Frequency-Domain Electrodermal Activity Index of Sympathetic Function

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Abstract— **A novel approach to quantify sympathetic function via the electrodermal activity (EDA) variability (EDAV) is proposed. EDAV involves power spectral density analysis of EDA data, focusing on the normalized power within the frequency band from 0.045 to 0.15 Hz, termed EDALFn. To test this index, orthostatic stress was induced on N = 10 subjects. Results showed a significant increase in the EDALFn when the subjects went from supine to standing positon. We also evaluated heart rate variability analysis on simultaneously measured electrocardiogram data. In contrast to EDALFn, the purported sympathetic measure (HRVLF and HRVLFn) did not show consistent increase in the spectral power at the above described frequency band when subjected to the orthostatic stress. Moreover, we found that EDALFn exhibited lower coefficient of variability when compared to the traditional time-domain measures of EDA indices.**

I. Introduction

Assessment of alterations in sympathetic tone is one of the major research fields in cardiovascular research. Sympathetic function has been demonstrated to have important pathophysiological, physiological and clinical relevance in many diseases [1].

Heart rate variability (HRV) is a low–cost technique based on electrocardiographic (ECG) signal beats detection and processing [2]. Power spectrum density (PSD) analysis of the HRV have been used to assess the autonomic nervous system [3]. The high-frequency (HF) components of HRV are known to be solely influenced by the parasympathetic system. The low–frequency (LF, 0.045–0.15 Hz) variability derives from the influence of the cardiac sympathetic nerves, but it is also influenced by the vagus, so the LF spectral power of HRV is not a reliable measure of sympathetic nerve traffic to heart.

Because of the need to fully elucidate the sympathetic system dynamics, analysis of electrodermal activity (EDA) has gain certain popularity in recent years [4]–[6]. This is due to that EDA reflects only activity within the sympathetic branch of the autonomic nervous system, because it is noticeable that there is no parasympathetic innervation of eccrine sweat glands. EDA is a measure with strong correlation to sweat production, defined as a measure of the changes in electrical conductance of the skin [7], [8].

There are two main time-domain measures based on EDA: the skin conductance level (SCL) and the nonspecific skin conductance responses (NS.SCRs). SCL and NS.SCRs are consistent with sympathetic nervous arousal. However, they are merely moderately positively correlated with each other, and highly variable between subjects [9]. Further development of EDA analysis, like EDAV, is needed.

Thus, the aim of this work is to examine if a similar association of LF components of HRV to sympathetic function also exists with the sudomotor function as measured by EDA. Specifically, we are interested in examining if EDAV analysis yields similar frequency dynamics at the HRV's prescribed LF range of 0.015-0.4 Hz. If such relation occurs, we can overcome the main disadvantage of LF domain analysis of HRV. To test our aim, we determined the EDA differences between baselines and induced orthostatic stress.

II. Materials and Methods

A. Protocol

Ten healthy volunteers (9 males, 1 female) of ages ranging from 18 to 36 years old (27.3 ± 6.39), weight 73.27 ± 10.11 kg, and height 175.62 ± 4.38 cm, were enrolled in this study. No gender–related differences have been reported for EDA or sympathetic function. Postural stimulation was induced using a straight forward protocol, including 4 minutes in supine position and 4 minutes standing. The experiment was carried out a quiet, dimly lighted room (ambient temperature, 26–27 °C), in order to avoid other interferences that might elicit sympathetic arousal. Before the test, the subjects were asked to stay in supine position for 5 min to procure hemodynamic stabilization. The study protocol was approved by the Institutional Review Board of The University of Connecticut and all volunteers consented to be subjects for the experiment.

ECG and EDA signals were simultaneously recorded, using the HP 78354A (Hewlett–Packard) and the GSR (ADINSTRUMENTS) module respectively. ECG electrodes were placed following recommendations to acquire Lead I and EDA electrodes were placed in index and middle finger for all subjects. Skin was prepared with alcohol before placing the electrodes. Signals were digitized using PowerLab system at 1 kHz, 12 bits resolution.

B. Signal processing

Fig. 1 shows an example of heart rate and EDA signals for a given subject. For HRV analysis, ECG signals were band–pass filtered (0.05–40 Hz) offline to reduce noise and motion artifacts. The R–waveform peaks were detected using a widely–used peak detection algorithm [10], [11], and HRV (RR segments) series were computed. The power spectra of HRV were calculated using Welch's periodogram method with 50% data overlap. The RR interval series were converted to an evenly time sampled signal (4 Hz) by cubic spline interpolation. A Blackman window (256) was applied to each segment and the Fast Fourier Transform was calculated for each windowed segment. Finally, the power spectra of the segments were averaged. Low–frequency (LF,

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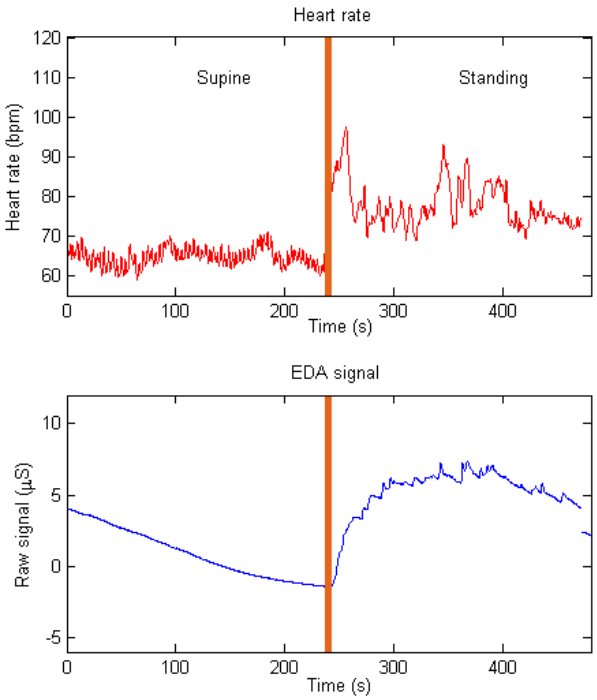

Figure 1. Changes on HRV and EDA for a given subject, as a result of orthostatic stimulation.

[ms2]) index (0.045 to 0.15 Hz) [3] and normalized LF (LF/Total power, [n.u., i.e. normalized units]) were computed.

For each subject, two minutes of post–transient tonic EDA signal were selected from each body position, starting from 30 seconds after the signal recording begins for supine and from 30 seconds after the subject changes position for standing stage. All EDA signals were down–sampled to 2 Hz and high–pass filtered (0.01 Hz) to eliminate any very–low–frequency trend and baseline. The power spectra of EDA signals were calculated using Welch's periodogram method with 50% data overlap. A Blackman window (128) was applied to each segment and the Fast Fourier Transform was calculated for each windowed segment. Finally, the power spectra of the segments were averaged. Total power was computed for every EDA signal. Low–frequency (LF, 0.045 to 0.15 Hz) power spectral components of EDA [µS2], as well as normalized to total power LF [n.u.] were computed.

SCL (µS) and NS.SCRs (times/minute) were also computed for the two minutes of tonic EDA signal selected of each body position, for each subject. Fig. 2 illustrates the process. To extract the tonic component of the EDA signals, a low–pass FIR filter with a cut–off of 0.0004 Hz was applied. The remaining signal (raw signal minus tonic component) was used to compute the NS.SCRs. SCL index was computed using the mean of the tonic EDA over the two–minute period. NS.SCRs were obtained manually for each minute and then averaged over the two–minute period. It is important to notice that for the measurement of NS.SCRs it is needed to define a minimum change in conductance to be considered as a response. When a second response occurred before completion of a response, two responses were count

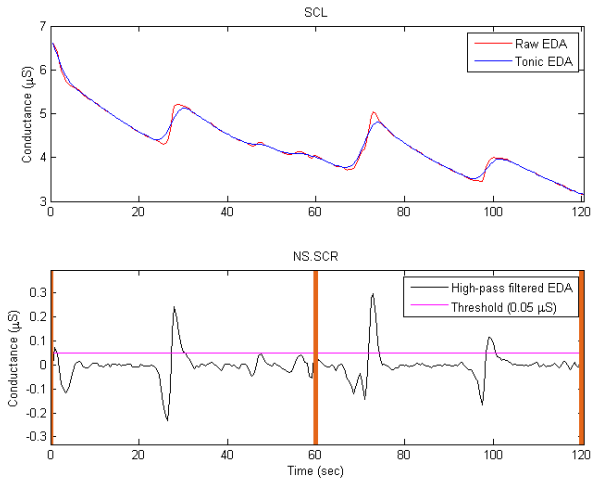

Figure 2. SCL and NS.SCRs. Measures of tonic EDA.

even though they overlapped. A threshold of 0.05 µS was used as recommended in the literature [4].

III. Results

Fig. 3 includes an example of power spectra of HRV series and EDA signal for a given subject. Notice the remarkable differences in the power spectra of HRV and EDA, between the different body positions. It is possible to see that HRV power spectra have components above 0.15 Hz. Those components are known to be related to parasympathetic function and usually go up to 0.5 Hz. For its part, EDA spectra have some components above 0.15 Hz, but they are less sensitive to body position.

Table I shows the computed indices for the supine and standing body positions. Information on the mean HR was included as well. One–tail paired t–test was applied for the full data set (i.e. HRV–LF, normalized HRV–LF, SCL, NS.SCRs, EDA–Total power, EDA–LF and normalized EDA–LF), in order to see if there are significant increasing on those prospective sympathetic markers. Comparisons between them were performed to evaluate whether those index can quantitatively differentiate the sympathetic arousal between the baseline and under orthostatic stress.

TABLE I. SYMPATHETIC FUNCTION INDICES

	Supine	Standing
	HRV indices	
HRV–LF (ms^2)	2.29 ± 1.68	16.66 ± 28.55
Normalized HRV–LF (n.u.)	0.228 ± 0.114	0.348 ± 0.195
Mean HR	65.99 ± 18.92	88.39 ± 14.47
	EDA indices	
Mean SCL (µS)	–2.166 ± 1.676	1.56 ± 3.64[a]
NS.SCRs (times/min)	0.65 ± 1.434	4.65 ± 3.75 [a]
EDALFn (n.u.)	0.137 ± 0.075	0.367 ± 0.212 [a]

a. Denotes statistically significant differences

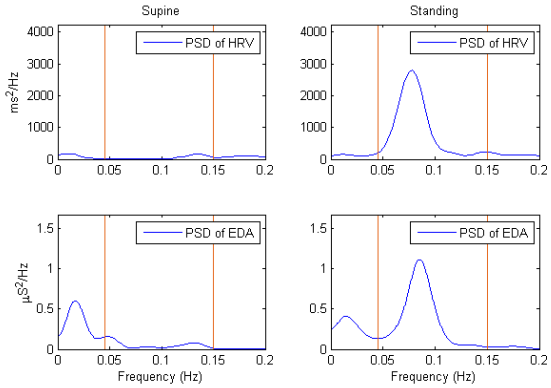

Figure 3. Power spectra of HRV (top) and EDA (bottom) for supine (left) and standing (right) positions for a given subject. Lines denote 0.045 and 0.15 Hz.

The t-test analysis was intended to measure of the power of the indices to distinguish between the postures. The statistically significant differences between stages indicate also the possibility to develop a sensitive and specific quantitative measure able to discriminate the physical stress induced by the different postures. HRV measures were not able to provide statistically significant differences between standing and supine (baseline). SCL and NS.SCRs exhibited the ability to provide significantly different measures between standing and supine. Normalized LF components of EDA were able to provide an index statistically different between baseline and orthostatic stress.

Beyond the differences between stages, high variability of EDA measurements has been a concern and an impediment to spread the use of this indices for assessing the general state of activation of the sympathetic system. Table 2 shows the ratio between the standard deviation and the mean for tonic EDA indices. The right most column is the average of such ratio. The aforementioned ratio is called coefficient of variability. Although NS.SCRs index showed a lower variability than SCL, both mean SCL and NS.SCRs exhibited relatively high variability values compared to normalized EDA-LF. Normalized EDA–LF (EDALFn) exhibited lower variability.

IV. Discussion

EDA provides functional information of sudomotor activity [7], [8], and sudomotor activity, in turn, is controlled by the sympathetic nervous system [12]–[14]. In practice, EDA is employed to measure sweat production because it is known that sweat gland activity modulates conductance of an applied current [4], [15], [16]. More recently, EDA has been used to measure the sympathetic activities [5]. However, due to the high variation of the time-domain measures of EDA, which are SCL and NS.SCRs, its use has not yet been widely deployed. The PSD of EDA variability (EDAV), similar to the PSD of HRV, is not yet a widely-adopted measure. To this end, the primary goal of this work was to test our hypothesis that if EDA does truly represent the cardiac sympathetic nervous activity, we should also see significantly elevated spectral power in the LF band (0.04-0.15 Hz).

TABLE II. Coefficient of Variation

	Supine	Standing	Average
Mean SCL	0.77	2.33	1.55
NS.SCRs	2.21	0.81	1.51
EDALFn	0.55	0.58	0.56

This frequency band has been widely accepted to represent the sympathetic function via the PSD of HRV analysis, when subjects were given stimuli to invoke the sympathetic nervous system. Indeed, with EDALFn, we found such evidence with induction of orthostatic stress which is known to invoke the central and cardiac sympathetic nervous dynamics [5].

The added benefit of analyzing EDA in the frequency domain can lead to less variability in the features estimated due to the inherent filtering properties of the frequency domain. More importantly, the EDA represents only the sympathetic nervous activities whereas the LF of the HRV exhibits both the parasympathetic in addition to the sympathetic tone. Thus, by combining both EDAV and HRV, there is the potential to identify separate dynamics of the sympathetic and parasympathetic nervous activities.

EDA–LF normalized index was significantly higher between supine and standing positions. That means that such index is able to quantitatively distinguish the difference in sympathetic arousal between the body positions. The fact that LF components are more suitable to discriminate between body positions indicates that such components are likely to be related to the overall sympathetic function, and that the remaining spectrum components are affected by thermoregulation and other processes.

Currently available techniques for sympathetic function assessment involve hemodynamic measurements [17], [18], adrenergic and ganglionic pharmacological blockade [19]–[22], noradrenaline measurement in urine or plasma [23]–[25], neurophysiological approach [26], [27], plasma noradrenaline kinetics [28], [29], heart rate variability (HRV) analysis [3], [30], [31] or imaging techniques [32]. Whether the high cost, the invasiveness of the technique, the impossibility to provide continuous monitoring, or the inability to separately assess sympathetic control prevent the widespread use of these techniques in sympathetic function assessment.

For its part, the presented frequency-domain technique has the potentiality to be used as the basis of a system to automatically and continuously assess sympathetic function. The transient that appears when changing position can be used as a trigger, 30 seconds can be provided to stabilize the signal and then 2 minutes of EDA can be used to evaluate the sympathetic function. Even though postural changes are a good application for sympathetic function assessment and a commonly used method to test and corroborate new techniques, it is necessary to perform autonomic blockade studies and experiments involving neuropathy-diagnosed subjects, to fully corroborate the sensitivity and specificity of the technique.

V. Conclusion

Based on the results, we can conclude that low frequency components power of EDAV is a suitable discriminant of central sympathetic arousal related to orthostatic stress. As a result, it is a potential quantitative measurement of sympathetic function. It is much more sensible than LF index of HRV for such a task, and exhibited a lower coefficient of variation, compared to other measures of tonic EDA.

References

[1] G. Grassi and M. Esler, "How to assess sympathetic activity in humans," *J. Hypertens.*, vol. 17, no. 6, pp. 719–734, Jun. 1999.

[2] J. T. Shepherd and G. Mancia, "Reflex control of the human cardiovascular system," *Rev. Physiol. Biochem. Pharmacol.*, vol. 105, pp. 1–99, 1986.

[3] "Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology," *Eur. Heart J.*, vol. 17, no. 3, pp. 354–381, Mar. 1996.

[4] W. Boucsein, D. C. Fowles, S. Grimnes, G. Ben-Shakhar, W. T. roth, M. E. Dawson, D. L. Filion, and Society for Psychophysiological Research Ad Hoc Committee on Electrodermal Measures, "Publication recommendations for electrodermal measurements," *Psychophysiology*, vol. 49, no. 8, pp. 1017–1034, Aug. 2012.

[5] R. Freeman and M. W. Chapleau, "Testing the autonomic nervous system," *Handb. Clin. Neurol.*, vol. 115, pp. 115–136, 2013.

[6] A. P. Colbert, K. Spaulding, A. Larsen, A. C. Ahn, and J. A. Cutro, "Electrodermal activity at acupoints: literature review and recommendations for reporting clinical trials," *J. Acupunct. Meridian Stud.*, vol. 4, no. 1, pp. 5–13, Mar. 2011.

[7] P. H. Ellaway, A. Kuppuswamy, A. Nicotra, and C. J. Mathias, "Sweat production and the sympathetic skin response: Improving the clinical assessment of autonomic function," *Auton. Neurosci.*, vol. 155, no. 1–2, pp. 109–114, Jun. 2010.

[8] M. Benedek and C. Kaernbach, "A continuous measure of phasic electrodermal activity," *J. Neurosci. Methods*, vol. 190, no. 1–5, pp. 80–91, Jun. 2010.

[9] A. Crider and R. Lunn, "Electrodermal lability as a personality dimension," *J. Exp. Res. Personal.*, vol. 5, no. 2, pp. 145–150, 1971.

[10] M. E. Nygårds and L. Sörnmo, "Delineation of the QRS complex using the envelope of the e.c.g," *Med. Biol. Eng. Comput.*, vol. 21, no. 5, pp. 538–547, Sep. 1983.

[11] C. Vidaurre, T. H. Sander, and A. Schlögl, "BioSig: the free and open source software library for biomedical signal processing," *Comput. Intell. Neurosci.*, vol. 2011, p. 935364, 2011.

[12] B. M. W. Illigens and C. H. Gibbons, "Sweat testing to evaluate autonomic function," *Clin. Auton. Res.*, vol. 19, no. 2, pp. 79–87, Apr. 2009.

[13] C. Setz, B. Arnrich, J. Schumm, R. La Marca, G. Tröster, and U. Ehlert, "Discriminating stress from cognitive load using a wearable EDA device," *IEEE Trans. Inf. Technol. Biomed. Publ. IEEE Eng. Med. Biol. Soc.*, vol. 14, no. 2, pp. 410–417, Mar. 2010.

[14] J. . Healey and R. W. Picard, "Detecting stress during real-world driving tasks using physiological sensors," *IEEE Trans. Intell. Transp. Syst.*, vol. 6, no. 2, pp. 156–166, Jun. 2005.

[15] J. T. Cacioppo, L. G. Tassinary, and G. Berntson, "Psychophysiological Science: Interdisciplinary Approaches to Classic Questions About the Mind," in *Handbook of Psychophysiology*, 3 edition., Cambridge England ; New York: Cambridge University Press, 2007.

[16] "Techniques in Psychophysiology. By I. Martin and P. H. Venables. (Pp. 669; illustrated; £19.50.) John Wiley & Sons: Chichester. 1980.," *Psychol. Med.*, vol. 10, no. 04, pp. 806–806, 1980.

[17] J. Brod, V. Fencl, Z. Hejl, and J. Jirka, "Circulatory changes underlying blood pressure elevation during acute emotional stress (mental arithmetic) in normotensive and hypertensive subjects," *Clin. Sci.*, vol. 18, pp. 269–279, May 1959.

[18] C. B. Thomas, J. A. Stanley, and M. A. Kendrick, "Observations on some possible precursors of essential hypertension and coronary artery disease. VII. The subjective reaction to the cold pressor test as expressed in the verbal response," *J. Chronic Dis.*, vol. 14, pp. 355–365, Sep. 1961.

[19] G. Grassi, S. Vailati, G. Bertinieri, G. Seravalle, M. L. Stella, R. Dell'Oro, and G. Mancia, "Heart rate as marker of sympathetic activity," *J. Hypertens.*, vol. 16, no. 11, pp. 1635–1639, Nov. 1998.

[20] S. Julius, A. V. Pascual, and R. London, "Role of parasympathetic inhibition in the hyperkinetic type of borderline hypertension," *Circulation*, vol. 44, no. 3, pp. 413–418, Sep. 1971.

[21] N. K. Hollenberg, D. F. Adams, H. Solomon, W. R. Chenitz, B. M. Burger, H. L. Abrams, and J. P. Merrill, "Renal vascular tone in essential and secondary hypertension: hemodynamic and angiographic responses to vasodilators," *Medicine (Baltimore)*, vol. 54, no. 1, pp. 29–44, Jan. 1975.

[22] B. Egan, R. Panis, A. Hinderliter, N. Schork, and S. Julius, "Mechanism of increased alpha adrenergic vasoconstriction in human essential hypertension.," *J. Clin. Invest.*, vol. 80, no. 3, pp. 812–817, Sep. 1987.

[23] M. Esler, G. Jennings, P. Korner, I. Willett, F. Dudley, G. Hasking, W. Anderson, and G. Lambert, "Assessment of human sympathetic nervous system activity from measurements of norepinephrine turnover," *Hypertension*, vol. 11, no. 1, pp. 3–20, Jan. 1988.

[24] R. D. Hoeldtke, K. M. Cilmi, G. A. Reichard, G. Boden, and O. E. Owen, "Assessment of norepinephrine secretion and production," *J. Lab. Clin. Med.*, vol. 101, no. 5, pp. 772–782, May 1983.

[25] I. J. Kopin, "Catecholamine metabolism: basic aspects and clinical significance," *Pharmacol. Rev.*, vol. 37, no. 4, pp. 333–364, Dec. 1985.

[26] A. B. Vallbo, K. E. Hagbarth, H. E. Torebjörk, and B. G. Wallin, "Somatosensory, proprioceptive, and sympathetic activity in human peripheral nerves," *Physiol. Rev.*, vol. 59, no. 4, pp. 919–957, Oct. 1979.

[27] W. Delius, K. E. Hagbarth, A. Hongell, and B. G. Wallin, "General characteristics of sympathetic activity in human muscle nerves," *Acta Physiol. Scand.*, vol. 84, no. 1, pp. 65–81, Jan. 1972.

[28] G. L. Brown and J. S. Gillespie, "The output of sympathetic transmitter from the spleen of the cat," *J. Physiol.*, vol. 138, no. 1, pp. 81–102, Aug. 1957.

[29] T. Bradley and P. Hjemdahl, "Renal extraction of endogenous and radiolabelled catecholamines in the dog," *Acta Physiol. Scand.*, vol. 126, no. 4, pp. 505–510, Apr. 1986.

[30] S. Akselrod, D. Gordon, J. B. Madwed, N. C. Snidman, D. C. Shannon, and R. J. Cohen, "Hemodynamic regulation: investigation by spectral analysis," *Am. J. Physiol.*, vol. 249, no. 4 Pt 2, pp. H867–875, Oct. 1985.

[31] A. Malliani, M. Pagani, F. Lombardi, and S. Cerutti, "Cardiovascular neural regulation explored in the frequency domain," *Circulation*, vol. 84, no. 2, pp. 482–492, Aug. 1991.

[32] D. S. Goldstein, "Clinical assessment of catecholaminergic function," *Stress Catecholamines Cardiovasc. Dis. N. Y. Oxf. Univ. Press*, pp. 234–286, 1995.