Temporal Dynamics of Multimodal Stress Biomarkers: ECG, EDA, and PPG

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Abstract

Early stress detection using wearable sensors requires understanding which modality responds first. Most studies compare stress versus baseline as static epochs, discarding temporal information. We investigated the temporal dynamics of ECG, EDA, and PPG stress biomarkers, including the anticipatory phase.

We analyzed the WESAD dataset (15 subjects) recorded during the Trier Social Stress Test. Six features were selected: heart rate (HR) and the root mean square of successive RR-interval differences (RMSSD) from ECG; skin conductance level (SCL) and skin conductance response (SCR) rate from EDA; and normalized pulse area and pulse skewness from PPG. In sliding 30-s windows we computed paired Cohen's d against a 5-min resting baseline, including the preceding transitions.

ECG responded earliest: heart rate rose already during the anticipatory transition before the formal stress onset (d = 1.6 during stress), an elevation not explained by movement (wrist accelerometry). EDA responded more slowly. PPG showed strong effects (pulse area d = −2.3), though with higher variability. Meditation produced a mirror-image pattern; amusement and baseline showed minimal effects, confirming stress specificity.

ECG captures the stress response earlier than EDA, emerging already during the anticipatory phase, and appears most suitable for early wearable stress detection.

1. Introduction

Wearable sensors allow stress to be monitored continuously and unobtrusively in daily life. The three modalities most commonly available in wearable devices are electrocardiography (ECG), electrodermal activity (EDA), and photoplethysmography (PPG). They reflect complementary physiological pathways: cardiac autonomic control [1], sympathetic sudomotor activity [2], and peripheral vascular tone [3], respectively.

Most studies of stress detection compare stress and baseline as static epochs [4,5]. This discards the temporal information in the transition into stress: the period immediately preceding a stressor, in which anticipation and preparation already engage the autonomic nervous system, is typically excluded from the analysis.

In this work we ask which modality responds first, and which responds most specifically, when stress develops. We study this on recordings from the Trier Social Stress Test (TSST), a standardized laboratory stressor; since the TSST has no sharp stimulus onset, we do not estimate absolute onset latencies but describe the relative order and temporal dynamics of the responses across modalities, using a sliding-window effect-size analysis that includes the transients preceding each condition.

The contributions are threefold: (i) the cardiac response precedes the electrodermal one, with PPG tracking cardiac dynamics with strong but variable effects; (ii) cardiac activation precedes the formal stressor onset in every subject, and wrist accelerometry shows that it is not driven by movement; (iii) specificity: amusement produces no comparable response and meditation a mirror-image pattern.

2. Methods

2.1. Data

We analyzed the WESAD dataset [6]: 15 healthy subjects recorded during a laboratory protocol built around the TSST [7]. The protocol comprised a baseline of about 20 minutes, a stress condition (TSST: a 5-min free speech followed by 5 min of mental arithmetic in front of a three-member panel), an amusement condition (funny video clips), and two guided meditations; the order of the stress and amusement blocks was counterbalanced across subjects. Transitions between conditions included self-report questionnaires; the TSST was additionally preceded by task instructions and a 3-min speech-preparation period, a documented anticipatory phase of the protocol.

Chest ECG was recorded at 700 Hz (RespiBAN), and a wrist device (Empatica E4) provided PPG at 64 Hz, EDA at 4 Hz, and acceleration (ACC) at 32 Hz.

Because the analysis concerns timing across modalities, we aligned the wrist signals to the chest ECG ourselves

instead of relying on the gesture-based synchronization of the dataset: beat-to-beat pairing of QRS complexes and PPG pulses was used to estimate a constant offset and a linear clock drift, followed by a spline correction of the residual, yielding sub-second timing accuracy.

2.2. Feature extraction

We extracted 57 features in sliding 30-s windows (5-s step): 26 heart rate variability features from ECG, 21 EDA features, and 10 morphological PPG features.

2.3. Feature selection

Six features were selected as those with the largest paired Cohen's d versus baseline (while remaining stable at rest), taking the two best per modality so that all three physiological pathways are covered: cardiac autonomic (ECG), sudomotor sympathetic (EDA), and peripheral vascular (PPG). They are the mean heart rate (HR) and the root mean square of successive RR-interval differences (RMSSD) from ECG; the skin conductance level (SCL) and skin conductance response (SCR) rate, i.e., the tonic and phasic EDA components; and the normalized pulse area and pulse skewness from PPG (Table 1).

Table 1. Selected features and their group-level effect size (paired Cohen's d vs. baseline) during the stress phase.

Modality	Feature	Cohen's d
ECG	HR (↑)	+1.6
	RMSSD (↓)	−0.9
EDA	SCL (↑)	+0.9
	SCR rate (↑)	+1.6
PPG	Pulse area (↓)	−2.3
	Pulse skewness (↑)	+1.0

2.4. Temporal analysis

The recordings were arranged on a common pseudo-protocol axis with four epochs: baseline (BL), TR_{Str} + stress (Str), TR_{Amu} + amusement (Amu), and TR_{Med} + meditation (Med), where TR_x denotes the transient immediately preceding condition x. So that every window contains all 15 subjects, each segment was trimmed to the shortest subject; transients were anchored to the condition onset and conditions to their start. Meditation occurred twice per subject, giving N = 30 meditation epochs.

The first 150 s of the baseline were discarded; the following 10 min are displayed in Fig. 1, and the next 5 min served as the reference. In every window we computed

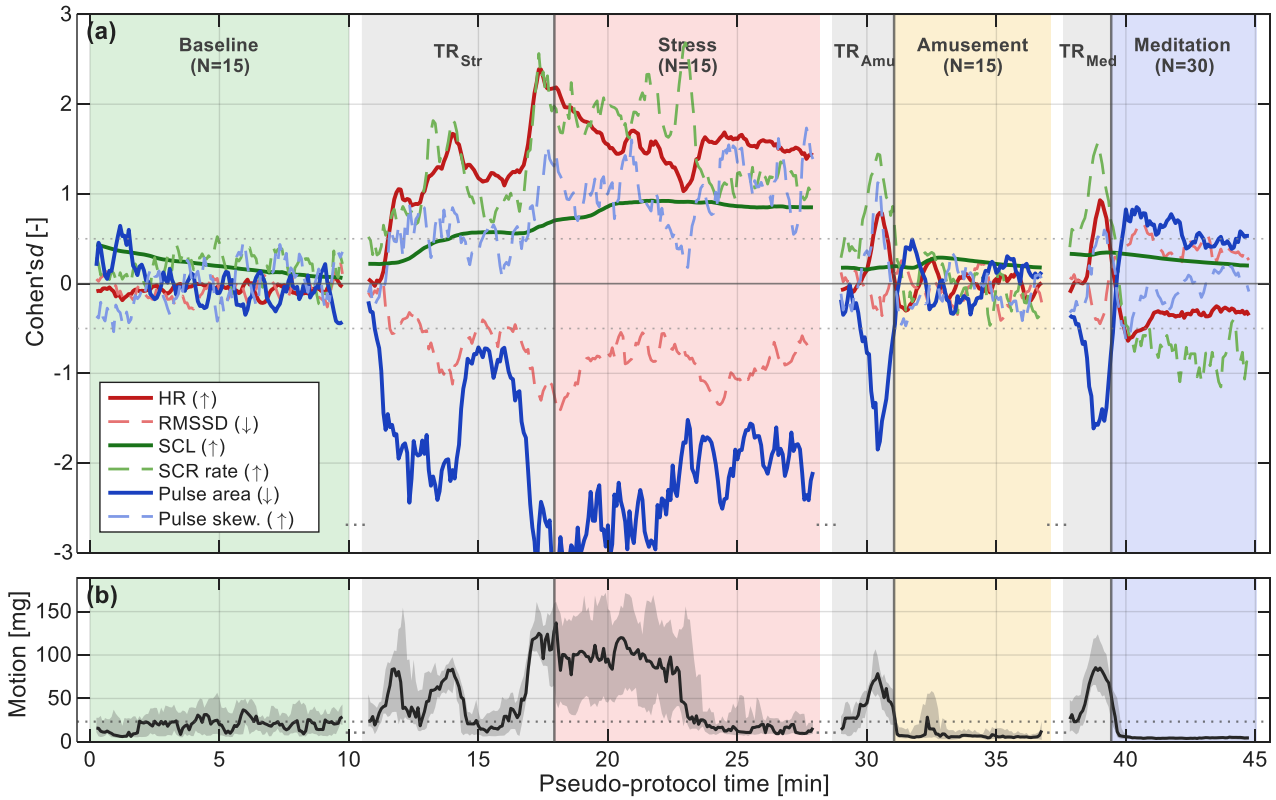

Figure 1. (a) Group-level effect size (paired Cohen's d vs. the 5-min baseline reference) of the six selected features on the pseudo-protocol axis (dots mark axis breaks, vertical lines the condition onsets). (b) Motion intensity. The line and shaded band show the median and interquartile range across subjects; the dotted line marks the 75th percentile of baseline motion.

the paired Cohen's d of the feature against this 5-min baseline reference (mean paired difference divided by the pooled standard deviation).

As a movement control, motion intensity was quantified from wrist accelerometry as the standard deviation of the acceleration magnitude $\sqrt{(x^2+y^2+z^2)}$ within each window, expressed in milli-g (1 mg = 10^{-3} g); the measure is independent of sensor orientation and is shown on the same time axis in Fig. 1b.

3. Results

3.1. Temporal ordering of modalities

ECG responded first (Fig. 1a). HR was elevated throughout the transient preceding stress (mean d ≈ 1.2 in TR_{Str}, peaking at 2.4 around the stress onset) and remained high during the stress phase (mean d = 1.6), while RMSSD decreased (d = −0.9), consistent with vagal withdrawal. The tonic SCL increased slowly and reached its plateau (d ≈ 0.9) only during the stress phase itself. The SCR rate was elevated already during the transient, but this proved to be nonspecific (Section 3.3).

PPG showed the largest effect magnitudes (pulse area d = −2.3, pulse skewness d = +1.0), closely tracking the cardiac dynamics, but also the largest between-subject variability of all modalities.

3.2. Pre-stressor response vs. movement

Motion was elevated in all transients, about 2–2.5 times the baseline level (Fig. 1b), reflecting questionnaires, repositioning, and postural changes. Critically, during the last 2–3 minutes before the stress onset motion returned to baseline (below the dotted threshold), while the HR effect remained at d ≈ 1.2. In contrast, in TR_{Amu} and TR_{Med} the cardiac deviations coincided with motion bursts and returned to zero when the movement stopped. The sustained pre-stressor elevation therefore cannot be attributed to movement or post-movement recovery.

3.3. Specificity and subject consistency

Across conditions, baseline and amusement produced no systematic effects (|d| < 0.3), confirming specificity for stress, whereas meditation produced a mirror-image pattern with four of the six features reversing sign.

At the level of individual subjects (Fig. 2), HR increased in TR_{Str} in 15 of 15 subjects (median +13.7 bpm; +18.7 bpm during stress). RMSSD was individually inconsistent: it decreased in only 10 of 15 subjects during stress, with an interquartile range of 48 ms against a median change of −15 ms. SCL was slightly above the reference in nearly all segments (11–14 of 15 subjects), so it is the magnitude of the change, not its sign, that carries

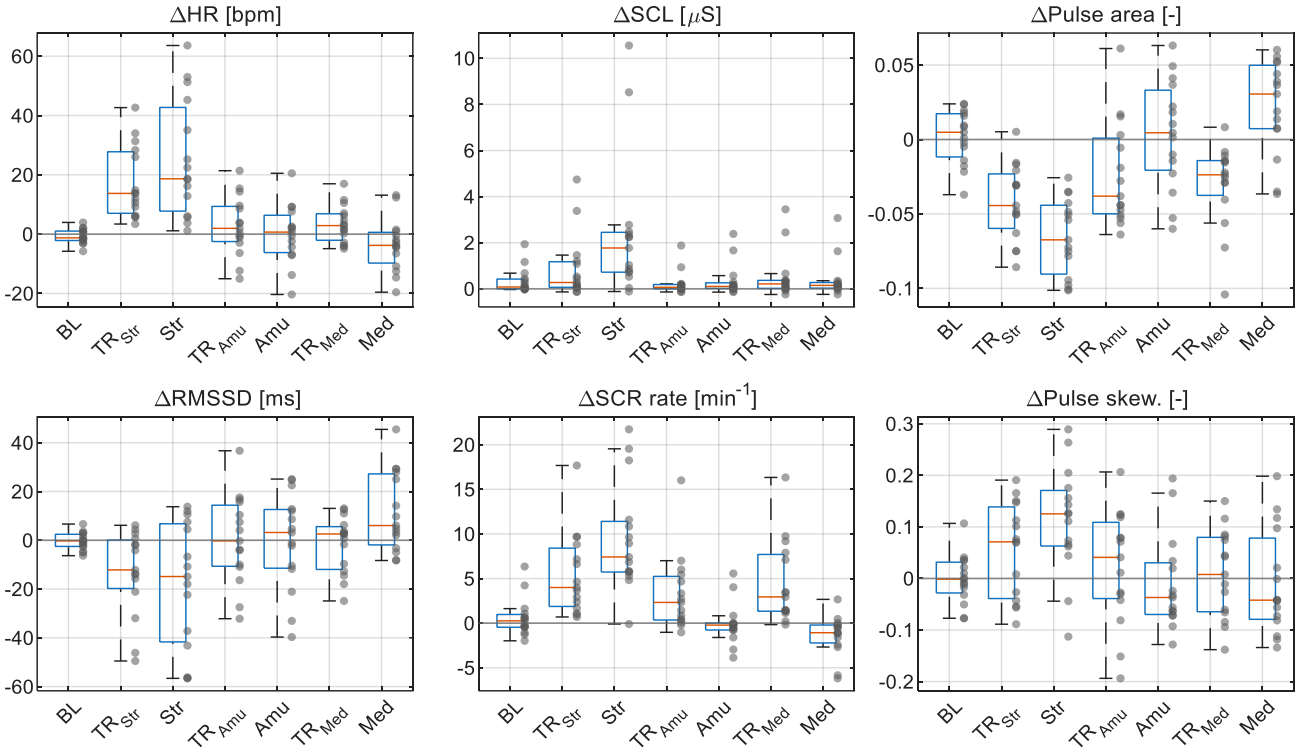

Figure 2. Per-subject changes relative to the baseline reference (Δ, native units) for the six selected features, grouped by modality into columns (ECG, EDA, PPG). Boxes show the median and interquartile range; each dot represents one subject (15 dots per segment; meditation values averaged over its two occurrences).

the information: +1.77 µS during stress. The SCR rate increased during stress (14/15) but also in every transient (15/15 before stress, 13/15 before amusement, 14/15 before meditation), i.e., even where no stressor followed.

PPG was directionally consistent also per subject: pulse area decreased in 15/15 subjects during stress and 14/15 already in TR_{Str}, and reversed during meditation in 12/15; pulse skewness was consistent only during stress (13/15).

4. Discussion

The observed order matches the physiology of the two pathways: the cardiac response, driven by rapid vagal withdrawal and sympathetic activation, changes within seconds, whereas tonic sudomotor activity accumulates over minutes; hence SCL did not reach its plateau until the stress phase itself. The small positive SCL offset seen in all segments more likely reflects slow electrode–skin hydration than arousal.

What causes the pre-stressor cardiac activation? Anticipation of the upcoming evaluation and the cognitive load of speech preparation are confounded by design [7]; for early detection it matters only that the response is psychogenic and precedes the stressor. The remaining explanations, movement and post-movement recovery, are ruled out: the response persisted in low-motion windows, whereas in the other transients heart-rate deviations tracked motion bursts and vanished with them (Sect. 3.2).

The SCR rate increased in every transient, including those after which no stressor followed: it conveys that "something is changing", not that "a stressor is approaching", consistent with the classical role of the SCR in the orienting response to novel or significant stimuli [2,8]. Our data cannot distinguish orienting, movement, and mild nonspecific arousal; on its own, phasic EDA is therefore unsuitable for specific early detection of stress, even though it reacts early.

PPG effects were strong and largely directionally consistent, in line with morphology-based stress assessment from PPG [5], but their magnitude varied widely between subjects, reflecting the motion sensitivity of wrist PPG and individual vascular reactivity. This volatility does not harm group-level detection but complicates individual thresholding and calibration.

The modalities differ sharply in wearability. Long-term ECG requires adhesive electrodes or a chest strap, with skin-irritation and comfort issues; smartwatch ECG is only an on-demand measurement requiring the other hand, ruling out continuous passive monitoring. PPG, in contrast, is acquired passively and continuously by virtually every wrist wearable. Since the key early marker is heart rate, wrist PPG can act as its carrier, with potential for warning of imminent stress or supporting its management.

Study limitations: a small sample (N = 15), a low wrist-EDA sampling rate (4 Hz), no sharp TSST onset (hence relative order, not absolute latencies), timing blurred by the 30-s windows, group-level effect-size curves, and incompletely documented transient content in the dataset.

5. Conclusion

ECG captures the stress response earliest: minutes before the formal stressor onset, in every subject, and independently of physical movement. EDA responds later and its phasic component is nonspecific during transitions; PPG responds strongly but with volatile magnitude. The cardiac channel is therefore the key to early stress detection from wearables, and for long-term wear it can be carried by wrist PPG through heart-rate estimation, whose further investigation is a priority.

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