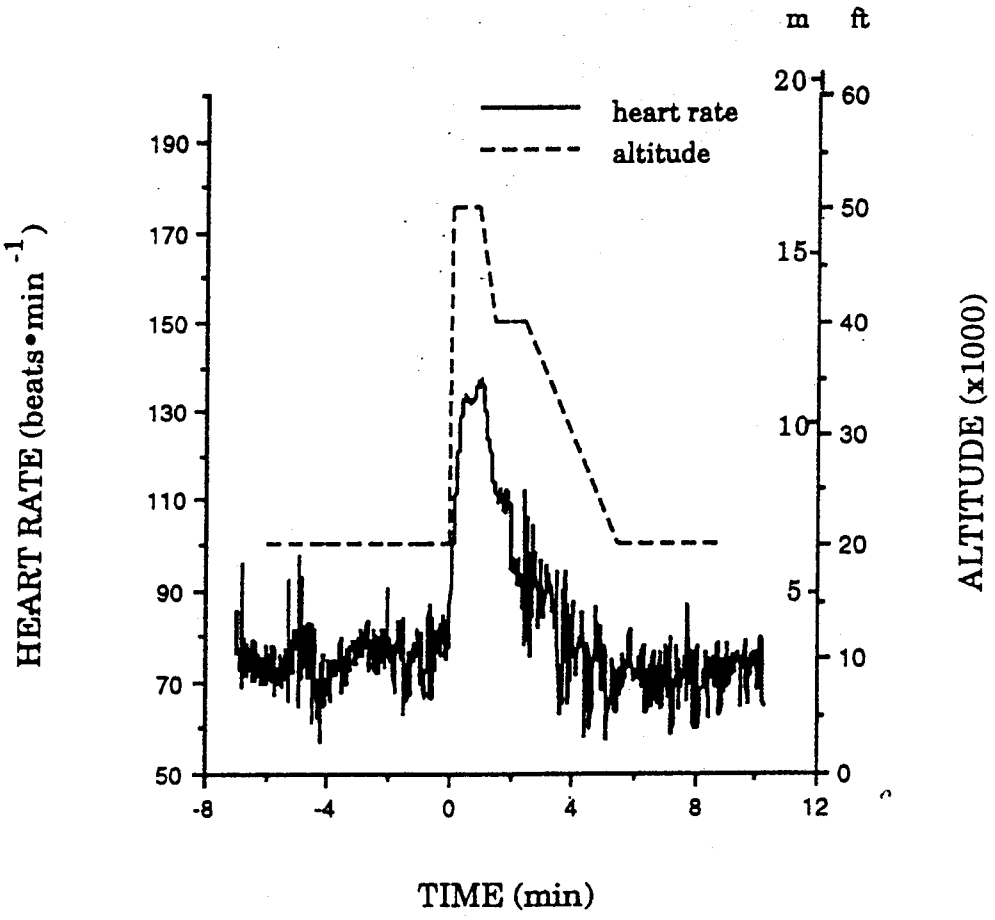

Figure 2. Typical heart rate response to RD [Breathing 93% oxygen with regulator in dilution mode].

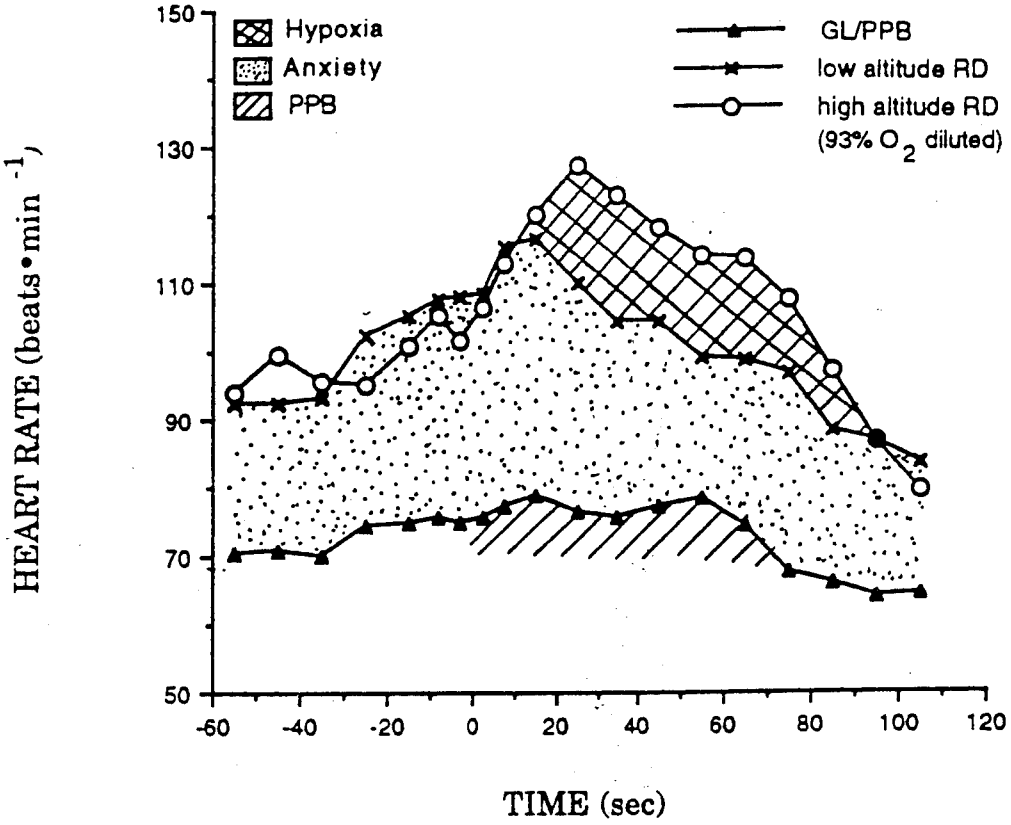

Figure 3. Heart rate response [means].

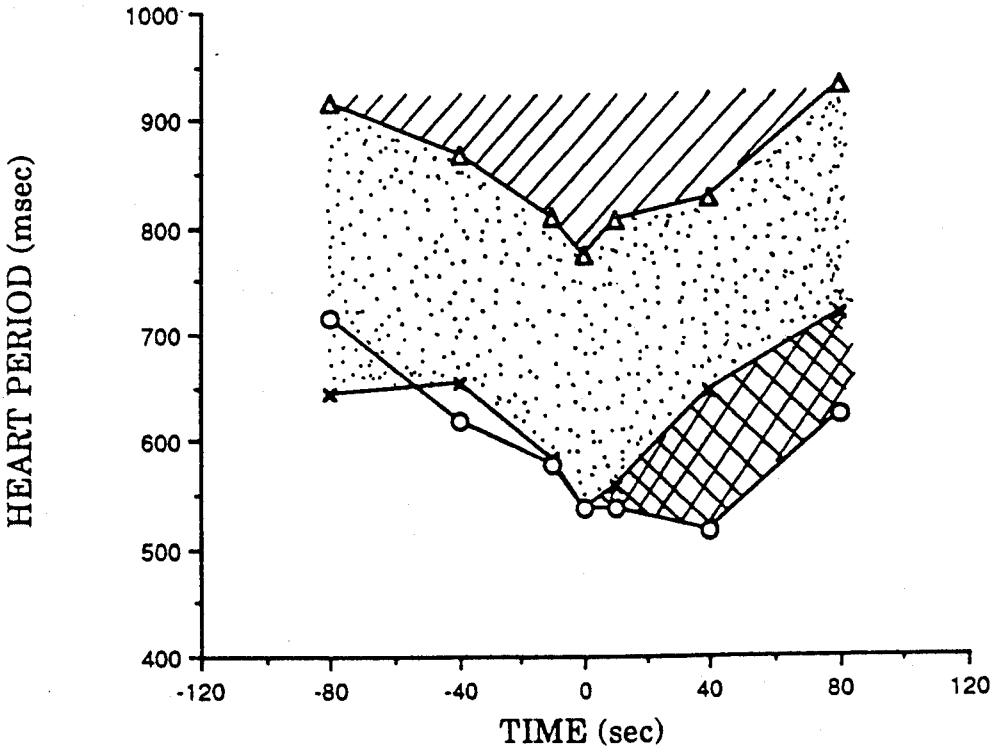

Figure 4. Heart period response [means].

increase in HR (bpm) is shown as a decrease in HP (ms). Again, the shaded regions represent the physiologic influences of PPB, anxiety, and hypoxia.

The physiologic effects of RD are dramatic, and, as expected, the cardiac rhythm parameters are also affected. The HP data (Figure 4) indicates that in the GL/PPB condition, there is a small amount of tachycardia, but with a rapid recovery to baseline. The low altitude RD condition results in a very similar curve, but at a much higher level of tachycardia, resulting from subject anxiety associated with the RD. The high altitude RD condition starts out at a similar magnitude and shape; however, it has a prolonged recovery phase presumably due to the reduced O_2 or hypoxia exposure for one min at 15,239 m (50,000 ft). During this time, the alveolar oxygen tension (PaO_2), as estimated by end tidal measurements, was 3KPa (22 mm Hg) immediately after RD and increased to 8 kPa (60 mm Hg) after descent to 12,191 m (40,000 ft), resulting in hypoxic hypoxia.

Heart period variance plots (Figure 5) reveal a sharp increase in variance co-occurring with the RD and a rapid return to normal for the GL/PPB and low altitude RD conditions. However, estimates of vagal tone by VTM (V) (Figure 6) do not show the sharp increase in variance occurring at RD as does Figure 5; thus the sharp jog may be artifact or a variability component not measured by VTM. After RD, a significant drop is seen in high altitude RD curve, reaching the lowest point at 40 s post-RD then recovering. This drop is increasingly evident in estimates of vagal tone by both VTM (Figure 6) and MEM (Figure 7).

Both HRV methods reveal a decrease in vagal tone with the onset of PPB at time zero; there is a return to pre-RD levels by 80 s. The low altitude RD curve reveals no clear trend, for the curve is slightly different for the two processing methods. Post-RD, the low altitude VTM curve, closely follows the PPB curve with a slight decrease in vagal tone and a more gradual slope to pre-RD levels. Whereas, the MEM low altitude curve decreases on the same path as the high altitude pre-RD curve, and increases after RD.

The ANOVA showed significant time, condition, and condition by time interaction effects for the HP, HPV, and estimates of vagal tone by the MEM method (V'). The ANOVA did not show a significant interaction of condition by time for the vagal tone results by VTM (V). However, subsequent t-tests for V and V' revealed significant condition differences at points 40 s and 80 s post-RD in which the high altitude RD shows a dramatic drop in vagal tone. These statistical results confirm a loss of vagal tone with exposure to high altitude and a slight decrease in vagal tone with PPB. No firm conclusions could be reached

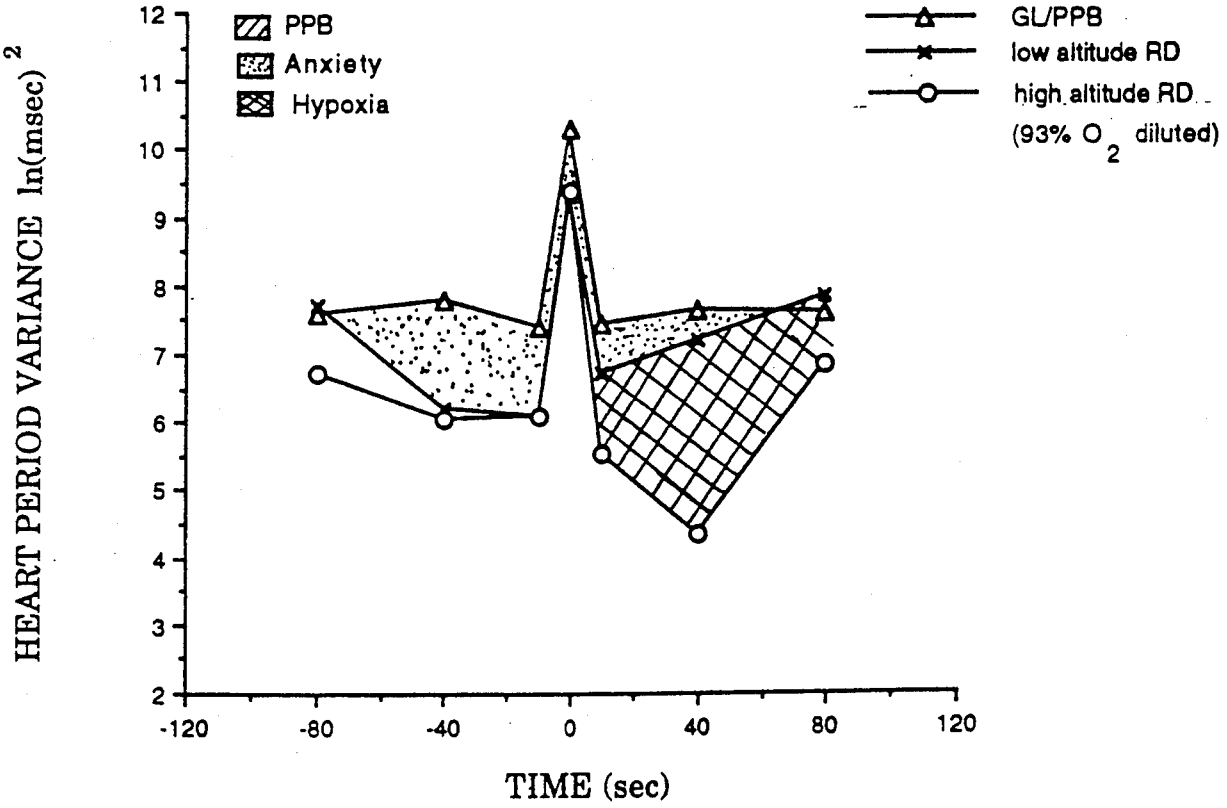

Figure 5. Heart period variance [means].

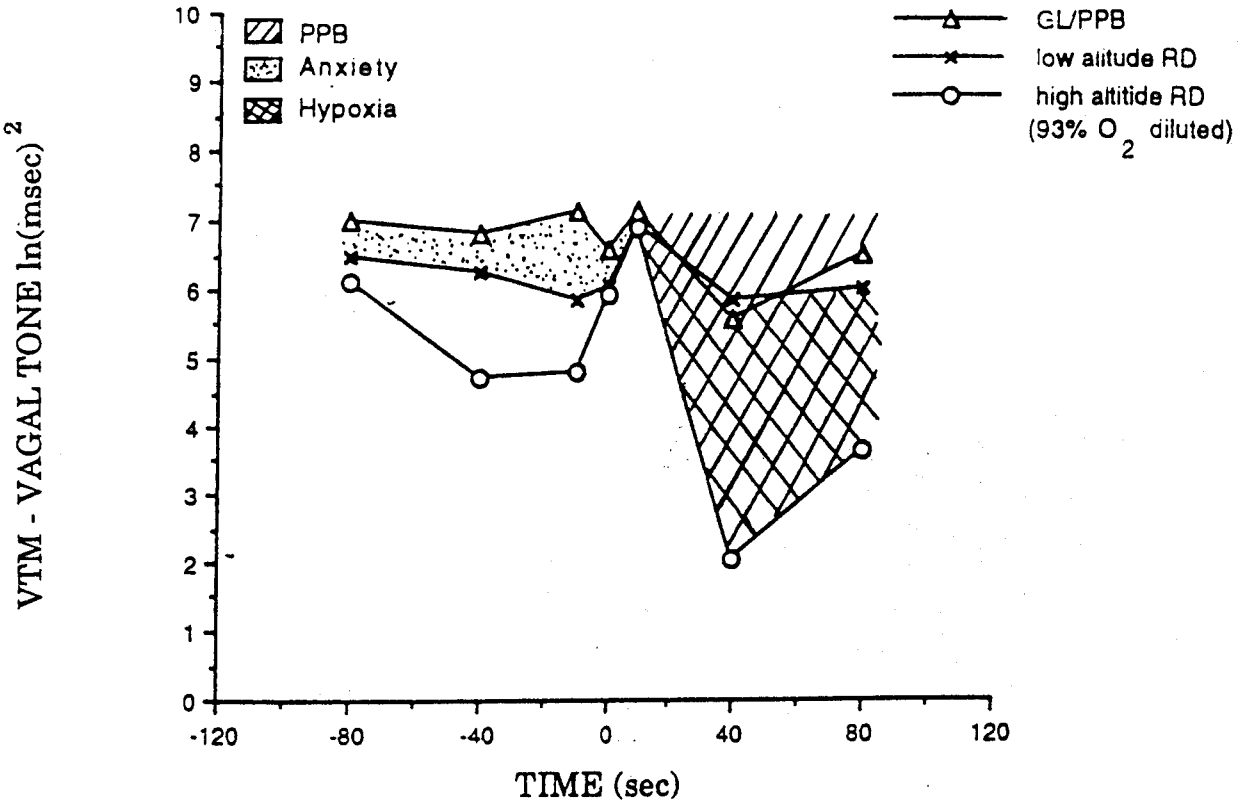

Fig. 6. Vagal tone measured by VTM [means].

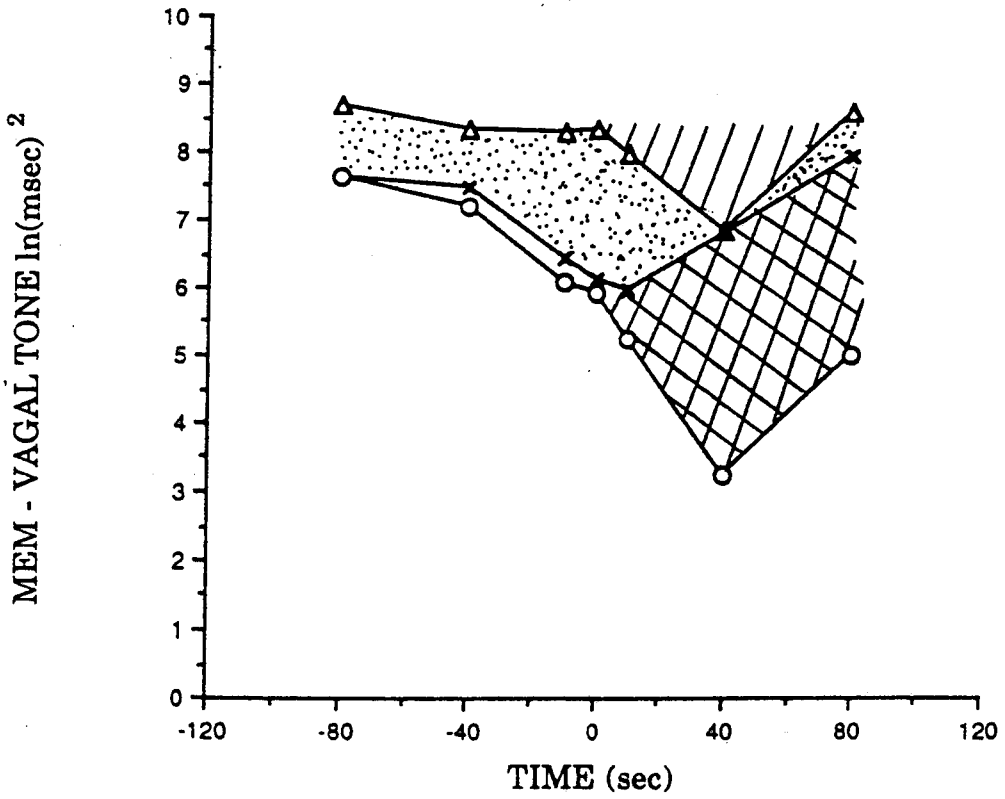

Fig. 7. Vagal tone measures by MEM [means].

regarding the low altitude RD profile on vagal tone because a different curve resulted from each processing method.

DISCUSSION

The resting heart rate of healthy individuals is influenced by many factors including age, gender, emotional state, temperature, and physical fitness. Tachycardia is part of the normal cardiovascular response to many environmental and physiological factors including exercise, fatigue and, of direct importance to this study, pressure breathing, emotional stress (anxiety), and hypoxia (8,10,12,36,37). During the high altitude RD, the effects of PPB, anxiety, and hypoxia occur simultaneously, producing tachycardia or a net increase in HR (decrease in HP) as shown in Figure 2. By performing the additional experimental profiles, we showed that the individual effects of PPB, anxiety, and hypoxia on HR could be isolated. By using two different estimates of HRV or vagal tone, we were able to confirm a loss of vagal tone with the onset of PPB and after RD in the high altitude RD profile (i.e., hypoxic exposure). To add perspective, it is important to review the individual cardiac responses and regulating mechanisms of each imposed stress.

Positive Pressure Breathing (PPB)

The cardiac response and regulating mechanisms during PPB can be explained in terms of normal respiration. Respiratory sinus arrythmia (RSA), previously described in this report as the rhythmic oscillation in HR that occurs with breathing, is caused by the autonomic vagal influences of the pulmonary stretch receptors. Increasing lung volume (i.e., on inspiration) results in increased pulmonary stretch which suppresses the reflex cardioinhibitory effects of the vagal nerves (i.e., reduced vagal tone), thus, producing tachycardia (6,10,17,25). This response is also referred to as the lung inflation reflex. Since PPB causes a continuous increase in end-expiratory lung volume, it might be expected to produce tachycardia (10).

The act of expiration against PPB requires considerable effort and is equivalent to mild exercise--another possible cause of tachycardia (30). Moreover, the cardiovascular dynamics of PPB itself play a role; peripheral pooling is followed by a reduction in venous return which leads to reduced right heart pressure, a fall in cardiac output, and a reflex vasoconstriction and tachycardia (10,36). Finally, the magnitude of tachycardia will also depend on the level

of PPB (and on the degree of thoracic and lower body counter pressure if used) (9,10).

The initial and predominant HR response to PPB is tachycardia caused by the reflex release of cardioinhibitory or vagal tone due to increased end-expiratory lung volume. The results from this study correspond well with the expected cardiac response and regulating mechanisms. The GL/PPB curve illustrated an increase in HR with the onset of PPB (Figure 3), and the decrease in vagal tone was revealed in both HRV processing methods (Figures 6 and 7).

Anxiety

Emotional stress has a potent effect on HR, as part of the normal "fight or flight" or defense reaction. A number of studies of people exposed to fear-inducing environments have confirmed that "the physiological correlates of the state of over-arousal induced by fear and anxiety include tachycardia and increased secretion of adrenaline and growth hormones" (11). Thus, any physiologic experiment in which the subject is exposed to a different and threatening environment will result in some degree of anxiety. Since an RD is an unusual and threatening experience, tachycardia from anxiety is an expected and reported HR repose (37).

This defense reaction analogous to that seen just prior to strenuous exercise and has been described as "an anticipatory acceleration just before work due to nervous influences from the cerebral cortex acting on the cardiac centers in the medulla" (30). Prior invasive experiments on the cardiovascular regulating mechanisms of this reaction show a combination of reflex general vasoconstriction and muscular vasodilatation. The baroreceptor response that would normally limit tachycardia was suppressed in the suprabulbular centers of the brain (25). This reaction is also described as a "reflex release from cardioinhibitory tone" or vagal tone (30).

Many studies have also been conducted using HRV or vagal tone measures to indicate pilot task induced stress or workload. These studies have shown reduced HRV during simulated and inflight pilot tasks; there was a greater HRV reduction in high workload levels which produced more severe mental stress (21,31,34,35,40).

The results from this study confirm the presence of tachycardia due to emotional stress or anxiety during RD, as illustrated by the low altitude curve in Figure 3 and an overall statistically significant interaction ($p=0.0092$) in the interbeat interval data. However, the results from the HRV analysis do not show a clear trend between the two

processing methods regarding the low altitude or anxiety curve. The data processed by VTM (Figure 6) did not show a statistically significant interaction of condition vs. time ($p=0.164$), whereas the data processed by MEM did show a statistically significant interaction ($p=0.0017$). Prior to the RD, the MEM results correspond well with cardiovascular regulating mechanisms described previously (i.e., a reflex release from cardioinhibitory tone), whereas the VTM results do not.

In recent reports, we concluded that the HRV measure showed less statistically significant levels than the HR measure, thus indicating that HRV was less sensitive to changes in a subject's cognitive state than HR measures. The papers suggested that "HRV may not provide any more information than simple HR" in assessing cognitive workload or mental stress (41,13). The HRV measure for assessing the anxiety effect during this RD experiment has not been found to reveal clear information on the autonomic regulating mechanisms of the heart. The conflicting results of the two processing methods on the same data leave open to question the value of HRV measures on this type of data.

Hypoxia

Hypoxic hypoxia caused by reduced PaO_2 induces respiratory and cardiovascular changes (8,36). The respiratory response, not evident until PaO_2 has fallen to about 8 kPa (60 mm Hg) as seen while breathing air above an altitude of 3,047 m (10,000 ft), is characterized by an increase in the rate and depth of breathing (12). Heart rate and cardiac output increase progressively in subjects breathing air above 1,828-2,438 m (6,000-8,000 ft), with a rise over resting levels of about 10% at 4,571 m (15,000 ft), 20-25% at 6,096 m (20,000 ft), and doubling at 7,619 m (25,000 ft) (8). In subjects breathing 100% oxygen, hypoxic stimulation of the heart is not seen before an altitude of about 12,191 m (40,000 ft) (36). At 11,582 m (38,000 ft), Air Force regulators deliver ABO at positive pressures to prevent a severe reduction in PaO_2. As previously mentioned, in our study the estimated PaO_2 was 3kPa (22 mm Hg) immediately after RD and 8kPa (60 mm Hg) after descent to 12,191 m (40,000 ft). These results confirm the presence of hypoxic hypoxia after RD.

The cardiovascular response and the regulating mechanisms resulting from hypoxia have been extensively researched and documented (3,8,14,16,17,18,20,25). Numerous invasive studies have shown the response to hypoxia resulting from an interplay of many opposing cardiovascular reflex mechanisms. These include effects from blood vessels, chemoreceptors, respiratory centers, heart muscle,

the central nervous system (CNS), and effects from increased release of catecholamines from the adrenal glands (16). The response from decreased PaO_2 cause vasodilatation and initiates the activation of the chemoreceptors, which produce an increased respiratory response and bradycardia. The secondary response from the stretch receptors in the lung (lung inflation reflex as noted in PPB) and atria and other baroreceptors produce tachycardia and an increase in cardiac output (25). Local hypoxia on the CNS affecting neurons of the medullary vasomotor centers also cause tachycardia and a sharp rise in peripheral resistance. A combination of the tachycardia caused by the respiratory response (reduced vagal tone) and the CNS response often override the effects of the initial bradycardia produced by the chemoreceptors (16,17,25). It has been shown that in the awake conscious animal there is a greater number of circulating catecholamines that also produce an increase in cardioacceleratory response (17).

The results from this experiment show tachycardia to be the overriding HR response to hypoxia (Figure 3). The fact that a significant decrease in vagal tone was shown by both HRV processing methods (Figures 6 and 7) illustrates the overriding effect of the parasympathetic or vagal respiratory response to hypoxia during this RD experiment.

Combined Cardiovascular Response

In this study, all of the external factors were considered to contribute to the observed consistent overall rise in heart rate during RD. Before decompression, the heart rate level was increased over the control resting level as a result of anxiety. A further "anticipatory" increase of 5 to 10 beats/min occurred just before RD, and a very rapid rise in rate occurred during and immediately after the RD. As HR increased, HRV or vagal tone was reduced during PPB and hypoxia. Thus, as shown by the HRV analysis, these changes are partially mediated by the reflex release of vagal or cardioinhibitory tone. As described in the literature, sympathetic responses from anxiety and hypoxia may also be present (17,25). The rise in HR reached a peak after 20 s during the period of combined PPB, anxiety, and hypoxic influences, and therefore stabilized before exhibiting a slight decline. Maintenance of increased HR during this time may have been due to increased cardioacceleratory tone with a loss of vagal inhibition, a similar response seen in exercise (30). A drop in HR and increasing vagal tone marks a recovery from the additive effects of PPB, anxiety, and hypoxia during the RD.

CONCLUSIONS

These data illustrate the individual influences of PPB, anxiety, and hypoxia during RD on the cardiovascular response and some of its regulating mechanisms. The two employed measures of heart rate variability, VTM and MEM, demonstrated the ability to noninvasively quantify some of the autonomic cardiovascular regulating mechanisms. We showed that these two measures were reliable indicators for verifying the parasympathetic or vagal response of PPB and hypoxia. Conflicting results regarding the anxiety component during RD leave room for question as to the use of these HRV measures for assessing emotional stress. However, these measures only examined a subset of the HRV frequency spectrum (the respiratory frequency range) which provides information only on the parasympathetic autonomic contributions. As suggested by other HRV investigators, analysis of other frequency ranges may provide more complete data on the autonomic regulation of the cardiovascular system (23). Subsequently, the MEM program now accommodates the investigation of other frequency ranges of the HRV spectrum.

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APPENDIX

Power Spectral Estimation by the Maximum Entropy Method

The following discussion was extracted from Chapter 12 of Numerical Recipes by W.H. Press, et al (1). The Maximum Entropy Method (MEM) of estimating the power spectrum is simply an alternative to the Fast Fourier Transform (FFT) method. The MEM employs the Wiener-Khinchin theorem from time series analysis which says that the Fourier transform of the autocorrelation function of a time series is equal to its power spectrum.

Thus, given the definition of the discrete Fourier Transform of an N equal interval sample representation of time series c(t) is

$$C_k = \sum_{j=0}^{N-1} c_j e^{2\pi i jk/N} \qquad k = 0,1,2,\ldots N-1 \text{ and } i = \sqrt{-1}. \tag{1}$$

The power spectrum of c(t) can be computed (with appropriate normalization) from the C_k's as,

$$P(0) = N^{-2} |C_0|^2 \tag{2}$$

$$P(f_k) = N^{-2} \left| |C_k|^2 + |C_{N-k}|^2 \right| \tag{3}$$

$$P(f_{N/2}) = N^{-2} |C_{N/2}|^2 \tag{4}$$

with,

$$f_k \equiv kN^{-1}\Delta t_s^{-1} = 2f_{N/2}kN^{-1} \qquad k = 0,1,2,\ldots,N/2 \tag{5}$$

where, Δt_s is the sampling interval.

Using,

$$z \equiv e^{2\pi i f \Delta t_s} \tag{6}$$

the entire complex frequency plane may be mapped onto the unit circle in the z-plane. Then, except for normalization convention, the estimate of the power spectrum of c(t) from N discretely sampled C_k can be written,

$$P(f) = \left| \sum_{k=-N/2}^{N/2-1} C_k z^k \right|^2. \tag{7}$$

But, equation (7) is not the true power spectrum which is actually given by the infinite power series,

$$P(f) = \left| \sum_{k=-\infty}^{\infty} C_k z^k \right|^2. \tag{8}$$

Equation (7) is known as the all zeros method of approximating Equation (8). That method essentially produces a polynomial approximation to the true spectrum. If there are sharp features in the spectrum or if it is desired to break up the spectrum into discrete bands it is preferable to

have a method which allows poles in the approximation rather than only zeros. We can derive such an expression by using the approximation,

$$P(f) \approx \frac{1}{\left| \sum_{k=-N/2}^{N/2-1} b_k z^k \right|^2} = \frac{a_0}{\left| 1 + \sum_{k=-N/2}^{N/2-1} a_k z^k \right|^2}. \qquad (9)$$

Where the a_k's and the b_k's can be determined from the condition that the first M+1 terms in the series in (9) must agree with the series in (8). This approximation is known as the all poles method and also as the Maximum Entropy Method or MEM. However, rather than compute the coefficients directly from (9) it is simpler to use the conclusion of the Wiener-Khinchin Theorem which says, in terms of the z-transform, that

$$\frac{a_0}{\left| 1 + \sum_{k=-N/2}^{N/2-1} a_k z^k \right|^2} \approx \sum_{j=-M}^{M} \varphi_j z^j. \qquad (10)$$

Where, φ_j, is the autocorrelation of C_k at lag j. The integer M, which is known as the "order" of the approximation can be any number up to N, the number of autocorrelations available. It turns out that the matrix equation leading to the solution of (10) can be written as a Toplitz matrix which has a symmetry which allows solution by a recursive algorithm. This solution method has been exploited by many researchers in solving autoregressive time series models (See, for example, Box and Jenkins, Time Series Analysis, Prentice-Hall, New York, 1978).

The subroutines MEMCOF and ELVMEM (taken from the subroutine library supplied with Numerical Recipes) implement the MEM method of spectral analysis. The method is often able to resolve peaks in a spectrum with fewer data points than required with the FFT (after spectral averaging). Both N and M are supplied by the user and by adjusting those values, the smoothness (sharpness) of the spectrum can be controlled. Moreover, the user is not restricted to integer powers of 2 as in most FFT algorithms. For further practical advice on the use of MEMCOF and ELVMEM, the reader is referred to Numerical Recipes.

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