Continuous Measurement of Finger Arterial Blood Pressure for Evaluating Noise-Induced Stress

Kontinuierliche Messung des arteriellen Fingerblutdrucks zur Bewertung von lärmbedingtem Stress

Bachelor thesis in the department of Electrical Engineering and Information Technology
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J. Helders

Abstract

Noise-induced stress is a prominent factor in modern daily life and poses a risk to mental and physical well-being. Due to low temporal resolution, blood pressure is rarely implemented in human stress detection. This work aimed to record a detailed timeline of the stress response to noise using a continuous non-invasive blood pressure (cNIBP) measurement device and assess its effectiveness as a stress indicator. An experimental study with 12 participants was conducted, combining a 2-back mental task with three noise conditions: road traffic noise at 58.12 and $52.12\,\mathrm{dB(A)}$ and ambient noise at $30.2\,\mathrm{dB(A)}$. The test consisted of a $60\,\mathrm{s}$ *Rest* phase, a $120\,\mathrm{s}$ *Trial* phase, and a $60\,\mathrm{s}$ *Recovery* phase. Systolic (sBP) and diastolic blood pressure (dBP), heart rate (HR), and interbeat intervals (IBI) were monitored with a cNIBP measurement device, the electrodermal activity (EDA) was recorded with a skin conductivity sensor, and psychological parameters were collected via a questionnaire. The measurement was split into 24 ten-second intervals and analysed through four comparisons to capture the dynamic of the stress response. Both sBP and dBP gradually increased during the *Trial* and were significantly elevated 30–$40\,\mathrm{s}$ after the onset of the stressor, followed by a sudden decrease within the first ten seconds of *Recovery*. The mean EDA level responded with an immediate increase in the first $10\,\mathrm{s}$, after which it slowly declined for the rest of the experiment. After noise exposure, the inspected EDA peak rate significantly decreased during *Recovery* compared to *Rest* and *Trial*. The HR spiked after the onset of noise at $58.12\,\mathrm{dB(A)}$, followed by a drop during recovery. The root mean square of successive differences (RMSSD), extracted from the IBI, and the psychological scores showed no significant differences throughout the experiment. In conclusion, it was shown that the BP and EDA were impacted similarly by the stressors of the experiment. The cNIBP recording provided detailed information on the recovery process and is a valuable addition to the field of human stress research. Meanwhile, the EDA had minimal latency when detecting the stressor's onset. The analysis of physiological and psychological results showed no evidence of a significant correlation between noise intensity and the magnitude of the stress response.

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Acronyms

AcH acetylcholine. 7

ACTH adrenocorticotropic hormone. 6, 7

ANS autonomic nervous system. 5, 6, 9

BP blood pressure. iii, 3, 6, 8, 12–14, 16, 17, 22, 25, 32, 50, 52–54, 56, 57

cNIBP continuous non-invasive blood pressure. iii, 2, 57

CNS central nervous system. 5

CRH corticotropin-releasing hormone. 6, 7

CVD cardiovascular disease. 7, 17

dBP diastolic blood pressure. iii, 8, 9, 13, 14, 39, 41, 51–53, 88–90

E epinephrine. 6, 7

ECG electrocardiography. 13, 14, 24

EDA electrodermal activity. iii, vi, 3, 9, 10, 12, 15, 16, 22, 23, 25, 31–33, 35, 44, 48, 50–52, 54, 56, 57, 97–99

GCs glucocorticoids. 6, 7

GSR galvanic skin response. 10, 12, 15, 16

HCU height correction unit. 19, 22

HPA hypothalamic–pituitary–adrenal. 1, 6–8

HR heart rate. iii, 6, 13, 14, 22, 25, 41, 51, 52, 54, 56, 91–93

HRV heart rate variability. 13, 15, 23, 24, 32, 33, 53, 56

IBI interbeat intervals. iii, 22–24, 33

ICG impedance cardiography. 14

ISI interstimulus interval. 11, 17, 18

ISO International Organization for Standardization. 20, 73

NE norepinephrine. 6, 7

PNS peripheral nervous system. 5, 6

PPG photoplethysmography. 8, 22, 24

PSNS parasympathetic nervous system. 6, 9, 24, 52

RMSSD root mean square of successive differences. iii, 24, 25, 32, 33, 44, 52–54, 56, 94–96

RTN road traffic noise. iii, 12, 14, 15, 17, 55

SAM sympathoadrenomedullary. 6–8

sBP systolic blood pressure. iii, 8, 9, 13, 14, 22, 39, 51–54, 56, 85–87

SCL skin conductance level. 9, 10, 15, 23, 31

SCR skin conductance response. 10, 23

SNS sympathetic nervous system. 6

STAI State Trait Anxiety Inventory. 21, 36, 38

TU Technische Universität. 17, 73

WNSS Weinstein Noise Sensitivity Scale. 21, 36–38, 50

1. Introduction

1.1. Motivation

Stress is a constant factor in modern day-to-day life. Whether at work or at home, we are exposed to various stressors. These stressors can occur in the form of performance pressure, interpersonal conflicts, health issues or noise. The European Environment Agency (EEA) estimates that 82 million people in urban areas in the European Union are exposed to road traffic noise levels of $55\,\mathrm{dB(A)}$ during the day-evening-night period [1]. Independent of the origin, all stressors trigger reactions in the human body, which sometimes happen unconsciously [2]. These stress responses can enhance performance and help us tackle challenges in the short term. However, prolonged exposure to chronic stress can have negative consequences for both physical and mental health. Research has shown that chronic stress impacts multiple systems in the body and can influence disease progression and recovery [2]. Persistent adaptions of the human body to stress, such as chronically elevated blood pressure, arteriosclerosis, or increased blood lipid levels, can increase the risk of cardiovascular disease [3] and stroke [4]. Additionally, prolonged activation of the hypothalamic–pituitary–adrenal (HPA) axis, a major system in the stress response, leads to increased secretion of the stress hormone cortisol. This weakens the immune system [5] and raises the risk of metabolic disorders like type 2 diabetes [6]. Furthermore, high cortisol levels are associated with insomnia and attention deficits [7], while anxiety, depression, burnout, and post-traumatic stress disorder are also connected to chronic stress exposure [8]. According to the German online database of Federal Health Monitoring (GBE)[9], in 2020, cardiovascular diseases cost €56.727 million, accounting for 13.3% of the overall cost for illness by healthcare providers, more than any other disease. The second highest cost was mental and behavioural diseases, which made up 13.1% of overall spending and cost €56.391 million. The many different adverse consequences of chronic stress and the cost associated with them demonstrate the need for preventative measures, starting with an early detection of stress.

In recent years, more and more focus has been put on the automatic detection of stress, and many studies have been conducted on the topic [10, 11]. Different modalities have been used to detect and classify the human stress response; some require complex experimental setups, while others fail to provide in-depth information and only paint a black-and-white picture. This is the case with the blood pressure measurements used most in research. They are usually done with conventional (automatic) sphygmomanometers, which operate based on oscillometry [12]. The process of acquiring blood pressure with such a device requires the inflation of a cuff on the upper arm, which takes about $40\,\mathrm{s}$ (deflation rate $3\,\mathrm{mmHg}$ per second [13], and normal systolic pressure of $120\,\mathrm{mmHg}$ [14]) and provides systolic, diastolic and mean arterial pressure once per measurement. Furthermore, repeated brachial artery occlusion due to short measurement intervals may induce changes in the measured pressure level [15]. Moreover, the European Society of Hypertension recommends a resting time of one minute between consecutive measurements [16]. This prohibits hemodynamic analysis with high temporal resolution, which makes blood pressure a modality unattractive for stress research, although it is affected by the body's response. However, this is not the case with continuous non-invasive blood pressure (cNIBP) measurements, which use a minimally obtrusive method for recording blood pressure changes on a beat-to-beat level. Because of these advantages, cNIBP-based measurements might provide additional insights into the human stress response. Yet, this has not been evaluated as a possible indicator of stress.

1.2. Objective

This work aims to examine the extent to which blood pressure, measured continuously via finger arterial pressure monitoring, can indicate noise-induced stress. For this, an experimental study will be designed and conducted in which participants are exposed to an acoustic stressor and perform a mental task. At the same time, their blood pressure and electrodermal activity are measured. A detailed time-series analysis will assess blood pressure's reliability as a stress indicator compared to the established gold standard, EDA. Additionally, the measured data will be combined into a collection and made available for further analysis.

The following research questions are proposed:

- How do the measurements change throughout the course of the experiment?

- Is the stress response influenced by different sound intensities regarding dynamic and overall amplitude?
- Does continuous blood pressure measurement provide a comparable insight into the human stress response to electrodermal activity?

1.3. Outline

This paragraph will provide an overview of the structure of this work. In Chapter 2, the foundations of this work will be presented. The terms “stress” and “stressor” will be defined and the concept of noise in this context is explained. Furthermore, the complex neurophysiological processes of the human stress response are laid out, followed by the technical background of the modalities used in this work. Finally, the concept of the *n-back task* is provided. Chapter 3 discusses and reviews the research done on this topic, with the focus electrodermal activity and blood pressure especially. Then, in Chapter 4, the methodology of the experiment conducted for this thesis will be presented, while Chapter 5 provides the detailed implementation of said methodology. The measurement results will be given in Chapter 6 and analysed in Chapter 7. Finally, Chapter 8 will conclude this work and give an outlook on future work.

2. Background

The following passages provide the necessary information to understand the next chapters. After defining stress, stressor and noise, the mechanisms behind the physiological stress response are highlighted. Furthermore, each measurement modality included in this work will be explained briefly.

2.1. Stress, Stressor and Noise

The term *stress* was coined by Hans Selye, who first used the word to describe the syndrome of "just being sick" and later defined stress as a general response to a demand upon the body [17]. Werdecker and Esch [2] characterise stress as possibly unconscious psychological, physiological or behavioural changes due to environmental and psychosocial stimuli, the so-called stressors. The German Federal Centre for Health Education (BZgA) specifies noise as a strong strain on an organism by internal or external stimuli [18]. Not every exposure to stress is necessarily bad. The body's performance increases with higher stress levels and reaches a maximum at the optimal stress level, after which the performance rapidly declines when it continues to rise. Up until the performance peak, this positive stress is called "eustress" (prefix *eu* from the Greek meaning good) [19]. This performance gain is crucial to ensure maximum survival [2, 11]. However, chronic exposure to a stressor and the constant onset and offset of stress, wears down the underlying stress response system, and the beneficial habituation capabilities now result in adverse pathophysiological processes that affect the mental and physical well-being [2, 20].
Stressors, as stress-invoking stimuli, can be divided into psychosocial and biogenic origins [19]. Solitude, over-taxation and interpersonal conflicts are, like most other stressors, psychosocial [2, 19]. These stimuli, real or imagined, do not directly elicit a physiological response and only do so after the brain identifies and interprets them as a stressor [19]. Biogenic stressors, in contrast, do not require cognitive appraisal, as they directly trigger

a neurological reaction. They are found in caffeine, nicotine, amphetamine, or painful stimuli like extreme heat or cold [21].
Noise, in literature generally defined as unwanted sound [22–26], can be either a biogenic or psychosocial stressor, depending on the characteristics. Examples of biogenic acoustic stressors are sounds with extremely high and fast-rising intensities, which invoke the acoustic startle response [27], an uncontrolled activation of multiple muscle groups resulting in a flinching motion [28, 29]. Many sounds depend on subjective judgment and thus are interpreted as noise by some, while others do not perceive the same sound as stressful. Because of this disparity in perception, Westman and Walters [27] further describe noise as sound that negatively affects the body.

Sound pressure levels are most commonly described in decibels (dB), a logarithmic unit representing the ratio of a sound wave pressure and a reference pressure. This reference is usually defined as the human hearing threshold and is designated as $0\,\mathrm{dB}$ on the logarithmic scale [30]. Due to the logarithmic characteristics of the decibel, a $10\,\mathrm{dB}$ increase corresponds to a doubling in perceived loudness, whereas a $3\,\mathrm{dB}$ increase is barely perceptible for the human ears [31]. Furthermore, sound levels are often weighted to better represent specific acoustic characteristics. A-weighted measurements ($\mathrm{dB(A)}$) are adapted to represent the human ear's non-linear response to sounds of the same intensity but varying frequency [32]. Most sounds that humans encounter in daily life are between $30\,\mathrm{dB(A)}$ and $100\,\mathrm{dB(A)}$ [31]. For example, to ensure a good night's sleep, sound levels as low as 35 to $40\,\mathrm{dB(A)}$ are required; a busy office can be $60\,\mathrm{dB(A)}$ and a busy road may be up to $75\,\mathrm{dB(A)}$ loud [31].

2.2. Physiological Stress Response

Since the stressor used in this work is noise, it needs to be identified as such before a bodily reaction is caused (see 2.1), which means that the basis of the physiological stress response lies in the nervous system. Therefore, the underlying structures and processes, as well as the subsequent effects on the human body, need to be addressed.
The human nervous system can be divided into two branches, the central nervous system (CNS), consisting of the brain and spinal cord, and the peripheral nervous system (PNS), which combines somatic and autonomic nervous system (ANS) [21]. When discussing the physical stress response, the limbic system is a highly interesting part of the brain. As the emotional control centre, it unifies multiple structures such as the hypothalamus, the pituitary gland and the amygdala, which all play major roles in the stress reaction [21]. The

spinal cord serves as the primary pathway for information within the entire nervous system, transmitting signals to and from the brain while participating in certain autonomous reflexes [21]. The part of the peripheral nervous system that takes up a big part of the stress response is the autonomic nervous system; it conveys the impulses necessary for maintaining homeostasis (e.g. thermoregulation, blood pressure and gastrointestinal function [33]). The ANS is further separated into sympathetic nervous system (SNS) and parasympathetic nervous system (PSNS). Both systems operate simultaneously, adjusting their discharge frequency and either enhancing or inhibiting tissue activity. This leads to typically opposing effects, enabling quick and precise control [33]. While the PSNS shows increased activity in phases of rest, where it is tasked with restorative metabolic functions, the SNS takes control in stressful situations, preparing the body for action [21, 33]. This is also known as the "fight-or-flight" reaction [2, 21, 33–35].

When a sound that potentially classifies as noise is introduced to the environment of the human body, it is first processed in the brain's auditory system and then transported from the auditory cortex to the amygdala for further analysis [29]. As a part of the limbic system, the amygdala is the highest level of the human stress response. After an introduced sound is deemed stressful or threatening, the subsequent reactions of the sympathoadrenomedullary (SAM) system and the hypothalamic–pituitary–adrenal (HPA) axis originate in the amygdala [21, 29]. The SAM system is put together by the hypothalamus, the nervous pathways of the SNS and the adrenal gland [21], a hormone-releasing gland located on top of each kidney. When activated by the amygdala, the hypothalamus innervates the sympathetic division of the ANS, which, for one, directly stimulates the end organs via the ganglions and also triggers the adrenal medulla to increase the release of the catecholamine norepinephrine and epinephrine. Since the adrenergic neurons of the SNS also secrete norepinephrine, the effect of the increase in adrenal hormone secretion and SNS activity is functionally the same and only differ in the onset latency and effect duration. The hormone secretion in the adrenal medulla is delayed by 20 to $30\,\mathrm{s}$; however, it lasts ten times longer than the direct innervation through the SNS [21]. Among the effects of the SAM system are increased arterial blood pressure, increased heart rate and increased secretion of the sweat glands [2, 21, 33].

As stated in the name, the HPA axis consists of the hypothalamus, the pituitary gland and, like the SAM system, the adrenal gland. Once stimulated, the neurosecretory cells in the hypothalamus release corticotropin-releasing hormone (CRH) into the portal system, which connects the hypothalamus and the pituitary gland. The CRH causes cells in the anterior pituitary gland to release adrenocorticotropic hormone (ACTH) into the circulatory system [2, 21, 29]. In the adrenal cortex, ACTH mainly stimulates the secretion of the glucocorticoids cortisol and corticosterone, which then regulate further hormone release via a negative feedback loop [35]. The activation of the HPA axis facilitates the

mobilisation of energy (e.g., glucose) and suppresses certain immune reactions to enable a sufficient “fight-or-flight” response [21, 35]. However, chronic exposure to stress leads to glucocorticoid resistance, which negatively affects feedback regulation and results in excessive inflammation [36], an increased risk of diabetes and obesity, cardiovascular disease, as well as anxiety and depression [21, 35].

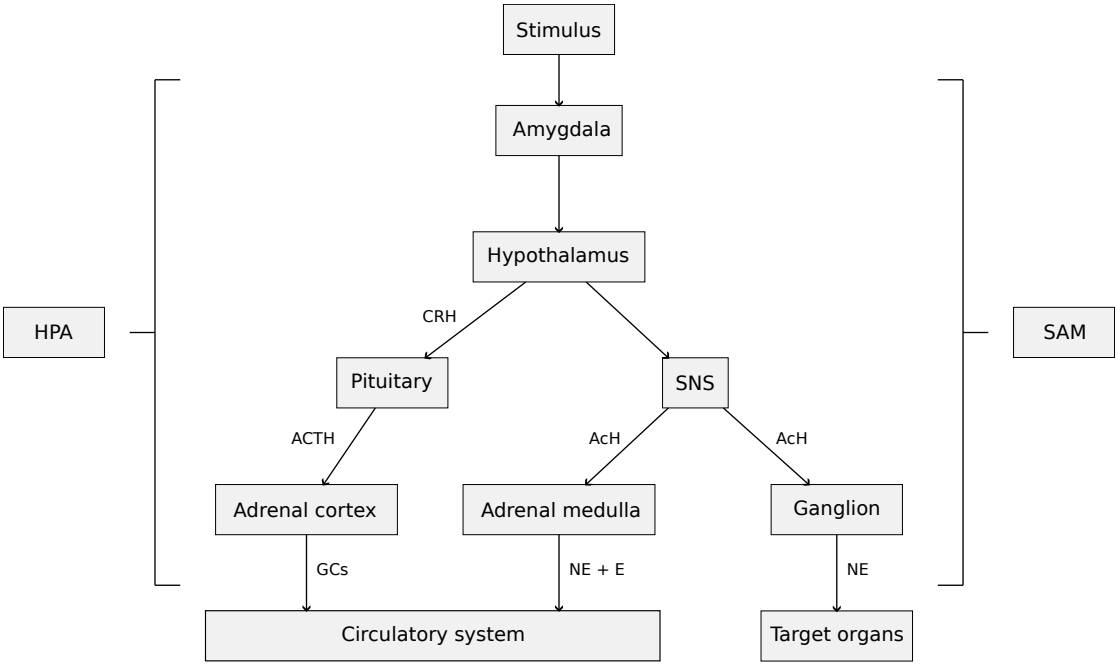


Figure 2.1.: Diagram illustrating the HPA axis and SAM system. The figure outlines the key components — hypothalamus, pituitary, adrenal medulla, adrenal cortex, and target organs — along with neurotransmitters and hormones such as corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), acetylcholine (AcH), norepinephrine (NE), epinephrine (E) and glucocorticoids (GCs). This highlights the body’s response to stress via neural and endocrine pathways.

Table 2.1.: Effects of HPA axis and SAM system activity.

HPA axis	SAM system
increased gluconeogenesis	pupil dilation
increased lipolysis	increased heart rate
increased urea production	peripheral vasoconstriction
immune suppression	skeletal vasodilation
	bronchodilation
	sweat gland stimulation
	inhibition of digestion

2.3. Finger Arterial Pressure Measurement

When measuring the finger arterial pressure, the goal is to minimise the gradient between intra and extra arterial pressure, the so-called transmural pressure [37]. This is done by applying the "volume clamp method", which was first introduced in 1973 by Czech scientist Jan Penaz [38]. By using a pneumatic finger cuff with a built-in photoplethysmography (PPG) sensor, consisting of a light source and photodetector, as well as a separate processing unit with a servo control system, the diameter of the unloaded vessel is determined by increasing the cuff pressure and analysing the PPG-waveform [37, 39]. The signals' shape and amplitude give insight into the state of the vascular wall. Once arterial and external pressure balance is reached, the artery is considered unloaded, and the necessary cuff pressure is used as a set point for the control system [39]. The control system then dynamically adjusts the cuff pressure to keep this condition. If the artery wall is unloaded, cuff and arterial blood pressure in the finger are the same, thus allowing continuous monitoring [39].
A recalibration algorithm called "Physiocal" is being carried out every 10 to $20\,\mathrm{s}$ until the signal reaches a stable condition, after which Physiocal is only done after 60 to $70\,\mathrm{s}$ [37, 39, 40]. The recalibration repeats the process mentioned above to account for changes in smooth muscle activity that change the vessel diameter and thus affect the pressure level inside the artery [37, 39]. Both the initial calibration and the Physiocal recalibration are shown in Figure 2.2.
The measured pressure then is internally processed by the device to represent central pressure conditions and provided as reconstructed systolic blood pressure (sBP) and diastolic blood pressure (dBP). Furthermore, if needed, a brachial calibration (BRAcal) can be performed, where brachial oscillometric BP-levels are taken to correct further the

reconstructed sBP and dBP values [41]. It has been shown that processed finger arterial pressure is an alternative to invasive continuous brachial artery measurement [37, 41] and satisfies the accuracy requirements of AAMI / ANSI / ISO 81060-2: 2013 [42] standards [40].

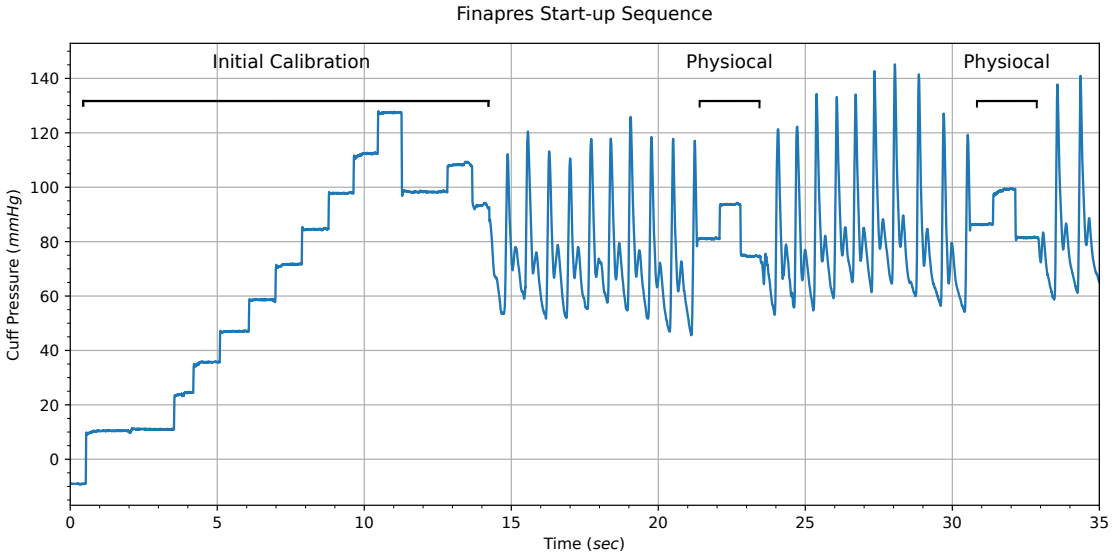
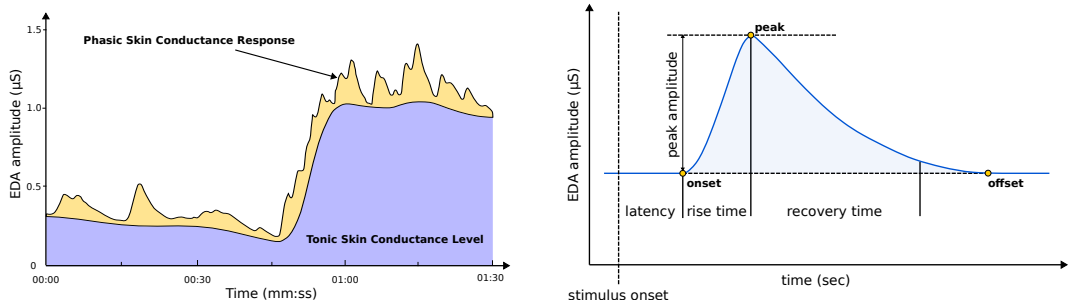


Figure 2.2.: Finapres startup sequence. In the first few seconds, the initial calibration takes place. Then, at expanding intervals, the Physiocal ensures the measurements are accurate. Inspired by Imholz et al. [37].

2.4. Electrodermal Activity

One of the most used methods for detecting stress in humans is the analysis of electrodermal activity (EDA) [10, 11]. By measuring differences in electric potential, resistance or conductance of the skin, information on ANS activity in the sweat glands can be gained [43]. Secretion in these glands is almost entirely influenced by sympathetic stimuli, with next to no antagonistic effect of the parasympathetic nervous system (PSNS) [43].
By applying two electrodes on the medial phalanges of two fingers and introducing a voltage or current, the skin conductance or resistance levels are measured [44]. The overall electrodermal activity is comprised of two parts, the skin conductance level (SCL) and the

skin conductance response (SCR). As implied by the term, the skin conductance level gives insight into the tonic conductance level of the skin, while the skin conductance response, also called galvanic skin response (GSR), provides information about the progression of skin conductance over short periods [43, 44]. Figure 2.3a illustrates how the overall EDA signal is comprised of the two components and Figure 2.3b depicts the different features of the skin conductance response. The latency, typically 1 to $5\,\mathrm{s}$ long, describes the duration from stimulus onset and the onset of the SCR. Peak amplitude refers to the amplitude difference between peak and onset, while rise time represents the time between the two. Finally, the recovery time refers to the duration from peak to offset, the recovery itself, which is much flatter in comparison to the steep incline of the SCR onset [45].



(a) An EDA signal split into tonic (SCL) and phasic (SCR) component.

(b) The different features of an event related skin conductance response.

Figure 2.3.: An illustration of example EDA data. Adapted from [45]

Apart from stress detection, EDA has found applications in behavioural medicine [43, 44], showing abnormalities in patients with depression, post-traumatic stress disorder and agoraphobia [46–48]. Furthermore, in some countries, EDA is part of polygraph testing for forensic medicine or criminal justice proceedings [43]. In addition to diagnostics, electrodermal activity biofeedback therapy can assist patients in gaining control over emotions related to sympathetic innervation of the skin and achieving a state of psychophysiological relaxation [43, 44]. It is reported that biofeedback therapy reduces the frequency of seizures in patients with drug-resistant epilepsy [49].

2.5. N-Back Task

The n-back task is a continuous recognition measure in which a participant is presented with a sequence of stimuli. It must determine whether a specific n-back rule is satisfied [50–53]. The subject has to press a button or key when the presented stimulus is the same as the one n-items back [53] (see Figure 2.4). The test difficulty generally scales with the value of n [50], while the duration of the stimulus presentation and the interstimulus interval (ISI) also contribute to the complexity. After completion, the participant's performance can be measured through accuracy or error rate and response time [50]. Various types of stimuli, such as letters, numbers, words, shapes or auditory signals, have been used in research [50–52], while the performance remained independent of the stimulus type [50]. The test was first published by Wayne Kirchner in 1958 [54] in a study on ageing, where he compared the short-term retention of rapidly changing information of elderly and young people. Since then, the n-back task has been a widely used test of working memory [50], which is needed to maintain and update small amounts of fast-changing information for a short time like reading, carrying a conversation or mental arithmetic [55–58].

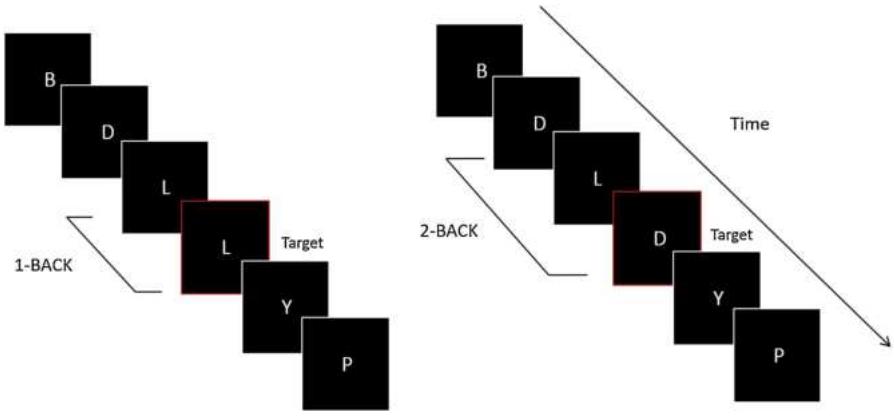


Figure 2.4.: Basic operation of the *n-back task*. Left: The second presentation of the letter "L" is correct in this 1-back example, as "L" was just presented one trial previously. Right: In this 2-back example, the second presentation of the letter "D" is correct. From Gilmour et al. [50].

3. Related Work

This chapter summarises work related to measurement methods, noise type or introduction, and cognitive tasks used in this study. Two reviews by Laufs et al. [10] and Giannakakis et al. [11] were chosen for further analysis to get an overview of publications that discuss physiological reactions to noise. Laufs et al. [10] reviewed 37 publications on physical stress reactions to acoustic stimuli, while Giannakakis [11] et al. based their review on 41 publications in human stress detection using biosignals.

Fifty-two publications shared similarities with this thesis by either measuring identical physiological features (BP or GSR), employing similar types of noise (RTN), or using comparable cognitive tasks. Of all the similar work, 36 used electrodermal activity as an indicator for stress and seven included blood pressure measurements in their study. Six introduced road traffic noise in their test, of which four are also part of the group that used BP and one that also used EDA. Three unique papers implemented a variant of the n-back task in their experiment. All included papers were evaluated for similarities based on the information provided in the reviews. These studies are listed in Table A.1.

3.1. Physiological Stress Detection

An important part of recording the bodily stress response is correctly gathering the vitals that stress influences most. This section thus provides an overview of publications that implemented blood pressure or electrodermal activity. The studies are presented with regard to their methods and noteworthy results.

3.1.1. Blood Pressure

In a 2010 study, Lee et al. [24] examined the response of heart rate variability as well as blood pressure to 5 min intervals of white noise at 50 dB(A), 60 dB(A), 70 dB(A) and 80 dB(A) volume levels. It is noteworthy that singular BP-measurements were only taken for volumes 70 dB(A) and 80 dB(A), resulting in no detailed time series analysis. No significant increases were recorded for blood pressure and heart rate parameters; only frequency features of HRV showed significant increases.

During a long-term (60 min) noise exposure experiment to study the effect of different noise types on men, Sim et al. [22] measured HRV, blood pressure and blood parameters while exposing the participants to 45 dB(A) traffic noise or speech or both a once. While HRV values were measured continuously, blood pressure measures and blood analysis were only determined before and after each session. Their results showed significant changes in HRV features while BP-levels only increased insignificantly. Because some frequency features indicated decreased stress levels while the participants were exposed to speech, the researchers proposed a possible beneficial effect of noise in an otherwise quiet environment.

To assess the cardiovascular and stress responses to noise with different frequency characteristics, Walker et al. [26] conducted an experiment which recorded ECG, blood pressure and saliva markers. After 10 min of acclimation and ten minutes of silence, the participants were exposed to either low-frequency (31.5 to 125 Hz) or high-frequency (500 to 2000 Hz) noise for 30 min, followed by 30 min of silence. The scientist measured BP-levels once directly before and after noise exposure, while the HRV features were extracted from continuous ECG monitoring. Although the scientists examined significant changes for HRV features, especially when low-frequency noise was used, neither salivary markers nor blood pressure values differed significantly depending on the noise.

Contrary to Walker et al. [26], a 2018 study by Lu et al. [59] showed significant blood pressure changes in an experiment which also focused on different frequency characteristics. They exposed young adults to 20 min recordings of different industrial noises, with rather low-frequency, rather high-frequency or an average of both, at 75 dB and 90 dB, while measuring BP and HR values before and after the noise was introduced. This resulted in a significant increase of systolic blood pressure and diastolic blood pressure for 90 dB industrial noise only.

In a paper on stress reaction to noise and cognitive tasks in an office environment, Kristiansen et al. [60] continuously measured blood pressure and electrocardiography (ECG)

during a four-phase experiment consisting of two rest phases, a mouse work task and an order judgement. They found no significant changes in their measurements, except diastolic blood pressure, which decreased during noise exposure. Though they used a continuous monitoring approach, no analysis regarding the dynamic of the results was made.

Gallasch et al. [61] conducted a comparison of road and rail traffic noise at $70\,\mathrm{dB(A)}$ and their impact on extra-aural effects. They continuously measured ECG, impedance cardiography (ICG) and blood pressure (BP) over a period of $100\,\mathrm{min}$, separated into three $20\,\mathrm{min}$ active phases where the mental task performed and the noise was introduced. Four $10\,\mathrm{min}$ rest phases made up the remaining test duration. The analysis was made only during noise, over $2\,\mathrm{min}$-intervals and showed trends of increased systolic blood pressure, yet significant changes were only recorded for railway noise and not for road traffic noise.

One of the few publications analysing the time-series response of blood pressure (BP) was conducted by Paunović et al. [62], they introduced road traffic noise (RTN) at $89\,\mathrm{dB(A)}$ for ten minutes to 130 young and healthy adults. The scientist used a thoracic bioimpedance device that recorded ECG, ICG and two variants of blood pressure (oscillometric and continuous). After conducting their three-phase experiment, consisting of a $10\,\mathrm{min}$ noise phase enclosed by two $10\,\mathrm{min}$ quiet phases, they computed the averages for every minute of their $30\,\mathrm{min}$ experiment. For the analysis, they compared each minute to the preceding minute, the first minute of the same phase, and the last minute before and after the noise exposure. Their results show a prompt and significant increase of BP and HR within the first minute of noise exposure, followed by a steady decrease throughout the rest of the experiment duration. This unique approach enabled a detailed time series analysis of the body's stress reaction through blood pressure measurements.

Table 3.1.: Overview of publications that implemented BP measurements.

Publication	Method	Type of Noise	Cognitive Task	Results
Lee et al. [24]	oscillometric	white noise	-	insignificant changes of BP
Sim et al. [22]	oscillometric	RTN + speech	-	insignificant increase of BP
Walker et al. [26]	oscillometric	unspecific HF + LF noise	-	insignificant changes of BP
Lu et al. [59]	oscillometric	industrial	-	significant increases at $90\,\mathrm{dB}$
Kristiansen et al. [60]	continuous	open plan office	-	overall no significant changes
Gallasch et al. [61]	continuous	RTN + railway	short-term memory	insignificant increase of BP during noise
Paunović et al. [62]	continuous	RTN	-	BP increases significantly within the first minute of noise

3.1.2. Electrodermal Activity

On the topic of electrodermal activity (EDA), much research has already been conducted [10, 11], though only few publications included acoustic stimuli, let alone road traffic noise (RTN). In an attempt to measure the effect of birdsong on perceived restorativeness, Suko et al. [63] conducted an experiment where participants were exposed to 2 min long sound samples. Both samples included birdsong, but only one had additional car noise included. The scientists measured electrodermal activity while introducing the sound at 61 dB for birdsong only and 63 dB for birdsong combined with car noise. Afterwards, the participants were asked to rate their perceived restorativeness on a scale. Their analysis of the SCL showed a significantly decreased recovery rate for birdsong with car noise compared to birdsong only. However, the subjective evaluation recorded with the perceived restorativeness scale showed no such trend, which is why the researchers implied that SCL measurements are able to detect the subconscious effect of background noise.

Another study on physiological stress recovery and the effect of noise on it was done by Alvarsson et al. [64]. They measured skin conductance level and high-frequency HRV components during an experiment which included three types of noise: nature sound at 50 dB, "high noise" (i.e. road traffic noise) at 80 dB, "low noise" which was the same as high noise but at 50 dB and ambient noise at 40 dB. Additionally, a speeded mental arithmetic task was presented to the participants to induce stress. The results showed a slower decrease of the SCL in periods where the "high noise" was presented, while the fastest drop occurred when natural sounds were played back. Contrary to the EDA feature, no significant changes were observed for the high-frequency HRV components. These findings, together with those of Suko et al. [63], suggest that positive and calming sounds can influence physiological recovery.

Trimmel et al. [65] examined how low-intensity aircraft and neighbourhood noise impact cognitive learning by measuring skin conductance while participants had to learn information from three texts on a philosophical topic. During the test, either a control background noise at 36 dB, a recording of passing aircraft at 48 dB or "neighbourhood-like" sounds at 45 dB were introduced. The increase in spontaneous skin conductivity fluctuations during aeroplane and neighbourhood noise suggested a higher physiological stress level, even for low-intensity background noises, though this effect was more pronounced for the aircraft pass-bys.

In 2021, Iadarola et al. [66] evaluated the galvanic skin response to three different acoustic stimuli. Their 10 min experiment consisted of a 5 min pre-stimulus baseline measurement, a 1 min stimulus and a 4 min post stimulus phase. The scientist chose the number of peaks

and the frequency band of the GSR signal for their analysis. During analysis, they noticed increased peaks for most participants when neutral (walking sounds) and unpleasant (screaming) noise was played. As for the frequency domain, their results showed that the power spectra were mainly located in the 0.4 to 1 Hz band, which is rarely observed in comparable publications.

3.2. Summary

The related work presented in the above paragraphs show that a lot of work has been done in human stress recognition, yet the implementation of blood pressure as an indicator for such is rarely described. The publications that included blood pressure measurements in their research seldom recorded significant changes when ordinary oscillometric methods were used. Although the use of continuous measurement methods did not always show significant changes, which may be due to too large analysis intervals, it was able to show a trend that would have gone unnoticed otherwise. Contrary to blood pressure, electrodermal activity is a well-established modality in the area of research.
It has been shown that using EDA features is a reliable approach when recoding the bodily stress reaction. These studies show the benefit of EDA measurements, as they are able to represent changes in sympathetic nervous activity with a high resolution.
The review of related work provided valuable information, which helped design the experimental setup and analysis in this work.

Regarding the in Chapter 1 proposed research questions, this chapter has shown that a high-resolution timeline analysis, as planned for this work, has not yet been conducted. Furthermore, no direct comparison of blood pressure and electrodermal activity measurements has been made to evaluate the use of continuous non-invasive blood pressure devices. This clearly indicates the research gap that this work addresses.

4. Methodology

This chapter will provide the methodology for the experiment conducted for this thesis. First, an overview of the study population and design will be given. Then the questionnaire used in the experiment and the preprocessing steps are presented. Finally, the approaches for the data analysis are provided.

4.1. Study Population and Design

For the experiment, four women and eight men, ages 21 to 27 years (mean 23.33, sd 2.42), all right-handed and non-smoking, volunteered to partake. The participants had no history of cardiovascular disease, such as chronically elevated blood pressure, and no hearing impairments. Furthermore, all participants confirmed that they do not take medication, which may influence the measurements and gave informed consent to the procedures in this experiment. The experimental setup and all methods were approved by the Technische Universität (TU) Darmstadt ethics committee (application EK 06/2025).

The experiment combined the exposure with road traffic noise (RTN) at three intensities and a version of the *n-back task* in a $120\,\text{s}$ trial. As a source for RTN, a two-minute excerpt (01:00 - 03:00 min) from a YouTube video containing footage of a busy road in Asia [67] was chosen. This was then played back via over-ear headphones (MMX 100, Beyerdynamic GmbH & Co. KG, Heilbronn, Germany) at one of three volumes: ambient background noise at $30.2\,\text{dB(A)}$ or road traffic noise at either $L_{eq,\,2\text{min}} = 52.12\,\text{dB(A)}$ ($68.22\,\text{dB(A)}$ max) or $L_{eq,\,2\text{min}} = 58.12\,\text{dB(A)}$ ($74.25\,\text{dB(A)}$ max), in the following described as *Vol. 0*, *Vol. 0.5* and *Vol. 1* respectively. Sound levels were measured and verified with a handheld sound level meter (Optimus+ Industrial, Cirrus Research plc, Hunmanby, United Kingdom). The *n-back task* was configured based on a pre-test conducted by Laufs et al. [68]. They concluded a configuration of $n = 2$, a stimulus duration of $500\,\text{ms}$ and an interstimulus interval (ISI) of $700\,\text{ms}$ was suitable for experiments such as the one presented, as it offers

a consistent cognitive load and is easily executed. For the stimuli, a set of 100 capital letters was compiled with a 50 % target percentage and a maximum consecutive target sequence of 2 for each volume. The participants had to press the laptop's space bar every time the letter shown on the screen matched the letter two items before. This was possible during the time the letter was presented and the ISI, amounting to a maximum available response time of 1.2 s. For more information on the *n-back task*, see Section 2.5.

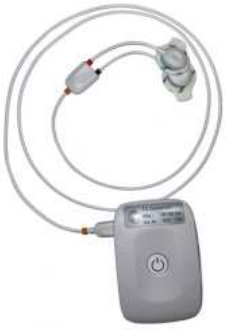
The test took place in an empty office inside the KISMED institute and was isolated in terms of external noise. Additionally, the blinds inside the room were closed, and only the light furthest away from the participants was turned on to avoid unwanted visual interference. After explaining the procedures and the motivation of the experiment thoroughly, the participants were given the consent form and the questionnaire to fill out. Once all necessary questions were answered, the measurement equipment was fitted and set up. The experiment was composed of three sessions, each with a different intensity of noise. This amounted to six presentation orders for the sounds, which were equally distributed among the participants. Each session consisted of a pre-measurement calibration phase, a 60 s *Rest* phase, with a 10 s countdown at the end, a 120 s *Trial* phase with noise and mental task, and a 60 s *Recovery* phase. During all phases, the participants were seated in front of the testing laptop and should remain still to avoid measurement errors due to movements. For an overview of the test setup, see Figures 4.1a and 4.1b.
After every completed session, another part of the questionnaire was presented (see Section 4.2 for further explanation), and a 5 min break was done. Both the sound introduction and the *2-back task*, were implemented in PsychoPy [69, 70], which is discussed in detail in Section 5.1. Furthermore, a step-by-step instruction for the experiment is provided in Section A.2.

(a) Experimental setup.

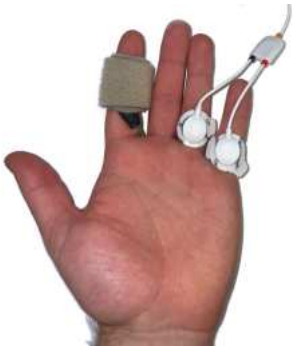
(b) Presenters view of the setup.

(c) *Finapres* patient unit.

(d) Applied *Finapres* device.



(e) *Biosignals* measurement dub and EDA sensor.



(f) Placement of cuff and electrodes on the hand.

Figure 4.1.: The experimental design and measuring devices used for this study. Pictures (a) and (b) show the test setup. The sensors are fitted to the left lower arm, and the test laptop is placed comfortably in front of the Participant. Photo (c) shows the *Finapres* control unit, the finger cuff, and the HCU. Figure (d) shows how the *Finapres* patient unit is applied to the Arm. Picture (e) presets the *Biosignals* measurement hub and electrodes. In (f), the placement of the sensors and cuff on the hand is shown.

4.2. Questionnaire

Various questionnaires and a set of general questions about personal information were combined into a survey to assess the participants' stress level and sensitivity to noise. After providing the personal information necessary for the measurement (age, height, weight; see Table 4.1), the participants were given a union of three questionnaires, which they were supposed to answer in part before the experiment and in part after each session. Although all questions included were initially expressed in English, translated versions in German were utilised for this experiment. The entire questionnaire is included in Section A.3.

Table 4.1.: Basic characteristics of the study population.

	MEAN ± SD	MIN	MEDIAN	MAX
Age	23.33 ± 2.42	21.00	23.00	27.00
Weight (kg)	76.83 ± 11.54	60.00	73.50	99.00
Height (cm)	182.08 ± 8.18	169.00	183.00	194.00
BMI	23.17 ± 3.20	20.06	22.40	29.76

Noise Annoyance - ISO/TS 15666:2021-05

The first part of the questionnaire was composed of a technical specification by the International Organization for Standardization (ISO).
It includes two questions, one on a verbal and the other on a numerical scale, to assess the participant's noise annoyance, thus providing context for the experiment's results. The verbal scale provided the options: not at all, slightly, moderately, very, and extremely, of which the last two were defined as highly annoyed. Meanwhile, on an 11-point numerical scale ranging from zero to ten, answers eight, nine, and ten corresponded to being highly annoyed [71, 72]. After scoring the answers, the percentage of highly annoyed individuals is provided, therefore giving insight into the noise annoyance of the study population. The German translation of the questions by T. Gjestland [73] was used for this experiment.

Weinstein Noise Sensitivity Scale

The next section of the questionnaire was the Weinstein Noise Sensitivity Scale (WNSS) First used by Neil D. Weinstein in a study on individual reactions to noise [74], the 21-question long Weinstein Noise Sensitivity Scale (WNSS) determines sensitivity to noise with respect to general noise and daily environmental sounds [75]. Each question is answered on a 6-point scale ranging from “strongly disagree”, representing 1, to “strongly agree”, representing 6. Rating the answers involved adding all scores after inverting those marked with “*”, resulting in an overall score between 21 and 126. Low scores mean lower sensitivity to noise, while higher values represent the opposite.
Again, a German translation by Zimmer and Ellermeier [76] was used for this survey.

State Trait Anxiety Inventory

Lastly the State Trait Anxiety Inventory (STAI) was included into the survey. The STAI, created by C. D. Spielberger [77] comprises two separate questionnaires analysing state and trait anxiety, which give insight into the participants’ stress level. State anxiety is influenced by the present circumstances, while trait anxiety represents a general tendency to be anxious [78]. Each part contains 20 items and is scored on a numerical scale from one to eight, ranging from “almost never” to “almost always” for the trait anxiety section and “not at all” to “very much so” for the state anxiety section. To record the effect of different volumes of noise, the state anxiety questionnaire was repeated after each session. Including all forty questions in the questionnaire was decided against due to limited time during the experiment, and a short version created and translated into German by J. Grimm [79] was chosen. This short version uses only ten items from each section but maintains the mentioned rating method. The scores were again calculated by inverting items marked with “*” and adding all corresponding values, which resulted in a minimum of ten and a maximum of eighty “points”. For further comparison, the raw results can be transformed into relative values. The cut-off scores are usually 0-30% for “no or low anxiety”, 30-40% for “moderate anxiety” and 40-100% for “high anxiety” [80].

4.3. Physiological Monitoring

Blood Pressure

The blood pressure recording was done with a hemodynamic monitoring system (*Finapres® NOVA*, Finapres Medical Systems B.V., Enschede, Netherlands) capable of measuring the blood pressure continuously on a beat-to-beat level. The theoretical foundation for the deployed measuring method is briefly explained in Section 2.3. After the cuff size was determined, the finger cuff was applied to the medial phalanx of the right index finger and the processing unit to the right lower arm, as described in the device handbook (see Figure 4.1d). Furthermore, a conventional brachial arm cuff was connected to the left upper arm. Once both pressure cuffs were applied to the participant, the device's height correction unit (HCU) was added to the setup. It is used to counteract changes in the height difference between the finger and the heart, which is why it was placed on the brachial cuff, as it is situated at heart level. After starting the measurement, one brachial calibration was conducted, where the reconstructed blood pressure is corrected according to the values measured with the brachial cuff. Once the session was done, the device provided an output file with the reconstructed systolic blood pressure and systolic blood pressure (*reSYS* and *reDIA* respectively), as well as the heart rate (HR) and interbeat intervals from the PPG sensor in the cuff, in following referred to as *Finapres* dataset. The data was originally sampled at $200\,\text{Hz}$; however, the reconstructed values were already downsampled to $1.25\,\text{Hz}$ in the export file.

Electrodermal Activity

The EDA was measured with a sensor and a 16-bit acquisition hub (Electrodermal Sensor and biosignalsplux Hub, PLUX wireless biosignals S.A., Lisbon, Portugal) at a sampling frequency of $500\,\text{Hz}$. The EDA-sensor was attached to the palmar proximal phalanges of the left little and ring finger using disposable, self-adhesive Ag/AgCl electrodes, and connected to the hub (see Figure 4.1f). A computer running the *OpenSignals (r)evolution* software (PLUX wireless biosignals S.A., Lisbon, Portugal) was linked to the acquisition hub via *Bluetooth®*. The recording was started and ended remotely in the software, preventing any passive influence on the measurement. After the acquisition, the gathered data was reviewed and exported for analysis. This export is in the following referred to as *Biosignals* dataset.

4.4. Preprocessing

After all the experiments were completed, the measurements were preprocessed prior to the analysis. Each session's data exports consisted of comma-separated values (CSV) files that included general information about the session and the data points. One of the goals during preprocessing was to compile a dataset that included all measurements from a single session. To achieve this, each individual dataset needed to be adjusted in size and sampling frequency. This was done by trimming each measurement according to the start and end of the experiment's initial and final phases using the corresponding timestamps. Doing so removed the pre-test data where the calibration occurred and ensured both the *Biosignals* and *Finapres* datasets were $240\,\text{s}$ long. Afterwards, the datasets were resampled to $10\,\text{Hz}$ and aligned based on their timestamps. Before this could happen, the *Biosignals* dataset and the IBI from the *Finapres* dataset required further processing to extract the desired features for the analysis. The preprocessing steps are shown in Figure 4.3.

Feature Extraction

A few features had to be extracted to perform a representative data analysis of the measurements, allowing comparison between skin conductance and continuous blood pressure as stress indicators. Based on the literature review in Chapter 3, two EDA features and one heart rate variability (HRV) were chosen in addition to the already internally processed *Finapres* data to represent the participant's stress level.

For the analysis of the electrodermal activity, the baseline-normalised EDA-level and the number of peaks per minute were chosen. The former was computed based on the first $50\,\text{s}$ of *Rest* by dividing the EDA with the mean value of said interval. To extract the peaks per minute, the relative peak height was calculated from the ratio of the phasic SCR and the tonic SCL. This provided a measure independent of the general tonic level, which varies depending on the participant and factors like the room temperature. The peaks were detected using a peak-finding algorithm. Only peaks with a height higher than 0.1 relative to the tonic level or a prominence greater than 0.02 were included in the detection. The peak prominence is represented by the difference to the bigger minimum of the right and left interval spanning from the potential peak to the next higher peak [81] (see Figure 4.2). Afterwards, the number of peaks was counted for each experiment phase and adapted to represent per-minute values.

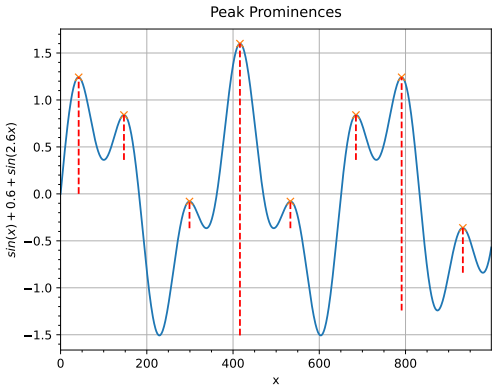


Figure 4.2.: Example for peak prominence. This example from the *SciPy* documentation [81] provides a visual explanation for the peak prominence parameter. Each vertical line represents the peak prominence for the corresponding peak.

For the HRV feature, the root mean square of successive differences (RMSSD) was chosen and computed from the *Finapres* IBI data. The RMSSD primarily reflects short-term changes in the HRV that are influenced by the parasympathetic nervous system [82]. The industry standards for time periods in HRV analysis are either 5 min or 24 h recordings of ECG or, in this case, PPG. Since the entire duration of the experiment was only approximately 7 min, including the pre-test calibrations, which were not included in the analysis, leaving 4 min of measurement data, not all existing HRV features were applicable. It has been shown that when using very short time frames, such as the 10 s intervals in this work, RMSSD was the most accurate at representing the standard 5 min measurement [83, 84]. The feature extraction process is detailed in Chapter 5.

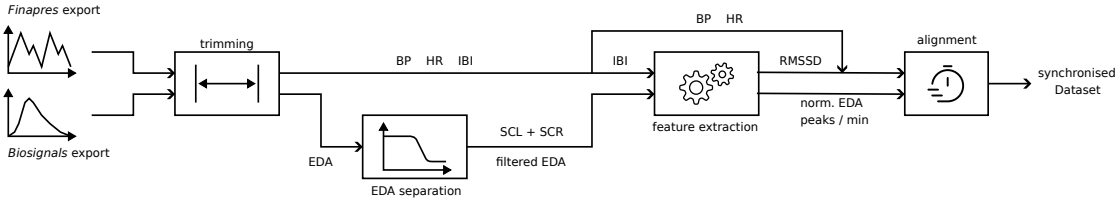


Figure 4.3.: The Preprocessing steps of the experiment, as described in Section 4.4.

4.5. Statistical Analysis

Two approaches were taken to analyse the BP, HR, RMSSD and normalised EDA measurements: an time series analysis and a volume analysis. For both approaches, the datasets were split into 24 ten-second intervals.

The time series analysis investigated the dynamic changes in the physiological features within one session. Here, the approach for a time series analysis conducted by Paunović et al. [62] was adopted, as it could show hemodynamic differences on a minute-to-minute level, which has not been done otherwise. Four comparisons were made to get information about the stress response throughout the test duration. The *consecutive* comparison observed the changes that occurred from one interval to the preceding one, resulting in 23 pairs. For the analysis within a phase, the *phase-start* comparison was conducted, where every interval was compared to the first interval of the same phase. This resulted in 5 pairs for the *Rest* and *Recovery* phase, while the *Trial* phase generated 11 pairs. Differences between all intervals since the start of the *Trial* phase and the last intervals of the *Rest* phase were bundled in the *pre-trial* comparison, which consisted of 18 pairs. Finally, the *post-trial* comparison examined the changes during the *Recovery* phase in relation to the last interval of the *Trial* phase, amounting to another 6 value pairs. For each comparison, the interval differences were calculated and checked for normality. A paired sample t-test was conducted to determine statistical significance when the differences were normally distributed. If the normality test was not passed, significance was assessed using a Wilcoxon signed-rank test.

The volume analysis compared both sessions with noise introduction to the one without to determine if the effect of the noise and *n-back task* was amplified depending on the volume. To account for baseline differences, the values were divided by the average of the first $50\,\mathrm{s}$ of the *Rest* phase, as the $10\,\mathrm{s}$ countdown could have influenced the values. Again, the difference between the noise session and the session without noise was calculated for each interval and then checked for normality. As with the time series analysis, depending on whether the data was distributed normally, either a paired sample t-test or a Wilcoxon signed-rank test was conducted.

Furthermore, the peak count per minute was analysed using similar approaches. Because these values were only calculated as per-minute values, a full time-series analysis was not possible, and instead, a comparison between each experimental phase was made. Similarly, the volume analysis was adapted for the peaks per minute analysis by comparing each phase of sessions with noise exposure to the same phase without an acoustic stressor.

The differences between phases or sessions were calculated and checked for normality. For normally distributed values, a paired sample t-test was conducted, while a Wilcoxon signed-rank test was used for non-parametric distribution.

Only the state anxiety test was chosen for further analysis, as these questions were repeated after each session, whereas the other parts of the questionnaire were only answered once at the beginning. The answers of both sessions with noise were compared to the baseline score without noise. Once again, the differences between each test score with noise and those without were computed, checked for normality and then statistically analysed. As for all the other approaches, a paired sample t-test or a Wilcoxon signed-rank test was conducted.

A p-value $p < 0.05$ was deemed significant for the entirety of the statistical analysis.

5. Implementation

In this chapter, the implementation of the test environment, of the data processing and of the data analysis will be described.

5.1. PsychoPy

The mental task and noise introduction were implemented in PsychoPy, an open-source *Python* package used for neuroscientific experiment design [69]. It also features a builder environment, which lets you construct complex psychological experiment setups via a graphical user interface (GUI). Inside this builder, the experiment then can be implemented using a flowchart-like approach and subsequently started in a separate full-screen application.

The program was structured into five routines: *Instructions*, *Rest*, *Trial*, *Recovery* and *End* (see Figure 5.1). Both the *Instructions* and the *End* routine did not contribute to the experiment's results, while the passive *Rest* and *Recovery* routines enclosed the only active *Trial* routine. When initialised, an input mask was shown on the screen, where

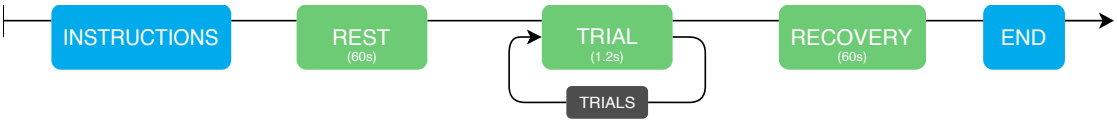


Figure 5.1.: This flowchart represents the *PsychoPy* experiment. The main part of the experiment only contained the three phases in the centre. *Rest* and *Recovery* stated their name, while the *Trial* routine included possible noise exposure and the mental task. It was repeated a total of 100 times by the *Trials* loop. The *Instructions* routine provided information on the *2-back task*, while the *END* routine stopped the program.

Table 5.1.: An excerpt of one of the conditions tables which provide the basis of the *2-back task*. The *thisLetter* column contained the letter sequence, while the other two columns marked the targets and the necessary answer for each column.

thisLetter	targets	corrAns
A	0	
O	0	
T	0	
O	1	space
T	1	space
U	0	
T	1	space
U	1	space
H	0	
⋮	⋮	⋮

the participant ID, the session number and the volume were configured and applied (see Figure 5.2). These configurations were made based on the ID's and presentation order given on the questionnaire. After the settings were confirmed, all routine components, such as on-screen text, key response observers, and custom code environments, were initialised by the program.

First, the *Instructions* routine started the experiment by briefly introducing the *2-back task*. This routine lasted indefinitely; however, if a space bar keypress was registered, it was forced to end, and the *Rest* routine started. This prompted the phase name on the screen; meanwhile, once $50\,\mathrm{s}$ had lapsed, a $10\,\mathrm{s}$ countdown started. The countdown was implemented by subtracting the current routine timer from the total routine duration for every frame during the last $10\,\mathrm{s}$.

The *Trial* routine, in combination with the *Trials* loop, was a crucial part of the *2-back task* implementation. It consisted of four components, which controlled the stimulus presentation, key response handling, and noise introduction. The loop retrieved information from the conditions table and iterated through its entries. A separate Excel table was created for each volume, containing the letters to be presented, as well as target markers and the correct key responses (see Table 5.1). The contents of a new row were read and passed to the *Trial* routine every repetition. Two components of the *Trial* routine were code environments: the *set_table* and the *play_sound* component. The *set_table* component used a *Python match* statement to determine the table identifier while the *play_sound*

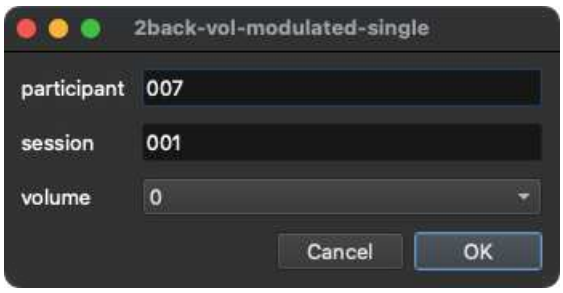


Figure 5.2.: The *PsychoPy* input mask provided three configuration options: participant ID, session ID and volume.

component used the built-in *psychtoolbox* sound engine to load the WAV file containing the noise and preset the volume. Both settings were based on the volume parameter in the input mask and initiated at the start of the experiment. The table identifier was needed for the *Trials* loop to choose the right conditions table for the *2-back task*. Once the *Trials* loop counter started ($trials.thisN = 0$) the playback was started until the loop reached its last repetition ($trials.thisN = 99$).
To present the stimulus, a text component was added to the routine. It showed the current letter passed by the *Trials* loop on the screen for $500\,\text{ms}$, after which the letter disappeared, and the screen went blank again. During the entire routine duration, the *key_resp* registered possible space bar presses. If a keypress was registered, it also checked whether the response was aligned with the current *corrAns* entry in the conditions table and tracked whether the response was correct. Every time a letter appeared on-screen, the current timestamp, key response time, and key response duration were saved in the output file. Once $120\,\text{s}$ had passed, the *Trial* routine ended, and the *Recovery* routine began. It had a similar structure as the *Rest* routine but did not include a countdown. After displaying the phase name and instructions to remain seated for another $60\,\text{s}$, the *End* routine announced the experiment's ending.
The program automatically handled the data export and saved a CSV file for each session. This file included the timestamps for every presented letter, the participant and session ID as well as the volume at which the noise was introduced. Furthermore, if the participant's *2-back task* performance during the session needed to be rated, the file provided whether the answer for each letter was correct.

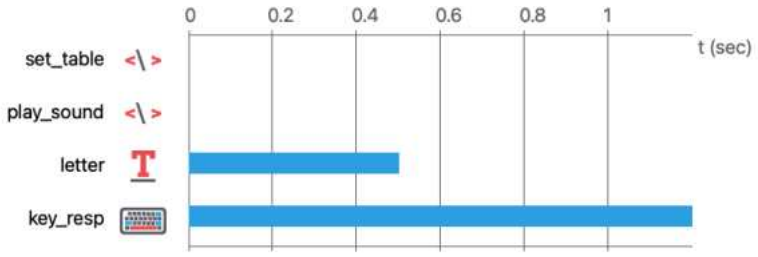


Figure 5.3.: The implementation of the *Trial* routine in *PsychoPy*.

5.2. Preprocessing

In order to perform all preprocessing steps, the data from each session was imported into *pyCharm* where it was read and converted to *Pandas DataFrames* [85] using *Python* 3.13.

5.2.1. Data Trimming

The first step was to trim the data according to the start and end of the experiment's initial and final phases using the corresponding timestamps in the *PsychoPy* export. Before further steps could be taken, these timestamps had to be reformatted to match the "datetime[64ns]" syntax, which was essential to the trimming and the alignment later on. After reformatting and adjusting the data type, the start of the experiment was determined by subtracting a time delta of $60\,\mathrm{s}$ from the first item in the timestamp column. This step included the $60\,\mathrm{s}$ period before the *2-back task*, during which the *Rest* phase occurred, and excluded the time before the measurement when the brachial calibration took place. The same process was applied to the last entry of the column to ascertain the overall end of the experiment, adding an extra $1.2\,\mathrm{s}$ to account for the duration of the final iteration. Both timestamps (*expStart* and *expEnd*) were then used as references for trimming the *Finapres* and *Biosignals* datasets.
In the *Finapres* export, the time axis was represented by the time since the measurement started. Since this prevented the start and end time calculation, a new column containing timestamps was created. After once again adjusting the format of the measurement start string in the *Finapres* file header to match the "datetime[64ns]" syntax, each row of the timestamp column was created by converting the time axis value to a timestamp with the measurement start as the origin using the *Pandas to_datetime()* method. Then, all rows before and after the *expStart* and *expEnd* were dropped from the DataFrame.

The *Biosignals* dataset required additional preparation, as neither the timestamps nor the time axis were readily accessible. After extracting the timestamp that indicated the measurement start from the file header, the time axis needed to be calculated before the dataset could be trimmed. Each row of the output file included the corresponding sample number (n), which, along with the sampling frequency (f_s) found in the header, was used to compute the time axis using Equation 5.1.

$$t = \frac{n}{f_s} \tag{5.1}$$

Like the Finapres dataset, the now calculated time axis represented the time since the start of the measurement, which was converted into timestamps and passed into a new column. Again, all rows before and after the *expStart* and *expEnd* were dropped from the DataFrame.
Figure 5.4 provides an example of the trimming process. Note that no other preprocessing has been done, and both datasets still contain raw measured data as it was exported from the device.

5.2.2. Feature Extraction

The *pyEDA Python* package [86] was used to extract the EDA features. It takes the raw signal, the signal's acquisition rate and the desired sampling frequency as inputs to compute a multitude of features, not all relevant to the work in this thesis. For this work, the EDA data was originally acquired at a rate of $f_s = 500\,\text{Hz}$ (in two cases $1000\,\text{Hz}$) and then downsampled to a sampling frequency of $f_s = 10\,\text{Hz}$. Along with the cleaned EDA-signal (*EDA_clean*), the phasic (*EDA_phasic*) and tonic (*EDA_tonic*) components are provided at the new frequency by the package. The cleaned EDA signal was then normalised to the mean value of the first $50\,\text{s}$ of the *Rest* phase using Equations 5.2 and 5.3. Even though the *pyEDA* package includes an option to detect and count the peaks in the input signal, this was not used, as the results showed severe shortcomings in the peak detection when the data was inspected. This led to the approach of "manually" detecting the peaks using the *find_peaks()* function included in the *SciPy signal* module [81] with certain arguments ensuring better detection overall. Before applying the function, the peak height of the phasic EDA relative to the tonic skin conductance level was calculated (see Equation 5.4), ensuring the data was independent of participant-specific factors. The algorithm was used once to find all peaks with a height greater than 0.1 relative to the tonic level and again to detect peaks with a prominence upwards of 0.02. Both data points were combined, making sure not to include duplicates, and then counted for each phase

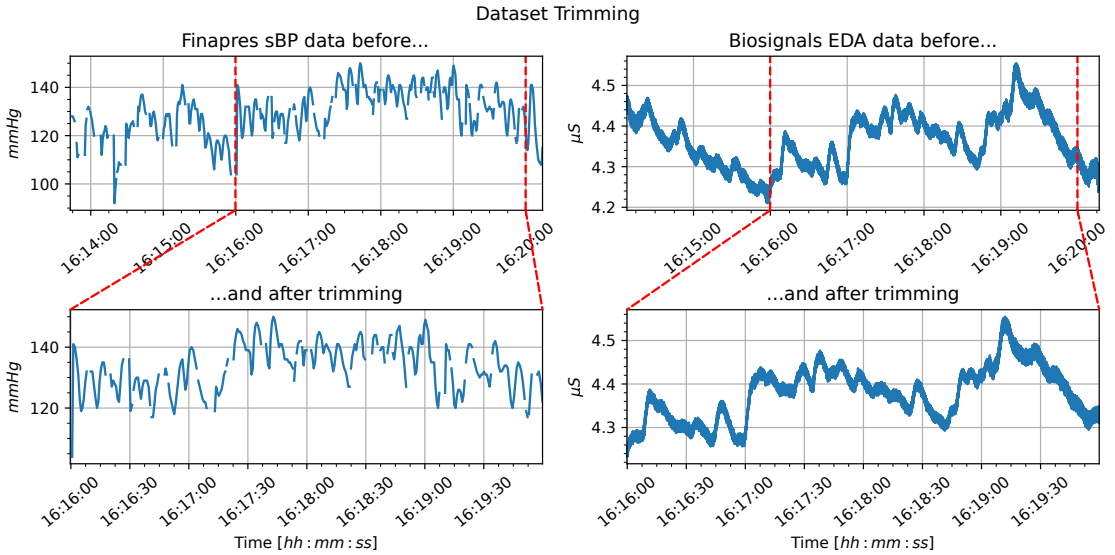


Figure 5.4.: This figure provides an example of the dataset trimming process. It compares the raw BP and EDA data before and after trimming according to the *expStart* and *expEnd* timestamps determined from the *PsychoPy* export file. The visible gaps within the *Finapres* graph result from active *Physiocal* calibrations, in which no reconstructed values are computed. The broad EDA graph is due to oscillations coming from the high sample frequency.

of the experiment. Because the *Trial* was twice as long as the *Rest* and *Recovery* phases, the peak count was halved to represent a by-minute parameter. The process of separating the EDA components, the normalisation of the cleaned signal and the peak detection is visualised in Figure 5.5.

$$\mathrm{EDA}_{\mathrm{baseline}} = \frac{1}{n}\sum_{i=0}^{n-1} \mathrm{EDA}_{\mathrm{filtered},\,i},\ n = 500 \tag{5.2}$$

$$\mathrm{EDA}_{\mathrm{norm},\,i} = \frac{\mathrm{EDA}_{\mathrm{filtered},\,i}}{\mathrm{EDA}_{\mathrm{baseline}}},\ i \in [0, 2400] \tag{5.3}$$

$$\mathrm{EDA}_{\mathrm{phasic\ rel.},\,i} = \frac{\mathrm{EDA}_{\mathrm{phasic},\,i}}{\mathrm{EDA}_{\mathrm{tonic},\,i}},\ i \in [0, 2400] \tag{5.4}$$

The computation of the HRV feature RMSSD was done via the *NeuroKit2* package [87]. It unites several different modules for neurophysiological signal processing and analysis. In

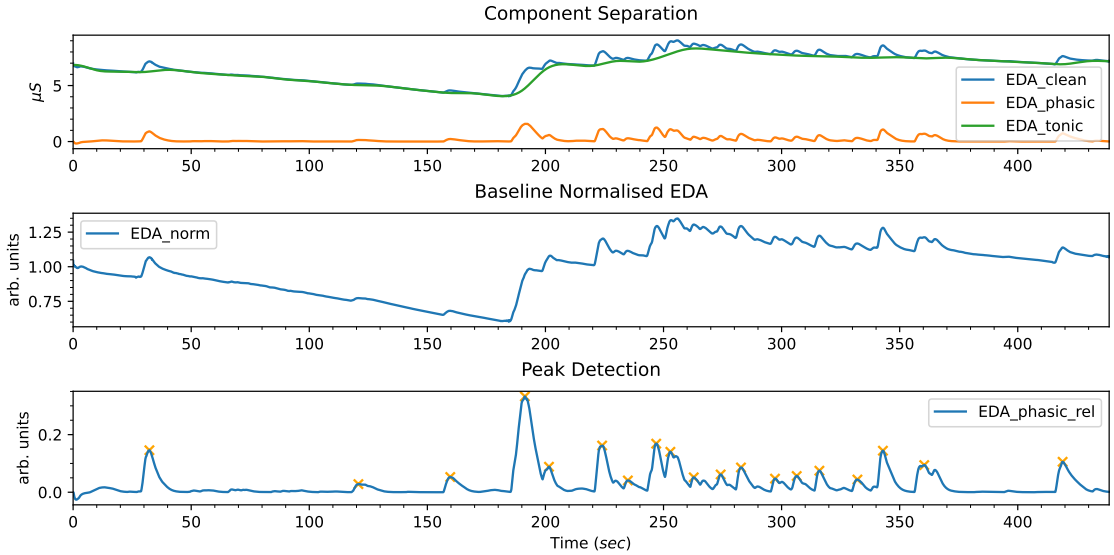


Figure 5.5.: The EDA signal processing steps. The top plot shows the separation of phasic (*EDA_phasic*) and tonic (*EDA_tonic*) components from the cleaned EDA-signal (*EDA_clean*). The bottom plot presents the result of the peak detection process.

this case, the HRV module was used as it contains the necessary function for the feature extraction. This module provides a single function for analysing all available features that can be extracted from pulse wave peaks and includes a convenience function that converts intervals into peaks. The latter is especially helpful, as the *Finapres* device only exported the interbeat intervals (IBI) from the PPG sensor in the cuff. After converting the IBI data to peaks using the *intervals_to_peaks()* functions, the signal was input into the *hrv_time()* function, which computed the time-domain features. However, because the function analyses the signal as a whole and thus only provides one value, the signal had to be split into the 10 s intervals proposed for the analysis, which resulted in 24 RMSSD values.

5.2.3. Alignment

Once all preprocessing steps were completed, the two *DataFrames* containing the measurements from *Finapres* and *Biosignals* export were combined using the *join()* function of *Pandas*. For this, the timestamp columns of the datasets were set as their indices, on

which they were then merged. By setting the method parameter “how” to “outer”, a union of both datasets was formed and ensured no data was lost. Because the *Finapres* data was only provided by the device with a sampling frequency of 1.25 Hz, it had to be upsampled to match the 10 Hz rate of the *Biosignal* dataset. This was achieved by resampling the already combined *DataFrame* to 10 Hz, using the *Pandas resample()* method. In addition to resampling the data, this method ensured that the dataset was trimmed within the experiment time frame by implementing an origin on which it was based. The *expStart* was used as the origin for the function. Furthermore, missing data points in the *Finapres* data were linearly interpolated using the *interpolate()* method included in the *Python* package.

After the alignment, additional columns containing the participant ID, the session type, the session volume and a new time axis were added to the *DataFrame* before it was saved in a CSV file. The steps mentioned above were repeated for each session and participant, resulting in 36 files in total.

5.3. Data Analysis Implementation

5.3.1. Column Selection

Since the measurements were separated into 36 files, they had to be selected and sorted according to the features and volumes. Like the preprocessing, this was done in Python. All files were searched and selected automatically using a searching algorithm based on the *walk()* method and the built-in *os* module. The files were identified with a unique filename snippet, read into a *Pandas DataFrame* and then appended to a list which combined all measurements. Then, all *DataFrames* were sorted based on their session type and volume. The next step involved selecting each column with a specific feature and volume or session type, and inserting it into a *dictionary* that ultimately contained nine *DataFrames*, one for each volume and session type. The measurements needed to be averaged over 24 ten-second intervals for the volume and interval analysis. For this, the *DataFrames* were split into sections of 100 rows for which the mean value of each column was calculated. The average of each row was then calculated to get the mean for all participants per interval, which was needed for plotting the data. At this point, the *DataFrame* were prepared for analysis and saved as CSV files for further analysis in the future.

5.3.2. Interval Analysis

For the interval analysis, the difference between each current interval and a reference point—either the previous interval, the first interval of the phase, the last interval of the *Rest* phase, or the last interval of the *Trial* phase—was calculated for all intervals and features. These differences were then checked for normality using the Shapiro-Wilk test from the *SciPy stats* module (*shapiro()*). The Shapiro-Wilk test tested the null hypothesis that the presented data is normally distributed [88]. If the test resulted in a p-value higher than the significance level $\alpha = 0.05$, the hypothesis was not discarded, and the data was assumed to be normally distributed. For normally distributed data, the significance was checked with another hypothesis test, the paired sample t-test. This tested the null hypothesis that the two samples (i.e. measurements of two intervals) have the same mean [89]. This time, if the p-value was below the significance level, the null hypothesis was discarded, and the data was assumed to be significantly different. For the implementation, the *ttest_rel()* function of the *SciPy* package was used. However, if the Shapiro-Wilk test did not confirm normality, a Wilcoxon signed-rank test was used to determine significance. The Wilcoxon signed-rank test is the non-parametric equivalent of the paired sample t-test [90]. It was implemented via the *wilcoxon()* function of the *stats* module and used the already calculated difference between intervals as input. All p-values, along with the overall mean values for all $10\,\mathrm{s}$ intervals, were combined into a new *DataFrame* that built the basis for the results tables (A.2-A.16). This process was repeated for each comparison and feature.

5.3.3. Volume Analysis

For the volume analysis, the features had to be normalised to the baseline for the volume analysis first. This step was skipped for the normalised EDA, as it was already given in relation to the baseline. The first $50\,\mathrm{s}$ were used as the baseline measurement. Similar to Equations 5.2 and 5.3, the average was calculated and the features were computed in relation to the baseline. Then, the differences between each interval at noise exposure and its counterpart at silence were calculated. Similarly to the interval analysis, these differences were checked for normality, and depending on the results of the Shapiro-Wilk test, either a paired sample t-test or a Wilcoxon signed-rank test was performed to assess significance. The p-values of the comparisons between sessions with noise and sessions without were combined in a *DataFrame* and subsequently saved in a CSV file.

5.3.4. Questionnaire scoring

The questionnaires were presented in paper form during the experiments. Since the questionnaires were filled out manually, the socio-demographic data and responses were entered into a *Python* script for processing. The script took the responses in the form and inverted all questions marked with an asterisk ("*") before scoring the surveys. Inversion had to be done as some questions were postulated to indicate higher sensitivity to noise or higher anxiety, while agreement with others would have meant the opposite. This was done with questions 1, 3, 8, 9, 12, 14, 15 and 20 of the WNSS, questions 3, 4 and 7 of the Trait part and questions 1, 4, 6 and 10 of the State section of the questionnaire. Then, the sum of all answers was calculated for each part of the questionnaire. Because a short version was used for the STAI, the scores of both State and Trait were converted to percentages using Equation 5.5.

$$\begin{aligned} raw &= sum\left(ans_i\right) \\ rel &= (raw - 10) \cdot \frac{100}{70} \end{aligned} \tag{5.5}$$

The calculated scores were then saved in a CSV file, along with the participant's general information. For the analysis, the scores of all participants were combined into a single *DataFrame* and processed with the *describe()* function of the package. This provides the mean, standard deviation, minimum, median and maximum values for all *DataFrame* columns (e.g. Age, Height or WNSS). To analyse the State Anxiety Inventory results for the influence of noise, the differences between the score for noise exposures and silence were calculated for all participants. Then, they were checked for normality using the Shapiro-Wilk test. The differences were distributed normally for the comparison of *volume 0* and *volume 0.5*, while they were not for the comparison of *volume 1* and silence. Based on this, a paired sample t-test was performed for the former and a Wilcoxon signed-rank test for the latter.

6. Results

In the following two sections, all results of the data analysis will be presented. Starting with the results from the questionnaires, where the noise annoyance test is evaluated, and both the WNSS and the Trait anxiety inventory are scored. Furthermore, the State anxiety test is scored and subsequently examined for noise influences on the score. Then, the in-depth analysis of the physiological measurements is presented for each volume. This includes the time-series comparisons, as well as the volume comparison for each feature.

6.1. Questionnaire Results

The two questions to assess the noise annoyance based on ISO/TS 15666:2021-05 standard resulted in none of the participants being classified as highly annoyed. Answers 'very' or 'extremely' on the verbal scale and 8, 9, or 10 on the numerical scale corresponded with highly annoyed. Only one participant answered with a 10 on the numeric scale, but this was most likely a misunderstanding, as the same person answered "not at all", representing the lowest weighted answer on the verbal scale.
The results of the remaining questionnaire sections are presented in Table 6.1. The Weinstein Noise Sensitivity Scale had a mean score of 49.83 out of 126 possible points, with a standard deviation of 14.40, while the Trait anxiety questionnaire had an average of 38.81% and a standard deviation of 18.23%.
When it comes to the State Anxiety Inventory, which was answered after each session, the mean score for *volume 0.5* was the highest with 32.74% and a standard deviation of 18.83%, while the average and standard deviation for *volume 1* and *volume 0* were $31.90 \pm 18.55\%$ and $29.29 \pm 13.83\%$ respectively. However, both increases in scores were insignificant compared to the score at *volume 0*. Furthermore, it is noteworthy that the standard deviation was larger when noise was included in the experiment.

Table 6.1.: This table presents the questionnaire results. The maximal possible score for the WNSS was 126, whereas the STAI scores were transformed into relative values. The rightmost column contains the p-values for the comparison with the baseline score at *volume 0*. A p-value below 0.05 was deemed significant.

	Mean ± SD	Min	Median	Max	Compared to Vol. 0 (p-value)
WNSS	49.83 ± 14.40	23	52	76	
Trait	38.81 ± 18.23 %	14.29 %	37.14 %	68.57 %	
State Vol. 0	29.29 ± 13.10 %	10.00 %	26.43 %	50.00 %	
State Vol. 0.5	32.74 ± 18.83 %	7.14 %	31.43 %	68.57 %	0.478
State Vol. 1	31.90 ± 18.55 %	2.86 %	29.29 %	61.43 %	0.581

6.2. Analysis of Physiological Measures

This section provides all results from the analysis of physiological features. The timeline analysis will be presented for each modality and volume, highlighting the significant differences for all four comparisons. Starting with the direct comparison, then the phase-start and pre-trial comparison, and finally the post-trial comparison (see Table 6.2 for explanation). Additionally, the results of the volume analysis are given at the end of each paragraph.

Table 6.2.: The explanation for each comparison made in the time series analysis.

Comparison	Explanation	Example
Consecutive	Compared each $10\,\text{s}$ interval to the previous $10\,\text{s}$.	$14 \rightarrow 13$
Phase-Start	Compared each $10\,\text{s}$ interval in a phase to the first $10\,\text{s}$ of the phase.	$5 \rightarrow 1 \mid 11 \rightarrow 7 \mid 21 \rightarrow 19$
Pre-Trial	Compared intervals 7-24 to the last $10\,\text{s}$ of *Rest*.	$16 \rightarrow 6$
Post-Trial	Compared intervals 19-24 to the last $10\,\text{s}$ of *Trial*.	$23 \rightarrow 18$

6.2.1. Systolic Blood Pressure

The dynamic changes of systolic blood pressure (sBP) for each volume are shown in Figure 6.1. Additionally, significant changes ($p < 0.05$) for each different comparison are marked. See Tables A.2, A.3 and A.4 for numeric means, standard deviations and p-values for all 10 s-intervals and volumes of the experiment.
When looking at the difference between consecutive intervals, all three sessions only occasionally revealed significant differences. For *volume 0*, a significant increase was recorded in interval 16, while the pressure significantly decreased in interval 20. For *volume 0.5*, the pressure significantly fell only in interval 19, which was also the case for *volume 1*. However, at *volume 1*, interval 10 was the only other section where the blood pressure rose significantly compared to the preceding interval. Compared to the phase-start, the pressure significantly increased for intervals 12, 13 and 16 to 18, while all intervals from 20 to 23 did show significant decreases at *volume 0*. Meanwhile, at *volume 0.5*, there were significant increases in intervals 5 and 6 compared to the start of *Rest*. Moreover, the systolic pressure increased in all intervals ranging from 11 to 16 and 18 compared to the start at interval 7. For *volume 1*, sBP significantly increased in sections 10 through 18 in relation to the start of the trial and significantly decreased from interval 21 until the end of the experiment compared to the start of *Recovery*. Compared to the last interval before the trial, the pressure significantly increased in intervals 10 to 13, 16, and 18 for *volume 0*, in intervals 10 to 18 for *volume 0.5* and in all intervals from 11 to 18 for *volume 1*. Finally, the pressure significantly decreased in all intervals for all volumes, except 19 at *volume 0*, compared to the last interval of *Trial*.
The volume analysis revealed an increase in systolic blood pressure for intervals 12 and 13 at *volume 0.5* compared to the session without noise. This is also presented in Figure 6.6.

6.2.2. Diastolic Blood Pressure

The dynamic changes of diastolic blood pressure (dBP) for each volume are shown in Figure 6.2. Additionally, significant changes ($p < 0.05$) for each different comparison are marked. See Tables A.5, A.6 and A.7 for numeric means, standard deviations and p-values for all 10 s-intervals and volumes of the experiment.
Compared to the previous interval, the pressure significantly increased only in intervals 4, 6 and 11 for *volume 0*, while no intervals did show significant increases for *volumes 0.5* and *1*. However, in interval 21 at *volume 0*, interval 19 at *volume 0.5* and intervals 19, 20 and 24 at *volume 1*, the diastolic pressure decreased significantly compared to the preceding 10 s. At *volume 0*, the dBP significantly increased in the last interval of *Rest* compared to

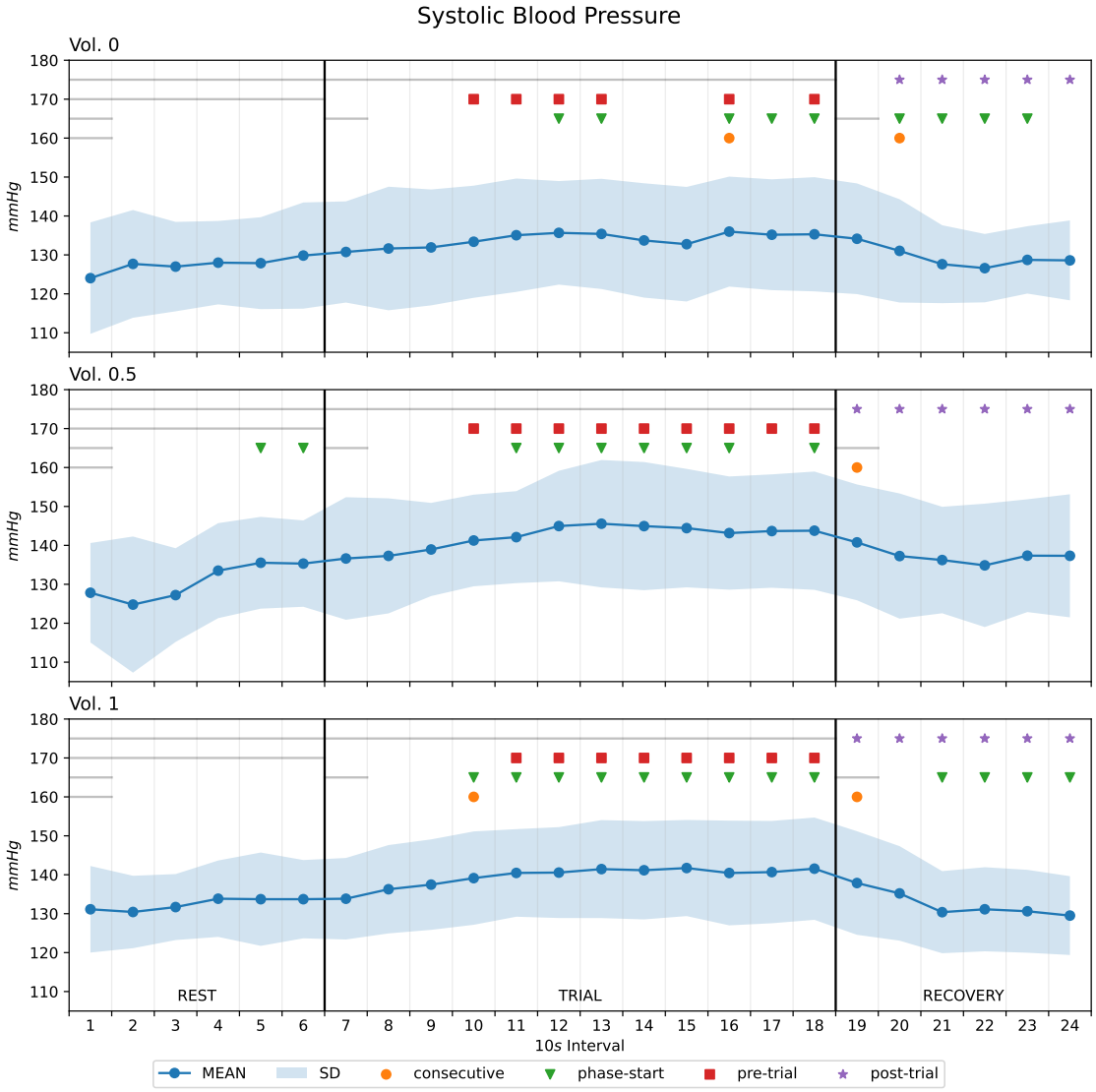


Figure 6.1.: The timeline changes in systolic blood pressure. The blue graph represents the mean values for each interval, while the light-blue area resembles the standard deviation. Furthermore, all intervals where significant changes occur are labelled with the corresponding marker. The orange circles represent consecutive comparisons, the green triangles indicate phase start comparisons, the red squares correspond to pre-trial comparisons, and the purple stars indicate intervals where significant changes are recorded compared to the last interval of the *Trial* phase.

the start of the phase, as well as in intervals 11, 12, 13, 16 and 18 compared to the first $10\,\mathrm{s}$ of the *Trial* phase. During recovery, significant decreases happened in intervals 21 and 22 compared to interval 19. For *volume 0*, the pressure increased significantly in all intervals 11 through 18, while at *volume 1*, it only increased in intervals 12 and 15-18. No evidence for changes compared to the last $10\,\mathrm{s}$ of *Rest* was found for *volume 0*, while the pressure significantly increased in all intervals from 10 to 18 compared to interval 6 for *volume 0.5*. Similarly, the dBP increased in intervals 11-18, except 13 at *volume 1*. While the blood pressure only decreased in intervals 21 and 22 when compared to the last section of the trial at *volume 0*, it significantly decreased in all intervals of the *Recovery* phase at *volume 0.5* and *volume 1*.
The volume analysis in Figure 6.6 shows that in intervals 23 and 24 at *volume 1*, the diastolic blood pressure decreased compared to *volume 0*.

6.2.3. Heart Rate

The dynamic changes in heart rate (HR) for each volume are shown in Figure 6.3. Additionally, significant changes ($p < 0.05$) for each different comparison are marked. See Tables A.8, A.9 and A.10 for numeric means, standard deviations and p-values for all $10\,\mathrm{s}$-intervals and volumes of the experiment.
The heart rate increased at *volume 0* in intervals 14 and 17 while it decreased in 3 and 21 compared to the interval before. At *volume 0.5*, only interval 12 did show a significant decrease in heart rate. Whereas, for *volume 1*, the heart rate significantly increased in intervals 8 and 17 and decreased in intervals 12 and 20, compared to the preceding $10\,\mathrm{s}$. Decreases occurred in intervals 3, 4, 16, and 18 at *volume 0* and 20-23 at *volume1* compared to the first interval of the phase. Only interval 8 did show significant increases at *volume 1* compared to the first $10\,\mathrm{s}$ of *Trial*, whereas no evidence for changes was found for *volume 0.5*. Compared to the last interval of the *Rest* phase, the heart rate decreased in interval 22 for *volume 0* and all intervals of the *Recovery* phase at *volume 0.5*. Whereas, for *volume 1*, in intervals 8 to 10, the heart rate increased significantly in relation to interval 6. Furthermore, in intervals 20 to 23 of *Recovery* at *volume 1*, the heart rate decreased compared to the last $10\,\mathrm{s}$ of the *Trial* phase, while no evidence for such changes was found for *volumes 0* and *0.5*.
When comparing *volume 0* and *1* to *volume 0*, the heart rate significantly decreased in intervals 23 and 24 at *volume 0.5*, while it increased in interval 10 at *volume 1* (see Figure 6.6).

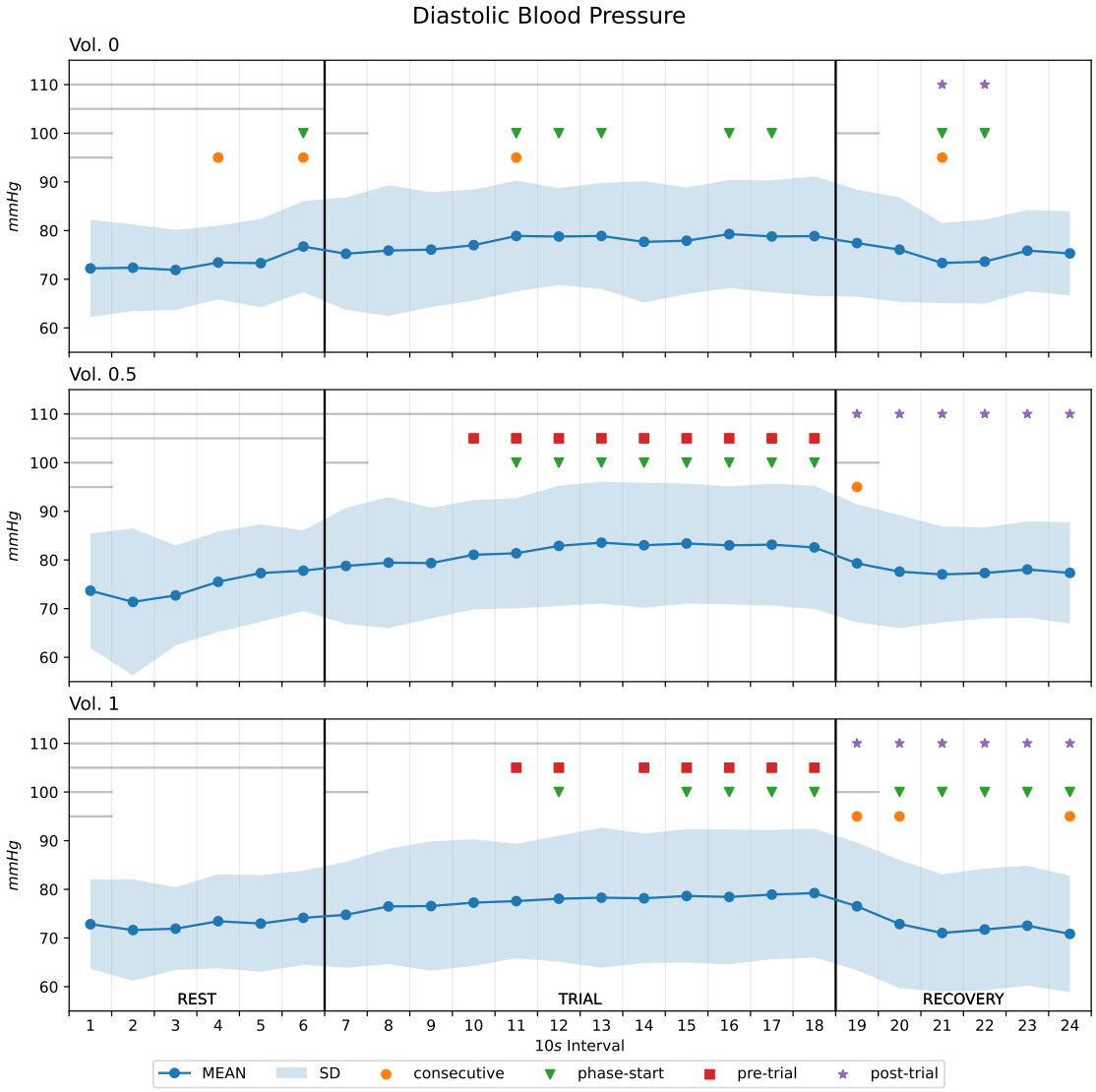


Figure 6.2.: The timeline changes in diastolic blood pressure. The blue graph represents the mean values for each interval, while the light-blue area resembles the standard deviation. Furthermore, all intervals where significant changes occur are labelled with the corresponding marker. The orange circles represent consecutive comparisons, the green triangles indicate phase start comparisons, the red squares correspond to pre-trial comparisons, and the purple stars indicate intervals where significant changes are recorded compared to the last interval of the *Trial* phase.

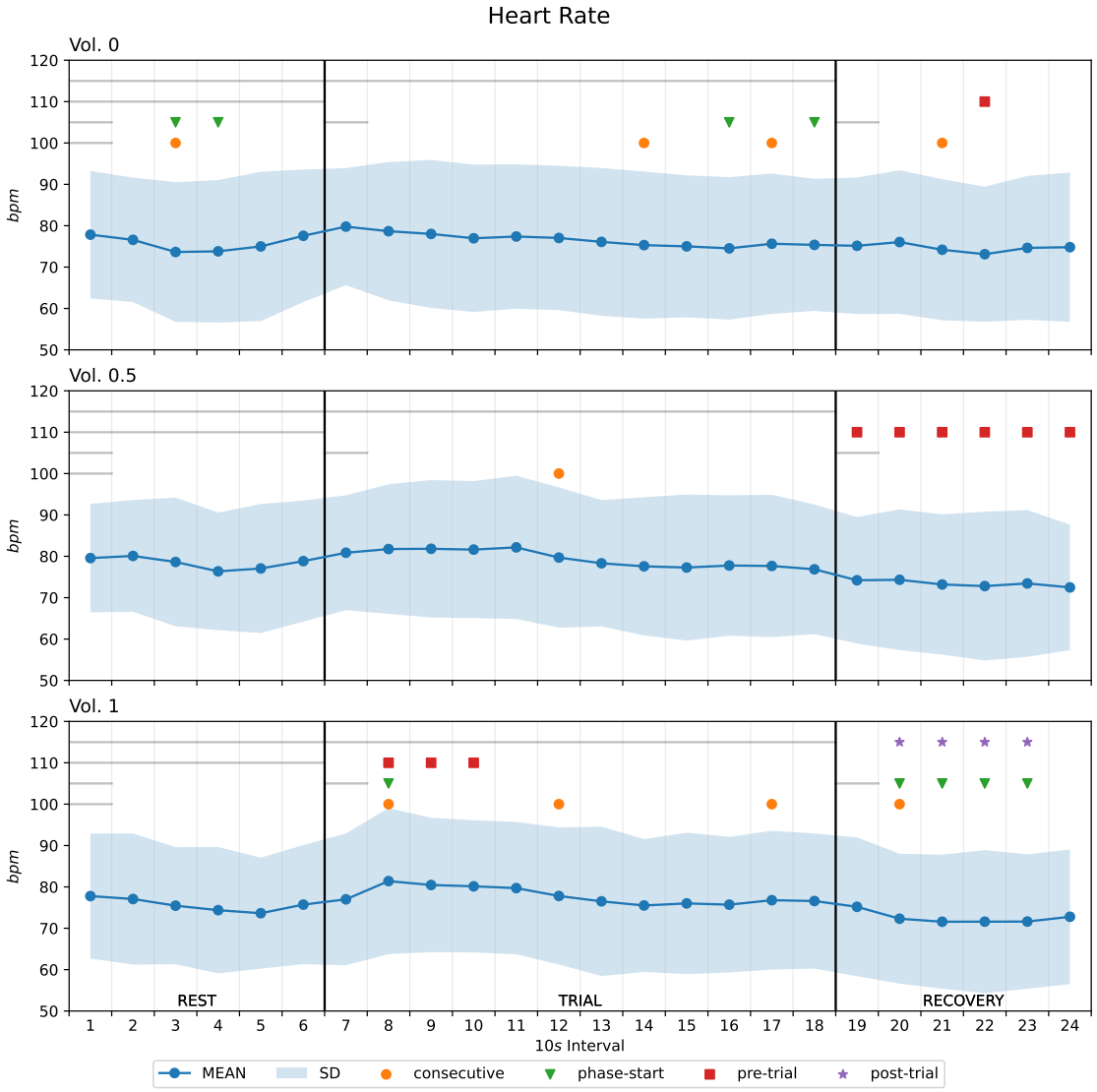


Figure 6.3.: The timeline changes in heart rate. The blue graph represents the mean values for each interval, while the light-blue area resembles the standard deviation. Furthermore, all intervals where significant changes occur are labelled with the corresponding marker. The orange circles represent consecutive comparisons, the green triangles indicate phase start comparisons, the red squares correspond to pre-trial comparisons, and the purple stars indicate intervals where significant changes are recorded compared to the last interval of the *Trial* phase.

6.2.4. Root Mean Squared Sequential Differences

The dynamic changes in the root mean square of successive differences (RMSSD) for each volume are shown in Figure 6.4. Additionally, significant changes ($p < 0.05$) for each different comparison are marked. See Tables A.11, A.12 and A.13 for numeric means, standard deviations and p-values for all 10 s-intervals and volumes of the experiment.
The RMSSD significantly increased in the second interval compared to the first for *volume 0*, in intervals 10 and 19 for *volume 0.5* and in interval 22 for *volume 1*. Compared to the first interval of each phase, significant increases only occurred in interval 2 at *volume 0* while decreases happened in interval 3 at *volume 0* and 21 at *volume 0.5*. The RMSSD only increased in interval 21 at *volume 0.5* compared to the last 10 s. Furthermore, in intervals 19 and 20, an increase compared to the last interval of the *Trial* phase happened at *volume 0.5*, while for *volume 1*, a significant decrease occurred in the last interval of the *Recovery* phase. Contrary to the timeline analysis, the volume analysis did not show evidence of a significant difference between noise exposure and quiet.

6.2.5. Baseline Normalised EDA Level

The dynamic changes of baseline normalised electrodermal activity (EDA) level for each volume are shown in Figure 6.5. Additionally, significant changes ($p < 0.05$) for each different comparison are marked. See Tables A.14, A.15 and A.16 for numeric means, standard deviations and p-values for all 10 s-intervals and volume of the experiment.
At *volume 0*, the normalised EDA level significantly increased in interval 7, while it decreased in intervals 4, 15, 18 and 21-23 compared to the preceding interval. For *volume 0.5*, the values increased in intervals 2, 7, and 8, whereas they decreased significantly in 15 and 20-23. At *volume 1*, intervals 7 and 8 show increased normalised EDA and decreased values for intervals 12, 13, 16, 18, 21, 22 and 23 compared to the previous 10 s. Compared to the phase start, the activity only significantly increased in intervals 2 and 8 at *volume 0.5* and intervals 8 and 9 at *volume 1*. Whereas it decreased in intervals 4, 5, 6 and 21 to 24 at *volume 0*, intervals 20 to 24 at *volume 0.5* and 21 through 24 at *volume 1*. Significant increases occurred in intervals 7-14 for *volumes 0* and *1*, while at *volume 0.5*, all intervals 7-13, except 11, did show significant differences compared to the last 10 s of the *Rest* phase. The normalised EDA significantly decreased in all intervals from 21 to 24 for all volumes. Although many intervals revealed significant differences when compared to intervals in the same session, no significant changes in normalised electrodermal activity (EDA) were found when comparing different volumes (see Figure 6.6).

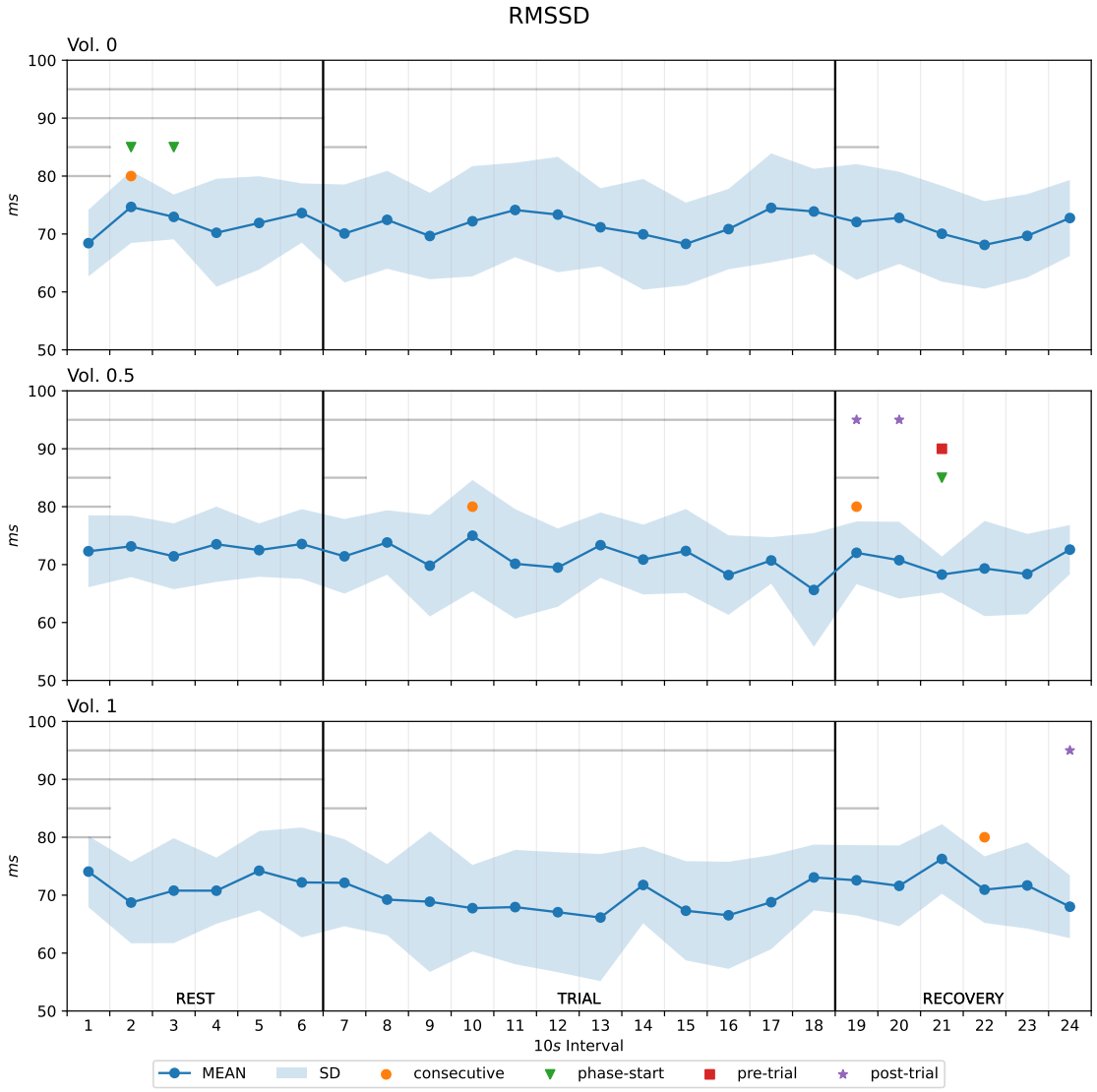


Figure 6.4.: The timeline changes in RMSSD. The blue graph represents the mean values for each interval, while the light-blue area resembles the standard deviation. Furthermore, all intervals where significant changes occur are labelled with the corresponding marker. The orange circles represent consecutive comparisons, the green triangles indicate phase start comparisons, the red squares correspond to pre-trial comparisons, and the purple stars indicate intervals where significant changes are recorded compared to the last interval of the *Trial* phase.

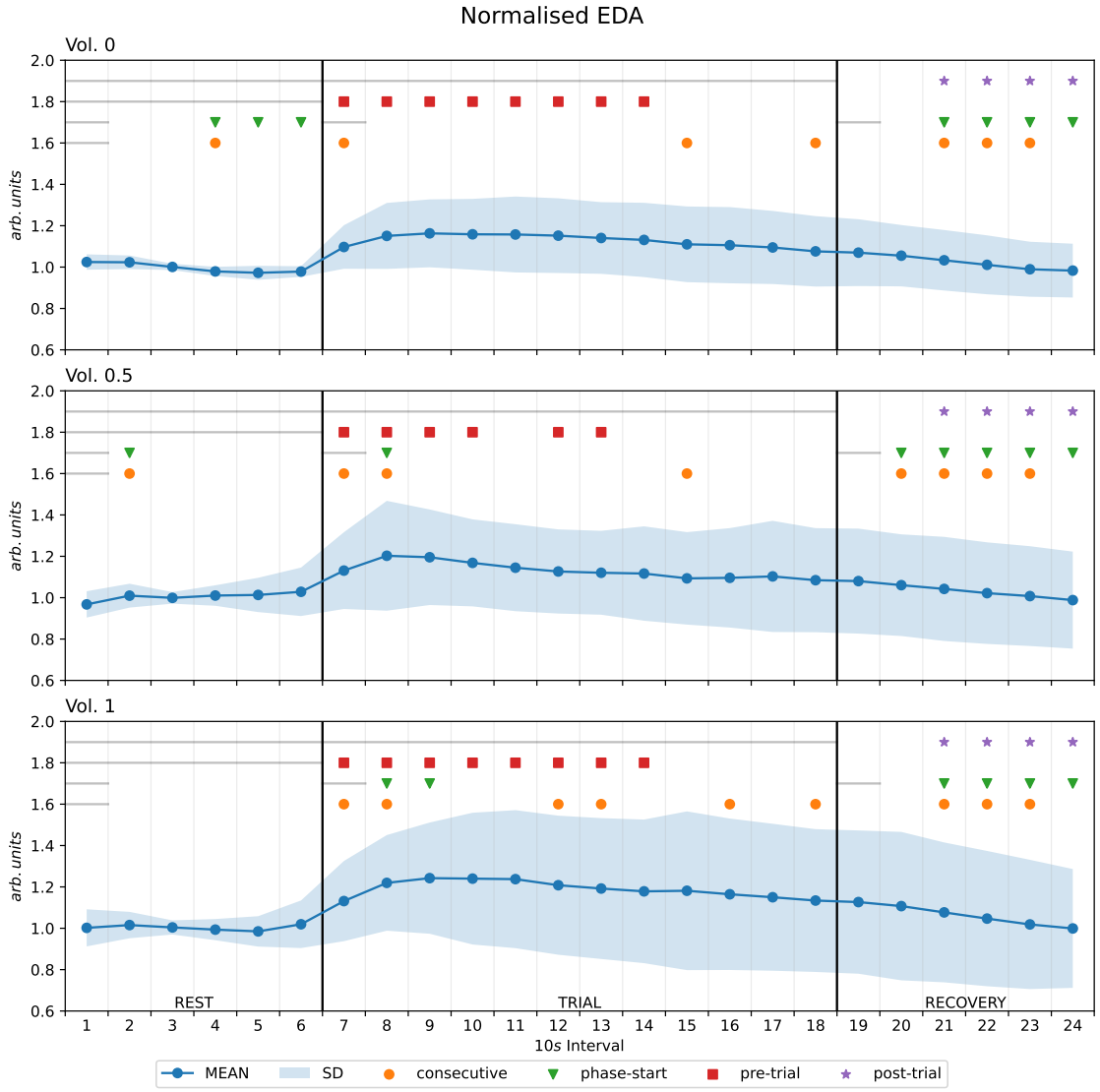


Figure 6.5.: The timeline changes of baseline normalised EDA level. The blue graph represents the mean values for each interval, while the light-blue area resembles the standard deviation. Furthermore, all intervals where significant changes occur are labelled with the corresponding marker. The orange circles represent consecutive comparisons, the green triangles indicate phase start comparisons, the red squares correspond to pre-trial comparisons, and the purple stars indicate intervals where significant changes are recorded compared to the last interval of the *Trial* phase.

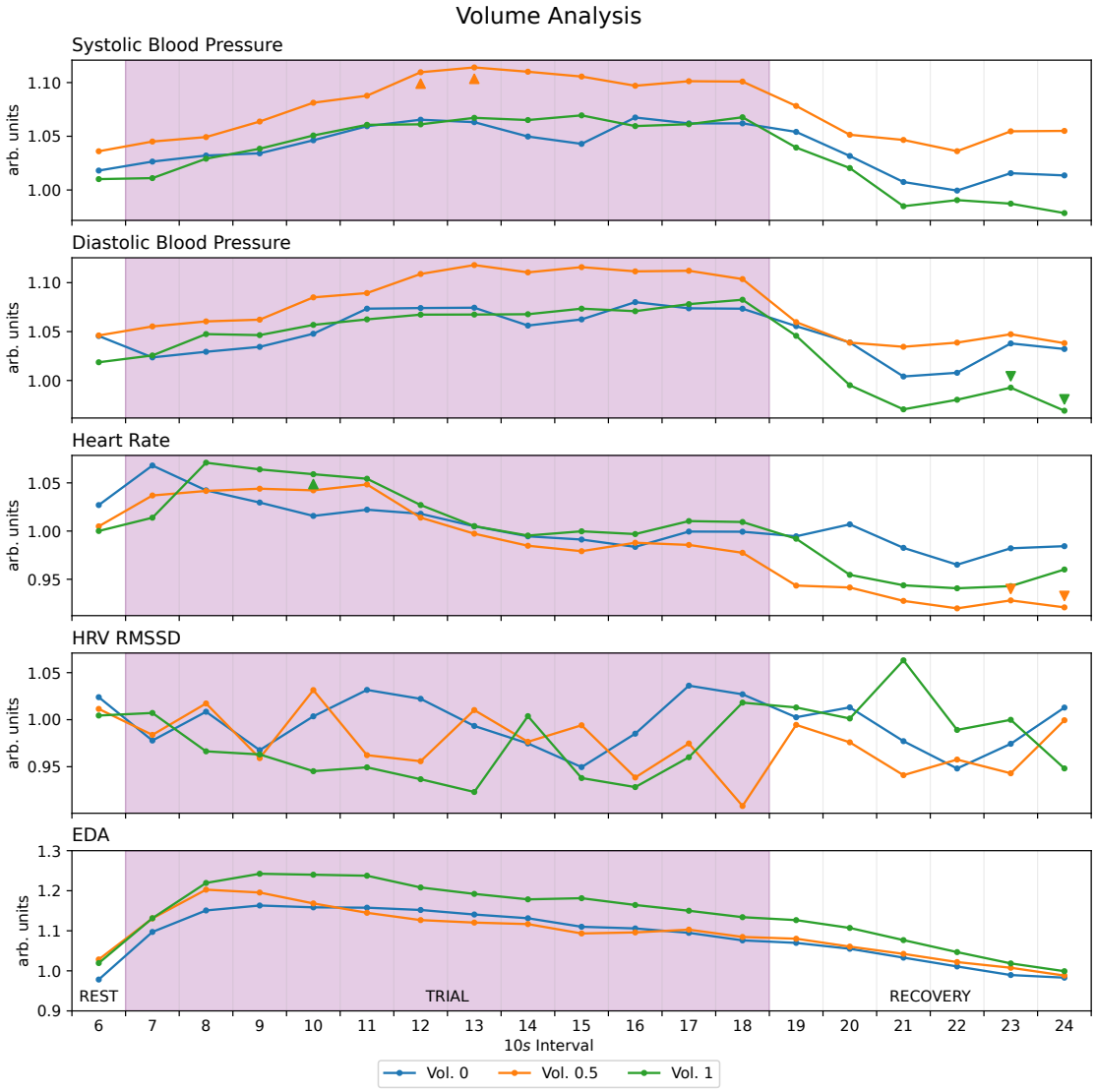


Figure 6.6.: The volume analysis of baseline normalised physiological features for volumes 0, 0.5 and 1. Significant differences in each interval compared to *volume 0* are marked by a triangle in the corresponding colour. The direction of the triangle indicates whether the value increases or decreases due to the noise. Tables A.17 and A.18 provide the numeric p-values on which this is based on.

6.2.6. EDA Peaks-Per-Minute

The results from the analysis of the EDA peaks-per-minute are presented in Table 6.3. At *volume 0*, the mean number of peaks slightly increases from 3.06 ± 2.68 during *Rest* to 3.25 ± 3.64 during *Trial*. Furthermore, during the *Recovery* phase, the average peak rate dropped to 1.92 ± 2.15 again. However, the changes were not significant when comparing the phases. Similar to *volume 0*, the amount of peaks per minute insignificantly increased from 3.83 ± 2.48 to 4.29 ± 3.12 going from *Rest* to *Trial* at *volume 0.5*. Again, the peaks dropped to 1.15 ± 1.45 after the trial, but this time the differences were significant compared to both *Trial* and *Rest*. Comparable results were found for *volume 1*. At this volume, the mean peak rate insignificantly rose from 3.92 ± 2.31 to 4.5 ± 3.44 during the *Trial* phase while significantly it decreased to 1.92 ± 1.83 during *Recovery*. Compared to *Rest*, the average peak rate also decreased significantly during the last phase of the experiment. Although some in-session comparisons revealed significant changes, no evidence for significant in- or decreases was found for the volume comparison.

Table 6.3.: The results of the peak rate analysis.

	Volume 0		
	Mean ± SD	Difference to *REST* *	Difference to *TRIAL* *
REST	3.08 ± 2.68	—	—
TRIAL	3.25 ± 3.64	0.801	—
RECOVERY	1.92 ± 2.15	0.073	0.110

	Volume 0.5		
	Mean ± SD	Difference to *REST* *	Difference to *TRIAL* *
REST	3.83 ± 2.48	—	—
TRIAL	4.29 ± 3.12	0.569	—
RECOVERY	1.15 ± 1.45	0.001	<0.001

	Volume 1		
	Mean ± SD	Difference to *REST* *	Difference to *TRIAL* *
REST	3.92 ± 2.31	—	—
TRIAL	4.50 ± 3.44	0.323	—
RECOVERY	1.92 ± 1.83	0.006	<0.001

	Difference to *Volume 0* *	
	Vol. 0.5	*Vol. 1*
REST	0.056	0.227
TRIAL	0.101	0.097
RECOVERY	0.339	>0.99

* p-values

7. Discussion

This chapter will discuss the results presented in Chapter 6. Each approach for analysing physiological measurements and their results will be reviewed separately. Furthermore, blood pressure and electrodermal activity will be compared to assess their capability to detect stress. In the end, the questionnaire results and the physiological measures will be compared to evaluate a possible disparity between physiological measures and subjective judgment.

Before discussing the analysis, a short contextualization of the questionnaire results is given. According to the noise annoyance assessment based on the ISO/TS 15666:2021-05, none of the participants were classified as highly annoyed. The average score for the Weinstein Noise Sensitivity Scale (WNSS) of 49.83 from possible 21-126 points (27.46%) suggests that the study population was rather insensitive to noise. The average recorded Trait Anxiety, which represents the general tendency to experience anxiety, was below 40%, indicating moderate anxiety among the participants during the time of testing.

7.1. Time Series Analysis

The time-series analysis incorporated a series of comparisons to shed light on the dynamic changes that occur during an experiment combining noise exposure and a cognitive task. The results have shown that in the first phase of the experiment, the *Rest* phase next to no significant changes in physiological measures occurred. In some cases, the measurement suggested relaxation during the phase. This was the case for the normalised EDA at *volume 0*, as it decreased towards the end of the first section compared to the start of the experiment. The heart rate did show similar signs of relaxation in the middle of *Rest* at *volume 0*. This may be due to higher stress levels at the start of the measurement and the curiosity or excitement that comes with it. Moreover, the participants' focus on the static screen while wearing the over-ear headphones, which isolated them acoustically, could

have had a calming effect and, therefore, reduced stress. However, some intervals did show significant increases in blood pressure right before the start of the *Trial* phase. At *volume 0.5*, the sBP increased significantly in the last 20 s compared to the start of the experiment. This could be since, for one, the anticipation of the *2-back task* and noise exposure may have led to increased stress, and for another, the 10 s countdown started in the last ten seconds of *Rest*. The latter may have amplified the anticipation and heightened the stress effect. Another indicator of the possible influence of the countdown is the increase of dBP in the last interval of rest compared to the start and the preceding 10 s at *volume 0*. Yet, the suggested effect is not examined for any other modality, which lessens the likelihood that the countdown significantly influenced the measurement.

Contrary to the *Rest* phase, multiple 10 s intervals revealed significant signs of stress during the *Trial* phase. This effect occurred fastest for the normalised EDA, which did show significant differences right after the trial started for every volume. Moreover, the increase in activity compared to the previous interval remained significant for the first 20 s of the trial only *volume 0.5* and *volume 1*. This could suggest that the addition of noise amplified the effect of the cognitive task on the participants. Furthermore, at *volume 1*, the EDA increased further in interval 9 compared to the start of the trial, which could mean that the higher intensity of noise had a greater effect on the participant's stress level compared to the lower volume and quiet. Otherwise, no evidence for significant changes compared to the first 10 s of *Trial* was found, suggesting that after the initial increase, the normalised electrodermal activity decreased again. This decrease, however, would have happened steadily in small steps as most intervals after the initial burst did not decrease significantly compared to the preceding interval. Similar timelines were recorded regarding significant differences for all volumes when comparing an interval and the last 10 s of *Rest*. Up until intervals 13 and 14, the EDA remained elevated compared to interval 6, which further supports the assumption that the values only decreased slowly after the sudden increase. Ten seconds after the start of the trial, in interval 8, the heart rate too had a significant increase compared to the preceding interval and the last interval of *Rest* at *volume 1*. The HR remained significantly higher during intervals 9 and 10. These results indicate that at *volume 1*, the heart rate increased only during the first seconds of the trial with a delay of approximately 10 s. For *volume 0* and *0.5*, significant changes only occurred occasionally, and no increases in connection to the start of the trial or noise were recorded. That means the heart rate may have only been affected by the sound at *volume 1*, while lower volumes and the n-back task did not influence it significantly.
As for systolic blood pressure, a significant increase was recorded for the first time in interval 10 for *volumes 0* and *0.5* and in interval 11 for *volume 1* compared to the last 10 s of *Rest*. These increases did not happen abruptly, as there was no significant difference

compared to the preceding intervals. Furthermore, the increase of sBP in intervals 10 to 18 at *volume 0*, as well as 11 to 16 and 18 at *volume 0.5*, also support this claim. This suggests that the systolic blood pressure levels rose only slowly but continuously throughout the noise exposure. The fact that at *volume 0*, the series of intervals with significant difference compared to the last interval of rest and the first interval of the trial was interrupted by sections without changes may suggest that the noise exposure amplified the stressful effect of the *2-back task*. The root mean square of successive differences did not show any signs of influence by either the cognitive task of the noise exposure. As the RMSSD is a feature that mainly represents the activity of the PSNS, an effect due to the trial would have been expressed by significant decreases in the values.

During recovery, most features show decreased values corresponding with relaxation and reduced stress. Contrary to the *Trial* where both BP parameters responded only after a 30–40 s delay, these features were the first to change in the *Recovery* phase. Immediately after the *Trial* stopped, sBP and dBP dropped significantly compared to the last 10 s of *Trial* for *volumes 0.5* and *1*. At *volume 0*, a significant decrease in relation to the *Trial* first occurred after another 10 s in interval 21. These differences persisted throughout the entire last phase of the experiment for all volumes, indicating a significant recovery effect. Both sBP and dBP showed no evidence for significant differences compared to the last interval before the *Trial*. This could indicate that BP levels decreased to similar levels as before the test. Although no clear evidence for the countdown's stressful effect was found, comparing the post-trial measurements to possibly already increased values would distort the result and undermine this suggestion. Moreover, the fact that only at *volume 1*, both systolic and diastolic pressure were consistently decreased during *Recovery* implies that the higher volume resulted in a prolonged relaxation response from the participants. The heart rate only decreased significantly compared to the *Trial* phase for *volume 1*. This, together with the prolonged recovery of sBP levels, indicates a link between noise intensity and hemodynamic recovery. Interestingly enough, for *volume 0.5,* during the entire *Recovery* phase, the HR values decreased to lower values than before the trial, which was not recorded by any other modality. This means that with the noise at *volume 0.5*, the participant may have started the experiment with an already elevated heart rate, which was not increased by the *2-back task* and the noise but decreased slowly to the point where the difference was significant compared to the *Rest* phase. The normalised EDA showed a significant and continuous decrease during the *Recovery* compared to the *Trial* for all volumes. However, this was not as abrupt as with the BP levels and only occurred 20 s after the end of the *Trial*. This might be due to the characteristics of the EDA measurement as the device measures the skin conductivity and, therefore, is influenced by the sweat gland activity. Although the sweat glands are entirely neurally activated, a decrease would only

be detected after some time as the skin surface remains moist. Again, the RMSSD fell short when it came to indicating clear signs of change during the *Recovery* phase.

On a separate note, the peaks per minute were not as responsive as the normalised EDA in detecting the onset of a stress response. The increase in the number of peaks from *Rest* to *Trial* did not reach significance. However, comparing the *Recovery* phase to *Trial* and *Rest* resulted in significant decreases for *volumes 0* and *1*. The decrease below the rate recorded for the *Rest* phase suggests that the participants were already in an increased stress state before the noise was introduced.

To summarise the time-series analysis, both normalised EDA and blood pressure were most affected by the experiment, followed by the heart rate. At the same time, the chosen HRV feature did not show clear signs of influence by the test. While the normalised EDA was the fastest measurement to respond to the stressors presented in the test sessions, the sBP and dBP were the first to show signs of recovery. Although the EDA and blood pressure were similarly affected by all volumes, the heart rate appeared to be influenced only by the onset of the noise at *volume 1*. This suggests that the increase in blood pressure might be caused by higher peripheral resistance rather than a chronotropic effect. These results concur with those of Paunović et al. [62] who influenced this approach by their study on timeline changes of blood pressure during noise exposure. Although they used 1 min intervals and longer exposure times in their analysis, their findings are reflected in the time-series analysis of this work. They reported significant increases during the first two minutes of noise exposure, equivalent to the entire noise exposure of this experiment, which did also show significant increases during noise. However, this experiment further shows that an increase might happen only after 30 s of noise. As for the recovery, the results of this work show a significant decrease right after the noise exposure ended. This is again also reported by Paunović et al. [62], who saw a significant decrease in blood pressure in the first minute after the noise exposure. Furthermore, their timeline analysis of heart rate shows a significant increase only in the first minute of noise for men. Although this experiment included women, it also showed increased heart rate only after the onset of noise, after which changes remained insignificant.

7.2. Noise Intensity and Stress Response Amplitude

The analysis of the physiological measurements regarding the influence of noise intensity on the stress response magnitude showed only a few significant differences among the features. The sBP increased significantly only in two intervals after $50\,\mathrm{s}$ of noise at *volume 0.5* compared to the quiet session. One interval showed an increased heart rate after $30\,\mathrm{s}$ for noise exposure at *volume 1*. When only looking at the course of the mean values and disregarding significance, both BP-features indicate the strongest influence of noise for *volume 0.5*, while silence and *volume 1* show similar behaviour over the course of the experiment. However, the mean normalised EDA suggests that noise at *volume 1* influenced the participants most, as higher levels are recorded throughout the experiment. The mean heart rate and RMSSD did not show a clear trend of amplitude differences depending on the volume. The results of the State Anxiety Inventory also did not provide significant differences depending on the noise intensity. However, the mean percentages increased in noise sessions and the highest value was recorded after noise at *volume 0.5*, which aligns with the results from the blood pressure measurements. Moreover, the increased standard deviation in noise exposure sessions compared to no-noise sessions suggests that some participants experienced heightened nervousness or agitation. To summarise, no significant differences or consistent trends linking noise intensity to the magnitude of the human stress response were examined.

7.3. Limitations

During the assembly of this work, several study limitations became evident. While this experiment provided insights into the dynamic stress response, its findings cannot be fully generalised to real-life situations. One key limitation is the study population. The Participants were predominantly young adults with average weight and blood pressure and without relevant pre-existing conditions, which limits the results' applicability to the general public. Additionally, the small sample size likely amplified individual variations in the outcomes. The study design also presented potential biases. Due to the way sound exposure was implemented, participants were aware of session order and volume, possibly introducing expectation bias. Gallasch et al. [61] reported that noise effects were strongest during the first exposure, suggesting a time-order effect that may have influenced our results. While the session order was reviewed for trends, further analysis was deemed unnecessary due to the small number of participants per sequence and

inherent dependency on individual characteristics. Furthermore, conducting all sessions in one sitting may have led to habituation, causing the strongest response to occur in the first trial, regardless of noise presence. Another limitation may have been the short duration and intensity of noise exposure. Compared to previous studies, the two-minute exposure to road traffic noise was relatively brief, potentially reducing or preventing a full stress response. Although the validated noise recordings represent typical road traffic noise intensities [1], a greater difference between noise levels could provide information about the insignificant trends observed in this study. Finally, measurement-related factors may have influenced the results. Although the blood pressure device was non-invasive, it was not entirely unobtrusive. The cuff applied continuous pressure to the participant's finger, creating a pulsating sensation, while the control system on the lower arm emitted a low-intensity rushing noise. Both factors could have affected the physiological measurements.

8. Conclusion

This work investigated the noise-induced stress response through non-invasive continuous finger arterial pressure measurement. Based on a comprehensive literature review, an experimental study was designed and implemented to induce stress via an acoustic stressor and a cognitive task. The physiological stress response was recorded with high temporal resolution, and an in-depth time series analysis was conducted.

One goal was to assess the dynamic changes of the physiological measurement throughout the course of the experiment. This was evaluated with a detailed time series analysis of 24 ten-second intervals. The results indicate that the autonomic reaction to stress is mediated differently by the recorded features. The blood pressure gradually increased once the test started. After $30\,\mathrm{s}$ to $40\,\mathrm{s}$, this increase became significant compared to the *Rest* period before the test. During the *Recovery*, both systolic and diastolic pressure abruptly decreased until a steady level was reached. On the contrary, the normalised electrodermal activity responded in a sudden spike in the first twenty seconds, after which a slight but continuous decrease was recorded. The heart rate was seemingly only influenced by the high-intensity noise, as a peak ten seconds after the onset and a decrease during *Recovery* was examined. The RMSSD, calculated form $10\,\mathrm{s}$-intervals, did not provide evidence to conclude a link between the experiment and the heart rate variability.

Furthermore, this work set out to examine the influence of different noise intensities on the dynamic and magnitude of the stress response. The results could not show a significant and consistent correlation between noise volume and the amplitude of the physiological reactions. The results of the systolic blood pressure suggest that noise at *volume 0.5* had the highest influence on the participants. The results of the other measurements do not support this claim, as amplitude differences due to noise were not recorded. However, differences in the dynamic response were observed for blood pressure and heart rate. For noise exposure sessions, the pressure levels significantly decreased immediately after the test. While in the session without noise, a significant drop compared to the trial was only detected after ten seconds. As mentioned above, the heart rate was only influenced by

the noise at *volume 1*, whereas the sessions at *volumes 0.5* and *0* did not show signs of influence by the test.

Finally, the continuous non-invasive blood pressure (cNIBP) was evaluated as an indicator for stress, compared to the electrodermal activity. This work has shown that the measurements of both blood pressure and electrodermal activity can detect the human stress response with high temporal resolution. The usage of cNIBP measurement devices, like the *Finapres® NOVA*, does provide novelty to the field of human stress research. Even though EDA did outperform the BP parameters in terms of latency, when it came to the dynamics of recovery, both systolic and diastolic pressure showed significant changes faster than the EDA. The results suggest that for detecting the onset of the human stress response, the electrodermal activity remains the better option. However, continuous blood pressure monitoring is advantageous when the interest lies in the dynamic changes during stress recovery.

Future research should rerun the proposed study with an increased population size to minimise individual effects and analyse a possible time-order effect. An extended noise exposure and recovery phase would further capitalise on the advantages of cNIBP measurements and provide detailed information on the dynamic of the human stress response. To better understand the relationship between noise intensity and stress response, future studies should use more distinctly varied noise levels.

Bibliography

[1] European Environment Agency. *Environmental noise in Europe, 2020*. Publications Office, 2020. DOI: 10.2800/686249.

[2] L. Werdecker and T. Esch. "Stress und Gesundheit". In: *Gesundheitswissenschaften*. Ed. by R. Haring. Berlin, Heidelberg: Springer, 2022, pp. 389–401. ISBN: 978-3-662-65219-0. DOI: 10.1007/978-3-662-65219-0_33.

[3] T. Münzel, M. Sørensen, T. Gori, et al. "Environmental stressors and cardio-metabolic disease: part II–mechanistic insights". In: *European Heart Journal* 38.8 (2016), pp. 557–564. DOI: 10.1093/eurheartj/ehw294.

[4] G. Kronenberg, J. Schöner, C. Nolte, et al. "Charting the perfect storm: emerging biological interfaces between stress and stroke". In: *European Archives of Psychiatry and Clinical Neuroscience* 267.6 (2017), pp. 487–494. ISSN: 1433-8491. DOI: 10.1007/s00406-017-0794-x.

[5] B. S. McEwen. "Neurobiological and Systemic Effects of Chronic Stress". In: *Chronic Stress* 1, 2470547017692328 (2017). DOI: 10.1177/2470547017692328.

[6] T. Münzel, M. Sørensen, F. Schmidt, et al. "The Adverse Effects of Environmental Noise Exposure on Oxidative Stress and Cardiovascular Risk". In: *Antioxidants & Redox Signaling* 28.9 (2018), pp. 873–908.

[7] T. Esch, G. B. Stefano, G. L. Fricchione, et al. "The role of stress in neurodegenerative diseases and mental disorders". In: *Neuro endocrinology letters* 23.3 (2002), pp. 199–208. ISSN: 0172-780X.

[8] M.-F. Marin, C. Lord, J. Andrews, et al. "Chronic stress, cognitive functioning and mental health". In: *Neurobiology of Learning and Memory* 96.4 (2011). Memory Impairment and Disease, pp. 583–595. ISSN: 1074-7427. DOI: 10.1016/j.nlm.2011.02.016.

[9] Gesundheitsberichterstattung des Bundes. *Total Cost of Illness in millions of Euro for Germany. Classification: years, sex, ICD-10, provider*. URL: https://www.gbe-bund.de/gbe/isgbe.indikatoren_set_sort?p_uid=gast&p_aid=7123319&p_sprache=D&p_help=2&p_indnr=66&p_ansnr=66165241&p_version=3&p_sortorder=d&p_dim_1=D.003&p_dw_1=1000004&p_dim_2=D.000&p_dw_2=3742&p_thema_id=26891&p_thema_id2=19200&p_thema_id3=19300&p_thema_id4=19370 (visited on 04/04/2025).

[10] C. Laufs, A. Herweg, and C. H. Antink. "Methods and evaluation of physiological measurements with acoustic stimuli-a systematic review". In: *Physiological measurement* 44.11 (2023). DOI: 10.1088/1361-6579/ad0516.

[11] G. Giannakakis, D. Grigoriadis, K. Giannakaki, et al. "Review on Psychological Stress Detection Using Biosignals". In: *IEEE Transactions on Affective Computing* 13.1 (2022), pp. 440–460. DOI: 10.1109/TAFFC.2019.2927337.

[12] J. E. Sharman, I. Tan, G. S. Stergiou, et al. "Automated 'oscillometric' blood pressure measuring devices: how they work and what they measure". In: *Journal of Human Hypertension* 37.2 (2023), pp. 93–100. ISSN: 1476-5527. DOI: 10.1038/s41371-022-00693-x.

[13] D. Zheng, J. N. Amoore, S. Mieke, et al. "How Important is the Recommended Slow Cuff Pressure Deflation Rate for Blood Pressure Measurement?" In: *Annals of Biomedical Engineering* 39.10 (2011), pp. 2584–2591. ISSN: 1573-9686. DOI: 10.1007/s10439-011-0347-9.

[14] A. J. Pappano and W. G. Wier. "9 - The Peripheral Circulation and its Control". In: *Cardiovascular Physiology (Tenth Edition)*. Ed. by A. J. Pappano and W. G. Wier. Philadelphia: Elsevier, 2013, pp. 171–194. ISBN: 978-0-323-08697-4. DOI: 10.1016/B978-0-323-08697-4.00009-5.

[15] G. Mahe, E. Comets, A. Nouni, et al. "A minimal resting time of 25 min is needed before measuring stabilized blood pressure in subjects addressed for vascular investigations". In: *Scientific Reports* 7.1 (2017), p. 12893. ISSN: 2045-2322. DOI: 10.1038/s41598-017-12775-9.

[16] G. Parati, G. S. Stergiou, R. Asmar, et al. "European Society of Hypertension guidelines for blood pressure monitoring at home: a summary report of the Second International Consensus Conference on Home Blood Pressure Monitoring". In: *Journal of Hypertension* 26.8 (2008). DOI: 10.1097/HJH.0b013e328308da66.

[17] H. Selye. “Stress without Distress”. In: *Psychopathology of Human Adaptation*. Ed. by G. Serban. Boston, MA: Springer US, 1976, pp. 137–146. ISBN: 978-1-4684-2238-2. DOI: 10.1007/978-1-4684-2238-2_9.

[18] G. Ernst, A. Franke, and P. Franzkowiak. “Stress und Stressbewältigung”. In: *Leitbegriffe der Gesundheitsförderung und Prävention. Glossar zu Konzepten, Strategien und Methoden*. Bundeszentrale für gesundheitliche Aufklärung (BZgA), 2022. DOI: 10.17623/BZGA:Q4-I118-2.0.

[19] G. S. Everly and J. M. Lating. “The Concept of Stress”. In: *A Clinical Guide to the Treatment of the Human Stress Response*. Ed. by G. S. Everly and J. M. Lating. New York, NY: Springer, 2019, pp. 3–18. ISBN: 978-1-4939-9097-9. DOI: 10.1007/978-1-4939-9098-6_1.

[20] B. Chu, K. Marwaha, T. Sanvictores, et al. “Physiology, Stress Reaction”. In: *StatPearls [Internet]*. [Updated: 2024 May 7]. [Accessed: 2025 Jan 24]. Treasure Island, FL: StatPearls Publishing, 2024. URL: https://www.ncbi.nlm.nih.gov/books/NBK541120/ (visited on 01/24/2025).

[21] G. S. Everly and J. M. Lating. “The Anatomy and Physiology of the Human Stress Response”. In: *A Clinical Guide to the Treatment of the Human Stress Response*. Ed. by G. S. Everly and J. M. Lating. New York, NY: Springer, 2019, pp. 19–56. ISBN: 978-1-4939-9097-9. DOI: 10.1007/978-1-4939-9098-6_2.

[22] C. S. Sim, J. H. Sung, S. H. Cheon, et al. “The Effects of Different Noise Types on Heart Rate Variability in Men”. In: *Yonsei Med J* 56.1 (2015), pp. 235–243. ISSN: 0513-5796. DOI: 10.3349/ymj.2015.56.1.235.

[23] A. Arjunan and R. Rajan. “Noise and brain”. In: *Physiology & behavior* 227 (2020), p. 113136. DOI: 10.1016/j.physbeh.2020.113136.

[24] G.-S. Lee, M.-L. Chen, and G.-Y. Wang. “Evoked response of heart rate variability using short-duration white noise”. In: *Autonomic Neuroscience: Basic and Clinical* 155.1 (2010), pp. 94–97. ISSN: 1566-0702. DOI: 10.1016/j.autneu.2009.12.008.

[25] K. Paunović, V. Stojanov, B. Jakovljević, et al. “Thoracic bioelectrical impedance assessment of the hemodynamic reactions to recorded road-traffic noise in young adults”. In: *Environmental Research* 129 (2014), pp. 52–58. ISSN: 0013-9351. DOI: 10.1016/j.envres.2014.01.001.

[26] E. D. Walker, A. Brammer, M. G. Cherniack, et al. “Cardiovascular and stress responses to short-term noise exposures—A panel study in healthy males”. In: *Environmental Research* 150 (2016), pp. 391–397. ISSN: 0013-9351. DOI: 10.1016/j.envres.2016.06.016.

[27] J. C. Westman and J. R. Walters. “Noise and stress: a comprehensive approach.” In: *Environmental Health Perspectives* 41 (1981), pp. 291–309. DOI: 10.1289/ehp.8141291.

[28] J. S. Yeomans and P. W. Frankland. “The acoustic startle reflex: neurons and connections”. In: *Brain Research Reviews* 21.3 (1995), pp. 301–314. ISSN: 0165-0173. DOI: 10.1016/0165-0173(96)00004-5.

[29] K. S. Kraus and B. Canlon. “Neuronal connectivity and interactions between the auditory and limbic systems. Effects of noise and tinnitus”. In: *Hearing Research* 288.1 (2012), pp. 34–46. ISSN: 0378-5955. DOI: 10.1016/j.heares.2012.02.009.

[30] P. Saha, M. U. Monir, S. M. Sarkar, et al. “18 - Noise pollution from oil, gas, and petrochemical industries”. In: *Crises in Oil, Gas and Petrochemical Industries*. Ed. by M. R. Rahimpour, B. Omidvar, N. A. Shirazi, et al. Elsevier, 2023, pp. 419–434. ISBN: 978-0-323-95154-8. DOI: 10.1016/B978-0-323-95154-8.00012-8.

[31] E. Murphy and E. A. King. “Chapter 2 - Principles of Environmental Noise”. In: *Environmental Noise Pollution (Second Edition)*. Ed. by E. Murphy and E. A. King. Second Edition. Boston: Elsevier, 2022, pp. 9–51. ISBN: 978-0-12-820100-8. DOI: 10.1016/B978-0-12-820100-8.00002-6.

[32] R. F. Weiner and R. A. Matthews. “Chapter 22 - Noise Pollution”. In: *Environmental Engineering (Fourth Edition)*. Fourth Edition. Burlington: Butterworth-Heinemann, 2003, pp. 423–446. ISBN: 978-0-7506-7294-8. DOI: 10.1016/B978-075067294-8/50022-1.

[33] L. K. McCorry. “Physiology of the autonomic nervous system”. In: *American Journal of Pharmaceutical Education* 71.4 (2007). ISSN: 0002-9459. DOI: 10.5688/aj710478.

[34] J. P. Henry. “Biological Basis of the Stress Response”. In: *Physiology* 8.2 (1993), pp. 69–73. ISSN: 1548-9213. DOI: 10.1152/physiologyonline.1993.8.2.69.

[35] J. A. Sheng, N. J. Bales, S. A. Myers, et al. “The Hypothalamic-Pituitary-Adrenal Axis: Development, Programming Actions of Hormones, and Maternal-Fetal Interactions”. In: *Frontiers in Behavioral Neuroscience* 14 (2021). ISSN: 1662-5153. DOI: 10.3389/fnbeh.2020.601939.

[36] A. Daiber, S. Kröller-Schön, K. Frenis, et al. “Environmental noise induces the release of stress hormones and inflammatory signaling molecules leading to oxidative stress and vascular dysfunction-Signatures of the internal exposome”. In: *BioFactors (Oxford, England)* 45.4 (2019), pp. 495–506. DOI: 10.1002/biof.1506.

[37] B. P. Imholz, W. Wieling, G. A. van Montfrans, et al. “Fifteen years experience with finger arterial pressure monitoring: assessment of the technology”. In: *Cardiovascular Research* 38.3 (1998), pp. 605–616. DOI: 10.1016/s0008-6363(98)00067-4.

[38] J. Pneaz. “Photoelectric measurement of blood pressure, volume and flow in the finge”. In: *Digest of the 10th International Conference on Medical and Biological Engineering*. 1973, p. 104.

[39] R. D. Boehmer. “Continuous, real-time, noninvasive monitor of blood pressure: Penaz methodology applied to the finger”. In: *Journal of clinical monitoring* 3.4 (1987), pp. 282–287. ISSN: 0748-1977. DOI: 10.1007/BF03337384.

[40] F. M. Systems. *Physiocal - finapres.com*. URL: https://www.finapres.com/technologies/physiocal (visited on 03/12/2025).

[41] I. Guelen, B. E. Westerhof, G. L. Van der Saar, et al. “Validation of brachial artery pressure reconstruction from finger arterial pressure”. In: *Journal of Hypertension* 26.26 (2008), pp. 1321–1327.

[42] International Organization for Standardization. *Non-invasive sphygmomanometers – Part 2: Clinical investigation of automated measurement type*. ISO 81060-2: 2013. Geneva, CH, 2013-04-18. URL: https://www.iso.org/standard/57977.html (visited on 03/12/2025).

[43] H. Critchley and Y. Nagai. “Electrodermal Activity (EDA)”. In: *Encyclopedia of Behavioral Medicine*. Ed. by M. D. Gellman. Cham: Springer International Publishing, 2020, pp. 741–744. ISBN: 978-3-030-39903-0. DOI: 10.1007/978-3-030-39903-0_13.

[44] N. Pop-Jordanova and J. Pop-Jordanov. “Electrodermal Activity and Stress Assessment”. In: *Prilozi (Makedonska akademija na naukite i umetnostite. Oddelenie za medicinski nauki)* 41.2 (2020), pp. 5–15. DOI: 10.2478/prilozi-2020-0028.

[45] M. Pedersen. *Galvanic skin response (GSR): The Complete Pocket Guide*. Feb. 2020. URL: https://imotions.com/blog/learning/research-fundamentals/galvanic-skin-response/ (visited on 03/18/2025).

[46] M. Birket-Smith, N. Hasle, and H. H. Jensen. "Electrodermal activity in anxiety disorders". In: *Acta Psychiatrica Scandinavica* 88.5 (1993), pp. 350–355. DOI: 10.1111/j.1600-0447.1993.tb03471.x.

[47] K. M. Williams, W. G. Iacono, and R. A. Remick. "Electrodermal activity among subtypes of depression". In: *Biological Psychiatry* 20.2 (1985), pp. 158–162. ISSN: 0006-3223. DOI: 10.1016/0006-3223(85)90075-7.

[48] B. O. Rothbaum, M. J. Kozak, E. B. Foa, et al. "Posttraumatic stress disorder in rape victims: Autonomic habituation to auditory stimuli". In: *Journal of Traumatic Stress* 14.2 (2001), pp. 283–293. DOI: 10.1023/A:1011160800958.

[49] Y. Nagai, L. H. Goldstein, P. B. Fenwick, et al. "Clinical efficacy of galvanic skin response biofeedback training in reducing seizures in adult epilepsy: a preliminary randomized controlled study". In: *Epilepsy & Behavior* 5.2 (2004), pp. 216–223. ISSN: 1525-5050. DOI: 10.1016/j.yebeh.2003.12.003.

[50] G. Gilmour, S. Porcelli, V. Bertaina-Anglade, et al. "Relating constructs of attention and working memory to social withdrawal in Alzheimer's disease and schizophrenia: issues regarding paradigm selection". In: *Neuroscience & Biobehavioral Reviews* 97 (2019), pp. 47–69. ISSN: 0149-7634. DOI: 10.1016/j.neubiorev.2018.09.025.

[51] C. Coulacoglou and D. H. Saklofske. "Chapter 5 - Executive Function, Theory of Mind, and Adaptive Behavior". In: *Psychometrics and Psychological Assessment*. Ed. by C. Coulacoglou and D. H. Saklofske. San Diego: Academic Press, 2017, pp. 91–130. ISBN: 978-0-12-802219-1. DOI: 10.1016/B978-0-12-802219-1.00005-5.

[52] N. S. de Boer, R. S. Schluter, J. G. Daams, et al. "The effect of non-invasive brain stimulation on executive functioning in healthy controls: A systematic review and meta-analysis". In: *Neuroscience & Biobehavioral Reviews* 125 (2021), pp. 122–147. ISSN: 0149-7634. DOI: 10.1016/j.neubiorev.2021.01.013.

[53] M. J. Kane, A. R. A. Conway, T. K. Miura, et al. "Working memory, attention control, and the n-back task: A question of construct validity". In: *Journal of Experimental Psychology: Learning, Memory, and Cognition* 33.3 (2007). DOI: 10.1037/0278-7393.33.3.615.

[54] W. K. Kirchner. “Age differences in short-term retention of rapidly changing information”. In: *Journal of Experimental Psychology* 55.4 (1958), pp. 352–358. ISSN: 0022-1015. DOI: 10.1037/h0043688.

[55] L. Padilla, S. C. Castro, and H. Hosseinpour. “Chapter Seven - A review of uncertainty visualization errors: Working memory as an explanatory theory”. In: *The Psychology of Learning and Motivation*. Ed. by K. D. Federmeier. Vol. 74. Psychology of Learning and Motivation. Academic Press, 2021, pp. 275–315. DOI: 10.1016/bs.plm.2021.03.001.

[56] L. E. Savidge and K. L. Bales. “Possible effects of pair bonds on general cognition: Evidence from shared roles of dopamine”. In: *Neuroscience & Biobehavioral Reviews* 152 (2023), p. 105317. ISSN: 0149-7634. DOI: 10.1016/j.neubiorev.2023.105317.

[57] B. J. Baars and N. M. Gage. “Chapter 10 - Thinking and problem solving”. In: *Cognition, Brain, and Consciousness (Second Edition)*. Ed. by Bernard J. Baars and Nicole M. Gage. Second Edition. London: Academic Press, 2010, pp. 344–369. ISBN: 978-0-12-375070-9. DOI: 10.1016/B978-0-12-375070-9.00010-3.

[58] J. M. Fuster. “Chapter 1 - The prefrontal cortex in the neurology clinic”. In: *The Frontal Lobes*. Ed. by M. D’Esposito and J. H. Grafman. Vol. 163. Handbook of Clinical Neurology. Elsevier, 2019, pp. 3–15. DOI: 10.1016/B978-0-12-804281-6.00001-X.

[59] S.-Y. Lu, C.-L. Lee, K.-Y. Lin, et al. “The acute effect of exposure to noise on cardiovascular parameters in young adults”. In: *Journal of Occupational Health* 60.4 (2018), pp. 289–297. ISSN: 1341-9145. DOI: 10.1539/joh.2017-0225-OA.

[60] J. Kristiansen, L. Mathiesen, P. K. Nielsen, et al. “Stress reactions to cognitively demanding tasks and open-plan office noise”. In: *International Archives of Occupational and Environmental Health* 82.5 (2009), pp. 631–641. ISSN: 1432-1246. DOI: 10.1007/s00420-008-0367-4.

[61] E. Gallasch, R. B. Raggam, M. Cik, et al. “Road and Rail Traffic Noise Induce Comparable Extra-aural Effects as Revealed During a Short-term Memory Test”. In: *Noise & Health* 18.83 (2016), pp. 206–213. ISSN: 1463-1741. DOI: 10.4103/1463-1741.189243.

[62] K. Paunović, B. Jakovljević, and V. Stojanov. “The timeline of blood pressure changes and hemodynamic responses during an experimental noise exposure”. In: *Environmental Research* 163 (2018), pp. 249–262. ISSN: 0013-9351. DOI: 10.1016/j.envres.2018.01.048.

[63] Y. Suko, K. Saito, N. Takayama, et al. “Effect of Faint Road Traffic Noise Mixed in Birdsong on the Perceived Restorativeness and Listeners’ Physiological Response: An Exploratory Study”. In: *International Journal of Environmental Research and Public Health* 16.24 (2019). ISSN: 1660-4601. DOI: 10.3390/ijerph16244985.

[64] J. J. Alvarsson, S. Wiens, and M. E. Nilsson. “Stress Recovery during Exposure to Nature Sound and Environmental Noise”. In: *International Journal of Environmental Research and Public Health* 7.3 (2010), pp. 1036–1046. ISSN: 1660-4601. DOI: 10.3390/ijerph7031036.

[65] M. Trimmel, J. Atzlsdorfer, N. Tupy, et al. “Effects of low intensity noise from aircraft or from neighbourhood on cognitive learning and electrophysiological stress responses”. In: *International Journal of Hygiene and Environmental Health* 215.6 (2012), pp. 547–554. ISSN: 1438-4639. DOI: 10.1016/j.ijheh.2011.12.007.

[66] G. Iadarola, A. Poli, and S. Spinsante. “Analysis of Galvanic Skin Response to Acoustic Stimuli by Wearable Devices”. In: *2021 IEEE International Symposium on Medical Measurements and Applications (MeMeA)*. 2021, pp. 1–6. DOI: 10.1109/MeMeA52024.2021.9478673.

[67] WEVA TV. *“Highway Heavy Traffic Sound Effect”*. Jan. 31, 2017. URL: https://youtu.be/YF677534Ayssi=rVuUTE5WoXhp7pzv&t=60 (visited on 02/10/2025).

[68] C. Laufs, A. Herweg, and C. H. Antink. “Listening test design for synchronous acquisition of physiological data and cognitive performance in disturbing noise”. In: *Fortschritte der Akustik - DAGA 2023*. Ed. by Deutsche Gesellschaft für Akustik e.V. Berlin, 2023, pp. 1202–1205. ISBN: 978-3-939296-21-8.

[69] J. W. Peirce. “PsychoPy — Psychophysics software in Python”. In: *Journal of Neuroscience Methods* 162.1-2 (2007), pp. 8–13. ISSN: 0165-0270. DOI: 10.1016/j.jneumeth.2006.11.017.

[70] J. W. Peirce, J. R. Gray, S. Simpson, et al. “PsychoPy2: Experiments in behavior made easy”. In: *Behavior Research Methods* 51.1 (2019), pp. 195–203. ISSN: 1554-3528. DOI: 10.3758/s13428-018-01193-y.

[71] C. Clark, T. Gjestland, L. Lavia, et al. “Assessing community noise annoyance: A review of two decades of the international technical specification ISO/TS 15666:2003”. In: *JOURNAL OF THE ACOUSTICAL SOCIETY OF AMERICA* 150.5 (2021), pp. 3362–3373. ISSN: 0001-4966. DOI: 10.1121/10.0006967.

[72] International Organization for Standardization. *Acoustics – Assessment of noise annoyance by means of social and socio-acoustic surveys*. ISO/TS 15666:2021-05. Geneva, CH, 2021-05-18. URL: https://www.iso.org/standard/74048.html (visited on 02/11/2025).

[73] T. Gjestland. “Standardized General-Purpose Noise Reaction Questions”. In: *12th ICBEN Congress in Noise as a Public Health Problem*. ID 2449. Zurich, 2017. URL: https://www.icben.org/2017/#6 (visited on 02/09/2025).

[74] N. D. Weinstein. “Individual differences in reactions to noise: A longitudinal study in a college dormitory”. In: *JOURNAL OF APPLIED PSYCHOLOGY* 63.4 (1978), pp. 458–466. ISSN: 0021-9010. DOI: 10.1037/0021-9010.63.4.458.

[75] D. L. Worthington. “Weinstein Noise Sensitivity Scale (WNSS)”. In: *The Sourcebook of Listening Research*. Ed. by D. L. Worthington and G. Bodie. Hoboken, NJ: John Wiley & Sons Inc, 2018, pp. 475–481. ISBN: 9781119102991. DOI: 10.1002/9781119102991.ch52.

[76] K. Zimmer and W. Ellermeier. “A German version of Weinstein’s noise sensitivity scale”. In: *Zeitschrift für Lärmbekämpfung* 44.4 (1997), pp. 107–110.

[77] C. D. Spielberger. *State-Trait Anxiety Inventory for Adults*. 1983. DOI: 10.1037/t06496-000.

[78] P. Skapinakis. “Spielberger State-Trait Anxiety Inventory”. In: *Encyclopedia of Quality of Life and Well-Being Research*. Ed. by A. C. Michalos. Dordrecht: Springer, 2014, pp. 6261–6264. ISBN: 978-94-007-0753-5. DOI: 10.1007/978-94-007-0753-5_2825.

[79] J. Grimm. “State-Trait-Anxiety Inventory nach Spielberger. Deutsche Lang- und Kurzversion.” In: *Methodenforum der Universität Wien: MF-Working Paper 2009/02* (2009). URL: https://empcom.univie.ac.at/methodenarchiv/ (visited on 03/22/2025).

[80] O. Kayikcioglu, S. Bilgin, G. Seymenoglu, et al. “State and Trait Anxiety Scores of Patients Receiving Intravitreal Injections”. In: *Biomedicine Hub* 2.2 (2017), pp. 1–5. DOI: 10.1159/000478993.

[81] P. Virtanen, R. Gommers, T. E. Oliphant, et al. “SciPy 1.0: Fundamental Algorithms for Scientific Computing in Python”. In: *Nature Methods* 17 (2020), pp. 261–272. DOI: 10.1038/s41592-019-0686-2.

[82] F. Shaffer, R. McCraty, and C. L. Zerr. “A healthy heart is not a metronome: an integrative review of the heart’s anatomy and heart rate variability”. In: *Frontiers in Psychology* 5 (2014). ISSN: 1664-1078. DOI: 10.3389/fpsyg.2014.01040.

[83] T. Thong, K. Li, J. McNames, et al. “Accuracy of ultra-short heart rate variability measures”. In: *Proceedings of the 25th Annual International Conference of the IEEE Engineering in Medicine and Biology Society (IEEE Cat. No.03CH37439)*. Vol. 3. 2003, 2424–2427 Vol.3. DOI: 10.1109/IEMBS.2003.1280405.

[84] M. L. Munoz, A. van Roon, H. Riese, et al. “Validity of (Ultra-)Short Recordings for Heart Rate Variability Measurements”. In: *PLOS ONE* 10.9 (2015), pp. 1–15. DOI: 10.1371/journal.pone.0138921.

[85] The pandas development team. *pandas-dev/pandas: Pandas*. Version 2024-09-20. 2020. DOI: 10.5281/zenodo.3509134.

[86] S. A. H. Aqajari, E. K. Naeini, M. A. Mehrabadi, et al. “pyEDA: An Open-Source Python Toolkit for Pre-processing and Feature Extraction of Electrodermal Activity”. In: *Procedia Computer Science* 184 (2021), pp. 99–106.

[87] D. Makowski, T. Pham, Z. J. Lau, et al. “NeuroKit2: A Python toolbox for neurophysiological signal processing”. In: *Behavior Research Methods* 53.4 (2021), pp. 1689–1696. DOI: 10.3758/s13428-020-01516-y.

[88] S. W. Looney and J. L. Hagan. “2 - Statistical Methods for Assessing Biomarkers and Analyzing Biomarker Data”. In: *Essential Statistical Methods for Medical Statistics*. Ed. by C. Rao, J. Miller, and D. Rao. Boston: North-Holland, 2011, pp. 27–65. DOI: 10.1016/B978-0-444-53737-9.50005-0.

[89] Y. Hasija. “Chapter 3 - Statistical methods in bioinformatics”. In: *All About Bioinformatics*. Ed. by Y. Hasija. Academic Press, 2023, pp. 43–75. DOI: 10.1016/B978-0-443-15250-4.00009-5.

[90] S. W. Scheff. “Chapter 8 - Nonparametric Statistics”. In: *Fundamental Statistical Principles for the Neurobiologist*. Ed. by S. W. Scheff. Academic Press, 2016, pp. 157–182. ISBN: 978-0-12-804753-8. DOI: 10.1016/B978-0-12-804753-8.00008-7.

[91] A. M. Abbasi, M. Motamedzade, M. Aliabadi, et al. “Combined effects of noise and air temperature on human neurophysiological responses in a simulated indoor environment”. In: *Applied ergonomics* 88, 103189 (2020). DOI: 10.1016/j.apergo.2020.103189.

[92] A. G. Airij, R. Sudirman, and U. U. Sheikh. “GSM and GPS Based Real-Time Remote Physiological Signals Monitoring and Stress Levels Classification”. In: *2018 2nd International Conference on BioSignal Analysis, Processing and Systems (ICBAPS)*. 2018, pp. 130–135. DOI: 10.1109/ICBAPS.2018.8527406.

[93] A. S. Anusha, J. Jose, S. P. Preejith, et al. “Physiological signal based work stress detection using unobtrusive sensors”. In: *Biomedical Physics & Engineering Express* 4.6, 065001 (2018). DOI: 10.1088/2057-1976/aadbd4.

[94] S. Cecchi, A. Piersanti, A. Poli, et al. “Physical Stimuli and Emotions: EDA Features Analysis from a Wrist-Worn Measurement Sensor”. In: *2020 IEEE 25th International Workshop on Computer Aided Modeling and Design of Communication Links and Networks (CAMAD)*. 2020, pp. 1–6. DOI: 10.1109/CAMAD50429.2020.9209307.

[95] W. Ellermeier, F. Kattner, E. Klippenstein, et al. “Short-Term Noise Annoyance and Electrodermal Response as a Function of Sound-Pressure Level, Cognitive Task Load, and Noise Sensitivity”. In: *Noise & Health* 22.105 (2020), pp. 46–55. ISSN: 1463-1741. DOI: 10.4103/nah.NAH_47_19.

[96] D. Giakoumis, A. Drosou, P. Cipresso, et al. “Using Activity-Related Behavioural Features towards More Effective Automatic Stress Detection”. In: *PLOS ONE* 7.9 (2012), pp. 1–16. DOI: 10.1371/journal.pone.0043571.

[97] A. Greco, G. Valenza, L. Citi, et al. “Arousal and Valence Recognition of Affective Sounds Based on Electrodermal Activity”. In: *IEEE Sensors Journal* 17.3 (2017), pp. 716–725. ISSN: 1530-437X. DOI: 10.1109/JSEN.2016.2623677.

[98] N. P. Giuliani, C. J. Brown, and Y.-H. Wu. “Comparisons of the Sensitivity and Reliability of Multiple Measures of Listening Effort”. In: *Ear and hearing* 42.2 (2021), pp. 465–474. DOI: 10.1097/AUD.0000000000000950.

[99] S. Han, R. Zhu, and Y. Ku. “Background white noise and speech facilitate visual working memory”. In: *The European journal of neuroscience* 54.7 (2021), pp. 6487–6496. DOI: 10.1111/ejn.15455.

[100] J. A. Healey and R. W. Picard. “Detecting Stress During Real-World Driving Tasks Using Physiological Sensors”. In: *IEEE Transactions on Intelligent Transportation Systems* 6.2 (2005), pp. 156–166. ISSN: 1524-9050. DOI: 10.1109/TITS.2005.848368.

[101] M. Hedblom, B. Gunnarsson, M. Schaefer, et al. “Sounds of Nature in the City: No Evidence of Bird Song Improving Stress Recovery”. In: *International Journal of Environmental Research and Public Health* 16.8 (2019). ISSN: 1660-4601. DOI: 10.3390/ijerph16081390.

[102] I. Holube, K. Haeder, C. Imbery, et al. “Subjective Listening Effort and Electrodermal Activity in Listening Situations with Reverberation and Noise”. In: *Trends in hearing* 20 (2016). DOI: 10.1177/2331216516667734.

[103] D. Hossain, S. M. Salimullah, R. Mahmudi, et al. “Cognitive Load Measurement Using Galvanic Skin Response for Listening Tasks”. In: *2019 4th International Conference on Electrical Information and Communication Technology (EICT)*. IEEE, 12/20/2019 - 12/22/2019, pp. 1–4. ISBN: 978-1-7281-6040-5. DOI: 10.1109/EICT48899.2019.9068854.

[104] S. A. Hosseini and M. B. Naghibi-Sistani. “Classification of Emotional Stress Using Brain Activity”. In: *Applied Biomedical Engineering*. Ed. by G. D. Gargiulo and A. McEwan. IntechOpen, 2011. Chap. 14. DOI: 10.5772/18294.

[105] D. Huysmans, E. Smets, W. de Raedt, et al. “Unsupervised Learning for Mental Stress Detection”. In: *Proceedings of the 11th International Joint Conference on Biomedical Engineering Systems and Technologies*. SCITEPRESS - Science and Technology Publications, 2018, pp. 26–35. ISBN: 978-989-758-307-0. DOI: 10.5220/0006541100260035.

[106] C. D. Katsis, N. Katertsidis, G. Ganiatsas, et al. “Toward Emotion Recognition in Car-Racing Drivers: A Biosignal Processing Approach”. In: *IEEE Transactions on Systems, Man, and Cybernetics - Part A: Systems and Humans* 38.3 (2008), pp. 502–512. DOI: 10.1109/TSMCA.2008.918624.

[107] M. Khalilzadeh, M. Homam, S. A. Hosseini, et al. “Qualitative and quantitative evaluation of brain activity in emotional stress”. In: *Iranian Journal of Neurology* 8 (Jan. 2010), pp. 605–618. URL: https://www.researchgate.net/publication/265379990_Qualitative_and_quantitative_evaluation_of_brain_activity_in_emotional_stress.

[108] K. H. Kim, S. W. Bang, and S. R. Kim. “Emotion recognition system using short-term monitoring of physiological signals”. In: *Medical & Biological Engineering & Computing* 42.3 (2004), pp. 419–427. ISSN: 0140-0118. DOI: 10.1007/BF02344719.

[109] H. Kurniawan, A. V. Maslov, and M. Pechenizkiy. “Stress detection from speech and Galvanic Skin Response signals”. In: *Proceedings of the 26th IEEE International Symposium on Computer-Based Medical Systems*. 2013, pp. 209–214. DOI: 10.1109/CBMS.2013.6627790.

[110] M.-h. Lee, G. Yang, H.-K. Lee, et al. “Development stress monitoring system based on personal digital assistant (PDA)”. In: *The 26th Annual International Conference of the IEEE Engineering in Medicine and Biology Society*. 2004, pp. 2364–2367. DOI: 10.1109/IEMBS.2004.1403685.

[111] J. Li, L. Maffei, A. Pascale, et al. “Effects of spatialized water-sound sequences for traffic noise masking on brain activities”. In: *The Journal of the Acoustical Society of America* 152.1 (2022), p. 172. DOI: 10.1121/10.0012222.

[112] C. Maaoui, F. Bousefsaf, and A. Pruski. “AUTOMATIC HUMAN STRESS DETECTION BASED ON WEBCAM PHOTOPLETHYSMOGRAPHIC SIGNALS”. In: *Journal of Mechanics in Medicine and Biology* 16.04 (2016), p. 1650039. ISSN: 0219-5194. DOI: 10.1142/S0219519416500391.

[113] C. L. Mackersie and N. Calderon-Moultrie. “Autonomic Nervous System Reactivity During Speech Repetition Tasks: Heart Rate Variability and Skin Conductance”. In: *Ear and Hearing* 37 Suppl 1 (2016), 118S–25S. DOI: 10.1097/AUD.0000000000000305.

[114] C. L. Mackersie and H. Cones. “Subjective and psychophysiological indexes of listening effort in a competing-talker task”. In: *Journal of the American Academy of Audiology* 22.2 (2011), pp. 113–122. ISSN: 1050-0545. DOI: 10.3766/jaaa.22.2.6.

[115] D. McDuff, S. Gontarek, and R. Picard. “Remote measurement of cognitive stress via heart rate variability”. In: *2014 36th Annual International Conference of the IEEE Engineering in Medicine and Biology Society*. 2014, pp. 2957–2960. DOI: 10.1109/EMBC.2014.6944243.

[116] D. J. McDuff, J. Hernandez, S. Gontarek, et al. “COGCAM: Contact-free Measurement of Cognitive Stress During Computer Tasks with a Digital Camera”. In: *Proceedings of the 2016 CHI Conference on Human Factors in Computing Systems*.

Association for Computing Machinery, 2016, pp. 4000–4004. DOI: 10.1145/2858036.2858247.

[117] J. Minguillon, E. Perez, M. A. Lopez-Gordo, et al. “Portable System for Real-Time Detection of Stress Level”. In: *Sensors (Basel, Switzerland)* 18.8 (2018). DOI: 10.3390/s18082504.

[118] M. Pedrotti, M. A. Mirzaei, A. Tedesco, et al. “Automatic Stress Classification With Pupil Diameter Analysis”. In: *International Journal of Human–Computer Interaction* 30.3 (2014), pp. 220–236. DOI: 10.1080/10447318.2013.848320.

[119] J. Radun, H. Maula, V. Rajala, et al. “Speech is special: The stress effects of speech, noise, and silence during tasks requiring concentration”. In: *Indoor Air* 31.1 (2021), pp. 264–274. DOI: 10.1111/ina.12733.

[120] P. Ren, A. Barreto, Y. Gao, et al. “Affective Assessment by Digital Processing of the Pupil Diameter”. In: *IEEE Transactions on Affective Computing* 4.1 (2013), pp. 2–14. DOI: 10.1109/T-AFFC.2012.25.

[121] M. Sadeghian, S. Yazdanirad, S. M. Mousavi, et al. “Effect of tonal noise and task difficulty on electroencephalography and cognitive performance”. In: *International journal of occupational safety and ergonomics : JOSE* 28.3 (2022), pp. 1353–1361. DOI: 10.1080/10803548.2021.1901432.

[122] G. A. Salzman, H. El, and T. P. Chang. “Impact of Environmental Noise Levels on Endotracheal Intubation Performance Among Pediatric Emergency Providers: A Simulation Study”. In: *Pediatric emergency care* 37.12 (2021), pp. 944–949. DOI: 10.1097/PEC.0000000000001831.

[123] C. Setz, B. Arnrich, J. Schumm, et al. “Discriminating Stress From Cognitive Load Using a Wearable EDA Device”. In: *IEEE Transactions on Information Technology in Biomedicine* 14.2 (2010), pp. 410–417. DOI: 10.1109/TITB.2009.2036164.

[124] N. Sharma and T. Gedeon. “Modeling a stress signal”. In: *Applied Soft Computing* 14 (2014), pp. 53–61. ISSN: 1568-4946. DOI: 10.1016/j.asoc.2013.09.019.

[125] G. Vila, C. Godin, S. Charbonnier, et al. “Pressure-Specific Feature Selection for Acute Stress Detection From Physiological Recordings”. In: *2018 IEEE International Conference on Systems, Man, and Cybernetics (SMC)*. 2018, pp. 2341–2346. DOI: 10.1109/SMC.2018.00402.

[126] J. Wijsman, B. Grundlehner, H. Liu, et al. “Towards mental stress detection using wearable physiological sensors”. In: *Annual International Conference of the IEEE Engineering in Medicine and Biology Society* (2011), pp. 1798–1801. DOI: 10.1109/IEMBS.2011.6090512.

[127] J. Wijsman, B. Grundlehner, H. Liu, et al. “Wearable Physiological Sensors Reflect Mental Stress State in Office-Like Situations”. In: *2013 Humaine Association Conference on Affective Computing and Intelligent Interaction*. 2013, pp. 600–605. DOI: 10.1109/ACII.2013.105.

[128] J. Zhai and A. Barreto. “Stress Detection in Computer Users Based on Digital Signal Processing of Noninvasive Physiological Variables”. In: *2006 International Conference of the IEEE Engineering in Medicine and Biology Society*. 2006, pp. 1355–1358. DOI: 10.1109/IEMBS.2006.259421.

[129] F. Zhou, X. Qu, J. Jiao, et al. “Emotion Prediction from Physiological Signals: A Comparison Study Between Visual and Auditory Elicitors”. In: *Interacting with Computers* 26.3 (2014), pp. 285–302. ISSN: 0953-5438. DOI: 10.1093/iwc/iwt039.

Glossary

ISO/TS 15666:2021-05 Technical specification by the International Organization for Standardization (ISO) dealing with the assessment of noise annoyance by means of social and socio-acoustic surveys [72]. 20, 37, 50

KISMED **K**ünstliche **I**ntelligente **S**ysteme der **Med**izin ist der führende Arbeitskreis an der TU Darmstadt der sich mit mit dem Thema KI in der Medizin befasst. 18

PsychoPy An open source software package written in Python primarily for use in neuroscience and experimental psychology research. 18, 27, 30

A. Appendix - Materials

A.1. Similar Publications

Table A.1.: Table with similar publications. These are papers in the realm of stress detection that used at least one of the methods included in this work.

Publications	EDA	BP	Similar Noise Type	N-Back Task
Abbasi et al. [91]				✔
Airij et al. [92]	✔			
Alvarsson et al. [64]	✔			
Anusha et al. [93]	✔			
Cecchi et al. [94]	✔			
Ellermeier et al. [95]	✔		✔	
Gallasch et al. [61]		✔	✔	
Giakoumis et al. [96]	✔			
Greco et al. [97]	✔			
Giuliani et al. [98]	✔			
Han et al. [99]	✔			
Healey et al. [100]	✔			
Hedblom et al. [101]	✔			
Holube et al. [102]	✔			
Hossain et al. [103]	✔			
Hosseini et al. [104]	✔			
Huysmans et al. [105]	✔			
Iadarola et al. [66]	✔			
Katsis et al. [106]	✔			

Continued on next page

Publications	EDA	BP	Similar Noise Type	N-Back Task
Khalilzadeh et al. [107]	✔			
Kim et al. [108]	✔			
Kristiansen et al. [60]		✔		
Kurniawan et al. [109]	✔			
Lee et al. [110]	✔			
Lee et al. [24]		✔	✔	
Li et al. [111]			✔	
Lu et al. [59]		✔		
Maaoui et al. [112]				
Mackersie et al. [113]	✔			
Mackersie et al. [114]	✔			
McDuff et al. [115]	✔			
McDuff et al. [116]				
Minguillon et al. [117]	✔			
Paunović et al. [62]		✔	✔	
Pedrotti et al. [118]	✔			
Radun et al. [119]				✔
Ren et al. [120]	✔			
Sadeghian et al. [121]				✔
Salzman et al. [122]				
Setz et al. [123]	✔			
Sharma et al. [124]	✔			
Sim et al. [22]		✔	✔	
Suko et al. [63]	✔			
Trimmel et al. [65]	✔			
Vila et al. [125]	✔			
Walker et al. [26]		✔		
Wijsman et al. [126]	✔			
Wijsman et al. [127]	✔			
Zhai et al. [128]	✔			
Zhou et al. [129]	✔			

A.2. Step-By-Step Instructions

1. Turn on all measuring devices, perform the zeroing of the HCU and reset the Finapres system time to be synchronised with the laptop. Connect the Ag/AgCl electrode to the EDA sensor and initialise the *Bluetooth*® connection between the laptop and the acquisition hub.
2. Welcome the participant, offer them a glass of water, and ask them to turn off all device notifications. Then, ask them to remove their jacket, hoodie, and all hand/arm jewellery. Ensure no pre-existing cardiovascular disease or hearing impairments are known.
3. Inform the participant about the scope of the experiment, the data gathered, and the study's goal. Make it clear that the experiment can be stopped at any time if the participant feels unwell. Their data is gathered anonymously and can only be discarded during and immediately after the experiment.
4. Have the participant answer the questionnaire up to 4.1 (including). Then, using the information on the first page of the questionnaire, set up the Finapres measurement.
5. Connect the *Finapres* cuff, the EDA sensors on the left hand, and the arm cuff on the right arm. Explain the sequence of the test phases and the instructions for the 2-back task.
6. Explain that a calibration period of about 2.5 min is needed for the BP device to be accurate and that during this time, the arm cuff will be inflated. (During this time, recording of EDA and BP is started, and the *PsychoPy* test is set up.)
7. After the brachial calibration is finished, the experimenter gives the signal to start the test by pressing the space bar.
8. Stop the recording of both devices approx. 10 s after the test is finished to ensure all data is gathered correctly. Ask the participant to answer the next page of the questionnaire. Meanwhile, export all the recordings.
9. Repeat steps 5, 6 and 7 twice with different settings for the *PsychoPy* test, ensuring not to forget the questionnaire and the data export.
10. Remove all sensors from the participant and disinfect them.

A.3. Questionaire

Questionnaire

Continuous Measurement of Finger-Arterial Blood Pressure for Evaluating Noise induced Stress

KIS*MED AI Systems in Medicine

Date: _____
Exp. ID: _____
Exp.Type: _____

TECHNISCHE UNIVERSITÄT DARMSTADT

Experiment ID.: _____

1. Persönliche Daten *(personal data)*

Geschlecht *(sex)*	☐ m *(m)*	☐ w *(f)*	☐ d *(nb)*
Alter *(age)*	_____ Jahre *(years)*		
Gewicht *(weigth)*	_____ kg (±0,5 kg)		
Körpergröße *(height)*	_____ cm		
Raucher:in *(smoker)*	☐ Ja *(yes)*	☐ Nein *(no)*	☐ k. A. *(N/A)*

2. Noise Annoyance (ISO/TS 15666)

Wenn Sie einmal an die letzten 12 Monate denken, wie stark haben Sie sich durch Verkehrslärm insgesamt gestört oder belästigt gefühlt?
Thinking about the last 12 months, how much does road traffic noise bother, disturb, or annoy you?

☐ Überhaupt nicht *(not at all)*

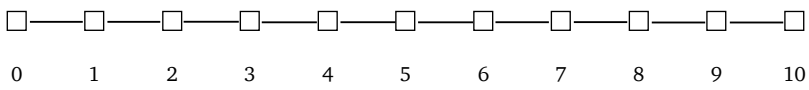
☐ Etwas *(slightly)*

☐ Mittelmäßig *(moderately)*

☐ Stark *(very)*

☐ Äußerst *(extremely)*

Es folgt eine Messlatte von Null bis Zehn, auf der Sie angeben können, wie sehr Sie Verkehrslärm insgesamt gestört oder belästigt hat. Wenn Sie sich äußerst gestört oder belästigt fühlten, wählen Sie die Zehn, wenn Sie sich überhaupt nicht gestört oder belästigt fühlten, geben Sie bitte die Null an, und wenn Sie irgendwo dazwischen liegen, wählen Sie eine Zahl zwischen Null und Zehn.
Wenn Sie nun an die letzten 12 Monate denken, welche Zahl zwischen Null und Zehn gibt am besten an, wie stark Sie sich durch Verkehrslärm insgesamt gestört oder belästigt fühlten?
Next is a zero to ten opinion scale for how much road traffic noise bothers, disturbs or annoys you. If you are not at all annoyed choose zero, if you are extremely annoyed choose ten, if you are somewhere in between choose a number between zero and ten.
Thinking about the last 12 months, what number from zero to ten best shows how much you are bothered, disturbed, or annoyed by road traffic noise?



Experiment ID.: _____

3. Weinstein Noise Sensitivity Scale

Nun folgt eine Reihe von Aussagen bezüglich individueller Reaktionen auf Lärm.
Nachdem Sie die Aussagen gelesen haben, kreuzen sie bitte das Kästchen an, welche ihr Level an Zustimmung widerspiegelt. Nutzen Sie die gegebene Skala:

Below are a number of statements addressing individual reactions to noise. After reading each statement, please circle the number that best represents your level of agreement with the statement. For each item, please use the following scale:

Stimme gar nicht zu *(strongly disagree)* 1 2 3 4 5 6 Stimme sehr zu *(strongly agree)*

	FRAGEN ***(QUESTIONS)***	**1**	**2**	**3**	**4**	**5**	**6**
1.	Es würde mir nichts ausmachen, an einer lauten Straße zu wohnen, wenn meine Wohnung schön wäre. *I wouldn't mind living on a noisy street if the apartment I had was nice. **	☐	☐	☐	☐	☐	☐
2.	Mir fällt Lärm heutzutage mehr auf als früher. *I am more aware of noise than I used to be.*	☐	☐	☐	☐	☐	☐
3.	Es sollte niemanden groß stören, wenn jemand ab und zu eine Stereoanlage voll aufdreht. *No one should mind much if someone turns up his or her stereo full blast once in a while. **	☐	☐	☐	☐	☐	☐
4.	Im Kino stört mich Flüstern und Rascheln von Bonbonpapier. *At movies, whispering and crinkling candy wrappers disturb me.*	☐	☐	☐	☐	☐	☐
5.	Ich werde leicht durch Lärm geweckt. *I am easily awakened by noise.*	☐	☐	☐	☐	☐	☐
6.	Wenn es an meinem Arbeitsplatz laut ist, dann versuche ich, Tür oder Fenster zu schließen oder anderswo weiterzuarbeiten. *If it's noisy where I'm studying, I try to close the door or window or move someplace else.*	☐	☐	☐	☐	☐	☐
7.	Es ärgert mich, wenn meine Nachbarn laut werden. *I get annoyed when my neighbors are noisy.*	☐	☐	☐	☐	☐	☐
8.	An die meisten Geräusche gewöhne ich mich ohne große Schwierigkeiten. *I get used to most noises without much difficulty. **	☐	☐	☐	☐	☐	☐
9.	Es würde mir etwas ausmachen, wenn eine Wohnung, die ich gerne mieten würde, gegenüber der Feuerwache läge. *It would matter to me if an apartment I was interested in renting were located across from a fire station. **	☐	☐	☐	☐	☐	☐
10.	Manchmal gehen mir Geräusche auf die Nerven und ärgern mich. *Sometimes noises get on my nerves and get me irritated.*	☐	☐	☐	☐	☐	☐
11.	Sogar Musik, die ich eigentlich mag, stört mich, wenn ich mich konzentrieren möchte. *Even music I normally like will bother me if I'm trying to concentrate.*	☐	☐	☐	☐	☐	☐

Experiment ID.: _____

12.	Es würde mich nicht stören, die Alltagsgeräusche meiner Nachbarn (z.B. Schritte, Wasserrauschen) zu hören. *It wouldn't bother me to hear the sounds of everyday living from neighbours (footsteps, running water, etc.). **	☐ ☐ ☐ ☐ ☐ ☐
13.	Wenn ich allein sein möchte, stören mich Geräusche von außerhalb. *When I want to be alone, it disturbs me to hear outside noises.*	☐ ☐ ☐ ☐ ☐ ☐
14.	Ich kann mich gut konzentrieren, egal was um mich herum geschieht. *I'm good at concentrating no matter what is going on around me. **	☐ ☐ ☐ ☐ ☐ ☐
15.	In der Bibliothek macht es mir nichts aus, wenn sich Leute unterhalten, solange dies leise geschieht. *In a library, I don't mind if people carry on a conversation if they do it quietly. **	☐ ☐ ☐ ☐ ☐ ☐
16.	Oft wünsche ich mir völlige Stille. *There are often times when I want complete silence.*	☐ ☐ ☐ ☐ ☐ ☐
17.	Motorräder sollten besser schallgedämpft sein. *Motorcycles ought to be required to have bigger mufflers.*	☐ ☐ ☐ ☐ ☐ ☐
18.	Es fällt mir schwer, mich an einem lauten Ort zu entspannen. *I find it hard to relax in a place that's noisy.*	☐ ☐ ☐ ☐ ☐ ☐
19.	Ich werde wütend auf Leute, die Lärm machen, der mich vom Einschlafen oder vom Fortkommen in der Arbeit abhält. *I get mad at people who make noise that keeps me from falling asleep or getting work done.*	☐ ☐ ☐ ☐ ☐ ☐
20.	Es würde mir nichts ausmachen, in einer Wohnung mit dünnen Wänden zu leben. *I wouldn't mind living in an apartment with thin walls. **	☐ ☐ ☐ ☐ ☐ ☐
21.	Ich bin geräuschempfindlich. *I am sensitive to noise.*	☐ ☐ ☐ ☐ ☐ ☐

Experiment ID.: _____

4. State Trait Anxiety Inventory

4.1. Trait Anxiety

Bitte geben Sie an, wie oft folgende Aussagen auf Sie zutreffen. Bei "fast nie" kreuzen Sie ganz links, bei "fast immer" ganz rechts an. Mit den Kästchen dazwischen können Sie abstufen.
Please indicate how often the following statements apply to you. Mark "almost never" on the far left and "almost always" on the far right. You can use the boxes in between to indicate varying degrees.

fast nie *(almost never)* 1 2 3 4 5 6 7 8 fast immer *(almost always)*

	FRAGEN ***(QUESTIONS)***	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
1.	Ich werde schnell müde. *I get tired quickly.*	☐	☐	☐	☐	☐	☐	☐	☐
2.	Ich verpasse günstige Gelegenheiten, weil ich mich nicht schnell genug entscheiden kann. *I miss favourable opportunities because I cannot decide quickly enough.*	☐	☐	☐	☐	☐	☐	☐	☐
3.	Ich bin ruhig und gelassen. *I am calm and composed. **	☐	☐	☐	☐	☐	☐	☐	☐
4.	Ich bin glücklich. *I am happy. **	☐	☐	☐	☐	☐	☐	☐	☐
5.	Ich neige dazu, alles schwer zu nehmen. *I am inclines to take things hard.*	☐	☐	☐	☐	☐	☐	☐	☐
6.	Mir fehlt es an Selbstvertrauen. *I lack self-confidence.*	☐	☐	☐	☐	☐	☐	☐	☐
7.	Ich fühle mich geborgen. *I feel secure. **	☐	☐	☐	☐	☐	☐	☐	☐
8.	Ich fühle mich niedergeschlagen. *I feel downhearted.*	☐	☐	☐	☐	☐	☐	☐	☐
9.	Unwichtige Gedanken gehen mir durch den Kopf und bedrücken mich. *Unimportant thoughts run through my mind and weigh me down.*	☐	☐	☐	☐	☐	☐	☐	☐
10.	Ich werde nervös und unruhig, wenn ich an meine derzeitigen Angelegenheiten denke. *I become nervous and restless when I think about my current concerns.*	☐	☐	☐	☐	☐	☐	☐	☐

Experiment ID.: _____ Session: _____

4.2. State Anxiety

Wie sehr treffen die folgenden Gefühlsbeschreibungen im Moment auf Sie zu? Kreuzen Sie das auf Sie passende Kästchen an. Es gibt keine richtigen oder falschen Antworten. Überlegen Sie bitte nicht lange und entscheiden Sie dann, wie stark das betreffende Gefühl im Moment bei Ihnen vorhanden ist.

Read each statement and select the appropriate response to indicate how you feel right now, that is, at this very moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

überhaupt nicht *(not at all)* 1 2 3 4 5 6 7 8 ganz und gar *(very much so)*

	FRAGEN ***(QUESTIONS)***	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
1.	Ich bin ruhig. *I feel calm. **	☐	☐	☐	☐	☐	☐	☐	☐
2.	Ich fühle mich angespannt. *I feel tense.*	☐	☐	☐	☐	☐	☐	☐	☐
3.	Ich bin aufgeregt. *I feel upset.*	☐	☐	☐	☐	☐	☐	☐	☐
4.	Ich fühle mich ausgeruht. *I feel at ease. **	☐	☐	☐	☐	☐	☐	☐	☐
5.	Ich bin beunruhigt. *I feel frightened.*	☐	☐	☐	☐	☐	☐	☐	☐
6.	Ich fühle mich selbstsicher. *I feel self-confident. **	☐	☐	☐	☐	☐	☐	☐	☐
7.	Ich bin nervös. *I feel nervous.*	☐	☐	☐	☐	☐	☐	☐	☐
8.	Ich bin überreizt. *I feel overwhelmed.*	☐	☐	☐	☐	☐	☐	☐	☐
9.	Ich bin besorgt. *I am worried.*	☐	☐	☐	☐	☐	☐	☐	☐
10.	Ich bin vergnügt. *I feel pleasant. **	☐	☐	☐	☐	☐	☐	☐	☐

Experiment ID.: _____ Session: _____

Wie sehr treffen die folgenden Gefühlsbeschreibungen im Moment auf Sie zu? Kreuzen Sie das auf Sie passende Kästchen an. Es gibt keine richtigen oder falschen Antworten. Überlegen Sie bitte nicht lange und entscheiden Sie dann, wie stark das betreffende Gefühl im Moment bei Ihnen vorhanden ist.

Read each statement and select the appropriate response to indicate how you feel right now, that is, at this very moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

überhaupt nicht 1 2 3 4 5 6 7 8 ganz und gar
(not at all) *(very much so)*

	FRAGEN ***(QUESTIONS)***	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
1.	Ich bin ruhig. *I feel calm. **	☐	☐	☐	☐	☐	☐	☐	☐
2.	Ich fühle mich angespannt. *I feel tense.*	☐	☐	☐	☐	☐	☐	☐	☐
3.	Ich bin aufgeregt. *I feel upset.*	☐	☐	☐	☐	☐	☐	☐	☐
4.	Ich fühle mich ausgeruht. *I feel at ease. **	☐	☐	☐	☐	☐	☐	☐	☐
5.	Ich bin beunruhigt. *I feel frightened.*	☐	☐	☐	☐	☐	☐	☐	☐
6.	Ich fühle mich selbstsicher. *I feel self-confident. **	☐	☐	☐	☐	☐	☐	☐	☐
7.	Ich bin nervös. *I feel nervous.*	☐	☐	☐	☐	☐	☐	☐	☐
8.	Ich bin überreizt. *I feel overwhelmed.*	☐	☐	☐	☐	☐	☐	☐	☐
9.	Ich bin besorgt. *I am worried.*	☐	☐	☐	☐	☐	☐	☐	☐
10.	Ich bin vergnügt. *I feel pleasant. **	☐	☐	☐	☐	☐	☐	☐	☐

Experiment ID.: _____ Session: _____

Wie sehr treffen die folgenden Gefühlsbeschreibungen im Moment auf Sie zu? Kreuzen Sie das auf Sie passende Kästchen an. Es gibt keine richtigen oder falschen Antworten. Überlegen Sie bitte nicht lange und entscheiden Sie dann, wie stark das betreffende Gefühl im Moment bei Ihnen vorhanden ist.

Read each statement and select the appropriate response to indicate how you feel right now, that is, at this very moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

überhaupt nicht 1 2 3 4 5 6 7 8 ganz und gar
(not at all) *(very much so)*

	FRAGEN ***(QUESTIONS)***	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
1.	Ich bin ruhig. *I feel calm. **	☐	☐	☐	☐	☐	☐	☐	☐
2.	Ich fühle mich angespannt. *I feel tense.*	☐	☐	☐	☐	☐	☐	☐	☐
3.	Ich bin aufgeregt. *I feel upset.*	☐	☐	☐	☐	☐	☐	☐	☐
4.	Ich fühle mich ausgeruht. *I feel at ease. **	☐	☐	☐	☐	☐	☐	☐	☐
5.	Ich bin beunruhigt. *I feel frightened.*	☐	☐	☐	☐	☐	☐	☐	☐
6.	Ich fühle mich selbstsicher. *I feel self-confident. **	☐	☐	☐	☐	☐	☐	☐	☐
7.	Ich bin nervös. *I feel nervous.*	☐	☐	☐	☐	☐	☐	☐	☐
8.	Ich bin überreizt. *I feel overwhelmed.*	☐	☐	☐	☐	☐	☐	☐	☐
9.	Ich bin besorgt. *I am worried.*	☐	☐	☐	☐	☐	☐	☐	☐
10.	Ich bin vergnügt. *I feel pleasant. **	☐	☐	☐	☐	☐	☐	☐	☐

Table A.2.: The time series analysis for sBP at Volume 0. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	reSYS (mmHg) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	124.029 ± 14.310				
	2	127.691 ± 13.840	0.325	0.325		
	3	126.992 ± 11.489	0.671	0.677		
	4	128.006 ± 10.716	0.347	0.424		
	5	127.877 ± 11.805	0.927	0.791		
	6	129.829 ± 13.636	0.332	0.152		
Trial	7	130.754 ± 12.995	0.573		0.573	
	8	131.643 ± 15.870	0.720	0.720	0.334	
	9	131.918 ± 14.884	0.881	0.557	0.210	
	10	133.382 ± 14.401	0.125	0.169	**0.049**	
	11	135.059 ± 14.560	0.069	0.051	**0.005**	
	12	135.667 ± 13.301	0.641	**0.022**	**0.020**	
	13	135.396 ± 14.153	0.624	**0.048**	**0.045**	
	14	133.713 ± 14.685	0.301	0.110	0.116	
	15	132.750 ± 14.716	0.368	0.431	0.345	
	16	135.986 ± 14.124	**0.047**	**0.021**	**0.003**	
	17	135.182 ± 14.232	0.733	**0.043**	**0.078**	
	18	135.301 ± 14.667	0.301	**0.030**	**0.030**	
Recovery	19	134.147 ± 14.216	0.330		0.164	0.330
	20	131.034 ± 13.262	**0.043**	**0.043**	0.910	**0.033**
	21	127.611 ± 10.009	**0.077**	**0.017**	0.594	**0.020**
	22	126.598 ± 8.782	0.289	**0.012**	0.422	**0.013**
	23	128.719 ± 8.652	0.076	**0.035**	0.754	**0.031**
	24	128.592 ± 10.258	0.950	0.067	0.424	**0.031**

Table A.3.: The time series analysis for sBP at Volume 0.5. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	reSYS (mmHg) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	127.828 ± 12.772				
	2	124.789 ± 17.482	0.578	0.578		
	3	127.226 ± 12.036	0.552	0.843		
	4	133.503 ± 12.204	0.088	0.128		
	5	135.531 ± 11.790	0.267	**0.015**		
	6	135.316 ± 11.091	0.882	**0.014**		
Trial	7	136.625 ± 15.742	0.588		0.588	
	8	137.296 ± 14.774	0.783	0.783	0.447	
	9	138.942 ± 11.965	0.346	0.401	0.470	
	10	141.251 ± 11.770	0.170	0.077	**0.002**	
	11	142.112 ± 11.808	0.397	**0.047**	**0.005**	
	12	144.962 ± 14.196	0.092	**0.005**	**0.005**	
	13	145.563 ± 16.362	0.596	**0.002**	**0.009**	
	14	144.944 ± 16.458	0.611	**0.008**	**0.025**	
	15	144.431 ± 15.214	0.676	**0.005**	**0.009**	
	16	143.155 ± 14.541	0.382	**0.014**	**0.028**	
	17	143.674 ± 14.563	0.543	0.064	**0.030**	
	18	143.770 ± 15.194	0.949	**0.008**	**0.012**	
Recovery	19	140.768 ± 14.873	**0.009**		0.087	**0.009**
	20	137.250 ± 16.078	0.051	0.051	> 0.999	**0.019**
	21	136.229 ± 13.675	0.636	0.102	0.791	**0.029**
	22	134.863 ± 15.845	0.303	0.085	0.920	**0.027**
	23	137.349 ± 14.483	0.077	0.204	0.611	**0.036**
	24	137.315 ± 15.806	0.980	0.179	0.850	**0.036**

Table A.4.: The time series analysis for sBP at Volume 1. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	reSYS (mmHg) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	131.132 ± 11.117				
	2	130.434 ± 9.302	0.631	0.631		
	3	131.697 ± 8.474	0.498	0.809		
	4	133.847 ± 9.810	0.086	0.424		
	5	133.722 ± 11.984	0.938	0.231		
	6	133.724 ± 10.052	0.999	0.319		
Trial	7	133.840 ± 10.469	0.955		0.955	
	8	136.277 ± 11.349	0.245	0.245	0.310	
	9	137.464 ± 11.618	0.503	0.084	0.209	
	10	139.135 ± 11.993	**0.034**	**0.046**	0.090	
	11	140.466 ± 11.250	0.219	**0.005**	**0.013**	
	12	140.558 ± 11.676	0.301	**< 0.001**	**0.009**	
	13	141.452 ± 12.583	0.416	**0.002**	**0.004**	
	14	141.156 ± 12.628	0.721	**0.004**	**0.006**	
	15	141.725 ± 12.345	0.494	**0.001**	**0.002**	
	16	140.449 ± 13.452	0.132	**0.013**	**0.019**	
	17	140.681 ± 13.141	0.812	**0.007**	**0.014**	
	18	141.558 ± 13.139	0.197	**0.002**	**0.003**	
Recovery	19	137.874 ± 13.304	**0.008**		0.103	**0.008**
	20	135.209 ± 12.136	0.106	0.106	0.522	**0.009**
	21	130.386 ± 10.542	0.063	**0.006**	0.147	**< 0.001**
	22	131.135 ± 10.798	0.320	**0.019**	0.236	**0.001**
	23	130.621 ± 10.643	0.680	**0.012**	0.250	**0.002**
	24	129.508 ± 10.120	0.459	**0.002**	0.087	**< 0.001**

Table A.5.: The time series analysis for dBP at Volume 0. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	reDIA (mmHg) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	72.23 ± 9.986				
	2	72.363 ± 8.921	0.956	0.956		
	3	71.897 ± 8.219	0.710	0.470		
	4	73.435 ± 7.581	**0.049**	0.850		
	5	73.308 ± 9.082	0.677	0.850		
	6	76.715 ± 9.361	**0.022**	**0.034**		
Trial	7	75.242 ± 11.569	0.37		0.37	
	8	75.892 ± 13.418	0.662	0.662	0.611	
	9	76.093 ± 11.812	0.874	0.542	0.668	
	10	77.003 ± 11.408	0.266	0.194	0.842	
	11	78.890 ± 11.385	**0.021**	**0.026**	0.064	
	12	78.778 ± 9.959	0.894	**0.025**	0.106	
	13	78.885 ± 10.898	0.622	**0.028**	0.148	
	14	77.679 ± 12.468	0.424	0.096	0.583	
	15	77.918 ± 10.945	0.804	0.121	0.531	
	16	79.285 ± 11.106	0.103	**0.023**	0.129	
	17	78.796 ± 11.515	0.628	**0.03**	0.289	
	18	78.856 ± 12.298	0.962	0.176	0.275	
Recovery	19	77.425 ± 10.990	0.133		0.709	0.133
	20	76.075 ± 10.744	0.144	0.144	0.764	0.058
	21	73.336 ± 8.242	**0.044**	**0.005**	0.129	**0.009**
	22	73.618 ± 8.634	0.633	**0.016**	0.179	**0.012**
	23	75.878 ± 8.368	0.082	0.267	0.615	0.113
	24	75.317 ± 8.631	0.666	0.16	0.559	0.076

Table A.6.: The time series analysis for dBP at Volume 0.5. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	reDIA (mmHg) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	73.682 ± 11.771				
	2	71.383 ± 15.101	0.536	0.536		
	3	72.713 ± 10.285	0.552	0.675		
	4	75.508 ± 10.311	0.079	0.492		
	5	77.302 ± 10.025	0.098	0.137		
	6	77.802 ± 8.292	0.707	0.051		
Trial	7	78.770 ± 11.935	0.518		0.518	
	8	79.451 ± 13.474	0.513	0.513	0.389	
	9	79.354 ± 11.371	0.939	0.685	0.360	
	10	81.066 ± 11.239	0.117	0.092	**0.019**	
	11	81.371 ± 11.333	0.578	**0.022**	**0.010**	
	12	82.896 ± 12.375	0.066	**0.006**	**0.010**	
	13	83.560 ± 12.509	0.331	**0.002**	**0.004**	
	14	83.020 ± 12.841	0.467	**0.001**	**0.013**	
	15	83.383 ± 12.329	0.686	**0.001**	**0.004**	
	16	83.003 ± 12.112	0.670	**0.006**	**0.010**	
	17	83.144 ± 12.524	0.833	**0.008**	**0.010**	
	18	82.579 ± 12.655	0.570	**0.020**	**0.011**	
Recovery	19	79.300 ± 12.148	**0.015**		0.357	**0.015**
	20	77.596 ± 11.596	0.129	0.129	0.903	**0.010**
	21	77.034 ± 9.853	0.970	0.155	0.620	**0.009**
	22	77.313 ± 9.346	0.654	0.180	0.748	**0.01**
	23	78.042 ± 9.918	0.324	0.393	0.876	**0.007**
	24	77.340 ± 10.391	0.445	0.217	0.762	**0.002**

Table A.7.: The time series analysis for dBP at Volume 1. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	reDIA (mmHg) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	72.831 ± 9.183				
	2	71.635 ± 10.436	0.453	0.453		
	3	71.916 ± 8.513	0.818	0.176		
	4	73.445 ± 9.666	0.190	0.578		
	5	72.976 ± 9.925	0.701	0.907		
	6	74.150 ± 9.697	0.430	0.504		
Trial	7	74.766 ± 10.888	0.670		0.670	
	8	76.495 ± 11.874	0.172	0.172	0.164	
	9	76.567 ± 13.275	0.941	0.199	0.214	
	10	77.270 ± 13.019	0.351	0.141	0.153	
	11	77.590 ± 11.756	0.742	0.051	**0.041**	
	12	78.071 ± 12.946	0.233	**0.017**	**0.037**	
	13	78.285 ± 14.390	0.802	0.068	0.056	
	14	78.164 ± 13.317	0.816	0.054	**0.048**	
	15	78.637 ± 13.713	0.556	**0.020**	**0.021**	
	16	78.443 ± 13.851	0.742	**0.039**	**0.032**	
	17	78.926 ± 13.299	0.480	**0.011**	**0.013**	
	18	79.229 ± 13.242	0.266	**0.005**	**0.007**	
Recovery	19	76.518 ± 13.114	**0.045**		0.256	**0.045**
	20	72.870 ± 13.178	**0.004**	**0.004**	0.572	**0.005**
	21	71.041 ± 12.06	0.234	**0.001**	0.095	**<0.001**
	22	71.749 ± 12.53	0.340	**0.008**	0.229	**<0.001**
	23	72.534 ± 12.325	0.319	**0.030**	0.439	**0.004**
	24	70.841 ± 11.947	**0.042**	**0.001**	0.140	**<0.001**

Table A.8.: The time series analysis for HR at Volume 0. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	HR (bpm) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	77.846 ± 15.403				
	2	76.586 ± 15.038	0.226	0.226		
	3	73.648 ± 16.891	**0.024**	**0.013**		
	4	73.814 ± 17.231	0.814	**0.034**		
	5	74.992 ± 18.060	0.281	0.113		
	6	77.563 ± 16.025	0.162	0.843		
Trial	7	79.778 ± 14.151	0.171		0.171	
	8	78.675 ± 16.719	0.450	0.450	0.453	
	9	78.015 ± 17.891	0.342	0.355	0.795	
	10	76.968 ± 17.821	0.244	0.171	0.738	
	11	77.390 ± 17.467	0.470	0.237	0.919	
	12	77.039 ± 17.437	0.515	0.225	0.789	
	13	76.095 ± 17.873	0.348	0.122	0.537	
	14	75.311 ± 17.781	**0.024**	0.080	0.369	
	15	75.011 ± 17.165	0.535	0.060	0.280	
	16	74.526 ± 17.227	0.315	**0.025**	0.152	
	17	75.649 ± 16.971	**0.018**	0.060	0.312	
	18	75.368 ± 15.965	0.745	**0.045**	0.297	
Recovery	19	75.143 ± 16.509	0.695		0.244	0.695
	20	76.050 ± 17.348	0.236	0.236	0.531	0.474
	21	74.195 ± 17.061	**0.011**	0.361	0.173	0.255
	22	73.117 ± 16.337	0.443	0.139	**0.002**	0.104
	23	74.642 ± 17.390	0.249	0.765	0.060	0.644
	24	74.811 ± 18.064	0.875	0.824	0.141	0.717

Table A.9.: The time series analysis for HR at Volume 0.5. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	HR (bpm) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	79.569 ± 13.125				
	2	80.091 ± 13.508	0.762	0.762		
	3	78.636 ± 15.558	0.970	0.684		
	4	76.369 ± 14.217	0.132	0.139		
	5	77.060 ± 15.597	0.627	0.569		
	6	78.839 ± 14.633	0.243	0.470		
Trial	7	80.861 ± 13.889	0.191		0.191	
	8	81.755 ± 15.661	0.694	0.694	0.143	
	9	81.819 ± 16.622	0.569	0.850	0.378	
	10	81.622 ± 16.562	0.852	0.519	0.429	
	11	82.163 ± 17.313	0.640	0.730	0.358	
	12	79.700 ± 16.960	**0.026**	0.724	0.762	
	13	78.315 ± 15.282	0.233	0.302	0.785	
	14	77.580 ± 16.700	0.365	0.181	0.513	
	15	77.288 ± 17.661	0.659	0.206	0.490	
	16	77.776 ± 16.973	0.477	0.282	0.662	
	17	77.668 ± 17.220	0.806	0.260	0.631	
	18	76.862 ± 15.666	0.566	0.184	0.411	
Recovery	19	74.212 ± 15.284	0.052		**0.034**	0.052
	20	74.326 ± 17.019	0.921	0.921	**0.035**	0.064
	21	73.193 ± 16.946	0.383	0.605	**0.008**	0.301
	22	72.797 ± 17.995	0.733	0.517	**0.005**	0.136
	23	73.448 ± 17.760	0.460	0.720	**0.010**	0.185
	24	72.479 ± 15.151	0.491	0.447	**0.007**	0.054

Table A.10.: The time series analysis for HR at Volume 1. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	HR (bpm) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	77.781 ± 15.099				
	2	77.074 ± 15.848	0.654	0.654		
	3	75.459 ± 14.156	0.229	0.303		
	4	74.378 ± 15.272	0.288	0.141		
	5	73.640 ± 13.421	0.537	0.082		
	6	75.720 ± 14.391	0.058	0.283		
Trial	7	76.992 ± 15.939	0.355		0.355	
	8	81.402 ± 17.652	**0.006**	**0.006**	**0.011**	
	9	80.461 ± 16.243	0.621	0.070	**0.003**	
	10	80.141 ± 15.992	0.807	0.064	**0.011**	
	11	79.709 ± 16.003	0.644	0.154	0.065	
	12	77.809 ± 16.575	**0.012**	0.674	0.176	
	13	76.508 ± 18.060	0.133	0.791	0.747	
	14	75.507 ± 16.059	0.519	0.355	0.912	
	15	76.015 ± 17.093	0.426	0.512	0.887	
	16	75.707 ± 16.391	0.606	0.313	0.995	
	17	76.782 ± 16.793	**0.046**	0.876	0.556	
	18	76.586 ± 16.334	0.301	0.807	0.656	
Recovery	19	75.191 ± 16.783	0.227		0.839	0.227
	20	72.314 ± 15.699	**0.005**	**0.005**	0.519	**0.001**
	21	71.583 ± 16.207	0.513	**0.001**	0.119	**0.001**
	22	71.602 ± 17.286	0.979	**0.014**	0.108	**0.002**
	23	71.621 ± 16.253	0.986	**0.001**	0.108	**0.002**
	24	72.780 ± 16.262	0.391	0.206	0.288	0.088

Table A.11.: The time series analysis for RMSSD at Volume 0. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase and 10 s interval		RMSSD (ms) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	68.416 ± 5.748				
	2	74.671 ± 6.222	**0.004**	**0.004**		
	3	72.950 ± 3.869	0.393	**0.030**		
	4	70.214 ± 9.321	0.261	0.589		
	5	71.907 ± 8.070	0.651	0.335		
	6	73.620 ± 5.115	0.322	0.087		
Trial	7	70.082 ± 8.463	0.311		0.311	
	8	72.446 ± 8.456	0.401	0.401	0.654	
	9	69.654 ± 7.465	0.271	0.879	0.122	
	10	72.197 ± 9.520	0.103	0.517	0.634	
	11	74.144 ± 8.162	0.970	0.210	0.829	
	12	73.352 ± 9.966	0.793	0.424	0.934	
	13	71.161 ± 6.748	0.542	0.693	0.340	
	14	69.942 ± 9.555	0.691	0.967	0.308	
	15	68.285 ± 7.137	0.415	0.530	0.086	
	16	70.847 ± 6.934	0.323	0.829	0.288	
	17	74.507 ± 9.408	0.081	0.292	0.788	
	18	73.886 ± 7.370	0.834	0.260	0.879	
Recovery	19	72.075 ± 9.992	0.549		0.626	0.549
	20	72.791 ± 7.963	0.836	0.836	0.622	0.449
	21	70.044 ± 8.278	0.348	0.598	0.293	0.283
	22	68.115 ± 7.567	0.555	0.342	0.104	0.121
	23	69.682 ± 7.196	0.628	0.520	0.221	0.271
	24	72.757 ± 6.565	0.324	0.843	0.698	0.608

Table A.12.: The time series analysis for RMSSD at Volume 0.5. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase and 10 s interval		RMSSD (ms) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	72.303 ± 6.204				
	2	73.148 ± 5.307	0.731	0.731		
	3	71.431 ± 5.684	0.305	0.746		
	4	73.511 ± 6.498	0.196	0.652		
	5	72.502 ± 4.607	0.505	0.931		
	6	73.551 ± 6.027	0.607	0.700		
Trial	7	71.425 ± 6.443	0.525		0.525	
	8	73.816 ± 5.569	0.403	0.403	0.926	
	9	69.805 ± 8.762	0.252	0.694	0.131	
	10	74.987 ± 9.616	**0.0**42	0.376	0.671	
	11	70.137 ± 9.443	0.128	0.750	0.242	
	12	69.484 ± 6.760	0.751	0.608	0.075	
	13	73.352 ± 5.650	0.059	0.583	0.920	
	14	70.870 ± 6.030	0.385	0.844	0.364	
	15	72.344 ± 7.248	0.970	0.798	0.488	
	16	68.190 ± 6.866	0.133	0.366	0.059	
	17	70.716 ± 4.016	0.226	0.766	0.290	
	18	65.619 ± 9.826	0.116	0.103	0.096	
Recovery	19	72.038 ± 5.417	**0.014**		0.850	**0.014**
	20	70.756 ± 6.638	0.543	0.543	0.448	**0.042**
	21	68.278 ± 3.132	0.174	**0.023**	**0.034**	0.329
	22	69.326 ± 8.220	0.682	0.259	0.297	0.163
	23	68.368 ± 6.933	0.608	0.128	0.175	0.280
	24	72.582 ± 4.249	0.062	0.820	0.718	0.050

Table A.13.: The time series analysis for RMSSD at Volume 1. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	RMSSD (ms) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	74.068 ± 6.143				
	2	68.725 ± 7.039	0.088	0.088		
	3	70.785 ± 9.059	0.324	0.350		
	4	70.777 ± 5.721	0.998	0.339		
	5	74.220 ± 6.851	0.274	0.953		
	6	72.201 ± 9.487	0.376	0.567		
Trial	7	72.141 ± 7.522	0.984		0.984	
	8	69.237 ± 6.155	0.370	0.370	0.420	
	9	68.871 ± 12.139	0.913	0.395	0.594	
	10	67.755 ± 7.465	0.760	0.233	0.298	
	11	67.943 ± 9.874	0.942	0.245	0.373	
	12	67.060 ± 10.363	0.655	0.193	0.275	
	13	66.139 ± 10.982	0.749	0.206	0.850	
	14	71.761 ± 6.626	0.086	0.891	0.902	
	15	67.312 ± 8.573	0.273	0.187	0.242	
	16	66.530 ± 9.246	0.797	0.120	0.166	
	17	68.790 ± 8.118	0.622	0.294	0.269	
	18	73.063 ± 5.675	0.172	0.754	0.751	
Recovery	19	72.568 ± 6.072	0.803		0.912	0.803
	20	71.610 ± 6.979	0.622	0.622	0.879	0.623
	21	76.250 ± 6.007	0.156	0.193	0.138	0.121
	22	70.952 ± 5.754	**0.020**	0.578	0.683	0.369
	23	71.681 ± 7.447	0.750	0.736	0.828	0.600
	24	68.010 ± 5.436	0.194	0.053	0.232	**0.017**

Table A.14.: The time series analysis for EDA at Volume 0. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	relative EDA (a.u.) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	1.024 ± 0.037				
	2	1.023 ± 0.033	0.622	0.622		
	3	1.001 ± 0.015	0.109	0.099		
	4	0.979 ± 0.022	**0.004**	**0.013**		
	5	0.972 ± 0.034	0.481	**0.030**		
	6	0.978 ± 0.026	0.356	**0.021**		
Trial	7	1.097 ± 0.105	**0.004**		**0.004**	
	8	1.151 ± 0.159	0.100	0.100	**<0.001**	
	9	1.163 ± 0.164	0.677	0.380	**0.001**	
	10	1.159 ± 0.171	0.677	0.569	**0.009**	
	11	1.158 ± 0.183	0.912	0.791	**0.002**	
	12	1.152 ± 0.181	0.276	0.791	**0.007**	
	13	1.141 ± 0.173	0.110	0.970	**0.007**	
	14	1.131 ± 0.180	0.283	0.677	**0.027**	
	15	1.110 ± 0.183	**0.002**	0.380	0.077	
	16	1.106 ± 0.184	0.129	0.301	0.151	
	17	1.095 ± 0.177	0.085	0.301	0.074	
	18	1.076 ± 0.170	**0.024**	0.266	0.116	
Recovery	19	1.070 ± 0.162	0.301		0.123	0.301
	20	1.055 ± 0.148	0.080	0.080	0.160	0.110
	21	1.033 ± 0.146	**0.004**	**0.014**	0.301	**0.019**
	22	1.011 ± 0.143	**0.005**	**0.007**	0.520	**0.011**
	23	0.990 ± 0.133	**0.026**	**0.007**	0.810	**0.009**
	24	0.983 ± 0.130	0.508	**0.016**	0.917	**0.019**

Table A.15.: The time series analysis for EDA at Volume 0.5. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	relative EDA (a.u.) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	0.967 ± 0.064				
	2	1.010 ± 0.058	**0.027**	**0.027**		
	3	0.999 ± 0.028	0.573	0.156		
	4	1.010 ± 0.050	0.530	0.210		
	5	1.013 ± 0.083	0.901	0.284		
	6	1.028 ± 0.117	0.362	0.234		
Trial	7	1.131 ± 0.186	**0.005**		**0.005**	
	8	1.203 ± 0.266	**0.042**	**0.042**	**0.001**	
	9	1.196 ± 0.231	0.826	0.118	**0.005**	
	10	1.168 ± 0.210	0.129	0.622	**0.034**	
	11	1.145 ± 0.210	0.168	0.677	0.110	
	12	1.127 ± 0.203	0.144	0.380	**0.034**	
	13	1.120 ± 0.203	0.417	0.424	**0.043**	
	14	1.117 ± 0.228	0.233	0.806	0.266	
	15	1.093 ± 0.224	**0.002**	0.507	0.569	
	16	1.096 ± 0.240	0.826	0.560	0.424	
	17	1.103 ± 0.269	0.791	0.266	0.470	
	18	1.085 ± 0.252	0.055	0.176	0.519	
Recovery	19	1.080 ± 0.254	0.554		0.850	0.554
	20	1.060 ± 0.246	**0.042**	**0.042**	0.733	0.130
	21	1.042 ± 0.252	**0.009**	**0.005**	0.519	**0.025**
	22	1.022 ± 0.246	**0.007**	**0.001**	0.339	**0.007**
	23	1.008 ± 0.241	**0.034**	**0.003**	0.266	**0.010**
	24	0.988 ± 0.235	0.086	**<0.001**	0.176	**<0.001**

Table A.16.: The time series analysis for EDA at Volume 1. The table shows the statistical evaluation of differences for each 10-second interval. All p-values < 0.05 were deemed significant.

Exp. Phase	and 10 s interval	relative EDA (a.u.) (Mean ± SD)	Compared to preceding interval	Compared to first interval of phase	Compared to last interval of *Rest*	Compared to last interval of *Trial*
Rest	1	1.002 ± 0.090				
	2	1.016 ± 0.064	0.472	0.472		
	3	1.004 ± 0.034	0.266	0.954		
	4	0.993 ± 0.051	0.434	0.838		
	5	0.985 ± 0.073	0.415	0.729		
	6	1.019 ± 0.115	0.233	0.761		
Trial	7	1.131 ± 0.194	**0.003**		**0.003**	
	8	1.219 ± 0.231	**0.004**	**0.004**	**0.001**	
	9	1.242 ± 0.269	0.910	**0.003**	**0.002**	
	10	1.240 ± 0.318	0.424	0.110	**<0.001**	
	11	1.238 ± 0.334	0.822	0.204	**<0.001**	
	12	1.208 ± 0.336	**0.007**	0.791	**<0.001**	
	13	1.192 ± 0.341	**0.005**	0.622	**0.001**	
	14	1.178 ± 0.347	0.052	0.424	**0.016**	
	15	1.181 ± 0.384	0.301	0.380	0.077	
	16	1.164 ± 0.366	**0.042**	0.380	0.110	
	17	1.150 ± 0.355	0.067	0.233	0.151	
	18	1.134 ± 0.345	**0.026**	0.233	0.266	
Recovery	19	1.127 ± 0.346	0.313		0.339	0.313
	20	1.107 ± 0.359	0.125	0.125	0.850	0.081
	21	1.077 ± 0.338	**0.028**	**0.009**	0.733	**0.011**
	22	1.047 ± 0.327	**0.001**	**0.002**	0.424	**0.002**
	23	1.019 ± 0.312	**<0.001**	**0.001**	0.233	**0.001**
	24	0.999 ± 0.287	0.107	**<0.001**	0.129	**<0.001**

Table A.17.: Volume analysis for *Volume 0.5*. The table shows the statistical evaluation of differences for each 10-second interval at *Volume 0* compared to silence. All p-values < 0.05 were deemed significant. Intervals 0-5 are not included, as they were used as the baseline reference.

Exp. Phase	and 10 s interval	reSYS	reDIA	HR	RMSSD	norm. EDA
Rest	6	0.381	0.982	0.360	0.670	0.233
Trial	7	0.471	0.367	0.513	0.925	0.424
	8	0.465	0.366	0.985	0.862	0.424
	9	0.733	0.336	0.738	0.622	0.491
	10	0.123	0.190	0.476	0.499	0.779
	11	0.088	0.566	0.450	0.182	0.728
	12	**0.021**	0.157	0.885	0.289	0.479
	13	**0.005**	0.108	0.746	0.424	0.521
	14	0.151	0.158	0.697	0.975	0.683
	15	0.057	0.120	0.580	0.211	0.590
	16	0.333	0.357	0.831	0.236	0.757
	17	0.144	0.168	0.497	0.229	0.853
	18	0.195	0.383	0.395	0.077	0.827
Recovery	19	0.316	0.869	0.110	0.470	0.822
	20	0.910	0.995	0.064	0.560	0.914
	21	0.110	0.195	0.244	0.511	0.861
	22	0.470	0.212	0.139	0.873	0.843
	23	0.176	0.622	**0.031**	0.629	0.750
	24	0.233	0.856	**0.001**	0.707	0.622

Table A.18.: Volume analysis for *Volume 1*. The table shows the statistical evaluation of differences for each 10-second interval at *Volume 1* compared to silence. All p-values < 0.05 were deemed significant. Intervals 0-5 are not included, as they were used as the baseline reference.

Exp. Phase	and 10 s interval	reSYS	reDIA	HR	RMSSD	norm. EDA
Rest	6	0.633	0.158	0.110	0.506	0.791
Trial	7	0.233	0.929	0.217	0.580	0.503
	8	0.884	0.499	0.363	0.379	0.174
	9	0.812	0.582	0.290	0.938	0.233
	10	0.823	0.658	**0.047**	0.281	0.301
	11	0.936	0.357	0.115	0.137	0.569
	12	>0.999	0.635	0.683	0.211	0.850
	13	0.380	0.640	0.995	0.299	>0.999
	14	0.399	0.525	0.976	0.596	0.791
	15	0.226	0.582	0.755	0.770	0.850
	16	0.648	0.666	0.587	0.310	0.970
	17	0.970	0.826	0.662	0.164	0.970
	18	0.773	0.754	0.339	0.683	>0.999
Recovery	19	0.530	0.791	0.947	0.843	0.850
	20	0.850	0.083	0.424	0.794	0.910
	21	0.910	0.124	0.733	0.106	0.850
	22	0.717	0.125	0.329	0.242	0.569
	23	0.135	**0.023**	0.174	0.546	0.519
	24	0.204	**0.007**	0.327	0.111	0.470