Removal of Respiratory Influences From Heart Rate Variability in Stress Monitoring

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***Abstract*—This paper addresses a major weakness of traditional heart-rate-variability (HRV) analysis for the purpose of monitoring stress: sensitivity to respiratory influences. To address this issue, a linear system-identification model of the cardiorespiratory system using commercial heart rate monitors and respiratory sensors was constructed. Subtraction of respiratory driven fluctuations in heart rate leads to a residual signal where the effects of mental stress become more salient. We experimentally validated the effectiveness of this method on a binary discrimination problem with two conditions: mental stress of subjects performing cognitive tasks and a relaxation condition. In the process, we also propose a normalization method that can be used to compensate for ventilation differences between paced and spontaneous breathing. Our results suggest that, by separating respiration influences, the residual HRV has more discrimination power than traditional HRV analysis for the purpose of monitoring mental stress/load.**

***Index Terms*—Heart rate variability, mental stress, respiratory sinus arrhythmia, system identification, wearable sensors.**

I. Introduction

A WEALTH of information about the state of the autonomic nervous system (ANS) can be obtained from an analysis of inter-beat intervals of the heart, commonly referred to as heart-rate-variability (HRV). The power spectrum of HRV shows a low-frequency band (LF: 0.04–0.15 Hz) reflecting sympathetic contributions (i.e., related to the "fight-or-flight" or stress response) and a high-frequency band (HF: 0.15–0.5 Hz) that is dominated by parasympathetic activity, which occurs when the body is at rest. For these reasons, the ratio of LF to HF power has been widely used as an index of sympathetic-to-parasympathetic balance [1] and of mental stress and mental load [2]. Measurements of HRV are also robust, relatively unobtrusive, and affordable with consumer-grade heart rate monitors (HRM), which makes them suitable for long-term ambulatory monitoring.

However, HRV can be influenced by factors other than mental stress, and one of the most influential short-term factors is respiration. For example, when a subject breathes slowly (i.e., below ten breaths per minute), both respiration and mental stress contribute to LF energy in the HRV power spectrum. Thus, when using HRV as a measure of stress it is critical to separate respiratory contributions to HRV from those that are due to the psychological stressors of interest. Despite this fact, however, respiratory influences are rarely considered in studies of HRV.

To address this issue, this paper proposes a method to detect mental stress from HRV that takes into account respiratory influences. Our approach consists of first modeling the effects of breathing on HRV through an autoregressive moving average model, and then subtracting respiratory-driven predictions from the HRV signal. The result is a residual signal that is dominated by activation in the sympathetic branch. In an earlier paper presented at the 2010 IEEE Sensors Conference [3], we established proof-of-concept for the proposed method using an experimental protocol where subjects had to follow a paced breathing signal. Here, we extend the method to consider spontaneous breathing, a necessary generalization for the method to be useful in ambulatory studies. For this purpose, we also propose a normalization technique to compensate for differences in lung volume between paced and spontaneous breathing.

This paper is organized as follows. Section II provides background on HRV analysis as a measure of autonomic cardiac control, and reviews the literature on modeling of the cardiorespiratory regulatory system. Section III describes the system-identification approach we use to model and reduce respiratory influences. Section IV describes the experimental protocol and wearable sensor system that was used to validate the method, while experimental results are provided in Section V, followed by a discussion and conclusion in Section VI.

II. Background

A. HRV as a Measure of Autonomic Cardiac Control

HRV analysis is a useful tool to assess cardiac autonomic function. HRV is influenced by gender [4], long-term factors such as aging and illness, and short-term factors such as mental stress, respiration, and physical exercise [1], [2]. Because of these properties, HRV has been employed in diverse areas ranging from psychological studies to clinical risk assessments.

Long-term factors are those that affect HRV for more than a month. As an example, HRV reduces with age due to loss of elasticity in the cardiac muscles [5]. Altered long-term HRV may also imply an increased risk of vascular failure, which could lead to death [6]. HRV is also associated with several forms of cardiac illnesses. Patients with coronary artery disease who exhibit reduced HRV are at an increased risk for cardiac mortality [7]. Myocardial infarction leads to decreased HRV

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at night because it reduces the body's ability to activate vagal dominance during sleep, which in turn is associated with higher risk for cardiac failure [8]. Lower HRV can also be a sign of autonomic dysregulation in the early stages of hypertension [9]. Finally, HRV has been linked to several mental disorders. As an example, depressed patients exhibit significantly lower beat-to-beat intervals and HF power, indicating decreased parasympathetic activity [10], and phobic anxiety, which can cause sudden cardiac death, is characterized by decreased HRV [11].

HRV is also influenced by mid-term factors, such as circadian rhythms. As an example, Huikuri *et al.* [12] analyzed HRV over periods of 24 h; their analysis showed a high variability before subjects woke up (reflecting a high vagal tone) and low values after the subjects were awake. In [13], Malpas and Purdie observed that HRV rises again during sleep, and the absence of these cycles in HRV can be an indication of cardiac failure. Ischemic stroke patients are another group that does not display these cycles due to cardiovascular autonomic dysregulation [14]. In contrast, people undergoing heavy physical training showed significantly more rhythm in parasympathetic activity during both day and night [15]. Parasympathetic activity is lower in NREM sleep than in REM sleep; an opposite trend is seen in sympathetic activity [16].

Short-term factors such as respiration and mental stress also have a heavy influence on HRV. Various forms of stressors (e.g., public speaking, mental arithmetic, or reaction-time tests) can lead to increased sympathetic activity and decreased parasympathetic activity [2]. As for emotion, anger leads to sympathetic activation, whereas appreciation produces a shift in HRV power spectrum towards the HF band; earlier studies [17] have also suggested that positive emotions lead to alterations in HRV. Physical activity is also a dominant confounding factor of HRV. During exercise, the HF component reflects an increased respiratory rate and the LF peak changes with the baroreceptor reflex [18]. In addition, exercise and passive tilt result in a short-term increase of HRV, which is related to increased HF power [19], [20].

Among the various factors that influence HRV, however, breathing is the most dominant one; increased breathing intervals result in increased inter-heartbeat intervals. At normal breathing rates, the effect of respiration on HRV begins during expiration and progresses slowly, though these influences are not so clearly evident at faster breathing rates [21]. This relationship between breathing and HRV is maximized at slow breathing rates of around 0.1 Hz [22]. Thus, given that respiration is one of most dominant factors influencing HRV, removing its influence from HRV is likely to increase the prominence of other factors.

B. Modeling of Cardiorespiratory Relationship

Computational modeling of the cardiorespiratory regulation system has been widely used in studies of HRV. In their seminal work [23], Saul and colleagues developed a transfer function model to explain the relationship between heart rate and respiration in various body postures (i.e., supine and tilt). Their results showed that the gain of the transfer function from respiration to heart rate is larger for the LF band than for the HF band, and that the gain is maximized at 0.1 Hz. To estimate parameters of the transfer function between instantaneous lung volume (ILV) and heart rate, Yana *et al.* [24] used a linear model where current heart rate was expressed as a linear combination of past ILV values; model coefficients were estimated by least squares. Other authors have explored the use of arterial blood pressure (ABP) as an additional input to the regulatory model [25], [26]. Although these multivariate cardiorespiratory models may be able to explain the hemodynamic behavior of the heart, measurement of ABP is invasive and therefore impractical in ambulatory settings.

HRV can be influenced by various breathing parameters such as changes in the concentration of carbon dioxide, respiration frequency, and ILV [27]. Among these parameters, however, work by Pöyhönen *et al.* [27] has shown that respiratory frequency is the most dominant contributor to HRV oscillations. In their study, a decrease in respiration rate induced an increase in the LF power of HRV in all subject groups, while changes in other breathing parameters showed only partial influences in a few subject groups. This result indicates that the change of respiration frequency is a more important influence in modulating HRV than changes in tidal volume.

III. Methods

A. Cardiorespiratory Model for Paced Respiration

We employed a special case of the autoregressive moving average with exogenous inputs (ARMAX) model [28]. An ARMAX model estimates the output of the system as a linear combination of previous inputs, outputs, and errors

$$y(t) = \sum_{\tau=1}^{n_a} a(t)y(t-\tau) + \sum_{\tau=1}^{n_b} b(\tau)x(t-n_k-\tau+1) + \sum_{\tau=1}^{n_c} c(\tau)e(t-\tau) + e(t) \quad (1)$$

where $y(t)$, $x(t)$, and $e(t)$ are the output, input, and error at time t, $a(\tau)$, $b(\tau)$, and $c(\tau)$ are their respective predictor coefficients , parameters n_a, n_b, and n_c are their corresponding model orders, and n_k is the delay before an input influences the output of the system. In our proposed model, the system output $y(t)$ is the heart period, whereas the input $x(t)$ is the respiration signal with sample delay $n_k = 1$ (exclude only current input).

If we regard the sum of all terms excluding input terms as a residual $r(t)$, the model can be represented as

$$y(t) = b(0) + \sum_{\tau=1}^{n_b} b(\tau)x(t-\tau) + r(t). \quad (2)$$

Thus, in our model, the current heart period is represented as a weighted sum of inputs from past respiratory measurements and a residual signal assumed to be related to stress. Model coefficients can be estimated through least squares as

$$\hat{b} = \arg\min_b \sum_t r^2(t) = \arg\min_b \sum_t (y(t) - \hat{y}(t))^2$$
$$\text{where } \hat{y}(t) = b(0) + \sum_{\tau=1}^{n_b} b(\tau)x(t-\tau). \quad (3)$$

Equation (2) can be expressed in matrix form as

$$y(t) = X_t\hat{b} + r(t) \tag{4}$$

where X_t is a tap delay of previous respiratory measurements ($X_t = [x(t-1), \ldots, x(t-2), \ldots, x(t-n_b), \ldots, 1]$), which leads to the pseudo-inverse solution

$$\begin{aligned} \hat{b} &= \left(X^T X\right)^{-1} X^T Y \\ \text{with } X &= [X_1 \quad X_2 \quad X_3 \quad \ldots]^T \\ \text{and } Y &= [y(1) \quad y(2) \quad y(3) \quad \ldots]^T. \end{aligned} \tag{5}$$

B. Normalized Model for Spontaneous Respiration

The above model assumes that ventilation is similar regardless of the respiratory conditions (i.e., forced versus spontaneous breathing). However, when subjects breathe following a pacing signal, the breathing volume tends to be larger (higher power) than that under spontaneous breathing [29]. This is problematic because modeling the cardiorespiratory transfer function requires a broadband respiratory signal, which can only be ensured if the subject follows a suitable pacing signal [23]. Thus, the resulting transfer function tends to underestimate HRV when the input is a spontaneous breathing signal. To account for this difference, we introduce a scaling factor α in the ARMAX model (2), which results in

$$y(t) = \alpha\left[b(0) + \sum_{\tau=1}^{n_b} b(\tau)x(t-\tau)\right] + r(t). \tag{6}$$

As before, we estimate ARMAX model coefficients through the pseudo-inverse solution $\hat{b} = \left(X^T X\right)^{-1} X^T Y$ using data from an initial calibration condition with paced breathing. Then, and for a given respiratory trace X_t under spontaneous breathing, we estimate HRV as $\hat{y}(t) = X_t\hat{b}$, and find the scale factors that minimize the error

$$\hat{\alpha} = \arg\min_{\alpha} \sum_t r^2(t) = \arg\min_{\alpha} \sum_t \left(y(t) - \alpha\hat{y}(t)\right)^2. \tag{7}$$

Currently, we employ an exhaustive search with a resolution of $\alpha = 0.01$ to find the optimal scale factor.

IV. Experimental

A. Wearable Sensors

To collect heart period and respiration data, we used a small and lightweight wearable sensor platform that we have developed for ambulatory monitoring of mental stress. Shown in Fig. 1, the system consists of a holster unit and a variety of wireless physiological sensors known to be correlated with mental stress. Namely, we capture HRV using a commercial heart-rate-monitor (HRM) (Polar WearLink+; Polar Electro Inc.), which our previous studies have shown provides comparable results to those obtained with electrocardiograms [30]. In turn, respiration can be measured with either a piezoelectric respiratory effort sensor (ultra piezo strap sensor 480420; Gereonics Inc.) or a pressure-based respiration sensor (SA9311M; Thought Technology Ltd.). Each respiration sensor has its advantages: the piezo sensor has a very low profile but is sensitive to motion artifacts, whereas the pressure-based sensor works well in the presence of body movements though at the expense of a larger package. As shown in Fig. 1, both respiration sensors are integrated with the HRM into a chest strap for added comfort.[1] Two additional wireless sensor modules allow synchronized measurement of electrodermal activity (i.e., skin conductance) and electromyography. Sensors wirelessly transmit to the holster unit using a low-power protocol (SimpliciTI, Texas Instruments Inc.). Since the focus of our study is the cardiorespiratory system, these two additional sensors were not used here.

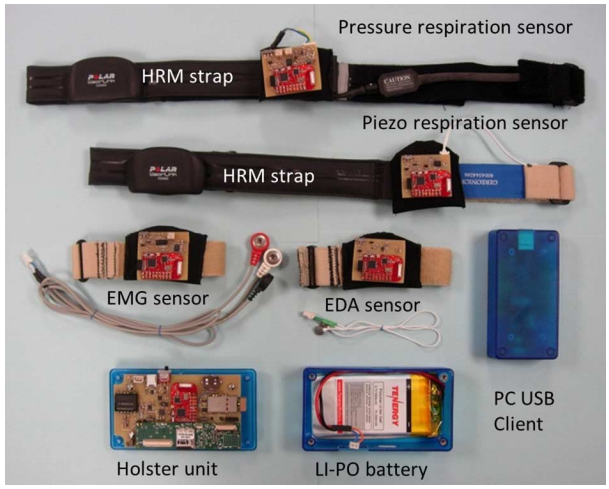

Fig. 1. Prototype of the current wearable wireless sensor system.

The holster unit contains an embedded Linux-centric platform (Verdex Pro; Gumstix, Inc.) for data storage (mini SD flash), real-time signal processing, and wireless networking. The holster unit also integrates a 3D accelerometer (LIS344ALH; STMicroelectronics, Inc.), a GPS unit (RXM-GPS-SR-B; Linx Technologies Inc.), a real-time clock unit (DS1308; Dallas Semiconductor, Inc.), a heart rate receiver module (Polar RMCM01; Polar Electro Inc.), and a wireless transceiver (Ez430-RF2500; Texas Instruments Inc.).

B. Experimental Protocol

To validate the suggested linear model for the purposes of detecting mental stress, we collected HRV and respiration data from four subjects on two experimental conditions (relaxation and mental stress), and an additional calibration phase from which the coefficients of the transfer function was estimated. Two different studies were performed: a study with paced respiration to test the basic ARMAX model in (2), and a second study with spontaneous breathing to test the normalized model in (6).

1) Paced Breathing: For the paced respiration study, subjects were asked to breathe following a pacing sound with a fixed period of 6.67 s for both experimental conditions (stress and relaxation); this pace ensured that respiratory influences affected both the LF and the HF components of the HRV power spectra. For the mental stress condition, subjects were asked to perform a dual task [31] consisting of target tracking and memory search

[1]The studies reported here were based on the pressure-based sensor.

Fig. 2. Computerized dual task test used to induce mental stress in the paced breathing study. Subjects are asked to track a moving target by moving the mouse. At the same time, subjects have to click the left-mouse button in response to a target letter being displayed (three letters were given at the beginning of the task).

Fig. 3. Computerized Stroop color word test used to induce mental stress in the spontaneous breathing study. Subjects are asked to press one of the four panels at the bottom of the screen according to the ink of the word being displayed (in this case, the subject is to press the RED panel).

tasks while following the pacing signal. For the breathing condition, subjects were asked to rest quietly while following the pacing signal.

During the dual-task test, subjects had to track a moving target by moving a small square with the mouse. At the same time, subjects also had to click the left-mouse button when one of three target letters was displayed on the screen. Subjects were asked to memorize these three target letters at the beginning of the task. Distractor letters (i.e., other than three target letters) were also randomly displayed; in the case of distractors, subjects were asked not to click the mouse. Subjects were provided instantaneous visual feedback about their overall performance by means of three error bars on the screen. A screen shot of the dual tracking test is shown in Fig. 2.

2) Spontaneous Breathing: For the spontaneous respiration study, subjects were not provided a pacing sound and instead were asked to breathe freely. For the mental stress condition, subjects were asked to perform a Stroop color word test (CWT) [31]; this ensured that our experimental findings were not constrained to a particular type of stressor. For the breathing condition, subjects were asked to rest quietly.

During the CWT test, subjects were shown one of four words (red, green, blue, or yellow) displayed with different ink colors, and had to click on one of four buttons according to the ink color (e.g., when presented with the word "red" in color blue, subjects were to select the button labeled as "blue"). A screen shot of the CWT is shown in Fig. 3. Each word was presented for 1000 ms, after which subjects had an additional 300 ms to respond; failure to respond within the total 1300 ms was treated as the same error as selecting the wrong button. To make the task more challenging, the test switched between two modes (congruent and incongruent) every 30 s. In congruent mode, the concept and the ink color were the same, e.g., the word "red" was presented in red. In incongruent mode, the concept and ink color were different, e.g., the word "blue" was presented in red. Subjects were provided with instantaneous feedback on their performance at the top of the screen.

3) Calibration: During an initial calibration phase, which was common to both paced and spontaneous breathing studies, subjects were asked to breathe following a sinusoidal pacing sound, with periods randomly drawn from a modified Poisson process [23]; this ensured that breathing signal had a nearly flat power spectrum over a broad range of respiratory frequencies. In a Poisson process, the distribution function (8) describes the probability of observing k events within at given time interval τ if the independent and identically distributed (i.i.d.) events occur at an average rate λ

$$P(X=k)=\frac{(\lambda\tau)^k e^{-\lambda\tau}}{k!};\quad k=0,1,2,\ldots. \tag{8}$$

From this, the interval τ between two consecutive events can be shown to follow the exponential density function (9):

$$\mathrm{f}(\tau)=\lambda e^{-\lambda\tau}\ \text{for}\ t>0. \tag{9}$$

In our case, we used a mean breathing period $1/\lambda = 3.66$ seconds to generate random breathing periods. As a modification to the traditional Poisson process, the minimum and maximum interval limits were set to 2 and 10 s, respectively; breathing periods outside this range were dropped to avoid discomfort (which may also induce stress).

Each of the three conditions (relaxation, stress, and calibration) lasted 5 min, and each subject ($n=4$) repeated the three conditions on three consecutive days. Respiratory signals were recorded at a sampling rate of 10 Hz. Then, RR tachograms and respiratory signals were uniformly resampled to a common 4 Hz rate, and band-pass filtered between 0.04 and 0.5 Hz to remove the VLF (very low frequency) component. The experiment protocol was approved by the Institutional Review Board at Texas A&M University, all subjects provided written informed consent for the study.

V. Results

A. Spectral Content in Cardio-Respiratory Signals

We conducted a preliminary experiment to illustrate the extent to which the HRV and respiratory signals share similar spectral content. For this purpose, we collected data from three different respiration settings: shallow breathing with a fixed 3.5-s period, deep breathing with a fixed 10-s period, and broadband breathing. For each condition, HRV and respiration signals were

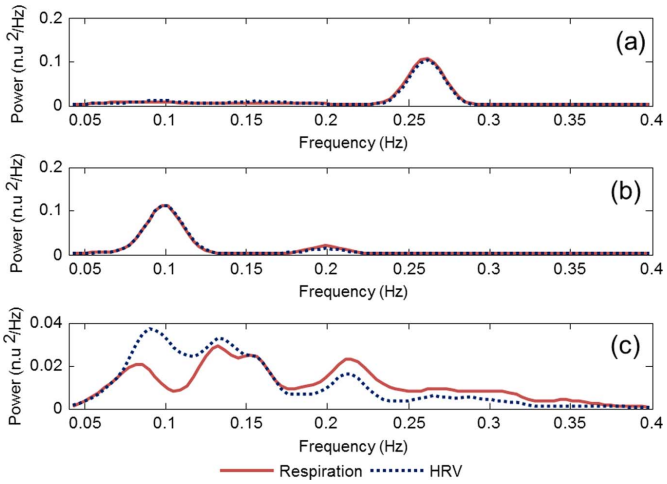

Fig. 4. Power spectra for HRV and respiration with: (a) fixed breathing period of 10 s, (b) fixed breathing period of 3.5 s, and (c) broadband breathing following a Poisson process. Data corresponds to subject #1.

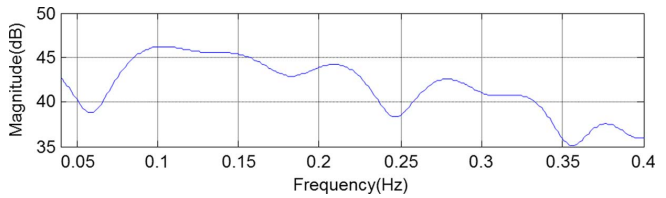

Fig. 5. Magnitudes of the transfer function for broadband breathing. Data corresponds to subject #1.

collected for 5 min. Subjects ($n = 4$) were asked to breathe following a pacing sound. In the two breathing conditions with a fixed period, when HRV is dominated by respiration, the normalized power spectrum of HRV shows high similarity to that of the respiratory signal. Fig. 4(a) and (b) shows results for subject #1; all subjects showed a similar pattern between HRV and respiration. These results indicate that, when breathing occurs over a narrow frequency band, HRV and respiration peak at the same frequency and have similar spectral content. When compared to fixed breathing, results from the broadband breathing condition [see Fig. 4(c)] show more noticeable differences between the two power spectra, though both signals display a peak at the same frequencies; as before, this pattern was observed on all subjects.

To gain further insight, we calculated the magnitude of the transfer function from respiration to HRV using the broadband respiratory data. Results are shown in Fig. 5 for one subject. The transfer function displays a maximum at a breathing frequency of 0.1 Hz, followed by a spectral slope from 0.1 to 0.4 Hz. This result is consistent with those reported in [23], and indicates that the HRV transfer function has higher gain in the LF band than in the HF band. As a consequence, HRV can be heavily influenced by respiration when breathing occurs within the LF band, which limits its diagnostic value for mental stress unless respiratory effects are accounted for.

B. Stress Detection With Paced Respiration

HRV spectra during the paced breathing study displayed a dominant peak at 0.15 Hz [see Fig. 6(b)], which followed the

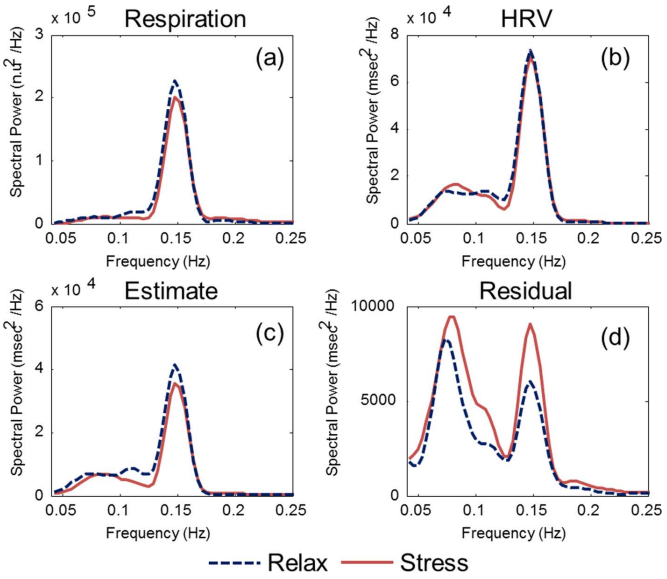

Fig. 6. Power spectrum of: (a) respiration signal (input), (b) HRV signal (output), (c) prediction by MA model, and (d) residual signal following paced breathing at 0.15 Hz. Data corresponds to subject #3.

respiration pacing signal [Fig. 6(a)]. These results indicate that, on the basis of the HRV signal alone, it is quite challenging to discriminate between the two experimental conditions (stress versus relaxation). We then used the derived ARMAX model to predict the HRV signal from the respective respiratory signal for both stressed and relaxed conditions. Power spectra for the predicted HRV signal and residual signal are shown in Fig. 6(c) and (d), respectively. The residual signal for the stress condition has more spectral power than that of the relaxed condition, which supports our hypothesis that residual analysis is more discriminatory than HRV analysis.

To confirm these findings, we compared the information content of the traditional HRV analysis and the residual analysis by means of a pattern classifier. First, we split the data recordings into 60-s windows with a 15-s shift, then extracted LF and HF power using Welch's method [32]. Each window was treated as a different sample, resulting in 71, 67, 70, and 71 windows for each subject, respectively (some windows had to be discarded due to noisy measurements). We trained a quadratic classifier [33] for each subject using data from two days and tested it on data from the remaining day; this process was repeated three times per subject, one for each day of data collection. The problem was setup as a binary classification problem, where the goal was to discriminate the mental stress conditions from the relaxation conditions. Classification results are summarized in Fig. 7. In all four subjects, classification performance of the residual signal outperformed that on HRV. In case of subjects 1 and 4, where HRV provides low classification rates, the residual signal outperformed HRV analysis by 72% and 75%, respectively. In the case of subjects 2 and 3, on which HRV tends to work better, the residual signal still improved classification performance by 11% and 22%, respectively. In addition, classification performance on the residual signal had significantly lower standard error within and between subjects.

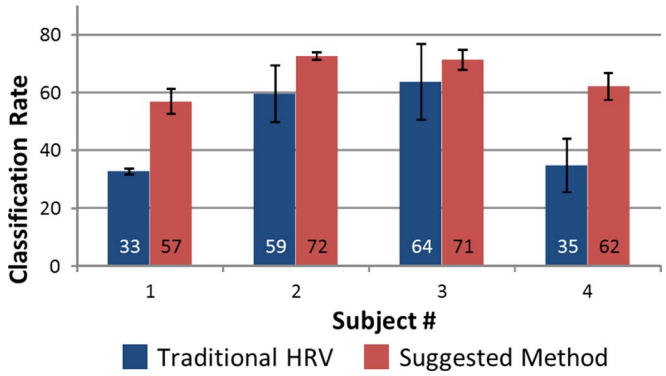

Fig. 7. Mean classification rate and standard error for HRV PSD and residual PSD when subjects breathe at a paced rate of 0.15 Hz.

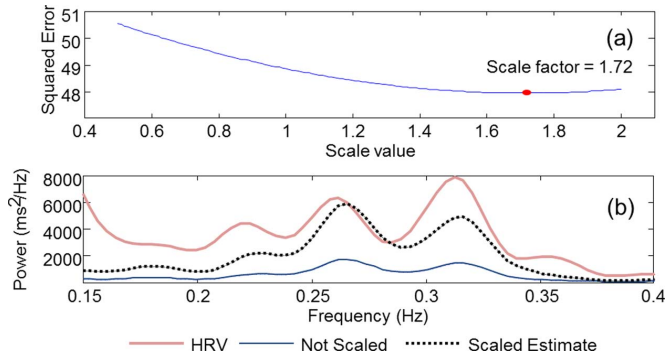

Fig. 8. (a) Mean squared prediction error as a function of the scaling factor. (b) Comparison of unscaled ($\alpha = 1.0$) and scaled ($\alpha = 1.72$) predictions. Data corresponds to subject #4.

C. Stress Detection With Spontaneous Respiration

The effects of the normalization gain (6) on HRV predictions from spontaneous breathing are illustrated in Fig. 8. Without normalization ($\alpha = 1$), the model underpredicts the HRV since the ARMAX parameters are based on a paced respiratory condition. Introducing the multiplicative scaling factor addresses this issue. Fig. 8(a) illustrates the sensitivity of the model with respect to the scaling factor; a minimum mean-squared error prediction is obtained for $\alpha = 1.72$ (data corresponds to subject #4).

Inspection of the respiratory signals indicates that subjects tend to follow different breathing patterns during the two experimental conditions: slow breathing when relaxed, and faster breathing when stressed (Fig. 9). Though this result shows that the respiratory signal itself may contain a significant amount of discriminatory information, it is important to note that respiration can be under voluntary control, which questions its validity as an objective measure of stress.

Results on the residual analysis are illustrated in Fig. 10 for a particular experiment with subject #4. The HRV signal [Fig. 10(b)] shows peaks at similar frequencies as the respiratory signal [Fig. 10(a)], but also at other frequencies that cannot be explained by the respiratory behavior. Subtraction of respiratory predictions shows that residual energy lies primarily in the 0.1 Hz band, a region that has been hypothesized to be a resonance of the cardiac system (see results in Section V-A).

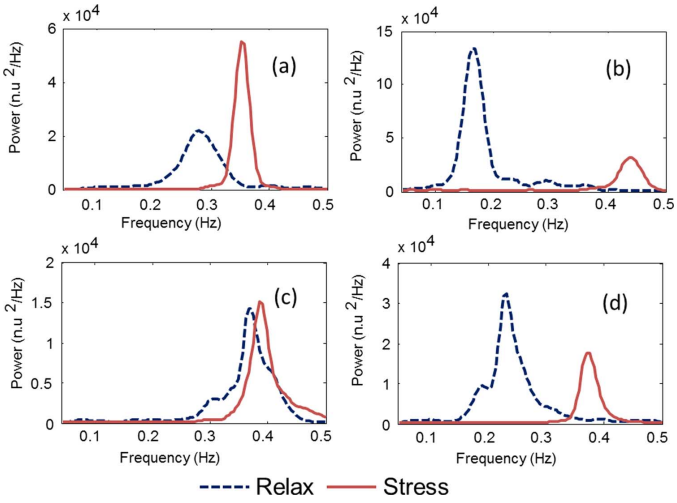

Fig. 9. Respiratory spectra for the four subjects under the two experimental conditions (relax versus stress). All subjects tend to breathe faster under stress. (a)–(d) Corresponds to subjects #1–4.

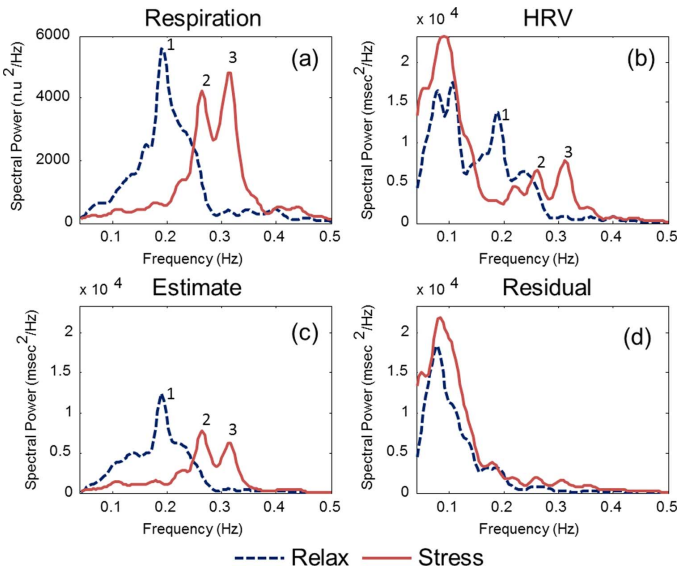

Fig. 10. Power spectrum of: (a) respiration signal (input), (b) HRV signal (output), (c) prediction by ARMAX model, and (d) residual signal under spontaneous breathing; data corresponds to subject #4. Numbers in (a) and (b) indicate corresponding peaks.

More importantly, the results in Fig. 10(c) and (d) suggest an alternative interpretation of our model. Whereas traditional HRV analysis decomposes the power spectral density into two fixed spectral bands (LF versus HF), our model suggests that HRV may be decomposed into two components: one that correlates with respiratory behavior [energy in Fig. 10(c)], and one that is orthogonal to it [energy in Fig. 10(d)]. The decomposition afforded by our model is advantageous because (unlike the LF/HF ratio) it is not affected by the particular breathing rate; what matters is the ratio between the HRV energy that is correlated with respiration (whatever the respiration rate may be) and the HRV energy that is uncorrelated with respiration. This interpretation is numerically assessed next.

Following the procedure described in Section V-B, we compared HRV analysis against residual HRV analysis through a pattern recognition experiment. Namely, we built a quadratic

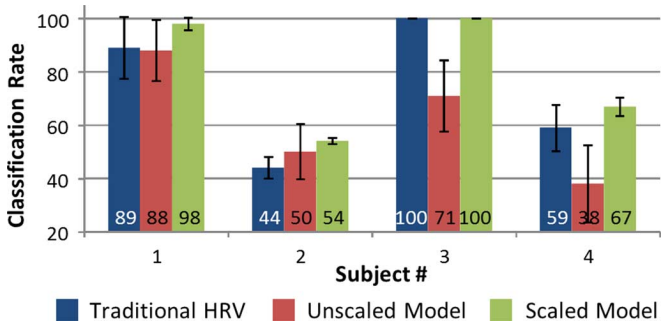

Fig. 11. Mean classification rate and standard error for HRV and residual analysis when subjects breathe spontaneously.

classifier to discriminate between stress and relaxation conditions on the basis of two HRV features (HF and LF power), and built a second classifier to discriminate between stress and relaxation conditions based on two residual HRV features: the predicted HRV power in Fig. 10(c) and the residual HRV power in Fig. 10(d). Fig. 11 summarizes the classification results. For subjects 1, 2, and 4 the residual features outperform the HRV PSD by 10%, whereas for subject 3 both methods obtained a 100% classification rate. In addition, classification results on the predicted/residual signal have significantly lower standard deviations than those based on raw HRV. This pattern of results was also observed with the results on paced breathing presented in Section V-B. To assess the relative contribution of the normalization stage, we also compared the normalized (scaled) model against the baseline model ($\alpha = 1$). Results in Fig. 11 show that the scaled model performs better for all subjects, leading to higher classification rates and lower variance than the baseline (unscaled) model.

VI. Discussion and Conclusion

The LF/HF ratio of HRV has been widely used as an index of autonomic nervous balance, and also as a measure of mental load/stress. Measurements of heart rate are robust, relatively unobtrusive, and affordable, so they have broad potential for long-term ambulatory monitoring. However, various confounding factors can also influence the LF/HF in addition to psychophysiological stress; among these, the effect of respiration is immediate and most dominant. At normal breathing rates (12–20 breaths per minute, on average), respiratory influences in HRV tend to fall within the HF band. However, breathing can often fall within the LF band, in which case the LF/HF ratio loses its diagnostics value. This issue is compounded by evidence that the cardiorespiratory system has a resonance in the frequency range of 0.1 Hz; thus, when breathing within the LF band, the effects of respiration tend to be amplified. Unless respiratory contributions are considered, HRV analysis may lead to erroneous results.

To address this issue, we have introduced a system identification method to compensate for respiratory influences. Our approach consists of modeling the effect to respiration on HRV with an ARMAX model; namely, given a tap delay of respiratory measurements, the model predicts the next heart period. In this fashion, the model can be used to subtract (linear) influences of respiration on HRV, which we hypothesized would make the effects of stress more salient on the resulting residual signal. To account for ventilation differences between paced breathing (which is required to obtain a broadband respiration signal for the ARMAX model) and spontaneous breathing, we also introduce a scaling factor that allows us to apply the learned ARMAX model in more natural settings where the subjects breathe freely. We validated our method using experimental data from four subjects, and our results show the superiority of the residual HRV signal over the traditional HRV approach, both during paced and spontaneous breathing conditions. Our modeling approach can also be interpreted as a decomposition of HRV energy into two orthogonal components: one that correlates with respiratory behavior and one that is orthogonal to it. This decomposition is advantageous because, unlike the traditional LF/HF ratio, it does not rely on fixed frequency bands but instead adapts to the respiratory rate of the subject.

Several directions of future works are being considered at the time of this writing. First, our study indicates that day-to-day variations are a major source of noise; data from subjects whose HRV remained stable across days yielded higher classification rates. Thus, practical calibration procedures are needed in order to compensate for differences from day to day. Second, the classification models tend to be user-dependent given the idiosyncrasy of physiological signals. Thus, more work is required in order to identify robust cardiorespiratory transfer functions that generalize across subjects. Third, the present study relied on a linear model to capture cardiorespiratory relationships. Nonlinear extensions (e.g., Volterra series) may allow us to capture the known nonlinear dynamics of the cardiorespiratory system [30]. Finally, we are in the process of conducting a large ambulatory study to test the ability of our model to detect mental stress in activities of daily living.

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