Institute of Sports Medicine

Department of Exercise and Health

Nocturnal Autonomic Activity after Sport-Related Concussion: A Home-Based Approach

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Declaration of Authorship

I hereby declare that the presented work is, to the best of my knowledge and belief, the result of my own research. Support during the research process or co-author contributions are presented for each publication. The work has not been submitted, either partly or completely, for a degree at this or another university. Ideas and formulations from other sources have been cited throughout the work.

I have read, understood, and accepted the PhD regulations (“Promotionsordnung NW”) from the 31st of March 2021 (AM.UNI.PB 10.21).

Date: 30.09.2025

Signature:

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Abstract

Sport-related concussion (SRC) is a common yet complex brain injury that may alter functional brain networks. This injury is characterized by heterogeneous symptoms and recovery trajectories, with most athletes returning to sport within four weeks. However, sleep-associated symptoms are frequently reported after SRC and have been linked to a longer recovery period. Clinical clearance for return to sport (RTS) is currently based on subjective symptom resolution, as objective physiological markers are lacking. This dissertation investigated whether nocturnal autonomic nervous system activity, measured in a home-based setting utilizing wearable technology, could serve as a neurophysiological marker for SRC recovery. Nocturnal autonomic parameters (heart rate, heart rate variability, and electrodermal activity) during and post RTS (3 weeks) and initial concussion symptoms of SRC athletes were collected and compared to matched control athletes. Results revealed a trend towards lower cardiac parasympathetic activity during RTS among SRC athletes, but not after RTS. Notably, athletes with prolonged RTS (≥ 28 days) exhibited significantly reduced cardiac parasympathetic and electrodermal sympathetic activity post RTS, suggesting changes in autonomic activity despite clinical symptom resolution. Whether these alterations contribute to or may be linked to prolonged recovery remains unresolved.

Zusammenfassung (DE)

Die sportassoziierte Gehirnerschütterung (SRC) ist eine häufige und komplexe Gehirnverletzung, die zur Beeinträchtigung funktioneller Hirnnetzwerke führen kann. Sie zeichnet sich durch heterogene Symptome und variable Erholungsverläufe aus, wobei die Rückkehr zum Sport (RTS) für Athlet:innen meist innerhalb von vier Wochen erfolgt. Schlafassoziierte Symptome sind dabei mit einem verlängerten Erholungsverlauf und einem erhöhten Risiko für anhaltende Symptome verbunden. Die klinische Freigabe zum RTS basiert primär auf subjektiver Symptomfreiheit, während objektive physiologische Marker fehlen. Diese Dissertation untersuchte, ob die nächtliche Aktivität des autonomen Nervensystems (ANS), gemessen im häuslichen Umfeld mittels Wearables, als neurophysiologischer Marker für die Regeneration nach SRC dienen kann. Erfasst wurden nächtliche ANS-Parameter (Herzfrequenz, Herzfrequenzvariabilität, elektrodermale Aktivität) während und drei Wochen nach RTS sowie initiale Symptome. Diese wurden zwischen SRC-Athlet:innen und gematchten Kontrollathlet:innen verglichen. Die Ergebnisse zeigten einen Trend zu reduzierter parasympathischer Aktivität bei SRC-Athlet:innen während des RTS. Athlet:innen mit prolongiertem RTS (≥ 28 Tage) wiesen eine verminderte kardiale parasympathische und elektrodermale sympathische Aktivität nach dem RTS auf. Ob diese Veränderungen Ursache oder Folge der verlängerten Erholung sind, bleibt bislang ungeklärt.

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List of Abbreviations

ACh	Acetylcholine
ANS	Autonomic nervous system
CAN	Central autonomic network
CBF	Cerebral blood flow
CNS	Central nervous system
CISG	Concussion in Sport Group
CT	Computed tomography
CV	Coefficients of variation
DAI	Diffuse axonal injury
DGSM	German Society for Sleep Research and Sleep Medicine
ECG	Electrocardiogram
EDA	Electrodermal activity
EDR	Electrodermal response
HF	High-frequency band
HR	Heart rate
HRV	Heart rate variability
IBI	Interbeat interval
LF	Low-frequency band
mTBI	Mild traumatic brain injury
meanEDA	Mean electrodermal activity
Mdn	Median
MRI	Magnetic resonance imaging
NREM	Non-rapid-eye-movement sleep
PNS	Parasympathetic nervous system
PPCS	Persistent post-concussive symptoms
PPG	Photoplethysmography
PTSD	Post-traumatic stress disorder
REM	Rapid-eye-movement sleep
RMSSD	Root mean square of successive NN interval differences

RTS	Return to sport
R^2	Coefficient of determination
SBP	Systolic blood pressure
SCAT	Sport Concussion Assessment Tool
SD	Standard Deviation
SNS	Sympathetic nervous system
SRC	Sport-related concussion
SRSS	Short Recovery and Stress Scale
SWS	Slow wave sleep
TBI	Traumatic brain injury
TST	Total sleep time
vmPFC	Ventromedial prefrontal cortex
WHO	World Health Organization

1 Introduction

"The concussion crisis has changed the face of sports as we know it and it has brought to surface the incredible importance of our brain health. The time is now for us to make our brain the number one priority so that education and awareness can take effect, and begin to change the way we approach the health of our athletes from youth to professionals."

(Ben Utrecht, former American football player)

Sport-related concussion (SRC) is a multifaceted subtype of mild traumatic brain injury (mTBI) that arises in the context of sports due to a direct or indirect transmission of biomechanical forces to the head (Patricios et al., 2023; Silverberg et al., 2023). Since the 1980s, reported incidences of SRCs in collegiate athletes have doubled (Hallock et al., 2023). Repeated exposure to SRC, which frequently occurs in contact sports, has been associated with severe neurological consequences later in life, including Alzheimer's, Parkinson's, and chronic traumatic encephalopathy (Gavett et al., 2011; Manley et al., 2017). However, the full impact of recurrent SRCs on long-term brain health remains unclear (Manley et al., 2017).

SRCs typically result from a direct blow to the head, neck, or body, transmitting forces to the brain, which can damage the brain's nerve fibers, a process known as diffuse axonal injury (DAI) (Patricios et al., 2023). This process can disrupt communication between different areas of the brain (Reinsberger, 2024). Standard imaging techniques, such as Magnetic resonance imaging (MRI), often fail to detect DAI. As a result, this rather functional than structural injury presents with a variety of symptoms that can be classified into the domains of neurocognition, affective/anxiety disorders, fatigue/sleep, headaches/migraines, as well as ocular and vestibular problems (Harmon et al., 2019). Therefore, diagnosing and managing SRC can be challenging, as individual clinical symptoms can change over time and lack specificity to concussion.

For athletes, the Concussion in Sport Group (CISG) recommends a six-step return to sport (RTS) protocol to guide recovery (Patricios et al., 2023). Medical clearance, which marks the completion of the RTS protocol, primarily relies on symptomatic resolution, as objective physiological markers are lacking (McCrory et al., 2017). In adults, the RTS protocol is typically completed within one month (DuPrey et al., 2022; Iverson et al., 2017; Leddy et al., 2017). However, 10 to 30% of athletes experience prolonged recovery and persistent post-concussion symptoms (Makdissi et

al., 2017). Initial symptomatology is the primary predictor of prolonged recovery (Iverson et al., 2017; Meehan et al., 2014), but sleep has been discussed as playing a mediating role in symptom expression and the recovery process (Hughes et al., 2022; Jaffee et al., 2015). Sleep and sleep-dependent synaptic plasticity play a crucial role in brain recovery (Giza et al., 2017). Hence, reporting sleep-associated symptoms is linked to longer recovery durations and a higher prevalence of persistent symptoms (DuPrey et al., 2022). It has been hypothesized that these persistent symptoms may be attributed to lasting alterations in brain function and networks (Makdissi et al., 2017).

Changes in the activity of the autonomic nervous system (ANS) after SRC have been frequently reported in the literature (Mercier et al., 2022) and are related to functional impairments in the regulatory centers of the ANS in the brain (Leddy et al., 2017; Pyndiura et al., 2020). The ANS plays a critical role in unconsciously adjusting homeostasis in the body through various organ system functions, including heart rate (HR), respiration, body temperature, blood pressure, and digestion (Jänig, 2007). Autonomic dysfunction after SRC is proposed to contribute to the symptoms experienced during the acute and post-acute stages of SRC (Purkayastha, Stokes, & Bell, 2019). In contrast to time-consuming and expensive advanced neuroimaging techniques, assessing autonomic parameters offers a non-invasive, practical, and cost-effective method for exploring neurophysiological recovery (Bishop et al., 2018; Senthinathan et al., 2017). It can be conducted in a home-based setting through wearable technology such as wireless wrist sensors (Coenen & Reinsberger, 2023; Tabor et al., 2023). Thereby, nocturnal recordings provide a convenient and standardized setting for ANS measurements (Buchheit, 2014), ensuring optimal data quality (Böttcher et al., 2022) and high participant compliance (Nasseri et al., 2020).

The mechanisms underlying autonomic dysfunction and concussion symptoms after SRC are not fully understood. They are hypothesized to result from injury-induced impairments to brain regions and networks involved in ANS regulation (Snyder et al., 2021). Investigating the interplay between autonomic activity, concussion symptoms, and sleep is crucial for advancing the understanding of the complex pathophysiology of SRC and its recovery trajectory**.** Therefore, this dissertation explores the potential of nocturnal ANS activity, measured in a home-based setting, as a neurophysiological marker for SRC recovery and its relationship with concussion symptoms.

2 Current State of Research

“Despite a significant increase in research dedicated to identifying and managing sport-related concussion, it remains one of the most complex injuries sports medicine professionals face.”

(Broglio et al., 2014)

According to reports from the World Health Organization (WHO), traumatic brain injuries (TBI) are rapidly becoming one of the leading causes of death and disability worldwide, posing a significant public health concern (Hyder et al., 2007). The vast majority of TBIs are classified as "mild" (approximately 90%), while only a minority of cases are considered "severe" (Giza et al., 2017). By the definition of the WHO, individuals with mTBI typically exhibit a Glasgow Coma Scale (a clinical scale used to evaluate the level of consciousness after brain injury) score ranging from 13 to 15. In contrast, individuals with moderate TBI have a score ranging from 9 to 12, and those with severe TBI have a score ranging from 3 (the most severe cases) to 8.

Concussions have been classified as a less severe type of mTBI, although there are no specific symptom profiles, diagnostic criteria, or objective markers to make a clear distinction between them. (Mayer et al., 2017). Giza et al. (2017) concluded: “Concussion may be considered a clinical syndrome included in the category of mTBI”.

Concussions are among the most common injuries in sports (Neselius & Brisby, 2014), and due to the complex and diverse nature of SRC (La Fountaine, 2018) managing the recovery process and determining the optimal timing for RTS can be challenging, as it relies on subjective symptom reporting (McCrory et al., 2017). Previous research has indicated that athletes may downplay their symptoms due to concerns about being sidelined for extended periods (Chrisman et al., 2013; Meier, Brummel, et al., 2015). Therefore, it is essential to employ objective measures to monitor physiological recovery (Harmon et al., 2019), to minimize the risk for secondary injuries (e.g., musculoskeletal injuries, repetitive concussion), which often follow SRC (Herman et al., 2017), persistent symptoms (Howell et al., 2020), longer recovery durations (Asken et al., 2016), and possible long-term consequences (Manley et al., 2017). One promising avenue for such objective assessment is the investigation of autonomic activity, which may offer insights into underlying neurophysiological recovery processes.

The purpose of this chapter is to provide relevant background information by initially explaining the pathophysiology of SRC (Chapter 2.1), followed by a description of concussion symptoms and their association with recovery. Subsequently (Chapter 2.2), the anatomy and physiology of the ANS are outlined, along with a summary of the measurement tools and parameters used to assess ANS function, culminating in a discussion of ANS dysfunction and its relationship with SRC symptoms. Finally (Chapter 2.3), the role of sleep as a modifier of SRC is examined, focusing on sleep problems and symptoms following SRC and the physiology of sleep.

2.1 Pathophysiology of Sport-related Concussion

SRC is a complex pathophysiological process in which biomechanical forces transmitted to the brain can lead to various neuropathophysiological disturbances (Patricios et al., 2023), including biochemical, metabolic, and structural changes that can impact brain functioning. Currently, the understanding of the acute pathophysiology of concussion is primarily based on rodent models. Although it is assumed that humans experience a comparable response to concussion, there is a lack of well-established evidence regarding the acute phase of injury (Churchill et al., 2017; Yoshino et al., 1991), but more prominent in the subacute phase (up to 3 months) and long-term (chronic) changes (over 3 months) (Giza & Hovda, 2014; Mayer et al., 2017).

Rapid forces can cause acceleration, deceleration, or rotation of the brain within the skull, resulting in the stretching and shearing of neuronal axons, glial cells, and cerebral blood vessels (Ling et al., 2015). Axons extending long distances from the cell bodies are particularly susceptible to stretching, which may result in DAI. DAI can lead to a chain of metabolic disturbances known as the “neurometabolic cascade” of concussion (Figure 1) (Giza & Hovda, 2014). Immediately after the injury, the excitatory neurotransmitter glutamate is released, contributing to changes in intra- and extracellular ionic flux (e.g., sodium, potassium, calcium, and chloride) (Romeu-Mejia et al., 2019). This results in impaired mitochondrial function while re-establishing ionic and cellular homeostasis, which demands high energy (adenosine triphosphate) consumption by the sodium-potassium pump. Additionally, there can be a temporary decrease in cerebral blood flow (CBF), which limits the delivery of glucose and oxygen to cells (La Fountaine, 2018). The disrupted balance of ions can cause a prolonged depolarization and hyperexcitability of the affected neurons, which may be associated with cell damage and death (Giza & Hovda, 2001).

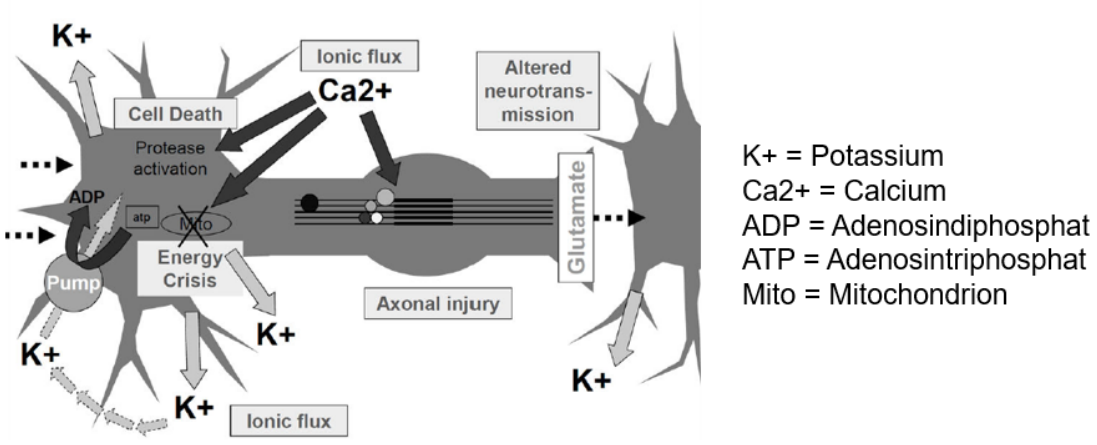

Figure 1 The neurometabolic cascade of concussion. Reproduced with permission from: Giza & Hovda, 2014.

This mismatch between energy demand and availability is causing a cerebral energy crisis, which can persist for seven to ten days after the injury (Giza & Hovda, 2014) and may lead to failure in neuronal function (Blennow et al., 2012). During this metabolic vulnerability period, a subsequent concussion can lead to an exacerbation of the metabolic imbalance and potentially result in more severe brain damage (e.g., malignant cerebral edema) or even mortality, described as the “second impact syndrome” (Romeu-Mejia et al., 2019).

DAI can further lead to disruption of axonal transport, resulting in reduced neurotransmission and axonal disconnections in the most severe form (Giza & Hovda, 2014). This may be the reason for the changes described in brain network connectivity following SRC (Murdaugh et al., 2018; Slobounov et al., 2011). While decreased functional connectivity (hypoconnectivity) is an expected consequence of disrupted neural communication, several studies have also reported functional hyperconnectivity, which describes increased synchrony or communication between brain regions (Chong & Schwedt, 2015). This pattern is thought to reflect a compensatory adaptation, whereby the brain recruits alternative networks or additional neural resources to maintain function in response to the localized injury (Hillary & Grafman, 2017). However, the specific brain networks involved and the directionality of these changes still need to be further investigated before firm conclusions can be made about hypo- and hyperconnectivity following concussion (Chong & Schwedt, 2015).

While neurometabolic (e.g., neurotransmission) (Vagnozzi et al., 2008) and physiological impairments (e.g., reduced CBF) (Meier, Bellgowan, et al., 2015) can last into the subacute phase (Leddy et al., 2017), a neuroinflammatory response

(inflammation within the nervous system) with microglia and astrocytes releasing proinflammatory cytokines (such as tumor necrosis factor- α, interleukin-6, interleukin-8, and interleukin-10) and chemokines that can cause further damage to neurons and other brain cells have been described to play an essential role for persistent neurodegeneration (Rathbone et al., 2015). Ongoing axonal degeneration and altered synaptic plasticity may also occur (Romeu-Mejia et al., 2019).

In summary, the pathophysiological changes associated with SRC occur in a diffuse manner, possibly affecting multiple brain regions, networks, and their communication. The heterogeneity of SRC can be attributed to the variability and individuality in which brain structures are impacted, as well as differences in the biomechanical characteristics of the injury, including the force of the impact, location, and direction of skull acceleration, deceleration, and rotational forces (Danielli et al., 2023; Langdon et al., 2020).

2.1.1 Symptoms and Clinical Recovery

SRC is characterized by a complex pathophysiological process (Chapter 2.1) that is associated with a broad range of clinical symptoms, which can be classified into the domains of neurocognition, affective and anxiety disorders, fatigue/sleep, headaches and migraines, as well as ocular and vestibular problems (including balance disorders) (Harmon et al., 2019). The wide range of these symptoms suggests that multiple brain regions may be affected by a single concussion (Danielli et al., 2023). However, significant variability exists among individuals in the onset, progression, and resolution of symptoms over time (Eagle et al., 2020; Patricios et al., 2023). Moreover, as reported symptoms are non-specific to concussion, they might be challenging to differentiate from pre-existing diseases, general fatigue, stress, and inadequate sleep (Hallock et al., 2023; Iverson et al., 2017). Therefore, symptoms can also be reported in healthy athletes (Mayer et al., 2017).

2.1.1.1 Clinical Assessment of Symptoms

The reporting of concussion symptoms represents the most sensitive indicator of an athlete's concussion status (Eagle et al., 2020; Harmon et al., 2019). Hence, diverse symptom checklists are devised for collecting SRC symptomology (Broglio et al., 2017). One of the most extensively utilized is the symptom checklist from the "Sport Concussion Assessment Tool" (SCAT) by the CISG (McCrory et al., 2017). The SCAT includes 22 symptoms on a 7-point Likert scale (0 = no symptom, 1-2 = mild symptom, 3-4 =

moderate symptom, and 5-6 = severe symptom), resulting in a max. symptom severity score of 132 (22x6) (Table 1).

Table 1 Symptom checklist of the SCAT5

	none	**mild**		**moderate**		**severe**	
Headache	0	1	2	3	4	5	6
“Pressure in head”	0	1	2	3	4	5	6
Neck pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like “in a fog“	0	1	2	3	4	5	6
“Don’t feel right”	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or anxious	0	1	2	3	4	5	6
Trouble falling asleep (if applicable)	0	1	2	3	4	5	6

While there are subtle variations among symptom scales, they all share common symptoms, including “Headache”, “Dizziness”, “Difficulty concentrating”, “Nausea”, “Fatigue”, “Trouble falling asleep”, “Drowsiness”, “Feeling slowed down”, and “Feeling in

a fog" (Broglio et al., 2017). For more effective management and treatment of this injury, symptoms are often categorized into clinical profiles (Kontos et al., 2019) (Table 2).

Table 2 Clinical profiles of concussion symptoms from Harmon et al. (2019)

Clinical Profile	Associated Symptoms
Cognition	Difficulty remembering, difficulty concentrating, confusion
Fatigue	Fatigue or low energy, feeling slowed down, trouble falling asleep
Anxiety/Mood	Nervous or Anxious, sadness, irritability, more emotional
Headache/Migraine	Headache, "pressure in head", neck pain, sensitivity to light, sensitivity to noise
Ocular	Blurred vision
Vestibular	Balance problems, nausea, or vomiting

Although certain athletes may exhibit a distinct clinical profile, it is more common for athletes to manifest multiple profiles concurrently. Given that symptoms align with more than one profile (Kontos et al., 2019), they are best conceptualized as interrelated and mutually reinforcing rather than as discrete manifestations of a single underlying pathology (Iverson, 2019). Among these, the fatigue/sleep cluster, which often includes symptoms such as "Trouble falling asleep", "Drowsiness", and "Fatigue or low energy", is said to play a critical role in post-concussion recovery, but this relationship has been insufficiently investigated (DuPrey et al., 2022).

2.1.1.2 *Clinical Recovery*

The severity of an athlete's initial symptoms in the first few days after injury is the most consistent predictor of recovery from SRC (Patricios et al., 2023). As symptoms typically improve rapidly within the first two weeks, most adult athletes recover from a clinical perspective (asymptomatic) within 10-14 days after injury (Harmon et al., 2019; McCrory et al., 2017). It is noteworthy that female athletes have been found to report more symptoms and experience a longer symptom resolution duration than male athletes (Bretzin et al., 2022; Covassin et al., 2013). These sex differences have been postulated to rely on alterations in neuroanatomy, CBF rates, hormones, and strength in neck muscles (Covassin et al., 2013; Koerte et al., 2020).

Unfortunately, several studies are showing that athletes from various sports tend to underreport and minimize concussion symptoms out of fear of being prevented from playing, underestimating the injury (severity), and due to a lack of SRC awareness and education (Delaney et al., 2018; Meier, Brummel, et al., 2015). Hence, defining clinical recovery solely based on a single variable, such as being symptom-free, may increase

the risk of making premature RTS decisions. The CISG recommends incorporating three elements into clinical evaluation and future research to determine recovery:

1. Evaluate symptom reports, including the resolution of concussion-related symptoms at rest, during cognitive activities, and following physical exertion.
2. Consider other relevant outcomes that affect ongoing symptoms or specific research questions (e.g., response to physical exertion)
3. Include measures of return to activity, such as return to learn and RTS.

(Patricios et al., 2023).

The proposed RTS strategy by the CISG consists of a six-step protocol that gradually increases the intensity and complexity of physical activity, ensuring that the athlete does not experience a recurrence of concussion symptoms. After an initial rest period of 24-48 hours, each step should take at least 24 hours, leading to a minimum time frame of one week from step one to step six (Table 3) (Patricios et al., 2023). Over the last two decades, reported RTS durations increased, with a recent meta-analysis reporting a mean duration of 19.8 days (Putukian et al., 2023). Typically, an athlete completes the RTS protocol within one month after the injury (Patricios et al., 2023).

Table 3 Return to sport (RTS) strategy. Modified from Patricios et al. (2023)

Step	Exercise strategy	Activity at each step	Goal
1	Symptom-limited activity	Daily activities that do not exacerbate symptoms (e.g., walking).	Gradual reintroduction of work/school
2	Aerobic exercise 2A-Light (up to approximately 55% maxHR) then 2B-Moderate (up to approximately 70% maxHR)	Stationary cycling or walking at slow to medium pace. May start light resistance training that does not result in more than mild and brief exacerbation of concussion symptoms.	Increase heart rate
3	Individual sport-specific exercise Note: If sport-specific training involves any risk of inadvertent head impact, medical clearance should occur before Step 3	Sport-specific training away from the team environment (e.g., running, change of direction, and/or individual training drills away from the team environment). No activities at risk of head impact.	Add movement, change of direction
4	Non-contact training drills	Exercise to high intensity including more challenging training drills (e.g., passing drills, multiplayer training) can integrate into a team environment.	Resume usual intensity of exercise, coordination and increased thinking
5	Full contact practice	Participate in normal training activities.	Restore confidence and assess functional skills by coaching staff
6	Return to sport	Normal gameplay.	

Athletes may begin step 1 (e.g., symptom-limited activity) within 24 hours of injury
maxHR: predicted maximal heart rate according to age (e.g., 220 - age).

Annual baseline testing, including symptoms (of the SCAT), has been proposed as a “best practice” model to aid with the diagnosis and RTS decision after SRC (Harmon et al., 2019). This has been established in some high-level sports exhibiting great SRC prevalence rates (such as soccer, American football, or basketball), but baseline values are often unavailable in other sports. The CISG emphasizes the consideration of functional outcomes and the ability of athletes to regain the same level of performance they had prior to the injury. Generally, if symptoms do not subside within two to four weeks after the injury, the athlete should be referred to a multimodal evaluation (Patricios et al., 2023).

Around 10 to 30% of athletes manifest a prolonged recovery process, exhibiting ongoing symptoms longer than the typical time frame of clinical recovery (> 10–14 days in adults) (Makdissi et al., 2013; McCrory et al., 2017). The percentage varies depending on the group investigated and the defined periods used to classify "prolonged”. In the literature, the heterogeneous definitions range from > 10 days, > 21 days, > 28 days / a month, to even over 2 months (Makdissi et al., 2017). According to the CISG, “prolonged/persistent post-concussive symptoms” (PPCS) are defined as symptoms lasting longer than four weeks, regardless of age (Patricios et al., 2023). This aligns with research using magnetic resonance spectroscopy, electrophysiological data, and neuropsychological assessments, which suggest that functional disturbances can persist for up to 30 days following a concussion (Kamins et al., 2017). Healthcare providers must determine whether persistent symptoms are due to ongoing concussion-related pathophysiology or indicate a separate underlying condition. These may include concurrent injuries or medical conditions like migraines, depression (Leddy et al., 2012), learning or attention disorders, as well as visual, ocular-motor, cervical, vestibular problems, or dysautonomia (Yeates et al., 2023). Risk factors for symptoms persisting beyond a month include subacute issues, such as depression and migraines, and a pre-injury history of mental health problems (McCrory et al., 2017). Younger age and female sex are also associated with a higher risk of prolonged symptomatology (Conder et al., 2020). Additionally, sleep problems have been identified as a potential contributor to extended recovery (Hughes et al., 2022). This topic will be further explored in Chapter 2.3.

2.1.2 Neurophysiological Measurement Tools

Recovery from SRC is a multifaceted process, with different symptoms often following distinct trajectories, which contradicts the common assumption that recovery is a singular, linear concept (Mayer et al., 2017). A more comprehensive understanding of the relationship between the underlying pathophysiology and clinical symptoms is essential for optimizing treatment and tracking recovery (Kamins et al., 2017). Numerous neurophysiological tools have been utilized to investigate SRC pathophysiology, including neuroimaging techniques (MRI, functional MRI, Diffusion Tensor Imaging, and Magnetic Resonance Spectroscopy), as well as hemodynamic (Near-Infrared Spectroscopy, Single Photon Emission Computerized Tomography, Arterial Spin Labeling) and electrophysiological measures (Electroencephalography (EEG), Event-Related Potential, Magnetoencephalography). However, there is no consensus on which tool should be employed for monitoring SRC recovery, as there is currently no gold-standard test recognized for displaying the severity and progress of SRC recovery (Bishop et al., 2018; Brown et al., 2023).

In research settings, advanced neuroimaging, fluid biomarkers, electrophysiological measures, and modalities that assess autonomic dysfunction demonstrate encouraging sensitivity in detecting acute neurobiological effects and tracking changes throughout the SRC recovery process (Patricios et al., 2023). Nonetheless, most of these methods are often impractical in clinical settings due to their high costs, specialized equipment, and time-consuming protocols (Lunkova et al., 2021). However, measurements of ANS activity, tracking possible autonomic dysfunction, offer a non-invasive, cost-effective method and even enable mobile applications through wearable technology (Bishop et al., 2018; Coenen & Reinsberger, 2023; Tabor et al., 2023). Therefore, data collection from much larger and more diverse populations can be conducted compared to studies utilizing traditional neuroimaging methods (Johnson & Picard, 2020).

Wearable technology refers to electronic devices worn on the body, typically as accessories (e.g., wristbands, smartphones, smartwatches, rings) (Nuuttila et al., 2025). These devices implement sensors that monitor physiological signals, such as HR, electrodermal activity, skin temperature, breathing rate, and movement (Schwartz & Baca, 2016). The key advantage of wearables lies in their ability to provide ambulatory, long-term assessments of vital signals during daily life under real-world conditions (Mühlen et al., 2021).

2.2 Autonomic Nervous System Activity for Monitoring Recovery

The ANS plays an essential role in maintaining the body's internal homeostasis, as it impacts, without conscious perception, most organ functions, including cardiovascular and respiratory control, thermal regulation, digestive processes, reproduction, and metabolic and endocrine physiology (Jänig, 2008). Therefore, parameters of the ANS have been applied to assess the recovery and adaptation of athletes in response to training (Bellenger et al., 2016), while also being clinically utilized to diagnose and evaluate various neurological diseases and disorders (Hilz & Dütsch, 2005).

2.2.1 Anatomy and Physiology of the Autonomic Nervous System

The ANS provides innervation to three types of tissues, influencing most tissues and organ systems in the body: glands, smooth muscle, and cardiac muscle. Consequently, the ANS influences nearly every part of the body (Bear et al., 2020). Traditionally, the ANS is anatomically classified into two divisions: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS), each consisting of central and peripheral components.

The SNS enables energy mobilization in the body in response to stress, excitement, and arousal ("fight or flight" response), while the PNS promotes restorative functions and restfulness ("rest or digest" response). A relative dynamic balance between the sympathetic and parasympathetic divisions can be observed in healthy organisms (Shaffer et al., 2014). In contrast, an imbalance, such as a predominance of one division, is associated with pathological conditions, maladaptation (Thayer et al., 2010), and increasing age (Abhishekh et al., 2013). Additionally, the enteric nervous system is the third division of the ANS, controlling the gastrointestinal tract (Chokroverty & Cortelli, 2021).

For the regulation of ANS activity and its adaptation to changing conditions (Sklerov et al., 2019), the central autonomic network (CAN) has been proposed as a functional "internal regulation system of the brain" (Benarroch, 1993). The CAN consists of multiple interconnected regions within the telencephalon, diencephalon, and brainstem (Shouman & Benarroch, 2022). Human neuroimaging studies explored the main cerebral and cerebellar regions involved in the CAN, which include the anterior and midcingulate cortices, the insula, the ventromedial prefrontal cortex (vmPFC), the thalamus, the amygdala, the hippocampal formation, and the hypothalamus (Beissner et al., 2013). The latter serves as the primary regulator of the ANS (Bear et al., 2020),

receiving input from higher cortical centers (the insula and prefrontal cortex), and transmitting information to the brainstem and spinal cord (Takahashi et al., 2015). Preganglionic sympathetic and parasympathetic neurons located in the gray matter of the brain or spinal cord serve as the primary conduits for the output of the CAN (Thayer et al., 2010). They are regulating various organs and their functions, such as the heart. Furthermore, sensory information originating from peripheral end organs is relayed to the CAN (Thayer & Brosschot, 2005).

Sympathetic preganglionic nerve fibers leave along the ventral roots of the thoracolumbar spinal cord (C8-T1 to L1–L2 segments). In contrast, the parasympathetic preganglionic nerve fibers stem from the cranial brain stem (3rd, 7th, 9th, and 10th (vagus nerve) cranial nerves) and the sacral spinal cord (S2-S4 segments) (Waheed & Vizzard, 2022). The preganglionic fibers are thinly myelinated for both divisions, and acetylcholine (ACh) serves as the preganglionic neurotransmitter. The sympathetic preganglionic fibers are relatively short, whereas the parasympathetic fibers are long. The postganglionic neurons in the ganglia outside the central nervous system (CNS) send unmyelinated nerves to innervate organs throughout the body (Gibbons, 2019). The sympathetic postganglionic fibers are longer, while the parasympathetic postganglionic fibers are relatively shorter. ACh is the postganglionic neurotransmitter for the parasympathetic division, whereas epinephrine/norepinephrine are the neurotransmitters for the sympathetic division. However, there are a few exceptions to this pattern of sympathetic postganglionic neurotransmission: sweat glands are innervated by postganglionic nerves through ACh (Hu et al., 2018), and the kidney through dopamine (Gibbons, 2019).

2.2.2 Measures and Parameters of Autonomic Activity

Several methods are used to assess ANS activity. In many neurological and internal diseases, evidence suggests that autonomic imbalance often manifests as increased SNS and decreased PNS activity (Thayer & Brosschot, 2005), which may lead to organ dysfunction (Khalid et al., 2019). Cardiac (HR and HRV) and sudomotor autonomic function (electrodermal activity) are commonly used to investigate ANS dysfunction (Hilz & Dütsch, 2005). Although they are obtained peripherally, they serve as indirect markers of CAN activity. Thereby, HRV is the most frequently studied marker of autonomic dysfunction after SRC (Brown et al., 2023).

2.2.2.1 Heart Rate Variability

HRV refers to the fluctuation in time intervals between successive heartbeats (called interbeat intervals (IBIs)) (Shaffer & Ginsberg, 2017). As the heart is under tonic autonomic control, with the sympathetic division associated with accelerating HR (shortening IBIs and reduced HRV) and the parasympathetic division in HR deceleration (longer IBIs and greater HRV) (Thayer & Lane, 2009), it serves as an indicator of the ANS to respond and adapt to external stimuli or psychological and physical challenges to homeostasis (Shaffer & Ginsberg, 2017). Parasympathetic activity immediately reduces HR (on a beat-by-beat basis) due to the rapid effect of synaptic released ACh, while sympathetic activity triggers the release of noradrenaline, which enhances cardiac contractility and HR. However, the action of noradrenaline is comparatively slower than that of ACh, resulting in a delay of approximately 5 seconds between the onset of sympathetic stimulation and the subsequent changes in HR (Draghici et al., 2016). Hence, abrupt fluctuations in HR are primarily influenced by the PNS (Shaffer, 2014; Johnson et al., 2019).

In a healthy state, changes in HR arise from dynamic, non-linear interactions between multiple physiological systems, exhibiting spatial and temporal complexity to provide flexibility in coping with changing conditions and environmental factors (Shaffer, 2014). This is why a healthy heart displays spontaneous oscillations in HR, while a diseased heart exhibits minimal variability under specific conditions (Thayer et al., 2012). “The heart is not a metronome” (Shaffer et al., 2014). Therefore, a higher HRV is widely regarded as a reliable indicator of cardiovascular health, reflecting increased adaptability and resilience within the ANS (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996).

In humans, HR is primarily under peripheral inhibitory control of the vagus nerve (the main nerve of the PNS) (Uijtdehaage & Thayer, 2000). Findings from pharmacological blockade and neuroimaging studies offer substantial evidence for the involvement of the prefrontal cortex in cardiac vagal function (Thayer & Lane, 2009). In safe conditions, the prefrontal cortex, especially the vmPFC, seems to tonically inhibit the "fear" or threat-related responses in the amygdala. On the contrary, in situations characterized by uncertainty and danger, key regions of the prefrontal cortex exhibit reduced activity. This hypoactive state leads to a lack of inhibition in sympathoexcitatory circuits, resulting in hyperactivation of the amygdala. Consequently, a decrease in the complexity of neurogenic rhythms and a reduction in HRV follow (Thayer et al., 2012). For this reason, HRV can serve as a measurable indicator, reflecting the function of the

heart's neural control (CAN) and its effective regulation in response to external factors and influences. This is defined by Thayer (2012) as the “Neurovisceral Integration Model”.

While the term “HRV” is often referred to as consisting of a single parameter, several different metrics of HRV capture various components of HRV (Hottenrott & Gronwald, 2014). Traditionally, HRV metrics are methodically classified into time-domain, spectral-domain, and non-linear parameters (Table 4) (Sammito et al., 2024). Time-domain parameters measure the level of HRV (the degree of variation in IBIs) observed over a fixed analyzing time (e.g., 1 minute to 24 hours). One of the most studied time-domain parameters is the root mean square of successive differences between normal heartbeats (RMSSD), as it reflects pure parasympathetic activity (Shaffer & Ginsberg, 2017).

Frequency-domain parameters determine the absolute or relative signal power within specific frequency bands, with high-frequency (HF) HRV predominantly reflecting parasympathetic activity. In contrast, lower frequencies (LF) (below approximately 0.15 Hz) involve a combination of both sympathetic and parasympathetic autonomic activity (Thayer et al., 2012). Non-linear parameters display the unpredictability and complexity of a series of IBIs (Shaffer & Ginsberg, 2017).

Table 4 Selection of HRV parameters. Modified from Shaffer & Ginsberg (2017) and Sammito et al. (2024)

Parameter (unit)	Description	Assigned ANS activity	Evaluation time
Time domain parameters			
SDNN (ms)	Standard deviation of NN intervals	No clear assignment	
SDANN (ms)	Standard deviation of the average NN intervals for each 5 min segment of a 24 h HRV recording	No clear assignment	Long-term recording, ideally 24 hours
SDNN index (SDNNI)	Mean of the standard deviations of all the NN intervals for each 5 min segment of a 24 h HRV recording	No clear assignment	Long-term recording, ideally 24 hours
pNN50 (%)	Percentage of successive NN intervals that differ by more than 50 ms	Parasympathetic	At least 2 min
NN50	The number of pairs of neighboring NN intervals that deviate from one another by more than 50 ms	Parasympathetic	At least 2 min
RMSSD (ms)	Root mean square of successive NN interval differences	Parasympathetic	Proposed: 5 min
Frequency domain parameters			
ULF (ms^2)	Absolute power of the ultra-low-frequency band (≤0.003 Hz)	No clear assignment	Ideally 24 hours
VLF (ms^2)	Absolute power of the very-low-frequency band (0.0033–0.04 Hz)	No clear assignment	At least 5 minutes, ideally 24 hours

LF (ms^2)	Absolute power of the low-frequency band (0.04–0.15 Hz)	Sympathetic and parasympathetic	At least 2 min
LFnu	Relative power of the low-frequency band (0.04–0.15 Hz) in normal units	Sympathetic and parasympathetic	
HF (ms^2)	Absolute power of the high-frequency band (0.15–0.4 Hz)	Parasympathetic	Proposed: 5 min
HFnu	Relative power of the high-frequency band (0.15–0.4 Hz) in normal units	Parasympathetic	
LF/HF	Ratio of LF-to-HF power	Sympathetic and parasympathetic	At least 5 minutes
Total power (ms^2)	Total power: total performance or total spectrum; corresponds to energy density between 0.00001 to 0.4 Hz	No clear assignment	
Non-linear parameters			
SD1 (ms)	Poincaré plot standard deviation perpendicular the line of identity	Parasympathetic	
SD2 (ms)	Poincaré plot standard deviation along the line of identity	Sympathetic and parasympathetic	
SD1/SD2	Ratio of SD1-to-SD2	Sympathetic and parasympathetic	
ApEn	Approximate entropy, which measures the regularity and complexity of a time series	No clear assignment	
SampEn	Sample entropy, which measures the regularity and complexity of a time series	No clear assignment	
DFA α1	Detrended fluctuation analysis, which describes short-term fluctuations	No clear assignment	
DFA α2	Detrended fluctuation analysis, which describes long-term fluctuations	No clear assignment	

ANS = autonomic nervous system; NN = normal-to-normal intervals

It is well established that HRV diminishes as one ages (Thayer et al., 2010) and that women exhibit increased vagal tone despite showing a higher mean HR than men (Shaffer & Ginsberg, 2017). Additionally, research has shown that the frequency of respiration can influence HRV. Inhaling accelerates the HR, while exhaling decelerates it. This physiological phenomenon is known as respiratory sinus arrhythmia (RSA). Therefore, HRV tends to increase when the respiratory frequency decreases (Draghici & Taylor, 2016).

In medical research, HRV has proven to be a valuable tool for predicting the occurrence of various health problems, including mental issues like stress, depression, anxiety, and post-traumatic stress disorder (PTSD), as well as physical disorders such as inflammation, chronic pain, diabetes, asthma, insomnia, fatigue, and concussion. These conditions often lead to increased sympathetic activity (Shaffer & Ginsberg, 2017), as they are associated with prefrontal hypoactivity (Thayer & Brosschot, 2005). Unhealthy lifestyle choices, such as insufficient physical activity and the intake of tobacco, alcohol, and drugs, have been further linked to autonomic imbalance and reduced parasympathetic activity. In the sport science field, HRV serves as a tool for

adapting training intensity, diagnosing and preventing fatigue, assessing overtraining, and determining aerobic capacity (Da Silva et al., 2015).

The gold standard for measuring HR and subsequently calculating HRV is through an electrocardiogram (ECG) (Sammito et al., 2024). In recent years, various wearables implementing pulse rate monitors using photoplethysmography (PPG) technology have been developed and have become widely available. PPG is a relatively inexpensive and practical technique for identifying changes in blood flow volume in peripheral blood circulation at the skin surface through the absorption and reflection of emitted light (Georgiou et al., 2018; Mühlen et al., 2021). It has been demonstrated that data obtained from wrist-worn PPG devices are sufficiently accurate for both HRV analysis and distinguishing between cases of sinus rhythm and atrial fibrillation (Tarniceriu et al., 2018).

2.2.2.2 Electrodermal Activity

While only a limited number of HRV parameters, such as RMSSD and the HF band (Table 4), are considered to primarily reflect parasympathetic activity, no single HRV parameter exclusively represents sympathetic activity. Therefore, another measurement tool is necessary to investigate both divisions of the ANS. Dawson et al. (2007) noted that “when ANS activity is of primary interest, electrodermal activity and heart rate are probably the two most common choices…”.

EDA (formerly called galvanic skin response) refers to changes in the electrical properties of the skin that occur as a result of SNS activity (Boucsein, 2012). Methodically, EDA can be differentiated into skin conductance or skin resistance, depending on the measurement method. When applying a direct current mode with a constant voltage to the skin's surface, EDA is called skin conductance (SC). This is the most widely applied method in psychophysiology (Boucsein et al., 2012). Therefore, EDA is often referred to as SC (Gersak, 2020). Conversely, it is identified as skin resistance (SR) in direct current mode with a constant current.

Changes in the conductance or resistance of the skin are caused by sweat secretion, which results from the activation of eccrine sweat (sudomotor) glands. These glands are innervated by sole sympathetic activity due to the distinctive postganglionic sympathetic transmitter ACh (see Chapter 2.2.1) (Boucsein, 2012). They are distributed throughout the entire skin, with the highest density in palmar and plantar regions (Critchley, 2002). Thus, EDA can be optimally assessed by placing wired and gelled electrodes on the fingers or palms (Bouscein, 1992). However, numerous studies

showed that EDA can be accurately measured in alternative locations, such as the forearm (van Dooren, 2012). Research using dry electrodes on the forearm (e.g., on the wrist as a wearable) has provided consistent and reliable ambulatory long-term EDA measurements (Poh, 2012).

The sympathetic activity involved in sweating can be related to various brain regions, including the cortex, basal ganglia, diencephalic structures such as the thalamus and hypothalamus, the limbic system, and several areas within the brain stem (Boucsein, 2012). In psychophysiology, EDA has found extensive application as a reliable indicator of SNS responses related to emotions, arousal (physiological and psychological activation of the organism through central nervous activation), attention, and in medical research for evaluating (psycho-)pathological conditions (such as epilepsy, anxiety, depression) (Boucsein et al., 2012). In emotion and arousal research, laterality in EDA measurements has been frequently described in relation to specific cortical and subcortical structures. Limbic regions, such as the amygdala and hippocampus, are associated with eliciting EDA ipsilaterally (on the same side of the body), whereas other regions, mainly the premotor cortex and basal ganglia, elicit EDA contralaterally (on the opposite side of the body). Therefore, the side of measurement should be consistent in an investigation, with the nondominant side primarily utilized (Picard et al., 2016). These lateralized effects are proposed to reflect the contribution of partially independent but interacting arousal systems: emotional (e.g., limbic regions), cognitive (e.g., prefrontal cortex), and motor (e.g., premotor cortex) (Picard et al., 2016).

EDA generally consists of two components that must be analyzed separately (Boucsein et al., 2012). The tonic component refers to the slowly changing baseline and characteristics of the EDA signal, the EDA level (EDL). Interindividual variation in EDL can be related to diverse constitutions and individual skin compositions (Reinsberger et al., 2015). Observing changes in EDA levels typically requires a minimum of 10 to 30 seconds (Boucsein, 2012). The phasic component consists of rapid transient changes, known as EDA responses (EDRs), that overlay the tonic activity in small waves (Figure 2) (Benedek & Kaernbach, 2010).

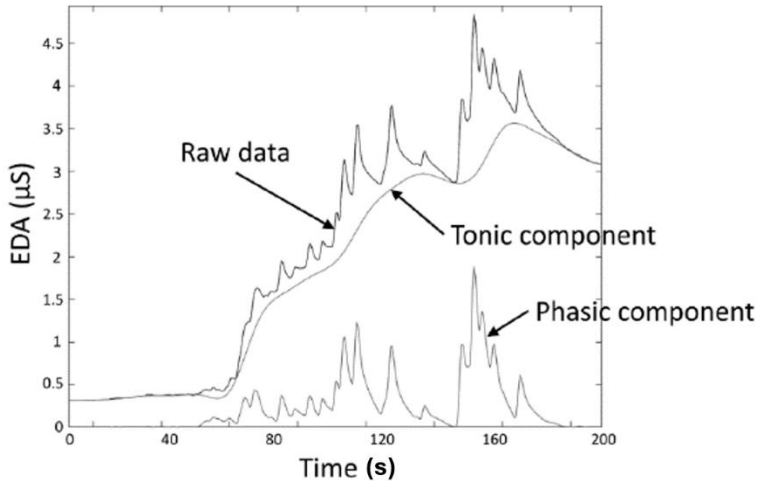

Figure 2 EDA signal and its decomposition into phasic and tonic components. Reproduced with permission from: Posada-Quintero & Chon, 2020

Thereby, EDRs can be categorized into stimulus-specific responses or non-specific responses (NS.EDRs), which can also emerge during sleep. Intraindividual differences in EDA are proposed to reflect situational levels of arousal or activation (Dawson et al., 2007), with higher levels of SNS arousal leading to increased EDA (Posada-Quintero & Chon, 2020). An overview of selected EDA parameters is displayed in Table 5.

Table 5 Selected EDA parameters. Modified from Bouscein (2012)

	Electrodermal activity	**Skin conductance**	**Skin resistance**
Method		Direct current, constant voltage	Direct current, constant current
Abbreviations			
In general	EDA	SC	SR
Tonic (level)	EDL	SCL	SRL
Phasic (response)	EDR	SCR	SRR
Nonspecific response	NS.EDR	NS.SCR	NS.SRR

EDA can exhibit higher values during the night than during the day (Johnson & Lubin, 1966), although one would expect a low level of arousal during sleep. These nocturnal “surges” of elevated EDA (Reinsberger et al., 2015), characterized by high-frequency patterns of EDA, are referred to as “sleep storms” (Sano & Picard, 2011). They are commonly observed during deep sleep stages and are associated with memory processes (Sano et al., 2014), although their underlying physiological mechanisms remain poorly understood (Onton et al., 2016).

Several internal and external factors must be considered for EDA measurements. Internal variables include age, gender, and ethnicity of the participants. Reduced phasic and tonic EDA activity has been linked to older age, possibly due to lower arousal levels, fewer active sweat glands, and changes in the CNS (such as decreased gray matter in brain regions involved in EDA). The influences of gender (e.g., hormonal fluctuations that occur monthly in women) and ethnicity (e.g., related to the reduction in active sweat glands as skin darkness increases) on EDA have shown less consistency. External factors include ambient temperature and humidity. Higher temperatures decrease skin resistance because sweat gland activity increases. Additionally, drugs and medications (e.g., those with anticholinergic effects that interfere with EDA's cholinergic mediation) and caffeine (which stimulates SNS activity) can also affect EDA (Boucsein et al., 2012).

2.2.3 Autonomic Dysfunction after Sport-related Concussion

The pathophysiology of SRC (see Chapter 2.1), particularly DAI, has been proposed to affect various brain areas, potentially leading to changes in communication within and between brain networks (Chong & Schwedt, 2015; Dobson et al., 2017). Subcortical structures associated with the CAN, such as the brainstem and hypothalamus, may be particularly vulnerable to rotational forces from biomechanical trauma. Their central location along the vector path of mechanical load makes them prone to shear and strain forces (Leddy et al., 2017; Snyder et al., 2021). Advanced neuroimaging studies have shown microstructural and functional changes in the brain following SRC (Churchill et al., 2017), especially in regions implicated in the CAN, including the cerebral cortex, midbrain, and brainstem (Pyndiura et al., 2020). Furthermore, it is increasingly recognized that with greater injury severity, the pathophysiological effects of concussion extend to deeper brain structures, such as the brainstem (Flores et al., 2023), potentially heightening the risk of autonomic dysfunction. An imbalance between the SNS and the PNS is thus hypothesized to reflect the neurophysiological state of SRC, with alterations in autonomic activity serving as possible markers of injury severity and recovery (Flores et al., 2023).

Changes in cardiac ANS activity, as measured by HRV parameters, have commonly been investigated after SRC at rest and during physical stress (e.g., exercise, orthostatic challenges, Valsalva maneuver) (Mercier et al., 2022). Thus, HRV has been suggested as a recovery marker for SRC (Hutchison et al., 2017; Senthinathan et al., 2017). However, findings during resting conditions have been conflicting, with some studies reporting changes in ANS activity in concussed athletes compared to controls

(Bishop et al., 2017; Hutchison et al., 2017; Purkayastha, Williams, et al., 2019; Senthinathan et al., 2017), while some did not detect this (Harrison et al., 2022; La Fountaine et al., 2009; Paniccia et al., 2018; Pyndiura et al., 2020). Other studies identified differences only during exercise (Abaji et al., 2016; Gall et al., 2004; La Fountaine et al., 2009) or physical challenges (Haider et al., 2020; Johnson et al., 2018). An overview of published studies investigating HRV in athletes after SRC is presented in Table 6.

Table 6 Studies investigating HRV in athletes after SRC

Author (year)	Sample	Timepoint(s) and measure condition	Results*: PNS parameters	Results*: SNS/PNS and non-linear parameters
Gall et al. (2004)	n = 14 athletes n = 14 controls	2 (rest: within 72 h after SRC, exercise: mean 5 days; around 1 week) rest (seated) exercise (cycling)	rest: no differences in HF during exercise: reduced HF at both time points	rest: no differences in SDNN, LF, LF/HF ratio during exercise: reduced LF at both time points
La Fountaine et al. (2009)	n = 3 symptomatic athletes n = 3 matched controls	2 (under 48 h after SRC; 2 weeks later) rest (seated) exercise (isometric hand grip contraction (IHGC))		reduced approximate entropy during IHGC at time point 1
Abaji et al. (2016)	n = 12 asymptomatic athletes n = 12 matched controls	1 (mean 95 days after SRC) rest (seated) exercise	rest: no differences in RMSSD during exercise: reduced HF	no differences in HR, LF, and SDNN during exercise: higher LF/HF ratio
Bishop et al. (2017)	n = 12 asymptomatic athletes n = 89 controls	1 (within 72 h after SRC) rest (seated)	reduced pNN50 / NN50	no differences in LF or LF/HF ratio
Hutchison et al. (2017)	n = 26 athletes n = 26 matched controls	3 (symptomatic; asymptomatic exercise phase; 1-week post RTS) rest (seated)	reduced HF (at all 3 time points)	reduced SDNN, higher LF/HF ratio at time point 3
Senthinathan et al. (2017)	n = 11 symptomatic athletes n = 11 matched controls	3 (symptomatic; asymptomatic exercise phase; 1-week post RTS) rest (seated, standing)	sitting: reduced HF norm values at time point 1	sitting: increased LF norm at time point 1; reduced sample entropy (during standing) over all 3 time points
Johnson et al. (2018)	n = 11 symptomatic athletes n = 10 controls	1 (within 10 days after SRC) rest (supine) face cooling (FC)	rest: no differences in HF and RMSSD; less increase in HF during the first minute of FC	rest: no differences in HR

Paniccia et al. (2018)	n = 29 youth athletes n = 15 matched controls	Individual (3-5 time points) Baseline measure (24 h, median: 93 days after injury) Individual follow-ups	baseline: no differences in RMSSD, pNN50, HF, and HFnu	baseline: no differences in HR and SDNN
Balestrini et al. (2019)	n = 65 recreational and competitive athletes n = 54 matched controls	SRC: Individual (weekly until medical clearance) Controls: 2 Rest (supine, seated, standing)	reduced RMSSD (seated) only in female athletes	higher HR (in each posture) on the first visit
Huang et al. (2019)	n = 23 symptomatic athletes n = 23 matched controls	1 (within 4 days after SRC) rest (seated) cognitive task	rest: reduced %HF	
La Fountaine et al. (2019)	n = 10 athletes n = 10 matched controls	2 (within 48 h after SRC, 1 week later) rest (seated)	no differences in HF	no differences in HR and LF
Purkayastha et al. (2019)	n = 31 symptomatic athletes n = 31 controls	3 (day 3, day 21, day 90 after SRC) rest (seated)	reduced RMSSD, pNN50 & HF at time point 1	
Haider et al. (2020)	n = 9 asymptomatic athletes n = 21 matched controls	1 (more than 1 year ago) rest (seated) face cooling (FC)	less increase in RMSSD to FC	reduced HR at rest; no differences in the LF/HF ratio
Pyndiura et al. (2020)	n = 41 asymptomatic athletes n = 72 control athletes	1 (median 3 years after SRC) rest (seated, supine)	no differences in HF	no differences in HR, LF, LF/HF ratio and SDNN
Memmini et al. (2021)	n = 15 asymptomatic athletes n = 18 controls (teammates)	1 (more than 6 months after SRC) rest (seated, pre and post-exercise)	pre-exercise: no differences in RMSSD, pNN50 post-exercise: The SRC group took longer to get to baseline values	pre-exercise: no differences in HR
Ellingson et al. (2022)	n = 30 athletes with HRV baseline values	1 (within five days after SRC) rest and controlled breathing protocol	no differences in HF and SD1 compared to baseline values	reduced SD2 and SDNN compared to baseline values no differences in LF and LF/HF ratio
La Fountaine et al. (2022)	n = 19 athletes n = controls	2 (within 48 h and 1 week of injury) rest (seated)	no differences in HF	no differences in HR, LF
Harrison et al. (2022)	n = 16 asymptomatic athletes n = 18 controls (teammates)	1 (mean: 2 years after SRC) rest cognitive test	rest: no difference in RMSSD during the cognitive test and after	rest and cognitive test: no difference in HR and SDNN

		cognitive test after exercise	exercise: higher RMSSD	during cognitive test and cognitive test after exercise: higher SDNN
Doucet et al. (2023)	n = 30 student-athletes n = 169 baseline controls	3 (baseline, 72 h after concussion and before returning to play) rest (2 min standing and 2 min sitting)	no significant effect of time was found for RMSSD, HF	no significant effect of time was found for VLF
Ji et al. (2024)	n = 26 adolescents n = 26 matched controls	2 (within 10 days after SRC and one month after RTS) rest (supine)	no significant differences in HF and RMSSD	
Haider et al. (2025)	n = 23 adolescents n = 24 controls	2 (within 10 days after SRC and after clinical recovery) face cooling (FC)	no significant difference in RMSSD	no significant difference in HR

**Cross-sectional results are stated for the concussed athletes in comparison to controls. (Exception: the study by Ellingson et al., 2022)*
PNS = parasympathetic nervous system; SNS = sympathetic nervous system

Possible confounders for the presented inconsistent results can be related to the heterogeneity in methods (HRV parameters, analysis duration, measurement condition, duration of measurement), study design (cross-section vs. longitudinal), subjects (age, sex, concussion history, other medical conditions and medications, sport and level of sport performance, fitness level), injury characteristics (definition of SRC, injury severity, time since injury) and the high intra- and interindividual variability in ANS activity. As baseline HRV values are not typically available in studies, a matched control group is often included for comparison. Although this approach incorporates reference values of healthy subjects, it does not account for intrapersonal individuality in ANS parameters. While Paniccia et al. (2018) did not find significant differences in youth SRC athletes compared to controls, they described an initial decrease (30-40 days after SRC) in parasympathetic activity (RMSSD, pNN50), followed by an increase until day 75. This highlights the necessity for longitudinal studies to obtain HRV values after the typical clinical recovery duration (1 month) from SRC and compare them to those acutely after SRC.

Although the literature (see Table 6) provides evidence of autonomic dysfunction after SRC, the confounding factors mentioned above must be considered. Due to the predominant representation of parasympathetic activity through HRV parameters (e.g., RMSSD, HF, pNN50/NN50), most research has focused on the parasympathetic division of the ANS. A tendency towards parasympathetic inhibition has been observed acutely after SRC, especially during physical challenges such as exercise (Patricios et al., 2023).

In contrast, sympathetic activity has been less consistently investigated (Pertab et al., 2018), with the LF/HF ratio being the primary metric examined. However, the accuracy of this ratio as an index of sympathetic dominance is debated, as the LF band reflects both parasympathetic and sympathetic activity (see Table 4) (Thomas et al., 2019). Consequently, it remains unclear whether autonomic dysfunctions observed after SRC are solely due to parasympathetic inhibition or involve increased sympathetic activity as well (Johnson et al., 2018).

Research conducted on various TBI severities suggests that there is an increased sympathetic activity following the injury (Khalid et al., 2019). Similarly, studies on SRC using cardiovascular metrics have generally reported increased SNS activity. For instance, La Fountaine et al. (2016) investigated athletes within 48 hours and the first week after SRC, examining changes in finger arterial pulse contours at rest and during a low-intensity isometric handgrip test. He described increased peripheral artery stiffness in SRC athletes compared to controls, representing a higher SNS activity. Further, Dobson et al. (2017) conducted autonomic tests on recreational athletes after SRC four times: 1) within 48 hours of injury; 2) 24 hours later; 3) one week after injury; and 4) two weeks after injury. Within 48 hours, SRC athletes exhibited increased resting systolic blood pressure (SBP), HR, and SBP perturbations due to enhanced SNS activity.

Research on SNS activity apart from cardiac metrics, such as investigations in EDA, has been rare. One study investigated EDA during standardized neurocognitive assessment of concussion between subjects with and without a history of concussion (Raikes & Schaefer, 2016). They found that subjects with a concussion history showed increased arousal (represented by larger phasic EDA) during a memory task than subjects without a concussion history. On the contrary, two other studies found hypoarousal (lower levels of EDA) in subjects with a history of TBI in a decision-making task (van Noordt & Good, 2011) and an executive function test (O'Keeffe et al., 2004). Since all these studies have been retrospective, the acute effects of SRC on EDA require further investigation.

These findings suggest that both branches of the ANS may be affected after SRC. Acutely, SRC appears to evoke an inhibition of parasympathetic activity and activation of sympathetic activity (Flores et al., 2023). The simultaneous investigation of HRV (parasympathetic activity) and EDA (sympathetic activity) using wearable devices can provide a more comprehensive understanding of the impact of SRC on both divisions of the ANS in a real-world setting, contrary to previously conducted research in the laboratory (Davis-Wilson et al., 2025).

Besides, the relationship between autonomic dysfunction and concussion symptoms has been poorly investigated. As SRC is related to a wide range of clinical symptoms and profiles (see Chapter 2.1.1), autonomic dysfunction is proposed as one of the contributing factors in the clinical presentation of headache, cognitive impairment, mood disorders, anxiety, and sleep problems (Purkayastha, Stokes, & Bell, 2019). Furthermore, autonomic dysfunction has been observed in other medical conditions (e.g., migraine, anxiety, chronic fatigue syndrome, depression, sleep problems), which can manifest similar symptoms as SRC (Pertab et al., 2018).

Research on moderate to severe TBI in humans and rodents has explored the relationship between pathophysiology (see Chapter 2.1) and symptoms (Hallock et al., 2023). Altered ionic flux in the brain has been proposed as a potential cause of migraines, headaches, and sensitivity to light or noise. At the same time, DAI and alterations in neurotransmission have been associated with cognitive impairments and slower processing and reaction times (Giza & Hovda, 2014). Furthermore, DAI in the hippocampus can lead to persistent deficits in memory consolidation and a reduced connection to other brain regions, including the prefrontal cortex and the amygdala (Wolf & Koch, 2016). Both structures are essential for central autonomic regulation.

Reductions in global and regional CBF post-injury have been found to correlate with diminished cognitive function and a longer RTS duration in college athletes, indicating a link between CBF and functional recovery (Meier, Bellgowan, et al., 2015), as the brain struggles to receive adequate oxygen and nutrients necessary for healing and normal function (Purkayastha, Williams, et al., 2019). Although the ANS is not solely responsible for cerebral perfusion and thus CBF, it plays a significant role. Post-injury hypoperfusion of the insular cortex, the central location for integrating sympathetic and parasympathetic activity, has been discussed as one possible mechanism behind ANS dysfunction (Purkayastha, Williams, et al., 2019).

Hypoconnectivity within the prefrontal cortex was found to be acutely present in post-injury athletes (within one week post-injury) compared to three weeks after SRC (Murdaugh et al., 2018). Damage to the prefrontal areas of the CAN can lead to the disinhibition of the central nucleus of the amygdala, typically under tonic inhibition of the prefrontal cortex (Thayer & Lane, 2009). This disinhibition, in turn, results in increased sympathetic activity due to the subsequent activation of sympathoexcitatory neurons in the ventrolateral medulla, affecting HR and HRV (see Chapter 2.2.2.1). Low HRV, representing hypoactivity of the prefrontal cortex, reflects impaired behavioral and cognitive adaptation following mTBI and has been linked to anxiety, depression, and

PTSD, common disorders after concussion (Purkayastha, Stokes, & Bell, 2019). Reduction in prefrontal cortex activity has also been observed in individuals experiencing sleep deprivation, revealing a possible link between SRC and sleep-associated symptoms following this injury (Kontos et al., 2019). In contrast, increased arousal has been considered another physiological mechanism behind sleep problems in mTBI patients (Wickwire et al., 2018), and similar mechanisms have been suggested for athletes with SRC (Considine et al., 2021). Symptoms such as irritability, difficulty concentrating, and hypervigilance closely resemble the hyperarousal dimensions seen in PTSD, further supporting the idea that elevated arousal may drive specific concussion-related symptoms (Polinder et al., 2018).

Neuroinflammation plays a crucial role in the pathophysiology of mTBI. It has been linked to various concussion symptoms, similar to those seen in conditions like headaches, chronic fatigue syndrome, and psychological issues such as depression and anxiety (Di Battista et al., 2020). Autonomic dysfunction can exacerbate neuroinflammatory responses, leading to prolonged symptoms and neurodegeneration, primarily due to heightened sympathetic activity (Purkayastha, Stokes, & Bell, 2019).

Currently, the complex relationship between autonomic dysfunction and concussion symptoms remains insufficiently understood, as many studies examining autonomic functioning after SRC did not report concussion symptoms (Mercier et al., 2022). However, concussion symptoms can, in part, be recognized as manifestations of autonomic dysfunction (Pertab et al., 2025).

2.3 Sleep as a Modifier of Sport-related Concussion

Sleep is a complex physiological process during which essential restorative processes occur, including cellular repair, clearing metabolic waste and toxins from the brain, the release of growth hormones, protein synthesis, and the consolidation of memories (Hauglund et al., 2020; Wickwire et al., 2018). Hence, it is the best recovery strategy for athletes (Halson, 2008). Considering the neurometabolic crisis and heightened energy demands after SRC, restorative processes such as sleep and synaptic plasticity during sleep play a crucial role in brain recovery (Jaffee et al., 2015). For this reason, it is not surprising that evidence advocates that sleep-associated problems following SRC are linked to longer recovery periods and more concussion symptoms (Gosselin et al., 2009; Hoffman et al., 2019b; Ludwig et al., 2019).

2.3.1 Sleep Problems and Sleep-associated Symptoms after Sport-related Concussion

Sleep-associated symptoms are among the most frequently reported symptoms following SRC, with 30 to 70% of athletes experiencing them (Stevens et al., 2022; Tkachenko et al., 2016). Although sleep problems (a general term encompassing both subjective symptoms and objective sleep changes) are commonly acknowledged as symptoms of SRC, the relationship between sleep and SRC remains poorly understood (Stevens et al., 2022). While subjective sleep complaints (the individual's perception of their own sleep and sleep duration, primarily collected through sleep questionnaires and diaries, as well as symptom checklists) after SRC, such as increased or decreased sleep duration, reduced sleep quality, trouble falling asleep or higher levels of daytime sleepiness are frequently reported in the literate, objective sleep disturbances (alterations in sleep architecture and sleep duration using actigraphy or polysomnography (PSG)) show less consistent results (Stevens et al., 2022).

Studies applying the gold-standard method, PSG, have been rare and have primarily investigated athletes with SRC months to years after the injury (Gosselin et al., 2009; Santos et al., 2020), thereby limiting the evidence of acute effects. Recently, Stevens et al. (2023) examined sleep (utilizing EEG) in ten subjects during the acute (within one week after injury) and subacute phases (eight weeks after injury) post-SRC with mild symptomatology. He found that SRC resulted in longer total sleep time (TST) and fewer arousals during both phases compared to published normative values, with significant improvements in sleep efficiency and reduced disruptions from the acute to the subacute phase. In contrast, sleep architecture did not show any changes. The increased TST and the reduced arousal index were argued to be a recovery mechanism by the brain to optimize rehabilitation. However, further research on different injury severity levels is necessary to validate these preliminary results. Research examining PSG in individuals with moderate to severe TBI during the acute phase reported increased slow wave sleep (SWS, a deep sleep phase during which axonal sprouting and synaptic remodeling occur) and more frequent awakenings (Wickwire et al., 2016).

Difficulties with sleep after SRC may arise due to the axonal damage caused, leading to the disruption of sleep and arousal centers in the brain and brain networks (Jaffee et al., 2015; Mollayeva et al., 2016). For clinical sleep disorders like insomnia, where patients struggle with initiating or maintaining sleep, early awakenings, or non-restorative sleep, hyperarousal has been proposed as an underlying mechanism (Calandra-Buonaura et al., 2016). This hyperarousal state is characterized by increased

activity in the arousal networks of the hypothalamus and brainstem, as well as their efferent projections to regions such as the medial prefrontal cortex and amygdala, which are involved in emotional regulation and stress responses (Miglis, 2016). The resulting dysregulation of these pathways can manifest as heightened sympathetic activity, reflected by elevated nocturnal HR and blood pressure, both of which are frequently observed in individuals experiencing autonomic dysfunction and disrupted sleep (Calandra-Buonaura et al., 2016). Interestingly, insomnia is the most common sleep disorder after mTBI (Wickwire et al., 2018), being three times higher than in the general population (Theadom et al., 2015).

Currently, sleep problems following SRC are often inadequately assessed. The symptom checklist of the SCAT includes the symptoms “Trouble falling asleep”, “Drowsiness”, and “Fatigue”, which are often merged into a sleep cluster (DuPrey et al., 2022). There are doubts about whether these appropriately represent possible sleep problems after SRC, since extended or shortened sleep durations are not documented (Stevens et al., 2022). However, studies analyzing this clustering have found positive correlations between sleep-associated symptoms and the recovery duration of adolescent athletes (DuPrey et al., 2022). Additionally, they have led to a higher number of concussion symptoms (Kostyun et al., 2015), highlighting the importance of sleep for SRC recovery. Still, as there is no consensus regarding sleep assessments after SRC, applying objective (e.g., accelerometry, polygraphy, or PSG) and subjective measures (e.g., sleep questionnaires, sleep diaries) has been proposed (Wickwire et al., 2016), depending on the clinical situation (Reinsberger, 2024).

In mTBI research, sleep disturbances have been associated with impairments in brain homeostatic processes (e.g., glutamate metabolism and cerebral glycogen storage) and the release of pro-inflammatory cytokines (e.g., interleukin-1β, interleukin-6, and tumor necrosis factor-α), resulting in diminished neuronal function and delayed recovery (Piantino et al., 2022). This reflects the role of sleep as a modifying factor, not just a symptom of concussion, in the management and recovery of SRC (Kontos et al., 2019). Further research is necessary to thoroughly understand the impact of SRC on sleep and vice versa, the relationship between concussion symptoms and physiological recovery, and the possible long-term effects of sleep problems on (neuro-)physiological health.

2.3.2 Sleep Physiology and Autonomic Activity

Sleep is commonly described as a phenomenon of the CNS, where the level of cortical activation determines a person's state of consciousness (Zambotti et al., 2018). It is a dynamically regulated process strongly influenced by homeostatic factors and circadian rhythms (Carley & Farabi, 2016). The ANS and sleep are closely related in terms of anatomy, physiology, and neurochemistry. Transitions between different sleep stages are primarily coordinated by the pons, basal forebrain areas, and other subcortical structures. The key neurotransmitters involved in these processes are noradrenaline (norepinephrine), serotonin, and ACh. The neuronal structures responsible for producing and distributing these neurotransmitters also serve as the central representation (CAN) of the SNS and PNS (Chokroverty & Cortelli, 2021).

The gold standard for objectively measuring and classifying sleep is PSG, which includes several measures, such as EEG, HR, and muscle and eye movement. Thereby, sleep can be categorized through changes and distinct features and patterns in electrical activity in the brain using EEG recordings (e.g., brain waves: delta 5–4 Hz; theta 4–8 Hz; alpha 8–12 Hz; beta 13–30 Hz, and amplitude) into two primary stages: rapid-eye-movement (REM) and non-rapid-eye-movement (NREM) sleep (Figure 3) (Zambotti et al., 2018).

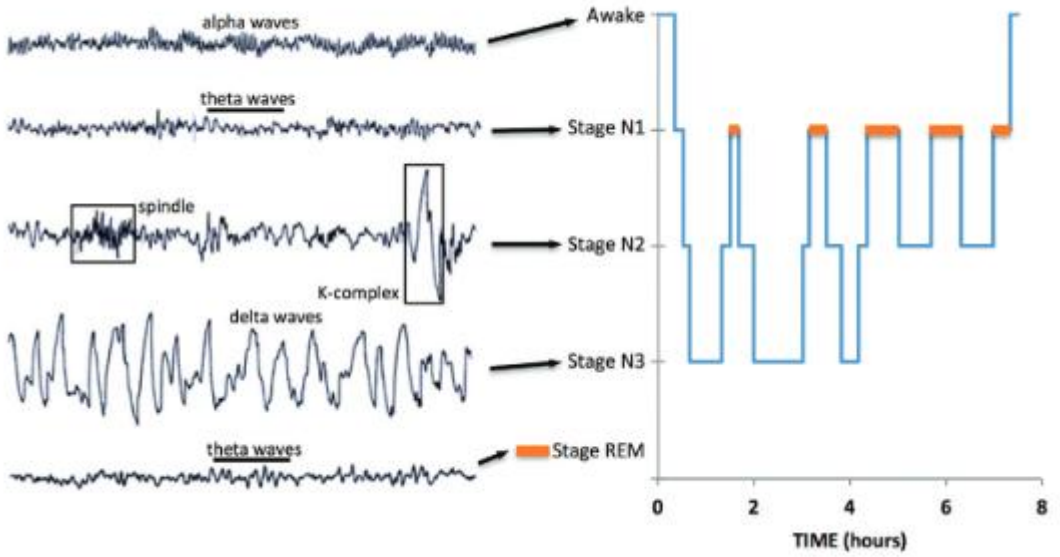

Figure 3 Typical EEG characteristics of sleep stages and wakefulness (left side) and temporal distribution of sleep stages throughout the night (right side). Reproduced with permission from: Carley & Farabi, 2016

A sleep cycle is characterized by alternating between these sleep stages, typically lasting for approximately 90 to 110 minutes. The duration of each stage within

the sleep cycle may vary, with NREM sleep usually occupying a greater proportion in earlier cycles and REM sleep increasing in duration during later cycles (Figure 3). The number of sleep cycles experienced at night can range from four to six, depending on the individual and their sleep duration (Bear et al., 2020).

During REM sleep (characterized by sharp theta waves), the body is immobilized, showing increased brain activity (e.g., in the pontine tegmentum, thalamus, basal forebrain, amygdala, hippocampus, anterior cingulate cortex, temporo-occipital areas) resembling wakefulness, while bursts of eye movements are associated with vivid dreaming (Bear et al., 2020). Sympathetic activity is proposed to be dominant during REM sleep, resulting in increased HR and respiratory rate.

NREM sleep, which accounts for most of the sleep time (approximately 75% of sleep), can further be classified into deeper sleep stages (NREM stages N1 to N3), with stage N3 often referred to as SWS (characterized by low-frequency, high-amplitude delta waves) or deep sleep. During SWS, high parasympathetic activity reduces HR, blood pressure, core temperature, respiratory rate, and energy expenditure. Conversely, the secretion of growth hormones is at its peak, playing a vital role in restoring neural and peripheral cellular functions (Halson & Juliff, 2017). Further, brain activity, particularly in subcortical regions (such as the brainstem, thalamus, basal ganglia, and basal forebrain), as well as in cortical areas (including the prefrontal cortex, anterior cingulate cortex, and precuneus), is reduced compared to wakefulness (Chouchou & Desseilles, 2014).

The nocturnal cardiovascular function has been well researched utilizing ECG recordings included in PSG. Still, the understanding of SNS activity during sleep remains incomplete, mainly due to constraints in measurement techniques (e.g., the invasiveness of microneurography poses challenges in assessing SNS without disturbing sleep, and HRV represents primary parasympathetic activity). Thus, it is essential to conduct studies that investigate both branches of the ANS during sleep (de Zambotti, 2018). As stated in Chapter 2.2.2.2, EDA has been proposed as a possible tool for investigating sleep invasively. Interestingly, nocturnal EDA (SCRs and SCL) exhibited heightened values during deep sleep stages (Sano et al., 2014), when parasympathetic activity is typically predominant. Until now, only a few theories have emerged to clarify why or how the rise in EDA occurs during SWS (Onton et al., 2016).

Due to technological progress in recent years, nocturnal ANS activity can be monitored with wearable technology in the natural (home) environment over extended periods (Zambotti et al., 2019). Wearables offer a convenient approach to record multiple

physiological signals (e.g., HR, EDA, skin temperature), thereby reducing some limitations (e.g., expenditure, laboratory setting) of traditional clinical sleep measurement methods (Nuuttila et al., 2025). This could help to investigate nocturnal autonomic activity over extended periods, offering supplementary insights into the recovery process of sleep itself (Nuuttila et al., 2021) and the exploration of the physiology behind sleep-associated problems (Schmid et al., 2021).

3 Research Aims and Questions

The primary aim of this dissertation was to investigate nocturnal autonomic activity (HR, HRV, and EDA) and its association with concussion symptoms in athletes during and post RTS after SRC, in comparison to healthy, matched control athletes in a home-based setting. Given the need for objective markers to manage and guide the RTS process (Patricios et al., 2023), assessing autonomic activity presents a promising, non-invasive approach for evaluating neurophysiological recovery (Pertab et al., 2018).

Sleep plays a critical role in post-injury brain recovery (Jaffee et al., 2015), yet sleep-associated symptoms are frequently reported and are proposed to be contributing to prolonged recovery durations and increased concussion symptoms (DuPrey et al., 2022). Given this interplay (Figure 4), examining autonomic activity, concussion symptoms, and sleep is essential for a comprehensive understanding of SRC recovery. Thereby, wearable technology enables a home-based, naturalistic assessment of sleep and associated autonomic activity, allowing for continuous monitoring without disrupting the athlete's daily routine (Tabor et al., 2023). In addition, sleep provides a standardized condition for measuring ANS activity (Buchheit, 2014).

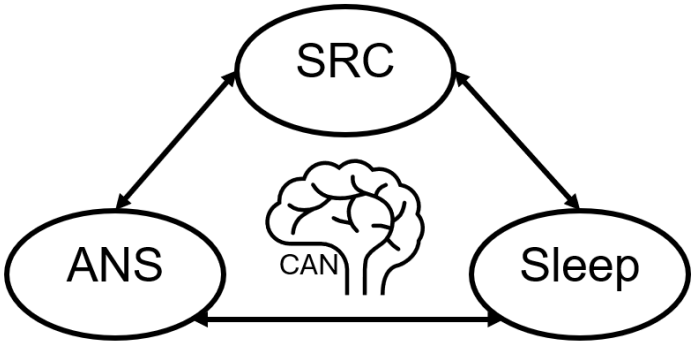

Figure 4 Schematic illustration of the interactions between sport-related concussion (SRC), the autonomic nervous system (ANS), and sleep, highlighting the central autonomic network (CAN) as a key integrative regulation center in the brain.

In summary, to elucidate the research aim, this thesis addresses the following research questions:

1) Are there differences in tonic and phasic **nocturnal sympathetic activity** (EDA) between SRC athletes during and post RTS and matched controls?

Furthermore, is sympathetic activity associated with the number and severity of initial concussion symptoms (SCAT5) and with subjective sleep quality? (see 4.3.2 research paper 1)

2) Are there differences in nocturnal **cardiac parasympathetic activity** (HR, RMSSD) between SRC athletes during and post RTS and matched controls?
   In addition, is parasympathetic activity associated with sleep-associated concussion symptoms (SCAT5)? (see 4.3.3 research paper 2)

3) Do SRC athletes with a **prolonged RTS differ from those with a regular RTS** in terms of nocturnal **autonomic activity** (EDA, HR, RMSSD) during and post RTS and initial concussion symptoms (SCAT5)? (see 4.3.4 research paper 3)

4 Publications and Results

Three original research publications contribute to this dissertation. These are based on a pilot study investigating nocturnal autonomic activity, initial concussion symptoms, and sleep of athletes during and post RTS after SRC, compared to matched control athletes.

The first publication compared nocturnal sympathetic activity between SRC and matched control athletes, while the second focused on evaluating nocturnal cardiac parasympathetic activity across both groups. Both papers additionally explored potential correlations between autonomic activity and concussion symptoms. The third publication investigated nocturnal ANS activity between SRC athletes with prolonged recovery times (> 28 days) and those with a regular recovery (≤ 28 days). Table 7 exhibits an overview of the publications.

Table 7 Overview of the three original publications

Paper (year)	Titel	Subjects included	Parameters analyzed	Nights included	Method ANS-Analysis
1 (2023)	Nocturnal Sympathetic Activity and Subjective Symptoms after Sport-Associated Concussion: a Pilot Study	SRC: n=18 Controls: n=18	-EDA (mean EDA, EDRs, sleep storms) -Concussion symptoms -Subjective recovery	-During RTS: the first four nights -Post RTS: single measurement (Post RTS2) -Controls: the first four nights	ANS: whole night (mean)
2 (2023)	Home-Based Measurements of Nocturnal Cardiac Parasympathetic Activity in Athletes during Return to Sport after Sport-Related Concussion	SRC: n=18 Controls: n=18	-HR, RMSSD -Concussion symptoms	-During RTS: all nights of the RTS -Post RTS: single measurement (Post RTS2) -Controls: all nights except the last one Post: last night	ANS: whole night (mean)
3 (2025)	Nocturnal Autonomic Activity in Athletes with regular versus prolonged Return to Sport after Sport-Related Concussion	SRC: n=17 Controls: n=17	-HR, RMSSD, EDA (CDA.Tonic, CDA.SCR, sleep storms) -Concussion symptoms	-During RTS: The first four nights -Post RTS: single measurement (Post RTS2) -Controls: the first four nights	HR & RMSSD: first 4-h (moving window function) EDA: first 4-h Sleep storms: whole night

CDA = continuous decomposition analysis; EDA = electrodermal activity; HR = heart rate; RMSSD = root mean square of successive differences; RTS = return to sport; SRC = sport-related concussion; SCR = skin conductance response.

The methodology will be elaborated upon in the following chapters, including detailed descriptions of the experimental procedures, participants, and key findings from each publication. The three papers vary slightly in terms of the subjects included, the

parameters analyzed, and the methods employed. The study design and the characteristics of the pilot study will be described first. As the three papers differ in the parameters examined and the analyses conducted, the statistical analysis section will be presented separately for each paper.

4.1 Methods

This pilot study received ethics approval from the Westphalia Medical Board (approval number: 2019-147-f-S). It was conducted between April 2019 and December 2021 in accordance with the Declaration of Helsinki. Participants provided informed consent prior to their participation in this study. The trial was registered at the German Clinical Trial Register (DRKS00019929).

4.1.1 Study Design and Participants

The study employed a longitudinal, prospective cohort study with a matched control group. Identification of SRC athletes was conducted through reports of (local) sports clubs, active screening of the news, and screening of patients attending the sports neurology clinic at the Institute of Sports Medicine at Paderborn University. To be included, SRC athletes required a clinical diagnosis of SRC. For every SRC athlete, a matched healthy control athlete was recruited based on the following criteria: sex, age, height, weight, sport, and level of expertise in their sport. When feasible, the control athlete was recruited from the same sports team/club as the SRC athlete.

Exclusion criteria for both groups comprised a history of cardiovascular, neurological, or dermatological conditions, mental or physical disabilities, diabetes mellitus, and pregnancy. Additionally, control athletes were excluded if they had experienced a concussion within the past year.

All participants received a wireless, wrist-worn multisensory device (wearable) at their inclusion appointment and were provided with standardized instructions on its proper use and operation. Furthermore, participants were instructed on how to wear the wearable device, and each one underwent a 5-minute baseline resting measurement in a quiet room while in a supine position. Participants were told to apply the wearable and conduct a 5-minute resting measurement (supine position) before and after their training sessions (in a quiet setting, within 30 minutes pre and post training), in addition to continuously wearing the wearable during nighttime (sleep). They were instructed and trained to place markers at night (when lying in bed trying to sleep) and in the morning (upon awakening) to generate identifiable timestamps in the raw data. Additionally, they

were asked to conduct a 5-minute resting measurement in bed before trying to sleep and immediately after awakening.

The wearable was worn on the non-dominant wrist during all measurements. Since each SRC athlete had an individual RTS process, the number of recorded nights and training sessions per athlete was heterogeneous. Not every night and training session of the RTS process was recorded due to various factors, including different time points of inclusion into the study, technical issues, participant sickness, lack of a quiet environment, insufficient time before and after training, and participant forgetfulness. Full recovery was defined as completing the last stage of the RTS protocol with medical clearance to return to normal competitive gameplay (Patricios et al., 2023). SRC athletes were requested to conduct one additional nocturnal recording one week (Post RTS1) and three weeks (Post RTS2) after completing the RTS. Control athletes were instructed similarly to SRC patients and wore the wearable device, if possible, for the same number of nights and training sessions as their matched athlete. Nocturnal data was investigated to answer the research questions. An overview of the study design is displayed in Figure 5.

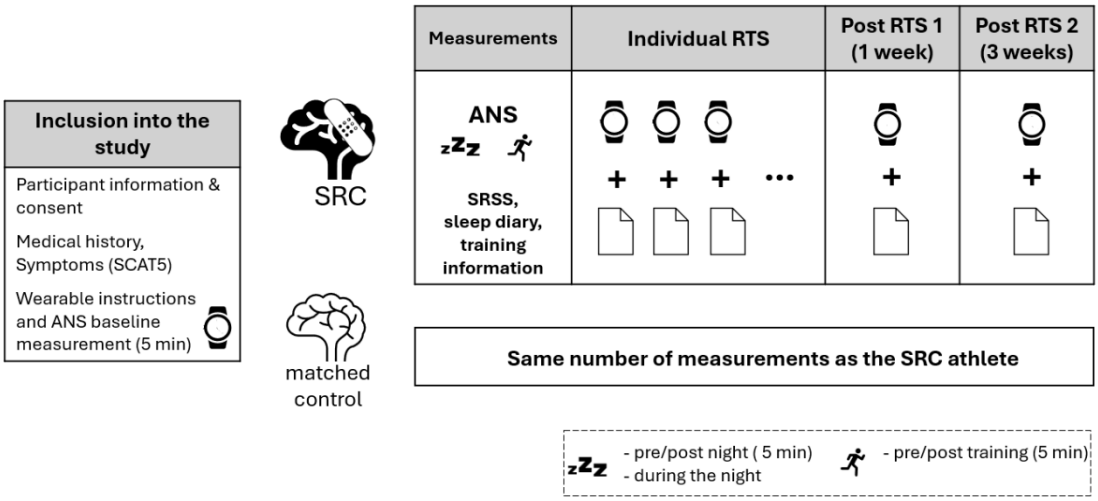

Figure 5 Schematic overview of the longitudinal, prospective cohort study. ANS = autonomic nervous system, RTS = return to sport, SRC = sport-related concussion, SRSS = Short Recovery and Stress Scale

4.1.2 Wearable Device

Data was recorded using a wearable device (Empatica® E4, Milan, Italy), designed for continuous, real-time monitoring of physiological signals and applied in various medical research fields (Empatica, 2022). It is certified as a class 2a medical

device according to Conformité Européenne standards. The E4 collects the blood volume pulse (BVP, sampling rate: 64 Hz) using PPG, EDA (4 Hz) by transferring a tiny, imperceptible electrical current between two stainless steel electrodes, thereby measuring electrodermal conductance, 3D-accelerometer data (ACC, 32 Hz), and skin temperature with an infrared thermopile sensor (4 Hz) (Figure 6). HR and HRV parameters can be calculated from the BVP signal (Empatica, 2022).

Previous research has demonstrated that during rest and nighttime measurements, the E4 consistently produced valid PPG data comparable to ECG, resulting in similar performance between the two devices in over 85% of the analyzed 24-hour data (McCarthy et al., 2016). Additionally, studies employing dry electrodes on the forearm have delivered consistent and reliable ambulatory long-term EDA measurements (Poh et al., 2010), which have been applied in sleep monitoring (Romine et al., 2019).

For data management, the E4 features internal storage that can save up to 60 hours of continuous data. Using Empatica's desktop application software (E4 Connect), data saved on the internal storage can be transferred to Empatica's cloud-based platform (Empatica Connect), enabling long-term data storage and viewing. Thus, each measurement is automatically assigned an individual session number, along with the date and the exact start time of the measurement. From Empatica Connect, raw data signals (BVP, EDA, ACC, temperature) for each measurement can be downloaded as CSV files. The device is powered by a rechargeable lithium polymer battery, providing up to 48 hours of continuous operation. A full recharge takes approximately two hours, depending on the battery's condition and the charger's output current (Empatica, 2022).

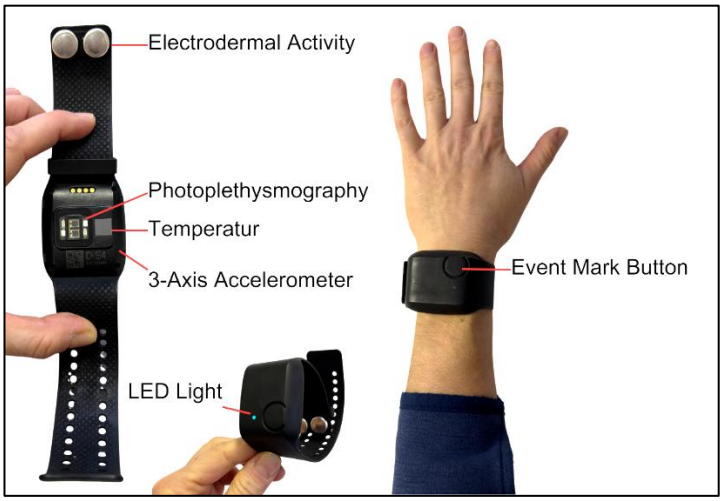

Figure 6 E4 wearable and its features

4.1.3 Data Preprocessing Procedure

Nocturnal BVP and EDA data were preprocessed using a custom-built script (Python, version 3.9.12; see Figure 7 for an example of a night measurement). The raw data was cut using this script to remove data unrelated to sleep. Sleep onset was defined as the first 10 minutes without movement (Kapella et al., 2017) after the last evening marker (Figure 7). This was visually determined based on accelerometry data. Wake-up time was approximated using the morning marker. Measurements were excluded from further analysis if the resulting BVP data segment was less than four hours long or if the measurement was interrupted due to the sensor's battery depletion, as confirmed by the sleep diary for validation.

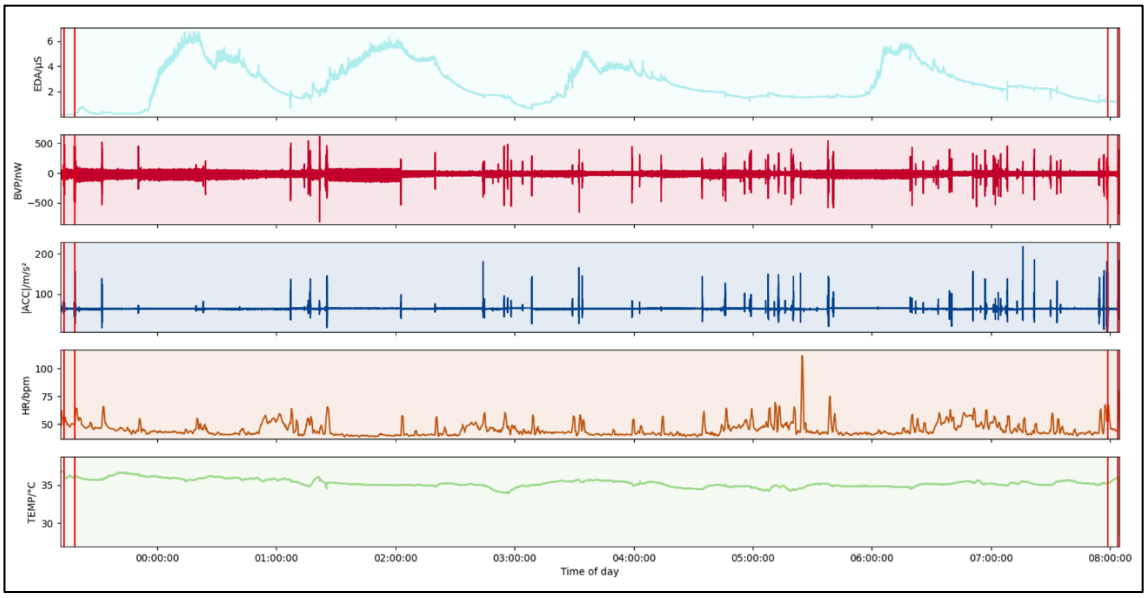

Figure 7 One night measurement visualized in the Python script. Channels: turquoise = Electrodermal Activity (EDA); red = Blood Volume Pulse (BVP); blue = Acceleration (ACC); orange = Heart Rate (HR) calculated from Inter-Beat-Intervals; green = skin temperature (TEMP). The horizontal red lines indicate the markers set by the subject (first one = start of the 5 min resting measure, second = trying to sleep, third = awakening in the morning, fourth = end of resting morning measure)

The nocturnal EDA segments were further detrended, low pass filtered (Butterworth filter, frequency: 0.4 Hz, filter order: 4), and smoothed with a factor of 9. Artifacts were visually inspected and replaced through interpolation (cubic spline or linear interpolation, Figure 8) within the custom-built script (Vieluf et al., 2019).

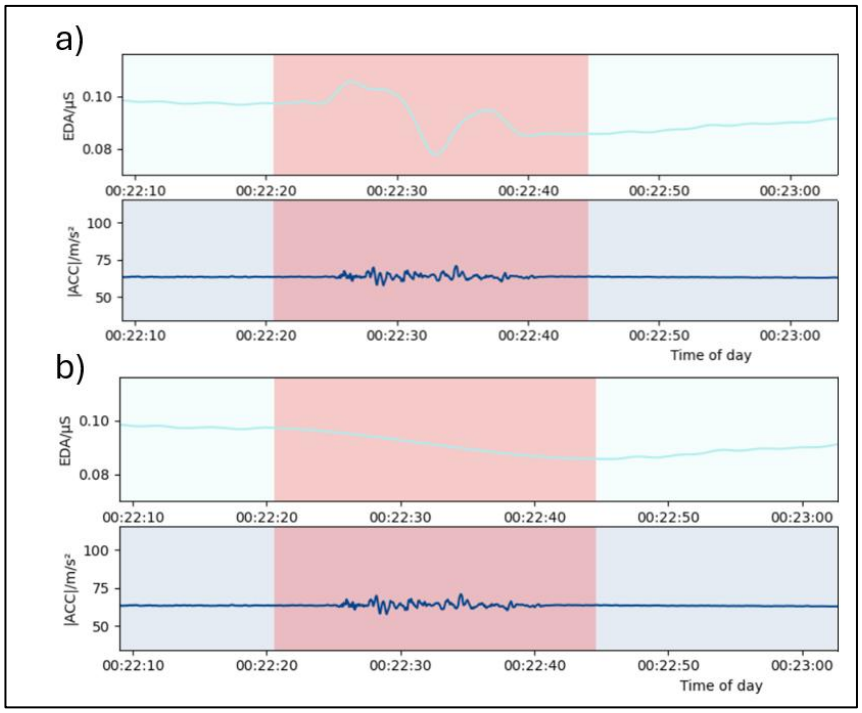

Figure 8 Electrodermal activity (EDA) artifact correction using cubic interpolation in the Python script: a) shows the raw data signal with the artifact, b) displays the signal after interpolation (cubic spline)

Nocturnal EDA was investigated in research papers 1 and 3.

In **paper 1**, tonic EDA (meanEDA) was calculated by averaging the signal over the entire night segment. For phasic EDA, EDRs (increase >0.02 µS/s) and sympathetic sleep storms (minimum 3 EDRs/30 s) (Sano & Picard, 2011) were automatically determined using a custom-built script (Python, version 3.9.12).

For **paper 3**, the software program LedaLab (version 3.4.9) was further applied to conduct a Continuous Decomposition Analysis (CDA) (Benedek & Kaernbach, 2010) to separate tonic (CDA.Tonic, characterized by a constantly, slowly varying baseline) and phasic EDA (CDA.SCR; characterized as rapid, situational adaptation to internal and external stressors: electrodermal responses; EDR: rise >0.02 µS/s (Boucsein, 2012)). LedaLab is designed to analyze skin conductance data and has been widely used in psychophysiological research. However, sympathetic sleep storms were determined by the custom-built script (Python, version 3.9.12, definition: minimum 3 EDRs/30 s) (Sano & Picard, 2011) throughout the night, as LedaLab does not calculate this parameter.

The BVP segments were preprocessed using Kubios HRV Premium (version 3.5.0, Biosignal Analysis and Medical Imaging Group, Kuopio, Finland). The pulse

acceptance threshold was set to 50%. Ectopic beats were removed by the automatic artifact correction algorithm and replaced by interpolated adjacent IBIs. The automatic noise detection (default setting: medium) of Kubios HRV Premium was applied to mark distorted IBI detections (e.g., caused by movement) as “noise segments” (Figure 9) (Tarvainen et al., 2021). These segments were visually inspected, and their lengths were adjusted if necessary. Noise segments were excluded from the analysis. Data containing more than 25% noise segments (effective data length < 75%) and data surpassing 10% of corrected IBIs were excluded from further analysis.

HR and RMSSD were analyzed within the final processed nocturnal BVP segments. RMSSD was selected for its established validity in reflecting parasympathetic activity, while also being less influenced by respiratory influences. HR was chosen as a broader marker that encompasses both sympathetic and parasympathetic influences (Laborde et al., 2017; Shaffer & Ginsberg, 2017). Both metrics have been validated for analysis over extended recording durations (van Lier et al., 2020).

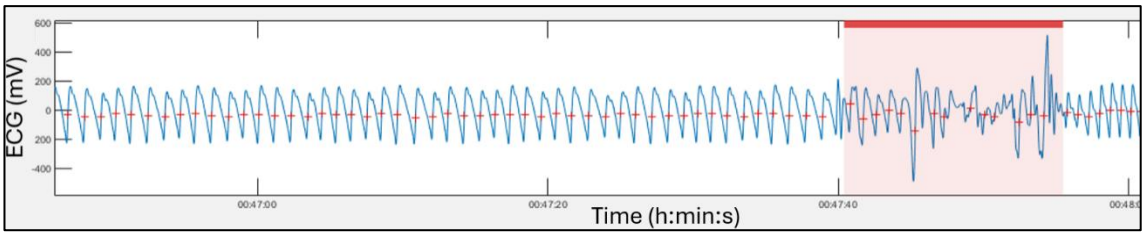

Figure 9 Blood Volume Pulse signal in Kubios Premium (version 3.5.0). The red cross mark indicates a detected beat. The automatic noise detection segment is marked in pink. Segments marked are excluded from the analysis. ECG = electrocardiogram

Nocturnal cardiac ANS activity was examined in papers 2 and 3.

In **paper 2**, HR and RMSSD were obtained from the whole nocturnal segment to reduce variation between different sleep stages (Herzig et al., 2017).

For **paper 3**, HR and RMSSD were assessed by calculating a mean over a continuous 4-h period using a moving window function (window width: 5 min, shift: 1 min) (Fenton-O'Creevy et al., 2012; Zhang et al., 2015) starting after the defined sleep onset. This was due to the implementation of the recommended 5-minute analysis time frame for HRV measurements (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996). As the early phases of sleep primarily consist of slow-wave sleep, measurements are less affected by body movements or other external influences (Brandenberger et al., 2005; Nummela et al., 2016).

An overview of the preprocessing steps is visualized in Figure 10.

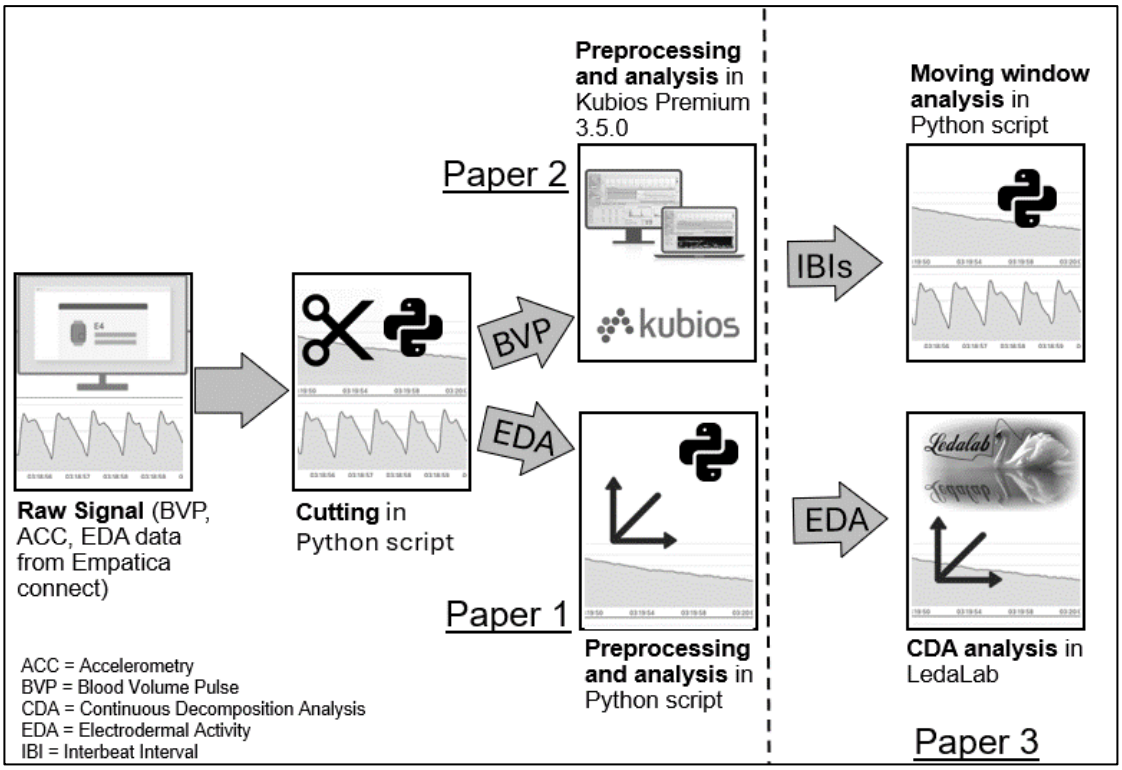

Figure 10 Autonomic nervous system data preprocessing steps

4.1.4 Medical History and Concussion Symptomology

All recruited athletes completed a standardized questionnaire to collect information on their anthropometric data, age, details of their sports participation (such as the type of sport and the number of years they participated), medical history (e.g., neurodevelopmental deficits like ADHD or neurological comorbidities such as epilepsy), and concussion history. The concussion history included whether the athlete had ever experienced a concussion, the number of times they had, and the date of their most recent concussion. Concussion symptoms and severity scores were obtained from the symptom checklist of the SCAT5 (see Chapter 2.1.1.1) (Manley et al., 2017; McCrory et al., 2017). The standardized questionnaire and the SCAT5 were applied to all participants once during the clinical neurological assessment (SRC athletes) or upon inclusion in the study (control athletes).

4.1.5 Sleep Diary and Subjective Recovery and Stress

Participants filled out a sleep diary and the Short Recovery and Stress Scale (SRSS) for each measurement night to gather subjective perceptions, information, and potential confounders related to the night measurements. The sleep diary is divided into two parts: one must be filled out in the evening, and the other must be completed in the morning after awakening. The SRSS was administered twice, before bedtime and again in the morning.

The short version of the sleep diary, developed by the German Society for Sleep Research and Sleep Medicine (DGSM), was used due to its higher compliance rate compared to the full version (Liendl & Hoffmann, 1999). This abbreviated diary consists of 14 items that capture various aspects of the participant's sleep experience and daily condition. Specifically, it records acute stress levels (on a scale from 1 = stressed to 6 = relaxed), daily performance levels (from 1 = good to 6 = bad), daily exhaustion (from 0 = none to 3 = very), bedtimes, wake-up times, sleep onset latency, daytime naps, the number of nocturnal awakenings, intake of alcohol or sleep medication before sleep, and perceived restfulness of sleep (on a scale from 1 = greatly restful to 5 = not restful at all). The DGSM sleep diary adheres to the recommended criteria for essential items in a sleep diary (Carney et al., 2012; Riemann et al., 2017) and has demonstrated adequate reliability and validity (Hoffmann et al., 1997).

The subjective recovery through sleep was assessed using the SRSS (Hitzschke et al., 2015). The SRSS comprises eight items, divided into four recovery and four stress scales. Responses are recorded on a Likert scale (from 0 = does not apply at all to 6 = fully applies), where athletes rate their agreement or the intensity of their experience for each item. The recovery scale includes physical performance capability, mental performance capability, emotional balance, and overall recovery, while the stress scale encompasses muscular stress, lack of activation, negative emotional state, and overall stress.

4.2 Statistical Analysis

Data were analyzed using SPSS (version 28, IBM Corporation, Armonk, New York, United States). Normal distribution was checked using Shapiro-Wilk. The level of significance was set at $p \leq 0.05$ for all tests.

Paper 1:

Group comparisons (concussed vs. controls) were performed for non-normally distributed data using Mann-Whitney U tests, while unpaired t-tests were used for normal distributed data. Coefficients of variation (CV) were calculated to assess individual and group-associated variability of the EDA data. Spearman correlations examined the relationship between concussion symptoms (number and severity) and meanEDA during RTS. Additional associations were assessed using Spearman correlations between sympathetic activity (meanEDA, EDRs, and sleep storms) and subjective recovery. The first four nights were averaged for each participant to investigate a standardized number of nights.

Paper 2:

Between-group comparisons (concussed vs. controls) for demographics, medical history, HR, and RMSSD were conducted using the Mann-Whitney U test. Effect sizes were calculated using Pearson's *r*. The CV was calculated for each athlete to assess the extent of nocturnal variation of HR and RMSSD. Between-group analysis for CV was based on the Mann-Whitney U test. The coefficient of determination (R^2) was calculated for each group (last RMSSD versus mean RMSSD) to analyze how well the last data (i.e., post RTS) fit the linear model of the mean. A higher R^2 indicates that the model (during RTS) better explains the variation in the dependent variable (post RTS). Uncorrected p-values, as well as adjusted p-values (adj., using the false discovery rate), were presented.

Paper 3:

Differences between SRC groups (regular RTS and prolonged RTS) and controls in demographics, concussion symptoms, and nocturnal ANS data (HR, RMSSD, EDA) were evaluated using the Kruskal-Wallis test. Post-hoc analyses were performed using Dunn-Bonferroni tests with Bonferroni corrections for multiple comparisons. The medical history (pre-existing conditions) was compared between SRC groups and controls using Fisher's Exact Test, which is appropriate for categorical data with small sample sizes. The percentage change of ANS parameters (during vs. post RTS) was calculated for SRC athletes. Comparisons between the SRC groups (regular vs. prolonged) were conducted with t-tests or Mann-Whitney U tests, depending on the normality distribution. The effect size Cohen's *d* was computed for normally distributed data, Pearson's *r* for non-normally distributed data, and the phi-coefficient for the result of Fisher's Exact Test.

4.3 Results

4.3.1 Participants' Characteristics

A total of 29 SRC athletes and 29 matched control athletes were recruited. Among these, six SRC athletes did not have an RTS (at least during the study period), one dropped out for personal reasons, one refused to wear the sensor at night, and two had insufficient data quality. Consequently, these ten athletes were excluded from further analysis. The characteristics and symptoms of the remaining participants are presented in Table 8 and Figure 11. For comparability, only data from the nineteen matched control athletes are included.

There were no group differences for age, weight, height, BMI, or number of previous concussions. SRC athletes reported a greater number of symptoms (Median (Mdn) = 7; U = 26.500, z = −4.548, $p < 0.001$, r = −0.737) and greater symptom severity (Mdn = 14; U = 14.000, z = −4.910, $p < 0.001$, r = −0.796) on the symptom checklist of the SCAT5 than controls (symptoms: Mdn = 0, severity: Mdn = 0).

Table 8 Participants' characteristics (n = 38)

	SRC athletes (n = 19)	**Control athletes (n = 19)**
Sex	m = 16, f = 3	m = 16, f = 3
Age (years)	23 ± 5	23 ± 5
Height (in cm)	184 ± 9	184 ± 10
Weight (in kg)	81 ± 12	80 ± 13
BMI (kg/m2)	24 ± 2	24 ± 2
Previous concussions (n)	1 ± 2	1 ± 1
Number of symptoms (SCAT5)	9 ± 5 *	2 ± 4
Symptom severity (SCAT5)	21 ± 16 *	2 ± 6
Sport	Soccer (8) Basketball (4) Handball (2) Am. Football (2) Ice Hockey (2) Modern Pentathlon (1)	Soccer (7) Basketball (5) Handball (1) Am. Football (4) Ice Hockey (1) Modern Pentathlon (1)

Data are presented as group means ± standard deviations and (number).
*Group differences were tested with the Mann-Whitney U-test. BMI = Body Mass Index, f = female, m = male, SRC = sport-related concussion, *p ≤ 0.05 = statistical significance*

Thirteen participants were high-league athletes, competing in the first, second, or third Bundesliga. Fifteen participants competed in the Regional- and Oberliga, eight in the Landes- and Kreisliga, and two at the national level (Modern Pentathlon).

The limited competitions in sports due to the COVID-19 pandemic influenced the RTS definition of four SRC athletes, which was based on being subjectively symptom-free while participating fully in training sessions. The mean RTS duration was 36 (± 43) days. On average, the PostRTS1 measurement was conducted 43 (± 43) days after SRC, and the PostRTS2 measurement was taken 57 (± 43) days post-injury. Due to the longer duration of potential recovery, the PostRTS2 measurement was considered representative of a healthy or fully recovered state for SRC athletes. Thus, it was selected for investigation in this study.

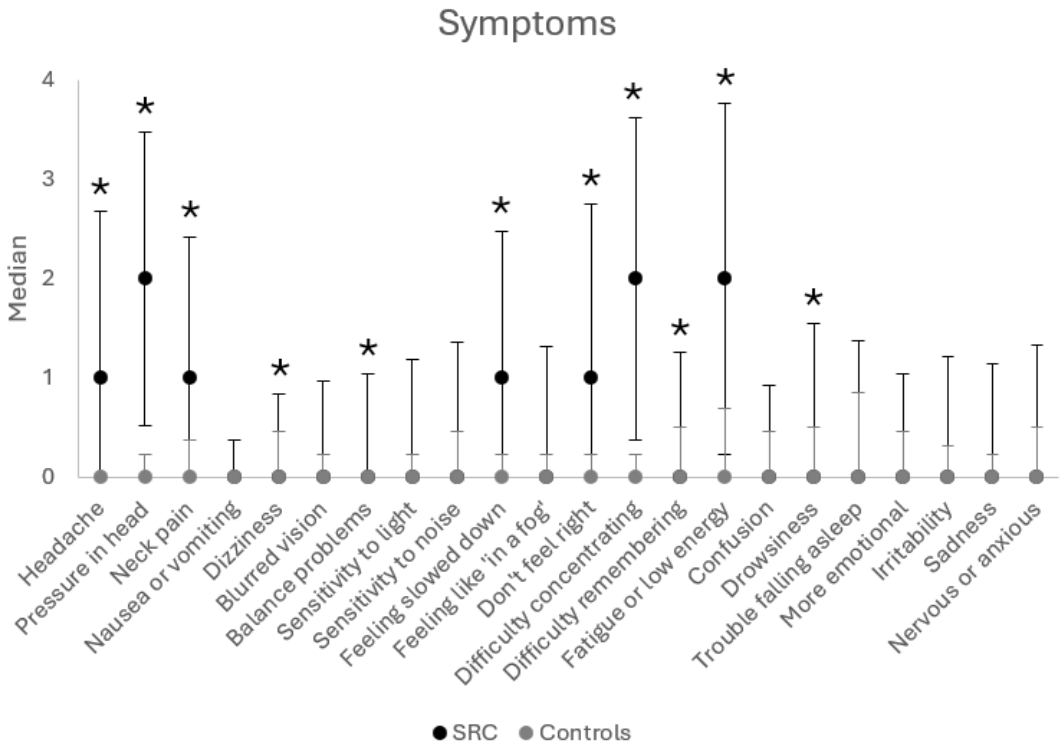

*Figure 11 Overview of the concussion symptoms (SCAT5) for sport-related concussion (SCR) and control athletes, *p < 0.05 (Mann-Whitney U-test)*

Because of variations in data quantity (e.g., the number of recorded nights, completed sleep diaries) and quality (e.g., a recording length of at least four hours, HRV: no more than 25% noise segments in a single measurement), the number of participants included in the three research publications varied.

4.3.2 Paper 1: Nocturnal Sympathetic Activity and Subjective Symptoms

Delling, A. C., Jakobsmeyer, R., Christiansen, N., Coenen, J., & Reinsberger, C. (2023). Nächtliche sympathische Aktivität und subjektive Symptome nach sport-assoziierter Concussion: eine Pilotstudie. B&G Bewegungstherapie Und Gesundheitssport, 39(02), 41–48. https://doi.org/10.1055/a-2023-7579

While cardiac parasympathetic inhibition in the ANS may persist beyond clinical recovery and is frequently described after SRC, there is a limited understanding of possible alterations occurring in the SNS. Due to the neurophysiological and psychological symptoms appearing after SRC, changes in the SNS are expected. Subjective sleep problems are a common symptom after SRC and have been associated with higher symptom severity scores and a longer recovery time. However, the complex interplay between SRC and sleep has not been sufficiently investigated, although sleep plays an essential role in SRC recovery. Therefore, nocturnal EDA was examined during and post SRC.

Eighteen athletes diagnosed with SRC and 18 matched controls wore a wrist sensor during sleep (SRC athletes during their individual RTS and three weeks post-RTS, controls conducted the same number of measurements as their SRC athlete). The study aimed to compare nocturnal tonic (meanEDA) and phasic EDA (EDRs, sleep storms) between these groups and further explore correlations with initial concussion symptoms (SCAT5) and subjective recovery after sleep (SRSS).

The results showed no significant differences in nocturnal EDA between SRC and control athletes during (meanEDA: SRC: 2.59 µS, Controls: 3.28 µS) and after RTS (meanEDA: SRC: 3.58 µS, Controls: 2.79 µS) (Figure 12). However, higher meanEDA during RTS was associated with a greater number of initial concussion symptoms (p = 0.025, r = 0.525) and an increased phasic EDA with lower subjective recovery after sleep (EDRs: p = 0.007, r = −0.642; EDRs/min: p > 0.001, r = −0.762; Sleep storms: p = 0.011, r = −0.616).

In summary, nocturnal EDA did not differ significantly between SRC athletes and controls, although higher EDA during RTS was associated with greater symptom burden and poorer subjective recovery, warranting further investigation with baseline measures and parasympathetic parameters.

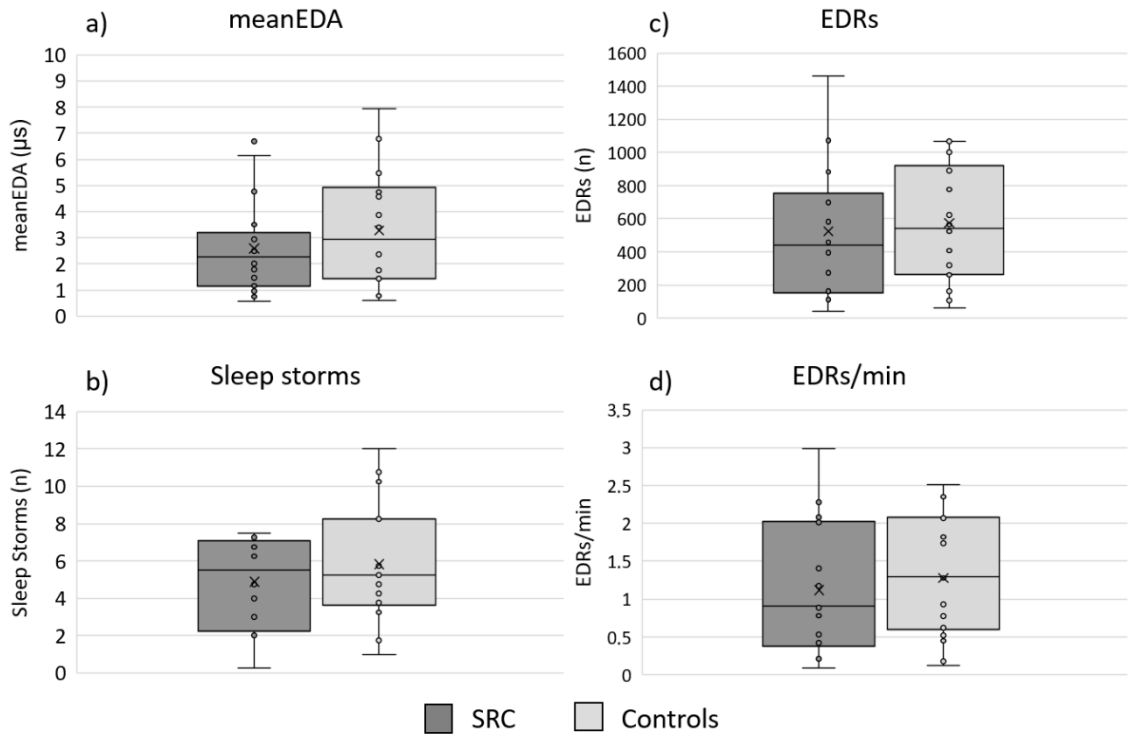

Figure 12 Nocturnal sympathetic activity of sport-related concussion (SRC) and control athletes during return to sport. EDA = electrodermal activity, EDRs = electrodermal responses

Author contributions

Anne Carina Delling, Nele Christiansen, Jessica Coenen, and Sebastian Schupp (in acknowledgment) contributed to the data collection process. The statistical analysis was conducted by Anne Carina Delling. Anne Carina Delling, Rasmus Jakobsmeyer, and Claus Reinsberger wrote the manuscript, and all authors commented on previous versions. All authors approved the final manuscript.

4.3.3 Paper 2: Nocturnal Cardiac Parasympathetic Activity and Sleep-associated Symptoms

Delling, A. C., Jakobsmeyer, R., Coenen, J., Christiansen, N., & Reinsberger, C. (2023). Home-Based Measurements of Nocturnal Cardiac Parasympathetic Activity in Athletes during Return to Sport after Sport-Related Concussion. Sensors, 23(9), 4190. https://doi.org/10.3390/s23094190

SRC often results in impaired autonomic functioning, which can persist beyond clinical recovery despite following RTS protocols. Parameters of HRV can be easily obtained using wireless wrist sensors to measure and monitor parasympathetic dysautonomia during SRC recovery. Sleep problems are common concussion symptoms that may lead to more extended recovery periods, as sleep is crucial for post-traumatic brain recovery. Sleep-associated symptoms may stem from SRC-induced disturbances in brain networks that affect sleep-wake regulation. However, studies on nocturnal HRV are limited. This study assessed nocturnal cardiac parasympathetic activity in SRC athletes during and after RTS. Further, the relationship between nocturnal parasympathetic activity and sleep-associated concussion symptoms (“Trouble falling asleep,” “Drowsiness,” and “Fatigue or low energy”) was investigated.

The study included 18 athletes diagnosed with SRC and 18 matched control athletes. Cardiac autonomic data (HR, RMSSD) was collected during RTS and three weeks post RTS. Nocturnal RMSSD was compared between the two groups and correlated with sleep-associated concussion symptoms.

During the RTS phase, the SRC group exhibited a lower median nocturnal RMSSD (Mdn = 77.74 ms, p = 0.021, r = −0.382, p adj. = 0.126) compared to control athletes (Mdn = 95.68 ms), suggesting reduced parasympathetic activity (Figure 13). Despite this trend, statistical significance was not retained after correcting for multiple comparisons. The reduction in HRV did not persist post RTS, indicating a possible recovery of autonomic function over time. Still, the coefficient of determination for RMSSD during and post RTS within the SRC athletes (R^2 = 0.764) did not display the same degree of dependency as the controls (R^2 = 0.914).

There was no difference in nocturnal HR between groups during (SRC: Mdn = 56 bpm, Controls: Mdn = 55 bpm, p = 0.515, p adj. = 0.567) or post RTS (SRC: Mdn = 55 bpm, Controls: Mdn = 55 bpm, p = 0.567, p adj. = 0.567).

Nocturnal RMSSD correlated positively with the sleep-associated symptom “Drowsiness” (r = 0.532, p = 0.023, *p* adj. = 0.046) in the SRC athletes during RTS.

SRC athletes showed a trend toward reduced nocturnal parasympathetic activity during, but not after, RTS compared to controls, with parasympathetic activity correlating with the symptom “Drowsiness”. In the future, monitoring restorative processes with a wearable during sleep may support SRC management.

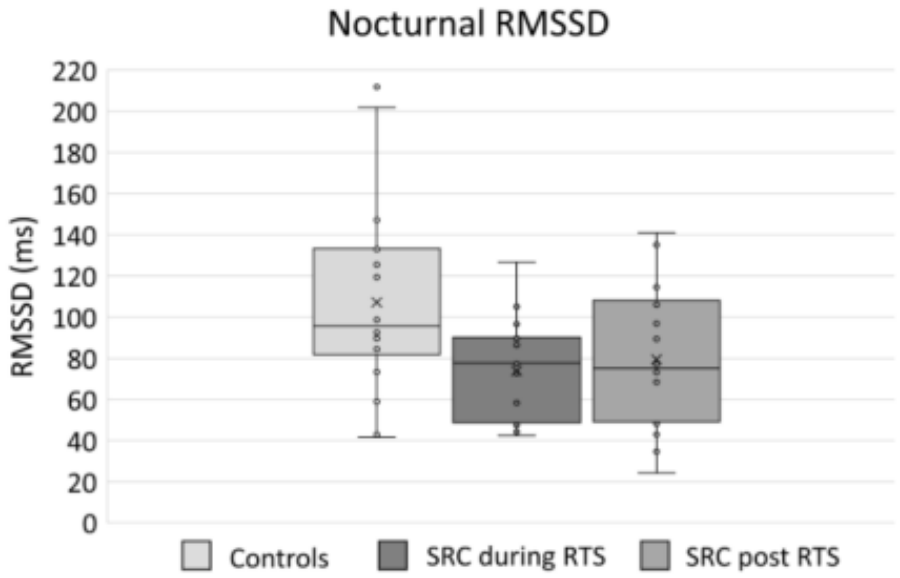

Figure 13 Nocturnal RMSSD for control and sport-related concussion (SRC) athletes during and post return to sport (RTS)

Author contributions

Anne Carina Delling, Nele Christiansen, Jessica Coenen, and Sebastian Schupp (acknowledged) assisted with data collection. The statistical analysis was performed by Anne Carina Delling. Anne Carina Delling, Rasmus Jakobsmeyer, Claus Reinsberger, and Jessica Coenen wrote the first draft of the manuscript, and all authors commented on previous versions. All authors approved the final manuscript.

4.3.4 Paper 3: Nocturnal Autonomic Activity in Athletes with Regular versus Prolonged Return to Sport

Delling-Brett, A. C., Jakobsmeyer, R., Coenen, J. & Reinsberger, C. (2025). Nocturnal Autonomic Activity in Athletes with regular versus prolonged Return to Sport after Sport-Related Concussion. *Scientific Reports*.

Clinical recovery after SRC is typically defined as full symptom resolution and usually occurs within four weeks (28 days) in most athletes. Despite the RTS protocol being widely accepted and applied, approximately 10 to 30% of athletes exhibit PPCS, which is associated with a prolonged recovery. Initial symptom number and severity may serve as predictors of prolonged recovery, and sleep-associated symptoms were linked to an increased risk of persisting symptoms. This study aimed to determine whether athletes with prolonged RTS initially present with more severe symptoms and exhibit persistent alterations in nocturnal ANS activity.

The sample included 17 SRC athletes and 17 matched controls. Based on the RTS duration, SRC athletes were categorized into two groups: regular RTS (rRTS, < 28 days, n = 10) and prolonged RTS (pRTS, ≥ 28 days, n = 7). Nocturnal ANS measures (HR, RMSSD, CDA.tonic, CDA.phasic, sleep storms) and initial concussion symptoms were evaluated and compared between groups during and post RTS (3 weeks after RTS). There were no significant differences between them in demographics, number of previous concussions, or medical history (Table 9). However, the RTS duration was significantly longer in the pRTS athletes (74 ± 53 days) compared to the rRTS athletes (13 ± 4 days, $t(6) = -3.009$, $p = 0.023$, r = 0.733).

Table 9 Characteristics of control, regular RTS, and prolonged RTS athlete groups

	Control athletes (n = 17)	**rRTS athletes (n = 10)**	**pRTS athletes (n = 7)**
Sex	m = 14, f = 3	m = 9, f = 1	m = 5, f = 2
Age (years)	23 ± 5	22 ± 3	24 ± 7
Height (in m)	1.84 ± 0.10	1.83 ± 0.06	1.85 ± 0.14
Weight (in kg)	81 ± 14	80 ± 9	83 ± 18
BMI (kg/m2)	24 ± 2	24 ± 2	24 ± 3
Previous concussions (n)	1 ± 1	1 ± 1	1 ± 2
RTS in days	/	13 ± 4	74 ± 53
Sport	Soccer (7) Basketball (4) Am. Football (4) Handball (1)	Soccer (7) Am. Football (2) Basketball (1)	Basketball (2) Handball (2) Soccer (1) Ice Hockey (1)

	Modern Pentathlon (1)		Modern Pentathlon (1)
Medical history (n)	Migraine (2)	Thyroid dysfunction (1) Depression (1)	Migraine (2) Depression (1) Learning disability (1) Thyroid dysfunction (1)

Data are presented as group means ± standard deviations and (number).
BMI = Body Mass Index, f = female, m = male, RTS = return to sport, rRTS = regular RTS, pRTS = prolonged RTS

Results demonstrated that pRTS athletes did not report more initial concussion symptoms than rRTS athletes. However, they described more “Dizziness”, “Feeling slowed down”, and “Balance problems”, while RTS athletes stated more “Drowsiness” than control athletes (see appendix). No significant differences were found in HR, RMSSD, and EDA among groups during RTS. However, the p-value was close to being significant for RMSSD with a moderate effect size, and further post-hoc analysis revealed a significantly reduced nocturnal RMSSD (Mdn = 70.88 ms, z = -2.433, p = 0.045, r = -0.496) in pRTS athletes compared to controls (Mdn = 92.71 ms) but not to rRTS athletes (Mdn = 86.31 ms, z = 1.426, p = 0.461, r = 0.345). Notably, post RTS RMSSD (Mdn = 51.77ms) was significantly lower in the pRTS group compared to both rRTS (Mdn = 91.43 ms, z = 2.524, p = 0.035, r = 0.612) and control group (Mdn = 92.71 ms, z = -2.747, p = 0.018, r = -0.560). Phasic EDA (sleep storms, Mdn = 3) was only reduced compared to the rRTS athletes (Mdn = 7, p = 0.046, r = 0.588).

Although no group differences were observed during RTS, athletes with prolonged RTS showed reduced nocturnal parasympathetic and phasic sympathetic activity after RTS, which may reflect either insufficient recovery or secondary consequences such as deconditioning or altered sleep physiology.

Author contributions

Anne Carina Delling-Brett, Rasmus Jakobsmeyer, and Claus Reinsberger conceptualized the research goals and aims for the project. Anne Carina Delling-Brett, Jessica Coenen, and Rasmus Jakobsmeyer contributed to the investigation and formal analysis. Anne Carina Delling-Brett and Rasmus Jakobsmeyer wrote the original draft, while Jessica Coenen and Claus Reinsberger critically reviewed and edited it. Claus Reinsberger additionally held the role of supervisor.

5. Discussion

The primary objective of this thesis was to address the need for objective markers to evaluate recovery and guide the individual RTS process following SRC. A process that currently relies on subjective symptom reporting. To achieve this, nocturnal autonomic parameters, including EDA (papers 1 and 3), HR, and HRV (papers 2 and 3), were investigated using a wearable device in a home-based setting in SRC athletes during and post RTS and compared to a cohort of healthy, matched control athletes. Additionally, the relationship between nocturnal autonomic activity and initial concussion symptoms (paper 1) was examined, with a particular focus on sleep-associated symptoms (paper 2). This was due to their connection with prolonged RTS and their interaction with concussion symptomatology. Further, the association between prolonged recovery and autonomic activity was investigated (paper 3).

5.1 Nocturnal Autonomic Activity after Sport-Related Concussion

Nocturnal HR and HRV were investigated in research paper 2, revealing a trend towards reduced cardiac parasympathetic activity, as indicated by decreased RMSSD values in SRC athletes compared to controls. This, however, did not reach statistical significance. The Neurovisceral Integration Model (Thayer et al., 2012) suggests that HRV serves as a means of quantifying the efficiency of neural communication between higher-order prefrontal structures and the body's regulatory systems (such as the cardiovascular system) (Laborde et al., 2017). A concussion is suspected to disrupt functional connectivity in the brain (Murdaugh et al., 2018), contributing to impaired cardiac autonomic function, which can be seen peripherally as abnormal HRV (Coffman et al., 2021; Ellingson et al., 2024). Current research on HRV after SRC has reported mixed findings, particularly during resting conditions (see Chapter 2.2.3, “Autonomic Dysfunction after Sport-related Concussion”). Nevertheless, nocturnal HRV measurements remain unexplored despite their potential to yield critical insights into sleep (Chouchou & Desseilles, 2014), the most crucial recovery phase of the body and brain (Costa et al., 2019; Myllymäki et al., 2012; Nummela et al., 2010; Nuuttila et al., 2025; Ramos-Campo et al., 2019).

The trend in reduced RMSSD did not persist three weeks after RTS, indicating a possible recovery of cardiac autonomic function. However, the coefficient of determination for RMSSD during and post RTS within the SRC athletes did not display the same degree of dependency as observed in the controls. For SRC athletes, 76.4%

(R^2 = 0.764) of the variability in RMSSD post RTS is explained by the model (during RTS), suggesting a moderate-to-strong relationship but not as strong as in the control group, where 91.4% (R^2 = 0.914) of the variance between their last and all the other measurements before is explained. This could potentially indicate incomplete recovery post RTS. Paniccia et al. (2018) examined long-term HRV in youth athletes (primarily females) after a concussion, reporting decreased cardiac parasympathetic activity (measured by pNN50, RMSSD, and HF) during the first 30–40 days post-injury. Parasympathetic activity increased by day 75 post concussion, although no significant differences were found at any point compared to the control group. This demonstrates that large intra- and intervariability in parasympathetic HRV parameters, as well as within-group variability, can distort the detection of potential group differences. Simultaneously, a trend can still be seen over time.

Looking at TBI research, HRV differences were mainly observed in moderate and severe TBI cases, while mild cases, including concussions, often showed HRV values similar to those of controls (Hilz et al., 2011). Therefore, in research paper 3, SRC athletes were divided into two distinct groups: athletes with a regular RTS (rRTS) and those with a prolonged RTS (pRTS). This enabled further investigation into the presumed severity of the injury and its effect on autonomic activity. Although no significant differences in nocturnal parasympathetic activity were found between rRTS, pRTS, and control athletes during RTS, a trend of reduced RMSSD was again observed in SRC athletes. Post-hoc analysis revealed a significantly diminished RMSSD for pRTS athletes compared to controls but not to rRTS athletes. Building on evidence that more severe SRC injuries are associated with the involvement of deeper brain regions, including central structures of the ANS, the CAN (Flores et al., 2023), the decreased RMSSD observed in pRTS athletes may reflect a more functionally severe injury compared to the rRTS athletes, resulting in greater cardiac ANS dysfunction (Hilz et al., 2011).

Post RTS, nocturnal RMSSD of pRTS athletes was significantly lower compared to rRTS athletes and controls. This finding may indicate insufficient physiological recovery in pRTS athletes from a likely more pronounced ANS dysfunction. In the literature, reduced cardiac parasympathetic activity has been observed in concussed athletes even after completing RTS protocols, weeks post-injury, and reaching asymptomatic states. This underscores that physiological disturbances can persist even after clinically determined recovery from SRC (Senthinathan et al., 2017; Hutchison et al., 2017; Abaji et al., 2016; Haider et al., 2020; Doucet et al., 2023). Although research on longitudinal cardiac autonomic functioning with a larger range of time since injury is

limited, persistent autonomic dysfunctions have also been reported in athletes with a history of concussion (Memmini et al., 2021). A recent meta-analysis concluded that a history of concussion is associated with reduced resting RMSSD and might therefore have long-lasting effects on the ANS (Wesolowski et al., 2023). In this study, 7 out of 19 SRC athletes reported a previous concussion injury. However, there was no significant difference in concussion history among the three groups.

The pRTS athletes were significantly included later in the study (more days since their injury). As a result, this group experienced more extended periods of limited training participation. This may have led to aerobic deconditioning, which is known to diminish parasympathetic activity (Mercier et al., 2022). Supporting this, a recent study on adults with PPCS (sport and other mechanisms of injury) reported lower levels of self-reported physical activity alongside significantly reduced resting parasympathetic metrics (e.g., pNN50, RMSSD, SDNN, SD1, SD2), and higher HR and BP when compared to healthy matched controls (Mercier et al., 2025). However, it is important to note that all pRTS athletes in the present study had resumed full training and competitive play for at least three weeks. This factor potentially mitigates the likelihood that observed reductions in parasympathetic activity were solely attributable to deconditioning, suggesting that autonomic dysregulation in this study may persist independently of physical fitness status.

Although the post RTS measurement was based on a single nocturnal recording, which is more susceptible to day-to-day variability compared to averaged values, nocturnal HR and HRV values have a high reliability in healthy endurance-trained athletes (Mishica et al., 2022). Nonetheless, in future research, individual pre-injury HRV baselines with personalized reference ranges (e.g., SD, CV, and the smallest worthwhile change) would allow a more accurate quantification of autonomic dysfunction after SRC. This approach may enable the detection of meaningful deviations from an athlete's normal variability and provides a reference for determining when HRV returns to pre-injury levels.

Nocturnal HR did not differ between the groups during RTS or post RTS, although pRTS athletes descriptively exhibited higher nocturnal HR compared to rRTS athletes. Since resting HR is primarily modulated by parasympathetic activity, an elevated resting HR typically indicates reduced parasympathetic modulation (Gourine & Ackland, 2019). Previous research on daytime resting HR after concussion concluded that there might be no significant difference between concussed and healthy athletes. However, ANS regulation during exercise is commonly impaired (Pelo et al., 2023), as studies have

demonstrated ANS dysfunction during states of heightened metabolic demand (Leddy et al., 2018). Recently, Coenen et al. (2024) reported increased connectivity in the CAN, as measured by EEG, in SRC athletes after exercise during their RTS, while no significant differences in exercise performance (e.g., HR) were found. This heightened connectivity may reflect compensatory neural mechanisms underlying altered cardiac autonomic regulation during and after exercise in the post-injury period.

The limited findings of significant differences in cardiac nocturnal autonomic activity between SRC and control athletes in this study may be attributed to the analytical approach, which averaged RMSSD and HR across all nocturnal recordings during the RTS phase (paper 2). Parasympathetic activity is expected to gradually normalize throughout the recovery process (Paniccia et al., 2018; Senthinathan et al., 2017), a trajectory that cannot be effectively captured through averaged data. Averaging inherently smooths out dynamic changes over time, reducing sensitivity to subtle shifts in parasympathetic activity, either across consecutive nights or within a single night. Nonetheless, this approach is widely acknowledged as a valid method to reduce day-to-day variability in HRV data (Plews et al., 2014) and is effective for identifying broader trends in HR and HRV (Mishica et al., 2022; Nummela et al., 2016). Additionally, whole-night means were calculated to enhance comparability across studies by minimizing methodological discrepancies (Mishica et al., 2022).

In contrast, paper 3 analyzed only the first four nights during the RTS of each athlete. By narrowing the time frame, this method likely minimized distortions introduced by the recovery process observed in the later stages of RTS. Additionally, HRV indices demonstrate enhanced reliability and diagnostic utility when averaged over multiple nights compared to single-night measurements (Le Meur et al., 2013; Plews et al., 2013). Thus, a minimum of three HRV recordings has been proposed to approximate the reliability and validity of weekly averages in trained athletes (Plews et al., 2014).

Moreover, in paper 3, only the first 4-h period of each night was investigated. As the early phases of sleep mostly contain SWS, measurements are less affected by body movements or other external influences (Brandenberger et al., 2005; Nummela et al., 2016). Nuuttila et al. (2022) compared HRV parameters derived from the whole night and the first four hours of sleep in runners, reporting good reliability in nocturnal HR and HRV in both methods, while the 4-h method showed a greater sensitivity to detect changes in homeostasis (increase in HR, decrease in natural log of RMSSD) following endurance exercise.

No significant differences in nocturnal sympathetic activity, as measured by EDA (tonic and phasic), were found between SRC and control athletes during and after RTS when analyzing the first four nights and the single post RTS night of the athletes (in paper 1). High individual variability was observed in the EDA parameters within both groups, consistent with previously reported data in healthy individuals (Sano et al., 2014). This variability could have contributed to the lack of significant group differences, masking underlying trends.

No differences in nocturnal EDA during RTS were found when SRC athletes were further distinguished by presumed injury severity, as defined by regular and prolonged RTS, in Paper 3. Post RTS, pRTS athletes displayed significantly lower numbers of sleep storms compared to rRTS but not to control athletes. As the post RTS measurement was a single measurement, this finding should be interpreted with caution due to the high day-to-day variability in sleep and EDA data.

Nocturnal EDA, both tonic and phasic parameters, may offer insights into underlying physiological and arousal states during sleep. However, its interpretation remains an area of ongoing research. Tonic EDA reflects constant, general arousal without any immediate response to stimuli. Phasic EDA captures short-term, stimulus-evoked fluctuations. These responses are temporally linked to discrete events or stimuli, demonstrating how the body reacts to specific environmental or psychological triggers. Research suggests that tonic EDA levels can indicate sleep quality and stress-induced changes, while phasic EDA often correlates with specific sleep phases (Boucsein, 2012). Increased EDRs during the night, referred to as "sleep storms" when they emerge in patterns, are predominantly observed during SWS. This phenomenon has been documented as a marker of SNS activity during deeper stages of sleep, suggesting that heightened phasic EDA may indicate increased SWS duration (Sano et al., 2014), which could be associated with higher subjective sleep quality (Gashi et al., 2022). In individuals after SRC, elevated phasic EDA may reflect an adaptive or compensatory increase in SWS, necessary for neurological recovery. This increase in SWS has been observed in moderate to severe TBI cases using PSG, but not consistently in mTBI (Mantua et al., 2018). Therefore, without PSG, the relationship between EDA and SWS remains speculative and warrants further research. A reduced number of sleep storms, as observed in this investigation among pRTS athletes, could be a sign of decreased SWS, which limits the most critical recovery phase of the body, possibly leading to diminished recovery.

A recent study by Stevens et al. (2023) utilized home-based PSG to assess sleep arousals in SRC athletes acutely (within one week of injury) and sub-acute (eight weeks post-injury). They reported a reduced sleep arousal index compared to normative data. Physiologically, these arousals are brief periods during which the brain transitions to a lighter stage of sleep, typically due to respiratory events, movement, or environmental stimuli, and are associated with SNS activation. While EDA is a well-established psychophysiological indicator of arousal during wakefulness (Meijer et al., 2023), its role as a marker of sleep-related arousal has yet to be determined. It can be hypothesized that a reduced phasic EDA, characterized by fewer EDRs and fewer sleep storms, may reflect a decrease in nocturnal arousals. Stevens et al. (2023) proposed that the reduced arousal may serve as a recovery mechanism utilized by the brain to rehabilitate itself. This could also be an explanation for the reduced sleep storms found in this investigation. However, the small sample size (n = 10) and the analysis of a single night of data in the study by Stevens et al. significantly limit its statistical power and generalizability.

The present study attempted to avoid this shortcoming by calculating an average across the first four nights (papers 1 and 3) for each subject, aiming to minimize individual day-to-day variability in EDA measurements. However, averaging across nights, as well as specific time periods (e.g., whole night in paper 1 or 4-hour segments in paper 3), may obscure nocturnal EDA dynamics and their association with sleep stages, which could yield further insights into the role of sympathetic activity during sleep. Future research should integrate EDA recordings with PSG to better elucidate the relationships between sympathetic activity, nocturnal arousals, sleep stages, and subjective recovery after sleep in healthy individuals, providing a foundation for studies with athletes after SRC.

In summary, across the three papers, nocturnal autonomic markers revealed a trend toward reduced cardiac parasympathetic activity in SRC athletes during RTS, while sympathetic activity remained unchanged. Although parasympathetic activity appeared to be normalized three weeks post RTS, subgroup analyses further indicated that a prolonged recovery (RTS) might be associated with reduced parasympathetic activity and fewer sympathetic sleep storms post RTS. It remains unclear whether these alterations contribute to prolonged recovery or are a consequence thereof.

Importantly, these findings do not establish nocturnal autonomic parameters as objective markers of neurophysiological recovery, but they highlight their potential relevance for future investigations. The inclusion of SRC athletes at varying time points post-injury and presumed injury severities introduced heterogeneity into the analysis,

reflecting diverse stages of recovery. This variability likely contributed to inconsistencies in autonomic outcomes and complicated the detection of significant group differences.

Future studies should therefore incorporate longitudinal and individualized approaches with multiple follow-up assessments from the acute phase of SRC through post RTS to track and characterize the progression of autonomic activity. In this study, the limited number of female participants precluded an investigation of sex-related differences in autonomic activity. Considering evidence that females generally exhibit higher resting cardiac parasympathetic activity (HF band) than men (Koenig & Thayer, 2016), but report more concussion symptoms and are prone to a prolonged recovery process after SRC (Iverson et al., 2017; Koerte et al., 2020), future research should explicitly address sex as a biological variable when investigating autonomic activity after SRC.

5.2 Relationship between Concussion Symptoms and Autonomic Activity

In this study, SRC athletes reported greater values in the concussion symptoms “Headache”, “Pressure in head”, “Neck pain”, “Dizziness”, “Feeling slowed down”, “Don’t feel right”, “Difficulty concentrating”, “Difficulty remembering”, and “Fatigue or low energy” in comparison to matched control athletes. After correcting for multiple testing, the data showed a trend towards elevated “Drowsiness” in SRC. These symptoms align with clinical profiles outlined by Harmon et al. (2018), encompassing the migraine/headache profile ("Headache" and "Pressure in Head"), cognitive profile ("Difficulty concentrating"), and fatigue profile ("Fatigue or low energy"), emphasizing the multifaceted nature of SRC symptoms. The mechanisms underlying symptom presentation after SRC remain poorly understood, mainly due to variability in symptom severity and type. However, autonomic dysfunction is proposed as a key factor contributing to symptoms such as headache, cognitive impairments, and fatigue/sleep disturbances (Purkayastha, Stokes, & Bell, 2019). Further, these symptoms closely resemble those observed in conditions such as migraines, anxiety disorders, chronic fatigue syndrome, and depression (Pertab et al., 2018).

When differentiating between pRTS and rRTS athletes, pRTS athletes exhibited more symptoms and higher symptom severity scores than rRTS and control athletes, although these differences did not reach statistical significance. Still, significant differences were observed for specific symptoms: rRTS athletes presented higher values for “Drowsiness” while pRTS athletes experienced more “Dizziness”, “Balance problems”, and “Feeling slowed down” compared to controls. This supports the

hypothesis that vestibulo-ocular and cognitive-related symptoms (Harmon et al., 2013) are linked to persistent post-concussive symptoms (Polinder et al., 2018; Tator et al., 2016). “Drowsiness” was also reported more often in a sample of collegiate athletes within 72 hours after concussion injury (Hoffman et al., 2019a). A study with children after non-sport-related mTBI found that “Drowsiness” and the prevalence of sleep disturbances decreased with increasing time since injury (Djukic et al., 2022). In this study, the symptom checklist of the SCAT5 was conducted significantly later for athletes in the pRTS group compared to those in the rRTS group. This may explain why increased “Drowsiness” was only observed in the rRTS group. Symptoms typically decline within two weeks post-injury (Collins et al., 2014), reducing the likelihood of significant group differences after just one week (Echemendia et al., 2023). This may explain the lack of significant differences in the number and severity of symptoms between rRTS and pRTS athletes.

Notably, in this study, nocturnal RMSSD during RTS correlated positively with the sleep-associated symptom “Drowsiness” in SRC athletes. However, this correlation was limited in clinical relevance due to the mild severity of "Drowsiness" (severity levels 1 and 2, except for one athlete who reported a severity level of 6) and its lack of significant difference from controls after adjusting for multiple testing. No significant correlation was found between RMSSD and other sleep-associated symptoms, including the significantly more frequently reported symptom "Fatigue or low energy". In the general population, fatigue is widely recognized as a key indicator of ANS dysregulation (Klimas et al., 2012). Both adults and children experiencing fatigue, including those with chronic fatigue syndrome, tend to exhibit increased sympathetic and reduced parasympathetic activation (Tanaka et al., 2015). Additionally, “Trouble falling asleep,” a symptom of insomnia often reported after mTBI (Wickwire et al., 2018), was not frequently stated within the SRC group. This suggests that sleep may not have been a significant issue for this athlete cohort. Nonetheless, the SCAT may not effectively capture specific types of sleep difficulties (Stevens et al., 2022). Sleep problems after SRC exhibit diverse patterns and timelines of presentation, varying significantly between individuals (Donahue & Resch, 2024; Donahue et al., 2024). Consequently, potential issues regarding sleep may have been overlooked in this investigation. Future research should integrate clinical sleep assessments when SRC is suspected to impact an athlete's sleep. Additionally, pre-injury sleep patterns and behaviors should be examined, as pre-existing problems may influence sleep after SRC and overall symptomatology (Donahue & Resch, 2024). Moreover, sleep disorders have been linked to altered nocturnal

autonomic activity, including reduced parasympathetic activity (as measured by HRV) and heightened sympathetic activity (Calandra-Buonaura et al., 2016), underscoring the need for further investigation into the relationship between sleep physiology and autonomic activity.

Research on the association between autonomic parameters and concussion symptoms remains limited. Many studies fail to report detailed results from symptom checklists, and variations in classifying symptoms into symptom clusters further complicate cross-study comparisons. Paniccia et al. (2018) reported a positive correlation between HRV in youth athletes and concussion symptoms, not analyzing specific concussion symptoms. Contrarily, Coffman et al. (2021) found that concussed adolescents (athletes and nonathletes) with greater HRV (SDNN and RMSSD) at rest reported lesser somatic symptom severity (“Headache”, “Nausea”, and “Dizziness”) at the subacute evaluation (3 to 15 days after injury). He suggests that this finding may be a compensatory mechanism (hypometabolic state) to conserve energy for brain recovery by increasing parasympathetic activity (reflected by increased HRV parameters). Ji et al. (2024) investigated 26 adolescents within ten days after SRC and concluded that lower HF was linked to more affective symptoms (“More emotional”, “Irritability”, “Sadness”, “Nervous or anxious”), while no differences in cardiac autonomic activity were found between SRC and matched control participants. Similar results have been found in mTBI patients, where low HRV, associated with hypoactivity of the prefrontal cortex, reflected impaired behavioral and cognitive adaptation and has been linked with anxiety, depression, and PTSD (Purkayastha, Williams, et al., 2019). Additionally, a moderate connection was reported between greater SNS activity (low-frequency systolic blood pressure variability) and increased total, cognitive, and fatigue symptom severity in adolescents with SRC (Ji et al., 2024).

In this investigation, although no significant differences in nocturnal EDA during RTS were found between SRC and control athletes, a higher nocturnal tonic EDA (meanEDA) during RTS in SRC athletes correlated with a greater number of initial concussion symptoms. As tonic EDA reflects ongoing, general arousal and sympathetic activity (Boucsein, 2012), this result may further emphasize the link between the SNS and concussion symptoms, even in the absence of group-level differences in autonomic activity. Research has also suggested a relationship between nocturnal EDA and memory-related processes, indicating its possible role in cognitive functioning. For instance, Sano et al. (2014) demonstrated a relationship between nocturnal EDA and memory consolidation in healthy individuals. The hippocampus, critical for memory

consolidation, forms connections with the prefrontal cortex, which is essential for higher-order cognitive functions such as decision-making, attention, and working memory (Jin & Maren, 2015). DAI in the hippocampus can disrupt these connections, leading to deficits in memory and cognitive impairments commonly reported after concussion and mTBI (Wolf & Koch, 2016). Interestingly, the amygdala and hippocampus, when directly stimulated with depth electrodes, elicit large SCRs (Mangina & Beuzeron-Mangina, 1996), further reinforcing the connection between these regions and electrodermal activity. The current study observed a significant difference in the symptom "Difficulty remembering" between SRC and control athletes. Nevertheless, the relation with nocturnal autonomic activity was not examined but should be investigated in future studies.

However, increased nocturnal phasic EDA (EDRs, EDRs per minute, sleep storms) during RTS was linked to lower subjective recovery after sleep in SCR athletes. In contrast, a higher tonic EDA (meanEDA) in the control group led to a reduced subjective recovery. This aligns with previous research suggesting that tonic EDA levels can indicate stress-induced changes in sleep quality (Boucsein, 2012). A heightened sympathetic activity, reflecting a state of hyperarousal, may impair sleep and therefore sleep-related recovery and restorative processes. This has been observed in individuals with autonomic dysfunction and disrupted sleep (Calandra-Buonaura et al., 2016).

From a methodological perspective, it is essential to note that concussion symptoms in this study were collected only once at the time of the athlete's inclusion. This single assessment does not account for potential changes or fluctuations in symptomatology over time. This limits the ability to draw robust conclusions about the relationship between concussion symptoms and nocturnal autonomic activity. Future research should integrate longitudinal symptom tracking alongside ANS activity to enhance interpretability and capture the progression and changes of symptoms. Digital technologies such as smartphone apps could support real-time symptom tracking, reducing recall bias and improving data reliability.

5.3 Limitations

To present the limitations of this study in a systematic and transparent manner, the PICOS framework was employed. It was developed to guide evidence synthesis and clinical trial reporting, providing a structured approach that ensures all relevant dimensions (Population/Participants, Intervention/Exposure, Comparison, Outcomes, Study design) of the study are critically addressed (Liberati et al., 2009).

The most pronounced limitation is the small sample size, which restricts the generalizability of the results and reduces statistical power, particularly given the high intra- and intervariability in ANS parameters, sleep per se, and the heterogeneity in SRC injury and severity. While most *participants* were elite athletes, performance levels ranged from local amateur (Kreisliga) to professional leagues (Bundesliga), potentially introducing heterogeneity in physical conditioning and affecting the PNS. Additionally, most participants were recruited from the sports neurology clinic at the Institute of Sports Medicine at Paderborn University and local elite sports clubs (e.g., soccer, basketball, football), which may have biased the sample toward more severe SRC cases and athletes involved in team sports (i.e., collision sports).

The *study design* also contributed to potential bias. Measurements were taken at different time points during the individual RTS for SRC athletes, as the date of inclusion varied after the concussion injury (range: 1–90 days post-injury). Therefore, potential acute autonomic alterations and initial concussion symptoms could have already recovered (in part) for the athletes who were not enrolled immediately post-injury. Concussion symptoms were only collected once during the participant's inclusion in the study. Thus, no development or changes in symptomology were tracked. Furthermore, the progression of RTS was not always medically supervised, particularly at the amateur level, where decisions were prominently made by the athlete and coach based on symptom resolution and full training participation. Therefore, the individual RTS processes are difficult to compare between SRC athletes. Although matched controls were used for *comparison*, reliance on (control) group-level data limits diagnostic sensitivity, given the large interindividual variability typically observed in autonomic parameters.

The COVID-19 pandemic further complicated this study by disrupting training schedules and competitions. RTS after SRC was defined as a return to regular training with medical clearance for four athletes, but without involvement in competition. Further, the classification of regular (≤ 28 days until RTS) versus prolonged RTS (> 28 days until RTS), as outlined in paper 3, relied on the completion of the RTS process. This approach inherently shares the same limitations, including variability in decision-making practices and the lack of standardized benchmarks for assessing readiness to return.

Sleep was estimated using accelerometry data, which, while not identical to PSG, has been shown to correlate strongly with PSG for assessing sleep duration (Regalia, 2021). Sleep onset was operationally defined as the first 10-minute interval without detected movement (Kapella et al., 2017). Although this represents a standardized

procedure, it cannot be confirmed that athletes were asleep during this initial period, introducing potential variability in the interpretation of nocturnal cardiac autonomic measures.

Autonomic parameters in this study were selected based on their validity in capturing parasympathetic activity (measured by RMSSD), sympathetic activity (measured by tonic and phasic EDA), as well as HR as a more global parameter, representing both sympathetic and parasympathetic activity (Laborde et al., 2017; Shaffer & Ginsberg, 2017). The applied wearable, the E4 sensor, has been validated for capturing HR and RMSSD (Schuurmans et al., 2020; van Lier et al., 2020). While it allows for the simultaneous recording of these metrics, the underlying physiological (organ) systems (e.g., heart and skin) operate on different time scales, complicating time-dependent analysis. From a network physiology perspective, the human body functions as an integrated network, where subsystems interact dynamically, with varying degrees of correlation, delay, and interaction (Lehnertz et al., 2020). In time-series analysis, these differences in temporal dynamics pose challenges for directly comparing or synchronizing EDA and HRV data using traditional linear methods. Phasic EDA captures more transient, immediate responses to stimuli, but with a latency of 1–3 seconds after the stimulus before the phasic SRC emerges. Tonic EDA reflects baseline arousal levels that evolve over 10 to 30 seconds or more (Boucsein, 2012). HRV, in contrast, encompasses both short-term fluctuations (e.g., via vagal withdrawal or respiratory sinus arrhythmia) and longer-term trends (e.g., fitness, lifestyle choices). Therefore, aligning these signals in a meaningful way is difficult. As a result, different analysis guidelines and time frames are proposed for HRV (see Task Force, 1996) and EDA (see Bouscein, 2012) (Lehnertz et al., 2020). Thus, EDA and HRV were investigated separately in the first two papers. In the third paper, both parameters were analyzed with the same analysis durations (first 4-h, except for sleep storms) to follow the guidelines and already established analysis protocols.

Future research may benefit from the integration of advanced multimodal analytical methods, such as Granger Causality, Multimodal Canonical Correlation Analysis, and Transfer Entropy, to better capture temporal dependencies and cross-system interactions. In parallel, machine learning (a subset of artificial intelligence) could complement these model-based approaches by enabling data-driven prediction of physiological dynamics and the discovery of complex, nonlinear patterns across time series and identifying subtle, clinically relevant patterns in large physiological datasets. Moreover, incorporating additional physiological parameters, which are often already

collected with wearables, such as skin temperature for thermoregulation assessment, blood pressure, and respiration frequency, could further refine the characterization of ANS interactions (Vieluf et al., 2021).

5.4 Practical Considerations of the Home-Based Approach

While this study represents the first attempt to investigate nocturnal autonomic parameters during recovery from SRC in a home-based setting using wearable technology, its pilot character must be emphasized. Therefore, the feasibility of applying a wearable device for continuous autonomic monitoring should also be evaluated (Thabane et al., 2010).

Wearables, such as the E4 sensor, offer a practical approach to collecting continuous physiological signals under naturalistic conditions, making them particularly suitable for longitudinal studies. This also minimizes participant burden while enhancing ecological validity, an important consideration given the demanding schedules of (elite) athletes. Among the 58 recruited athletes, only one refused to wear the sensor at night. One athlete developed a rash after wearing it every night for four weeks and subsequently reduced usage to once per week. Another participant was excluded from papers 2 and 3 due to ectopic beats affecting HRV analysis. Despite these challenges, 58 athletes recorded 1004 nights, of which 902 nights (89.84 %) displayed usable HRV data and 904 nights (90.04%) produced usable EDA data. These findings underscore the feasibility and reliability of home-based monitoring using a wearable device.

However, several challenges were identified. Technical issues, including the susceptibility of PPG and the EDA electrodes to motion artifacts, loss of contact (e.g., the sensor is not worn tightly enough), and contact pressure (e.g., the sensor is worn too tightly), can affect data accuracy. Environmental influences, such as temperature, lightning, and humidity, can also impact data (Böttcher et al., 2022; Mühlen et al., 2021). To address these issues, the study focused on nocturnal recordings, as nighttime conditions are more standardized and less affected by external stimuli (e.g., noise, light), and body movement, thus providing higher data quality for assessing ANS activity compared to daytime settings (Buchheit, 2014; Schuurmans et al., 2020). Moreover, nighttime recordings are expected to result in higher data completeness due to a lower risk of users unintentionally stopping the recording (Böttcher et al., 2022). Nonetheless, one participant failed to correctly turn on the sensor, which was detected after the first sensor exchange, resulting in a one-week data loss.

Additionally, practical considerations such as battery life, memory capacity, and the deterioration of EDA electrodes must be taken into account for studies utilizing wearables in a home-based setting. The E4's internal storage allows for 60 hours of data, and its recording capacity covers approximately 48 hours, necessitating weekly sensor exchanges by the investigator (based on an average sleep duration of 8 hours per night) and charging at least every five days. As battery performance declines over time, some devices require more frequent charging, increasing user effort. Moreover, the E4's lack of a display, relying solely on LED notifications, made handling less intuitive. Unfortunately, this occasionally led to missing data when the device was not properly activated or charged, or when technical issues (e.g., storage problems) appeared. While the E4’s sampling frequencies fall short of clinical standards, wearable devices must balance data resolution, memory, and battery limitations. Higher sampling frequencies enhance resolution, but they also increase data volume, which can quickly exhaust memory and battery resources. Implementing cloud-based architectures that enable real-time data transmission without local storage may represent a strategy to mitigate these limitations.

Long-term recordings, such as sleep monitoring, generate substantial amounts of data that require extensive preprocessing. For this study, manual artifact correction for EDA (Tronstad et al., 2022) and visual inspection of BVP (HR and HRV) signals was necessary (Mühlen et al., 2021). These processes are time-intensive and prone to human error despite adherence to standardized analysis protocols. Advances in machine learning have already shown promise in distinguishing artifacts from physiological signals, particularly for EDA (Gashi et al., 2020; Taylor et al., 2015). However, the real-time implementation of artifact handling in wearable devices remains unvalidated (Tronstad et al., 2022). The integration of machine learning models in the future could optimize automated data processing and analysis, thereby increasing the effectiveness of wearable technology for medical applications and big data investigations (Zhang et al., 2023). However, because physiological data constitutes highly sensitive health information, its collection, storage, and processing are subject to stringent data protection regulations that must be strictly adhered to.

Another challenge is ensuring participant compliance in home-based studies. This study mainly investigated elite athletes, whose demanding schedules and multiple commitments can impact adherence. To address this, several strategies were implemented to improve compliance, including providing clear instructions and handouts on the study and the wearable device used, emphasizing the importance of the research,

offering prompt feedback on device usage and technical issues, and conducting regular check-ins to ensure consistent engagement.

6 Conclusion and Outlook

This dissertation contributed to a growing body of evidence and understanding of the interplay between neurophysiology and recovery, assessing nocturnal ANS activity in athletes during and post RTS after SRC. Utilizing a wearable device, this pilot study investigated autonomic activity (HR, RMSSD, and EDA) in a home-based setting during the most important recovery phase: sleep.

SRC athletes exhibited a trend towards reduced nocturnal cardiac parasympathetic activity during RTS compared to matched controls, a difference that was not present three weeks post RTS. However, athletes with a presumed more severe injury (defined by a prolonged RTS: ≥ 28 days) showed significantly lower parasympathetic activity post RTS compared to those with regular RTS (< 28 days) and controls, possibly indicating persistent reductions in parasympathetic activity. Additionally, pRTS athletes exhibited decreased nocturnal phasic sympathetic activity post RTS compared to rRTS athletes. It remains to be elucidated whether these differences are a cause (indicating insufficient recovery after SRC, a more severe functional injury, or dysautonomia) or may be a consequence (e.g., physical deconditioning, changes in sleep physiology) of prolonged RTS.

The implications of EDA regarding nocturnal arousal and sleep stage dynamics remain to be clarified. Nocturnal EDA in SRC athletes during RTS correlated positively with initial concussion symptoms and negatively with subjective recovery after sleep, while no significant differences to control athletes were found. Furthermore, nocturnal parasympathetic activity correlated positively with the sleep-associated symptom “Drowsiness” in SRC athletes, though the mild symptom severity in this cohort limited its clinical relevance. Given the limitations of SCAT5 in assessing all aspects of sleep, sleep-related impairments may have been insufficiently gathered in this study.

Clinical evaluation after SRC should emphasize classifying symptom domains to individualize interventions. If the sleep domain is affected, incorporating clinical sleep assessments is essential to identify and characterize sleep-related problems, as these can influence recovery trajectories, symptom persistence, and nocturnal autonomic activity. Future research should investigate the relationship between sleep and nocturnal autonomic activity in healthy individuals to establish baseline physiological patterns. By integrating (portable) PSG with EDA and HRV measurements, the occurrence and distribution of nocturnal EDA can be assessed, as well as the dynamic characteristics and interactions of autonomic activity across sleep stages and their association with

subjective sleep quality. These findings can serve as a reference framework for identifying deviations in clinical conditions, such as after SRC.

Moreover, longitudinal studies with multiple measurements during RTS and post RTS, are essential to capture intraindividual changes in nocturnal autonomic activity, concussion symptoms, and sleep throughout the recovery period (physiological and symptomatic) and beyond. Wearable technology can play a critical role by enabling the collection of home-based sleep and physiological data during real-life conditions. Ideally, pre-injury baseline values are available to enable direct comparison with post-injury measurements, allowing for more accurate assessment of changes in autonomic parameters, sleep, and symptoms following SRC. However, the complex nature, heterogeneity, and individuality of SRC injuries, symptom expression, autonomic activity, sleep, and personal physiology, along with their potential interactions, pose significant challenges for research and statistical approaches, especially in small sample studies. These factors may have contributed to the inconsistent findings in this research field and this study.

In the future, machine learning may be a promising solution for managing, preprocessing, and analyzing extensive multimodal data. Machine learning algorithms may facilitate the automated analysis of temporal patterns, cross-system interactions, and individualized trajectories. These advanced analytical methods hold potential to improve the characterization of autonomic dysfunction in SRC and support more personalized, data-driven RTS processes. The limitation of the current definition of clinical recovery for athletes, focusing solely on symptom resolution for evaluating clearance for physical activity and competition, underscores the need for more objective assessments. Given the high rates of musculoskeletal and re-injuries following SRC, the primary aim for athletes, coaches, and medical staff should extend beyond mere RTS, focusing instead on restoring or even exceeding pre-injury performance levels.

In summary, this thesis highlights the potential of assessing nocturnal autonomic parameters in a home-based setting to gain insights into neurophysiological recovery following SRC. At the same time, it underscores the need for multimodal, longitudinal approaches to track the individual progression and interaction of the ANS, concussion symptoms, and sleep after SRC. This could enhance the precision of RTS decision-making and foster a more individualized, objective approach to concussion management, ultimately improving athlete care, performance, and long-term health.

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Appendix

Table Concussion Symptoms (paper 3)

Concussion symptoms of control, regular RTS, and prolonged RTS athlete groups, presented as median (± SD)

	Control athletes (n = 17)	rRTS athletes (n = 10)	pRTS athletes (n = 7)
Symptom number	1.00 (± 4.43)	6.50* (± 5.24)	12.00* (± 4.23)
Symptom severity	1.00 (± 6.39)	11.50* (± 15.02)	28.00* (± 15.10)
Headache	0.00 (± 0.49)	1.50* (± 1.43)	3.00* (± 1.98)
Pressure in head	0.00 (± 0.53)	1.50* (± 1.25)	3.00* (± 1.62)
Neck pain	0.00 (± 0.39)	1.50 (± 1.71)	1.00 (± 1.13)
Dizziness	0.00 (± 0.49)	0.00 (± 0.70)	1.00* (± 1.07)
Balance problems	0.00 (± 0.00)	0.00 (± 0.97)	0.00* (± 1.22)
Feeling slowed down	0.00 (± 0.24)	0.00 (± 1.41)	1.00* (± 1.70)
Don’t feel right	0.00 (± 0.24)	1.00* (± 1.27)	3.00* (± 2.14)
Difficulty concentrating	0.00 (± 0.24)	1.00* (± 1.65)	3.00* (± 1.00)
Difficulty remembering	0.00 (± 0.53)	0.50 (± 1.25)	1.00 (± 1.38)
Fatigue or low energy	0.00 (± 0.72)	2.00* (± 1.63)	3.00* (± 1.86)
Drowsiness	0.00 (± 0.56)	1.50* (± 1.84)	0.00 (± 0.98)

RTS = return to sport; rRTS = regular RTS; pRTS = prolonged RTS; * $p < 0.05$ different to controls (Kruskal-Wallis-test with post hoc test)

Original research articles

1. **Delling,** A. C., Jakobsmeyer, R., Christiansen, N., Coenen, J. & Reinsberger, C. (2023). Nächtliche sympathische Aktivität und subjektive Symptome nach sport-assoziierter Concussion: eine Pilotstudie. *Bewegungstherapie und Gesundheitssport. 39,* 41 – 48.

2. **Delling**, A. C., Jakobsmeyer, R., Coenen, J., Christiansen, N. & Reinsberger, C. (2023). Home-Based Measurements of Nocturnal Cardiac Parasympathetic Activity in Athletes during Return to Sport after Sport-Related Concussion. *Sensors*, 23(9), 4190.

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3. **Delling-Brett**, A. C., Jakobsmeyer, R., Coenen, J. & Reinsberger, C. (2025). Nocturnal Autonomic Activity in Athletes with regular versus prolonged Return to Sport after Sport-Related Concussion. *Scientific Reports*.

“Maybe it’s good we don’t know what will happen next in our stories, because if we did, we might not turn the page. Or we might skip ahead and never experience the good that comes out of the hard moment we’re living through.”

(Simone Biles)