Heart rate, heart rate variability, and GSR in relation to motion sickness caused by lateral vehicle motion

Kenny Lie



Heart rate, heart rate variability, and GSR in relation to motion sickness caused by lateral vehicle motion

by

K.K. Lie Hok Lien

in partial fulfilment of the requirements for the degree of

Master of Science in Mechanical Engineering

at the Delft University of Technology, to be defended publicly on Thursday January 24, 2019 at 10:00 AM.

Supervisor: Thesis committee: Dr. ir. Riender Happee Ir. Tugrul Irmak Dr.ir. Daan M. Pool COR, 3ME, TU Delft COR, 3ME, TU Delft C & S, AE, TU Delft

An electronic version of this thesis is available at http://repository.tudelft.nl/.



Acknowledgements

This thesis marks the end of my Master of Science programme at the Delft University of Technology. I would like to take this oppurtunity to show my gratitude to those that have assisted me in myriad of ways and made this research possible.

First of all, I would like to thank Dr. Ir. Riender Happee and Ir. Tugrul Irmak for supervising me throughout the research project. Their expert knowledge, devotion and immense patience pushed me to achieve more than I thought I could. A special thanks goes to Ir. Yunyi Li for collaborating with me and conducting the experiments together.

Secondly, I want to say thanks to all my friends that have supported me closely over the years. They have provided me the advice, encouragement and entertainment when needed and also at times when not needed.

Finally, I would like to express my heartfelt gratitude and love to my mom, dad, and brother for their unconditional love and support.

This research helped me grow and understand myself better in many ways (in particular my motion sickness susceptibility), and I know that this journey will benefit me greatly in my future career and in my life.

Kenny Lie Delft, January 2019

Summary

Self-driving cars is considered the next major step in the automotive industry and with automation in passenger vehicles, the driver can benefit from the freed up time for leisure or work, as he or she becomes the passenger. However, this is only possible if the drivers are comfortable during automated driving. The major issue here, is that the susceptibility of motion sickness (MS) increases significantly when the driver does not have his or her eyes on road, this susceptibility needs to be minimized. However, motion sickness is not clearly understood and assessing it has been traditionally done qualitatively with questionnaires. Because of the highly individual and subjective nature of motion sickness and its symptoms, it is hard to quantify it accurately by questionnaires. To minimize and prevent motion sickness, it is beneficial to measure it quantitatively in different ways in addition to the traditional questionnaire.

This thesis explores and investigates the use of physiological measurements, ECG and GSR, to relate motion sickness in a realistic driving experiment. Despite motion sickness not being quite understood, the sensory conflict theory is the generally accepted one explaining it. In short, when visual and the vestibular signals do not match, it will cause the human body to react with motion sickness. That is why sometimes people get sick in the car, while they are reading a book. The motion of the car do not reflect the stationary book one is seeing. It is known that the central nervous system (CNS) integrates the visual and vestibular information, but it also regulates (the symptoms of) motion sickness. In the literature, metrics like heart rate (HR), heart rate variability (HRV) (although debatable) and the galvanic skin response (GSR), are employed to try capture the physiological changes that occur when motion sickness develops. These signals can be measured relatively easy with ECG and GSR. Mixed results have been found regarding the use and its accuracy of GSR, HR and HRV to predict MS. More importantly, the studies are mostly done with laboratory experiments that do not (fully) reflect or relate to motion sickness in (automated) vehicles and had a relative small sample size.

Hence, a road test is conducted for this study with a Toyota Prius on a closed road. A slalom course was driven with a speed 25 km/h to reach lateral accelerations up to 0.4G with a lateral frequency of 0.175 Hz. This frequency and velocity has been chosen, because it is known that people are the most sensitive for MS of frequencies near 0.2 Hz and this velocity reflects urban driving. 23 participants took part of the experiment and had their ECG, GSR and their MISC (Misery scale, an illness rating) recorded during the drive. The experiment lasted until MISC rating 7 (=medium nausea) or either 30 minutes was completed. The ECG (HR, LF/HF ratio) and GSR (skin conductance level SCL, skin conductance response SCR) recordings were then compared to the MISC ratings to see if there was a significant difference between the participants who got sick and stopped at MISC 7 (sensitive) and the participants who did not get sick (non-sensitive). To enable data comparison, the means were calculated for the intervals of 1 minute prior to the test until the start (baseline), first 2 minutes of the test, middle 2 minutes.

There was a near significant effect between the HR and the sensitive and non-sensitive group (p = 0.073). The HR showed a decreasing trend as the experiment went on for both groups, however the sensitive group started with an average HR of 87 bpm and a larger variance versus 83 bpm. The sensitive group also displayed stronger negative trend of the HR over the course of the experiment. Time was found to be a significant factor on the HR (p < 0.001). This trend was also approximated by a linear fitting algorithm that took different data points into account. Both groups share the same pattern; a quick initial rise in HR followed by a gradual decrease over time.

As for the HRV, there was a significant difference between the LF/HF ratio within the sensitive group. However, the LF/HF ratio showed a negative trend over time as MISC increased, which was the opposite, as found in similar studies in HRV literature.

Both the SCL and SCR also did not show a significant difference between the two groups. Only with the SCL time was found to have a significant effect (p < 0.05).

The results show little support correlating motion sickness or even to distinguish sensitive and nonsensitive groups with HR, HRV or GSR data. It might be beneficial to categorize people into different sensitivity profiles for MS susceptibility to make HR information more useful as there is too much of individual differences. Currently, there were few to none road tests done regarding motion sickness. It appears that physiological measurements for predicting MS in vehicles are not as straightforward and do not translate well from other types of laboratory tests to realistic road tests. Not to mention that HRV has become a controversial metric in the recent decade that might need to be re-evaluated for use.

A larger sample size, adding a control trial without slaloming, different frequencies and velocities can be tested as well to get a better grasp of MS. Future research can focus more on road tests to explore and investigate the physiological changes and measurements thereof associated with MS, so it can be understood and prevented in self-driving cars.

Table of Contents

1.	Intro	duction	1		
1	1.1 Motion sickness				
	Visua	Il-vestibular incongruences	2		
	Vehicle motion				
	Meas	suring and evaluating comfort and motion sickness with questionnaires	5		
1	.2 1	Physiological measurements	8		
	Hear	t rate	8		
	Hear	t rate variability	9		
	GSR.		. 10		
1	.3 -	Thesis objective & Research questions	. 14		
2.	Meth	ods	. 16		
2	.1 Expe	eriment	. 16		
	Parti	cipants and Procedure*	. 16		
	Road	and Vehicle*	. 17		
	Moti	on*	. 19		
	Visio	n*	. 21		
2	.2 1	Notion Sickness Measurements*	. 22		
	Mise	ry Scale (MISC)	. 22		
	Moti	on Sickness Assessment Questionnaire (MSAQ)	. 23		
2	.3 I	Physiological measurements	. 25		
	ECG -	– Electrocardiography	. 25		
	GSR -	– Galvanic Skin Response	. 31		
	Statis	stics	. 34		
	Flow	chart of data process	. 34		
3.	Resu	lts	. 35		
	3.1	Vehicle motion	. 35		
	3.2	ECG	. 39		
	3.3	GSR	. 53		
4.	Discu	ission	. 58		
	Hear	t rate	. 58		
	Hear	t rate variability	. 59		
	GSR.		. 60		
	The e	experiment and the physiological measurements	. 61		
5.	Conc	lusion	. 63		
6.	Recommendations				
Refe	erence	s	. 65		

Appendix A: MOTION SICKNESS SUSCEPTIBILITY QUESTIONNAIRE	68
Appendix B: Experiment Briefing	73
Appendix C: MOTION SICKNESS ASSESSMENT QUESTIONNAIRE (MSAQ)	75
Appendix D: Heart rate plots	
Appendix E: GSR	

^{*} JOINTLY WRITTEN WITH YUNYI LI (RELATIONS BETWEEN VISUAL CONDITIONS, HEAD MOTION AND MOTION SICKNESS IN VEHICLE PASSENGERS, 2018)

List of figures

FIGURE 1-1 EMPIRICA	ALLY DERIVED RELATIONSHIP OF MSI (percent emesis within two hours) to wave frequency and aver	AGE
ACCELERATION	I IMPARTED DURING EACH HALF-WAVE CYCLE OF VERTICAL SINUSOIDAL MOTION	4
FIGURE 1-2 RESULTIN	NG FREQUENCY WEIGHTINGS THAT IS NORMALIZED FOR MILD NAUSEA BEING COMPARED AGAINST VERTICAL	
ACCELERATION	I WEIGHTINGS WF DEFINED IN BS-6841 (SOLID THIN LINE). ASYMPTOTIC WEIGHTING = SOLID THICK LINE;	
NORMALIZED N	MILD NAUSEA INCIDENCE: BLACK TRIANGLES	5
FIGURE 1-3: ISO-263	31 FREQUENCY WEIGHTED ACCELERATIONS FOR MOTION SICKNESS COMFORT	5
FIGURE 1-4: SCHEMA	ATIC REPRESENTATION OF A QRS WAVE (WIKI, 2018)	8
FIGURE 1-5: ANATON	MY OF THE ECCRINE SWEAT GLANDS IN THE SKIN (DAWSON, SCHELL, & FILION, 2007)	11
FIGURE 1-6: SIMPLE	CIRCUIT DIAGRAM OF A GSR device with a constant current approach. Resistor 1 is the skin	
CONDUCTANCE	E. (LEDALAB, 2018)	11
FIGURE 1-7: SCR (DA	AWSON, SCHELL, & FILION, 2007)	13
FIGURE 2-1: DAYS IN	ITERVAL BETWEEN THE TWO TRIALS FOR THE SAME PARTICIPANT (MEAN: 8.4 DAYS; SD: 4.8 DAYS)	17
FIGURE 2-2: TU DELF	ft's driverless Toyota Prius being manually driven on the experiment road – (a) road; (b) vehicl	E 18
2-3: (A)		20
FIGURE 2-4B: DESIGN	NED VEHICLE MOVEMENT FOR ONE SLALOM – (A) LATERAL ACCELERATION; (B) PATH	20
FIGURE 2-5: VEHICLE	MOVEMENT DURING EXPERIMENT	21
FIGURE 2-6: EYES-OF	F-CONDITION IN THE VEHICLE	22
FIGURE 2-7: TMSI M	Ловіта	25
FIGURE 2-8: PLACEM	IENT OF THE ECG ELECTRODES. ONLY V1, V2 AND V3 WERE USED	26
FIGURE 2-9: QRS WA	Ανε (wiki, 2018)	27
FIGURE 2-10: EXAMP	PLE OF A DETRENDED ECG PLOT WITH UNDETECTED PEAKS BY THE ALGORITHM AT 100.75 AND 101.5 SECON	DS.
		27
FIGURE 2-11: USING	RR PLOT TO INSPECT AND LOOK FOR UNIDENTIFIED PEAKS. UNIDENTIFIED PEAKS WILL SHOW A SPIKE IN THE R	R
PLOT WHILE MI	IS-IDENTIFIED PEAKS WILL SHOW A NEGATIVE SPIKE.	28
FIGURE 2-12: TYPICA	AL HEART RATE DATA WITH THE ACCOMPANYING MISC OVER TIME, CALCULATED FROM THE RR PEAKS. PLOT	
SHOWS PARTIC	CIPANT 10 WITH EVES-ON-ROAD (VISION) CONDITION WHO COMPLETED THE 30 MINUTES.	29
FIGURE 2-13: EXAMP	PLE OF A HR VS TIME PLOT WITH THE RAW UNSMOOTHED (INSTANTANEOUS) HR (LIGHT BLUE), SMOOTHENED	HR
BLUE, EITTED H	HR (DASHED RED) AND THE AVERAGE HR AT 4 INTERVALS.	
FIGURE 2-14: TYPICA	A LECHE ERECTIENCY PLOT FOR PARTICIPANT 10	
FIGURE 2-15: FLECTR	RODE PLACEMENT ON THE PALM OF THE HAND WITH AMBLI BLUESENSOR N	32
FIGURE 2-16: GSR M	APPING ACOLIRED BY TESTING THE UNIT	32
FIGURE 2-17: PLOT C	THE PROCESSED DATA WITH ITS TONIC AND PHASIC COMPONENT OVER TIME. THE BLACK LINES INDICATE TH	F
START AND FNI	D OF THE TRIAL	- 33
FIGURE 2-18: FLOWO	CHART OF THE DATA PROCESSING AND ANALYSIS	34
FIGURE 3-1: CAR LON	NGITUDINAL LATERAL AND VERTICAL ACCELERATION IN TIME FOR A 6-MIN TRIAL (RULE - RAW DATA: RED -	54
		35
FIGURE 3-2: POWER	AND CLIMINATIVE POWER OF CAR LONGITUDINAL LATERAL AND VERTICAL ACCELERATIONS IN ERECUENCY FOR	
6-MIN TRIAL	AND COMOLATIVE FOWER OF CAR LONGITUDINAL, LATERAL AND VERTICAL ACCELERATIONS IN TREQUENCITOR	36
		50
	CE MISC AT EVERY MINITE FOR DATH CONDITIONS	27
	SE WISC AT EVENT MINUTE FOR BOTH CONDITIONS	37 r)
AND 10 19 (D	INVERTINGE FOR 10 PARTICIPANTS IN TWO VISUAL CONDITIONS, PARTICIPANT 1-9 PROMITOP TO BOTTOM (LEFT	. <i>),</i>
AND 10-10 (RI	IGHT)	50
FIGURE 5-5. DUXPLU	TOF THE MEAN FIR AT DIFFERENT TIME POINTS FOR THE SENSITIVE AND THE NON-SENSITIVE GROUPS. PRE -	20
PRESTART OR B	SASELINE, INS = NON-SENSITIVE GROUP (BLUE), S = SENSITIVE GROUP (RED)	39
FIGURE 3-0:BUXPLUI	I WITH THE MEAN FIR AT DIFFERENT TIME POINTS FOR THE NON-SENSITIVE (A) AND SENSITIVE GROUP (B). FIG	JURE
3-7: (B)		40
FIGURE 3-8: PLOT OF		41
FIGURE 3-9: BOXPLO	IS SHOWING THE DISTRIBUTION OF THE HEART RATES AT EVERY MISC RATING INTERVAL FOR THE NON-SENSIT	IVE
(A) AND SENSIT	IIVE GROUP (B)	42
FIGURE 3-1U: (B)		43
FIGURE 3-11: AVERA	GE HEART RATE OF ALL PARTICIPANTS AT EVERY MINUTE INTERVAL GROUPED INTO ALL, SENSITIVE AND NON-	
SENSITIVE CATE	EGORY	43
FIGURE 3-12:TOTAL	NUMBER OF SENSITIVE PARTICIPANTS AT EVERY MINUTE BEFORE REACHING MISC 7.	44
FIGURE 3-13: (A)	FIGURE 3-14: (B)	45

FIGURE 3-15: ALL NON-SENSITIVE TRIALS DEPICTED WITH CORRESPONDING MISC	45
FIGURE 3-16: ALL SENSITIVE TRIALS DEPICTED WITH CORRESPONDING MISC	46
FIGURE 3-17: BOXPLOT OF THE MEAN HR AT DIFFERENT TIME POINTS FOR THE SENSITIVE AND THE NON-SENSITIVE GROUP	'S. THE
YELLOW BOXES ARE CALCULATED WITH THE FITTED HR LINE. PRE = PRESTART OR BASELINE, NS = NON-SENSITIVE GF	ROUP (BLUE),
S = SENSITIVE GROUP (RED).	47
FIGURE 3-18: RESULTING PLOT OF LINEARLY FITTED HEART RATES FOR EVERY INDIVIDUAL TRIAL. TRANSPARENT RED LINE IN	DICATES
SENSITIVE TRIAL AND BLUE LINE A NON-SENSITIVE ONE. THE THICKER, OPAQUE LINES REPRESENT THE AVERAGED LINE	E PER GROUP.
	47
FIGURE 3-19: AVERAGE LINEARLY FITTED LINE PLOT TOGETHER WITH MEAN HEART RATES PER MINUTE FOR BOTH GROUPS.	48
FIGURE 3-20: THE MEAN LF/HF RATIO AT FOUR TIMEPOINTS (BASELINE, BEGIN, MID, END) FOR BOTH GROUPS	
FIGURE 3-21: BOXPLOTS SHOWING THE MEAN LF/HF RATIO OF THE TWO GROUPS INDIVIDUALLY (A)(B) FIGURE 3-22: (B)	49
FIGURE 3-23: THE MEAN LF RATIO AT FOUR TIME POINTS (BASELINE, BEGIN, MID, END) FOR BOTH GROUPS	51
FIGURE 3-24: THE MEAN HF POWER AT FOUR TIME POINTS (BASELINE, BEGIN, MID, END) FOR BOTH GROUPS.	52
FIGURE 3-25: THE MEAN (NORMALIZED) SCL RATIO AT FOUR TIME POINTS (BASELINE, BEGIN, MID, END) FOR BOTH GROU	PS 53
FIGURE 3-26: AVERAGE NORMALIZED SCL PER TRIAL PLOTTED OVER TIME POINTS. BOLD BLUE LINE REPRESENTS THE MEAN	N SCL OF ALL
THE NON-SENSITIVE TRIALS	
FIGURE 3-27: AVERAGE NORMALIZED SCL PER TRIAL PLOTTED OVER TIME POINTS. BOLD RED LINE REPRESENTS THE MEAN	SCL OF ALL
THE SENSITIVE TRIALS	
FIGURE 3-28: THE MEAN (NORMALIZED) SCRS AT FOUR TIME POINTS (BASELINE, BEGIN, MID, END) FOR BOTH GROUPS	55
FIGURE 3-29: AVERAGE SCR PEAKS PLOTTED OUT CHRONOLOGICALLY PER TRIAL FOR THE NON-SENSITIVE GROUP. ONE OL	JTLIER CAN BE
DISTINGUISHED IN THIS PLOT. THE AVERAGE MEAN EXCLUDING THE OUTLIER IS SHOWN AS BOLD BLUE LINE	
FIGURE 3-30: AVERAGE SCR PEAKS PLOTTED OUT CHRONOLOGICALLY PER TRIAL FOR THE SENSITIVE GROUP. BOLD RED LIN	IE
REPRESENTS THE AVERAGE OF ALL THE TRIALS AT GIVEN TIME POINT	
FIGURE 3-31: THE CORRECTED MEAN (NORMALIZED) SCRS AT FOUR TIME POINTS (BASELINE, BEGIN, MID, END) FOR BOTH	I GROUPS
WITHOUT OUTLIER.	

1. Introduction

Automation within the automotive industry is currently regarded as the major game changer of this time. Several car makers are venturing into automation and implementing different levels of automation into their cars or commercial vehicles. During (highly) automated driving, the driver gets to utilize his free time for leisure or work. Additionally, it would be even better to adopt various seating (even rearward facing) positions within the car cabin to enhance the experience of the driver, who will now become a passenger. This is only a viable option, if the driver remains comfortable and safe in the car. Comfort, or rather discomfort, predominantly points to motion that cause motion sickness and uncomfortable or even painful vibrations. This thesis research, will focus on motion sickness above other discomforts, as this could be the main factor that may hinder users from enjoying automation fully in the near future. If motion sickness is better understood, it enables automation systems to drive in a certain way to increase comfort, but first it is necessary to understand how and why motion sickness develops, how it could be assessed and ultimately keep motion sickness at a minimum or none at all. The assessment and possible predictions of motion sickness in particular will be put under scrutiny in this thesis.

In this chapter, the underlying background will be explained, starting with the theories behind motion sickness and how it has been assessed in most literature and in practice. The next section consists of alternative (physiological) ways, with electrocardiogram (ECG) and galvanic skin response (GSR), which has been used to quantify motion sickness in other types of experiments or fields that might shed more light onto motion sickness in realistic driving scenarios. Afterwards, the research objective and questions will be outlined together with this reports structure.

1.1 Motion sickness

Technological advancements in the last hundred years have led to innovations in transport. New ways of transport have emerged, and novel vehicles have been designed to make our lives more accessible. All moving vehicles (or even simulators), ranging from cars, trains, sea vessels, to airborne vehicles, are extending the envelope of motions and different environmental stimuli that the passengers experience. With this, there are many different kinds of sicknesses that fall into the general category of 'motion sickness', such as air-sickness, car-sickness, sea-sickness and space-sickness. While (Golding J. F., 2006) uses the term self-driving carsickness for motion sickness in (semi-)autonomous driving, this report will be using the term carsickness as many of the current references are based on conventional (non-autonomous) driving or simulator tests.

Typically, carsickness symptoms may vary from, cold sweating, increase in salivation, drowsiness, headache, pain, anxiety, nausea, vomiting. Not every episode of motion sickness will develop with the same symptoms, even with similar stimuli. Another symptom that is often not recognized, is the sopite syndrome (Lacker, 2014). This syndrome develops during short but highly provocative stimuli or prolonged exposure to low intensity stimuli and causes drowsiness and persistent fatigue. According to the studies of (Matsangas and McCauley, 2014), the sopite syndrome can persist from hours to days depending on the exposure of the stimuli and causes the subject to feel apathetic, bored and irritable. These symptoms might last when nausea has not yet occurred or even when it already has subsided.

Another issue is the individual differences in motion sickness susceptibility. Different studies have tried to find and isolate predictors of individual susceptibility. The only reliable predictors were found to be age and sex. Infants and young children are not susceptible to motion sickness and from the age of 6, motion sickness susceptibility increases until around 10 years. Women also tend to be more susceptible to motion sickness than men (Turner, 1999).

Even with these predictors, there are still a large variation between individuals, but developing symptoms usually takes ten to twenty minutes (O'Hanlon, 1974). Another important aspect is that

people react to motion sickness differently. Some are more prone to vomiting and feel total relief for a period of time, while others have a higher threshold to vomit, but therefore feel nauseous for a longer time. Others might even vomit and feel partial to no relief at all and are disabled for the rest of the time. Habituation to self-driving carsickness is assumed to be less evident than in seasickness or other types of motion sickness, because there is not much time and space for the passenger to accommodate to the motions as the travel duration in cars is relatively shorter. While this might show the symptoms and share some insights on the susceptibility of motion sickness between individuals, this thesis focusses on comfort during automated driving. The main goal is to prevent self-driving carsickness from developing. In the next part, the major causes leading to self-driving carsickness will be discussed.

Visual-vestibular incongruences

Self-driving carsickness often develops from a visual-vestibular incongruence. There is an incongruence between what the drivers or passenger sees and what one feels. Internally, a conflict arises between the transduced signal and the predicted signal. For example, reading a book in a moving car or sitting in a rearward facing seat, will often induce or aggravate motion sickness, because the internal predictions are biased towards the book being stationary due to the stationary visual surroundings, while the perception from the vestibular sensors tell otherwise. This is the most commonly accepted theory and also known as the sensory conflict theory (Reason J., 1978).

One common evolution-based hypothesis on why motion sickness occurs, or rather the symptoms, that seems consistent in different studies and yet controversial, states that the vestibular organs together with the central nervous system (CNS) act like a toxin detector (Treisman, 1977). The vestibular organs detect and sense the spatial orientation, linear acceleration, maintaining balance and stabilize eye movement. This information will be processed by the brain constantly. Upon detecting incongruence or unexpected patterns of visual and vestibular inputs, the human body thinks that a neurotoxin might have been ingested, creating symptoms like nausea and vomiting to avoid ingesting more toxins with food avoidance and removing it by vomiting. According to this theory, motion sickness is an evolutionary defense mechanism in humans, which is unintentionally triggered by the external environment.

Besides the visual-vestibular organs sensing a mismatch between visuals and the movement of the head, the semi-circular canal and otoliths (sensing rotational and linear accelerations) can also cause motion sickness in a similar way. These are stimulated by the Coriolis effects acting on the (tilted) head, independent of eyes open or closed. Also, during linear accelerations, the gravitational vector should always be pointing 'downwards' with respect to the reference frame of the head, if not, motion sickness might occur. Tests were conducted in centrifuges and moving base simulators for linear accelerations.

In order to look at the causes of motion sickness a systematic way, (Stott, 1986) integrated the theory and proposed a simple set of rules that if broken, will lead to motion sickness:

- Rule 1. Visual-vestibular: motion of the head in one direction must result in motion of the external visual scene in the opposite direction;
- Rule 2. Canal-otolith: rotation of the head, other than in the horizontal plane, must be accompanied by appropriate angular change in the direction of the gravity vector;
- Rule 3. Utricle–saccule: any sustained linear acceleration due to gravity, has an intensity of 1g and defines 'downwards.'

In the paper of (Bles, Bos, de Graaf, Groen, & Wertheim, 1998), they even went a step further. There, it is proposed to integrate all the previously mentioned different sensory mismatches leading to MS into one single model. This model is an adaptation and extension of the model in (Oman, 1982). The

so-called subjective vertical theory, needs only the difference between the subjective vertical and the sensed vertical (by the sensory organs) to calculate the problematic vector causing motion sickness. Even though the subjective vertical theory might sound simple, it still takes sensory information into account and combining it all makes it complex. The models were applied and mathematically fitted to data of (O'Hanlon, 1974) (Griffin, 1991), that consists of vertical motions on a moving base (ship-) simulator and its contribution to seasickness. The vertical motion influence was also further compared to real ship motions. It was found that the subjective vertical theory matched the experimental data and corresponded to the (vertical) motion sickness incidence in (Griffin, 1991) (Bos & Bles, 1998). The research attempted to identify the periodic vertical motion that is seen as the primary factor leading to motion sickness in ships, aircraft and land vehicles.

On a side note, even though the experimental data acquired from ships matches the motion sickness incidence, most of the automotive related motion sickness papers heavily rely on these ship-based data. It is known that movement ranges, thus acceleration, between the two vehicles are different.

Knowing that the subjective, perceived motion is critical to motion sickness, usually drivers who have control over the motion will not get sick as easily, contrary to the passengers. The driver can employ compensatory movements like tilting the head to align with the gravito-inertial force, thus decreasing lateral acceleration and the onset of motion sickness. In the study of (Osth, Eliasson, Happee, & Brolin, 2014), driver anticipatory postural responses during braking were investigated with numerical human body models (HBM) and test subjects. The drivers were found to be maintaining their initial posture better during self-initiated braking maneuvers than during autonomous braking actions. During self-initiated braking, the driver would brace themselves voluntarily by having their foot on the brake pedal, extending the hip and thigh and plantarflexes the ankle with relative high muscle efforts. This also leads to lower levels of forward head displacement compared to autonomous braking, where the driver cannot anticipate and prepare for the deceleration.

For self-driving cars, withdrawing from the driving tasks means less anticipation of the car movement by control and by not having visuals of the roads, it will even further increase the likelihood of getting motion sick. In a similar way, motion sickness also occurs when passengers or drivers watch a display while the vehicle is moving. This is called visually induced motion sickness (VIMS) and is also caused by visual and vestibular conflicts (Bos, Bles, & Groen, 2008).

Vehicle motion

It is safe to say that an aggressive driving style will result in excessive body motion and will lead to discomfort. In a short track study, drivers preferred lower accelerations level with eyes off road compared to manual driving, when performing a lane change (Lange, Maas, Albert, & Bengler, 2013). By reducing the jerk from 2.9 to 1.3 m/s³, while maintaining accelerations up to 1.8 m/s² during braking from 120 to 80 km/h, it improved the general well-being, comfort but also safety of the driver in an automated vehicle (Festner, 2016). While participants found the high jerk and accelerations 'annoying', it however did not worsen the performance significantly in additional tasks, like monitoring, reading and writing numbers. However, (prolonged) smooth motions may trigger more sickness than jerky motions.

From the last section, several major factors and underlying mechanisms that cause discomfort and motion sickness were highlighted. Previously, it was mentioned that motion sickness was investigated for seasickness first. The acquired vertical ship motion data was used to create a function that would describe the percentage of emesis (vomiting) within two hours versus the vertical wave frequency and the acceleration imparted during each half-wave cycle. This function with the percentage of emesis is called the 'Motion sickness incidence' (MSI) and is shown in figure 1-1. The graph shows that the MSI is the highest around 0.2 Hz and this region should be avoided to prevent motion sickness. Even though the authors mention that this preliminary model has its limited practical use at its time, because of the ignoring of important factors like exposure period and acclimatization to motion. But in subsequent studies like (Bos & Bles, Modelling motion sickness and subjective vertical mismatch detailed for vertical motions, 1998) (Griffin, 1991) (Golding J. F., 2006) (Golding & Gresty, 2005), it was shown that

the peak around 0.2 Hz in MSI matched other experimental data and theories also for other degrees of freedom (Diels & Bos, 2015). It also formed the basis for the (vertical vibration components of the) ISO 2631 standard, showing guidelines and (comfort) evaluation of human exposure to whole-body vibrations.



figure 1-1 Empirically derived relationship of MSI (percent emesis within two hours) to wave frequency and average acceleration imparted during each half-wave cycle of vertical sinusoidal motion

In the current standards related to whole body vibrations and comfort (ISO, 1997), it is noted that it is not specifically addressing cars or ships but machines in general, the overall vibration discomfort and also the incidence of motion sickness can be estimated and predicted with a summation of different frequency weighted accelerations across the seat, back and the feet. The ISO standard suggests that the frequency range from 0.5 Hz to 80 Hz has the most impact on health, comfort and perception, while the range from 0.1 Hz to 0.5 Hz affected motion sickness (figure 1-3). The frequency weightings are developed by experiments and measurements that reflect the sensitivity to different directions and different frequencies of the vibrations in different locations. It is applicable to motion to the human body through the supporting surfaces: the feet of a standing person, buttocks, back and feet of a seated person or the supporting area of a recumbent person. So, dependent on the posture, the vibrations will be felt differently. Similarly, in the study of (Golding, Markey, & Stott, 1995), the low frequency horizontal movement was twice as sickening as the vertical movement (p < 0.05 to p < 0.050.0001) especially with the upright sitting posture compared to supine. That the frequency and the direction of the accelerations impacted the MSI was once again shown in the study of (Donohew & Griffin, 2004). 120 subjects divided into 6 groups were exposed up to 30 minutes to sinusoidal lateral movements at one of the six frequencies (0.0315, 0.05, 0.08, 0.125, 0.16, 0.20 Hz) and during the experiment, subjects were required to provide motion sickness ratings at intervals of 1 minute. The results show that mild nausea caused by lateral oscillation can be predicted with the frequency weightings shown in figure 1-3. Acceleration frequency weightings ranging from 0.0315 to 0.25 Hz causes similar mild nausea, while frequency weightings from 0.25 to 0.8 Hz it reduces 12 dB per octave. This is different than the frequency weightings that are used in (British Standards Institution, 1987). However, this may not be applicable where roll motion is present during lateral oscillation as this has not been accounted for.



figure 1-2 Resulting frequency weightings that is normalized for mild nausea being compared against vertical acceleration weightings Wf defined in BS-6841 (solid thin line). Asymptotic weighting = solid thick line; Normalized mild nausea incidence: black triangles



figure 1-3: ISO-2631 frequency weighted accelerations for motion sickness comfort

Measuring and evaluating comfort and motion sickness with questionnaires

In the experimental studies related to comfort and motion sickness, assessments thereof are often based on subjective measures. Test subjects are required to fill in a questionnaire at the end of the

experiment, ranging from performing tasks in simple test rigs, prototype seats or real-life driving tests and asking them to qualitatively and quantitatively rate certain stimuli to be comfortable or not.

What makes the assessment of comfort or motion sickness difficult is that it is a subjective parameter and that (physiological) mechanisms of certain conditions are not yet well understood (like motion sickness). Furthermore, there are often multiple cross-linked factors that might influence or induce a sensation or symptom.

As mentioned earlier, there exist many comfort and discomfort studies on human factors and ergonomics, but there is no clear universal way or a course of action. For example, (Ulherr & Bengler, 2017) compared and discussed the discrepancies of the current state of the art of seating (dis)comfort studies. Every finding is very specific and individual making it complicated to compare or interpret. The authors argue that, even with the recent surge of publications in 2016 about sitting comfort and or discomfort, there is still no clear universal standard for comfort and discomfort, whether it should be a single scale entity or separate etc. Another argument is the evaluation of comfort and or discomfort. Do these terms mean the same for different experimenters and subjects? Studies that are conducted in other countries and in other languages still use the term comfort and discomfort in published papers, while in the native language of the subjects and authors do not have the equivalent translation of these terms. It gives rise to uncertainties, for not knowing whether the subjects understand what they are supposed to evaluate and this issue has not been adequately mentioned in literature. So, besides different experimental designs and approaches, the possibility that subjects (mis)understand comfort and discomfort causes studies to be difficult to compare with each other. With the above study, the authors hope to instigate new agreements and standards for comfort and discomfort, assessment tools and experiment designs for seat comfort. Knowing this, it can be said that this issue is not only for seat design and sitting comfort, but also for other fields of ergonomics such as motion sickness where comfort and discomfort need to be evaluated.

The Reason and Brand Motion Sickness Susceptibility Questionnaire (MSSQ)

The most well-known and widely used questionnaire targeting motion sickness and its individual differences is the 'Motion Sickness Susceptibility Questionnaire (MSSQ)' by (Reason & Brand, 1975). This extensive questionnaire aims to predict individual susceptibility to motion sickness caused by a variety of stimuli, and is not limited to in-car situations but also on other land, sea and airborne (even space) vehicles. The MSSQ aids researchers in determining the individual differences in motion sickness in relatively uncontrolled environments and also the unavailability of facilities to perform controlled tests in laboratories. This questionnaire is based on the earlier mentioned aspects causing motion sickness, i.e. toxin-detector hypothesis, sensory conflict theory etc. It consists of questions related to one's history of feeling nauseous, sickness or vomited as a child and as adult in various situations. However, the original MSSQ stems from 1975 and the reference norms have never been officially published. Therefore, an improved MSSQ was proposed (Golding J. F., 1998). The redesigned MSSQ provides new (and only adult) reference norms and was validated with data of motion and nonmotion induced nausea stimuli. In short, "the MSSQ is good at predicting who will be motion sensitive, but less efficient at identifying motion-resistant individuals." Still, according to the results, the revised adult reference norms were almost identical to the unpublished norms of the original questionnaire, thus the revised version was deemed reliable and may be used as a direct replacement of the original. Following its predecessor study, the MSSQ has been shortened even more (MSSQ-Short) (Golding J. F., 2006). Similar to the previous study, new data and motion tests have been added (as well as, chemotherapy, post-operative nausea and vomiting (PONV)) to re-evaluate and simplify the questionnaire. Results show that it still correlates in accordance with the original lengthy results and the revised one. The conclusions on this MSSQ-Short are similar to the revised questionnaire of 1998 and maintain its reliability while reducing the time needed to fill out.

The Motion Sickness Susceptibility (History) Questionnaire (MSSQ) (Griffin & Howarth, 2000)

Another questionnaire that has been used extensively and validated is the MSSQ by (Griffin & Howarth, Motion sickness history questionnaire., 2000). Similarly to the one by (Reason & Brand, 1975), the questionnaire consists of questions related to one's exposure to motion in different forms of transport and the occurrence of illness, but unlike the former, it includes a list of specific symptoms (feeling hot, headaches, change of skin color, mouth-watering, drowsiness, dizziness, nausea, vomiting) that one has experienced in the PAST year. The questionnaire allows for experimenters to select participants based on their individual variations in susceptibility to motion sickness. Furthermore, this questionnaire provides a thorough manual to assess the measures relating to motion sickness susceptibility with the scores acquired from the questionnaire. The calculation of said measures will not be further explained here and can be found in the (Griffin & Howarth, Motion sickness history questionnaire., 2000). The questionnaire can be found in appendix A.

Similar to some extent, the motion sickness assessment questionnaire (MSAQ) (table 2-5) is a different symptoms checklist that participants need to fill out after the stimulus exposure. The questionnaire divides symptoms in different categories. More information can be found in Section 2.2.

Illness Rating Scale

For motion sickness, there is another type of questionnaire that measures the severity of motion sickness according to symptoms on an illness rating scale. Several illness ratings scale exists, like the 7-point illness rating (IR) scale (Griffin & Howarth, 2000), and the 11-point misery scale (MISC) (see section 2.2 and MISC, table 2-4, (Bos, MacKinnon, & Patterson, 2005)).

The subjective illness ratings are a widely used measure to assess motion sickness in time during experiments. It gives insight on how symptoms of motion sickness progress and subside during experiments. However, there are no standard rules on the interval of measurements and depends on experiment setup. As motion sickness symptoms can develop rapidly depending on conditions, the timing of giving a rating sets the resolution and accuracy and in between the rating, some gradual onset of other symptoms are not captured.

Furthermore, there is the subjective aspect of experiencing and rating the symptom. For example, when does mild nausea (illness rating of 3) progress to mild-to-moderate nausea (illness rating 4)? Even with words describing the feeling and symptoms that comes with the questionnaires, it is up to the participants' judgement.

1.2 Physiological measurements

Heart rate

When motion sickness starts to develop, a multitude of (autonomic) physiological responses occur, ranging from feeling nauseous, headache, cold sweating, increase in blood flow in deeper blood vessels, skin pallor, but also changes in heart rate (Golding J. F., 2016). It often starts with an increase in heart rate followed by a rebound decrease according to (Benson, 2002). Also, it has been reported in multiple studies that heart rate increases when exposed to nauseogenic motion (Holmes & Griffin, 2001), especially during nausea. In the studies of (Cowings et al, 1990) and (Stout et al, 1995), both reported a positive correlation between subjective ratings of nausea and heart rate. (Holmes & Griffin, 2001) did a similar study with an optokinetic drum, where they compared HR and the heart rate variability (HRV) to each of the subjective sickness ratings. The findings are consistent with the aforementioned studies, that there is a significant increase in HR with increase in severity of nausea. HR, together with the HRV, provides an alternate source to measure what is going on in the human body and could be a predictor for motion sickness. If this is the case, measuring of heart rate can easily be done with a non-invasive electrocardiogram (ECG). The ECG detects electric potential that occurs during the de- and polarization of the ventricles in the heart (figure 1-4). Capturing the adjacent QRS complexes over time, results in the heart beat over time. This cardiac cycle provides oxygenated blood to the human body and the rate thereof depends on physiological states like stress, strenuous activities, emotions, nausea, but can also stem from cardiac problems.



figure 1-4: Schematic representation of a QRS wave (wiki, 2018)

Furthermore, HR fluctuates with respiration rate and is heavily dependent on the respiratory frequency and the depth of ventilation (Grossman & Taylor, 2007). The integration of respiratory and cardiovascular responses is a complex interaction of neural, central, humoral and mechanical feedback mechanisms in the body.

In short, while measuring HR to detect motion sickness with simple ECG might seem promising and easy, HR by itself is a tricky motion sickness indicator, as seen from the above contradictory examples and the intertwined physiological mechanisms. With current technology, capturing heart rate data with ECG means you can also acquire HRV data simultaneously.

Heart rate variability

The term heart rate variability (HRV) refers to various methods and metrics that analyses the rate of change of the (heart) beat-to-beat intervals over time (R-R interval, R coming from the QRS peaks). HRV analysis has been widely used to investigate the autonomous nervous system and many studies regarding motion sickness have used this as a tool to assess motion sickness. While the origins of motion sickness are not yet well understood, it is known that the symptoms of motion sickness, e.g. sweating, stomach awareness, skin pallor etc., are manifestations from the autonomic nervous systems (ANS).

The ANS is a division from the peripheral nervous system (PNS) and consists of visceral motor nerve fibers that regulate smooth muscles, cardiac muscles and glands. Smooth muscles are found (in the walls of) the hollow organs, for instance stomach, bladder, but also arteries and veins. It regulates bodily functions and is also referred to as an involuntary nervous system. The ANS is subdivided into the parasympathetic nervous system (PSNS) and the sympathetic nervous system (SNS). In short, the PSNS is responsible for the 'rest-and-digest' and 'feed-and-breed' functions, i.e. stomach, colon, reproductive systems, while the SNS stimulates the 'fight-or-flight' response functions. Both act together, as a suppressing agent or a stimulating one, and are always active in the body to maintain homeostasis (Marieb & Hoehn, 2007). One of SNS function is activating the sweat glands, which is of use for measuring motion sickness with the GSR, described in the next subchapter. During fight-or-flight or even stress response, large quantities of epinephrine is released and causes an increase in heart rate, cardiac output, gastrointestinal vasoconstriction and many more, to prepare the human for imminent danger.

A broadly recognized, yet debatable, metric that is used for HRV analysis in the frequency domain. It is split into a low frequency (LF) power band and a high frequency power band (HF). (There is also an ultra low (ULF) and a very low frequency band (VLF), but is not addressed here, as it is less relevant for this study.) The LF component (0.04 - 0.15 Hz) is addressed to the SNS activity, and the HF component (0.15 - 0.4 Hz) is linked to both the SNS and the PSNS activity (Schaffer & Ginsberg, 2017) (Reyes del Paso, Langewitz, Mulder, & van Roon, 2013) (Quintana & Heathers, 2014). Often the LF/HF ratio will be used to show an overall increase of the sympathetic tone at the onset of motion sickness in various studies, e.g. (Holmes & Griffin, 2001) (Himi, et al., 2004) (Lin, Lin, & Chiu, 2011) (Ohyama, et al., 2007) (Sjors, Dahlman, Ledin, Gerdie, & Falkmer, 2004). It may estimate the ratio of SNS and the PSNS activity.

However, there are several factors that need to be taken into account when using HRV. Respiration is inherently coupled with HR and therefore, also the HRV. It has been shown that the HRV can be manipulated by (forcefully) changing breathing pattern (Quintana & Heathers, 2014). The LF band is affected by breathing from $\sim 3 - 9$ breaths per min, while the HF band (also known as the respiratory band) is affected by breathing from $\sim 9 - 24$ bpm (Schaffer & Ginsberg, 2017).

As mentioned before, the popular proposed method above of using the LF/HF ratios to represent 'sympathovagal balance' has been challenged many times in the literature. Either LF or HF has been questioned whether it truly describes the SNS or PSNS activity as they claim (Billman, 2013). The link between LF power and sympathetic nerve activation is considered very poor. Both sympathetic and parasympathetic nerve activities are very complex (non-linear) mechanisms that are confounded with the mechanical effects of respiration and accompanying heart rate. So it is impossible to attribute it to the LF/HF power with any degree of certainty. Another argument against the metric LF/HF power is the mathematical one. Changes in the LF/HF ratio can be due to either LF power getting larger or HF getting smaller, or by a combination of the two. So, identical LF/HF ratios could be caused by doubling of the sympathetic nerve activation with a constant parasympathetic activation or same sympathetic activation but halved parasympathetic activation. This obviously makes a huge difference when interpreting the ratios as in cardiac autonomic balance. As LF and HF covers a lot of complex physiological and cardiac (overlapping) responses, the HRV does not infer much or anything at all.

While it may seem pretty controversial to keep using this metric, many relevant motion sickness studies did use and try to investigate whether LF/HF might be an indicator to motion sickness (e.g. (Farmer, et al., 2015)). The study of (Mullen, Berger, Oman, & Cohen, 1998) is an example where they found that motion sickness is not reflected in a change in the frequency spectrum between sick and non-sick groups where participants where rotated in a seat and need to perform a task. However, in the rotating seat experiment of (Doweck, Gordon, & Shlitner, 1997), there was difference in the power spectrum (in particular near 0.075 to 0.15 Hz) between the sick participants during the sickening motion and rest.

GSR

One of the symptoms that occur when people are developing motion sickness is (cold) sweating (Reason & Brand, 1975) (Golding & Gresty, 2005). As electrodermal activity or skin conductance (GSR) can be measured, many researches have been done to see whether this physiological measurement can be an indicator for motion sickness. Skin conductance measurements are non-invasive and relatively easy to be measured. However, measuring motion sickness through skin conductance is not simple. Sweating is an autonomic response of the body to various internal and external inputs, and especially to emotional and cognitive ones. Some of the sweating might be due to stress or linked to thermoregulation of the body. Because of this property, extracting information regarding motion sickness is difficult and experiment setups will play an important role. Before getting into the details of previous studies, its working principles will be explained.

The human body has many sweat glands that serve a multitude of purposes to support life. They regulate core body temperature, maintain water balance in the body, emotional states such as embarrassment, fear and stress also cause gland activation. Electrodermal activity has been found to be influenced by psychosocial events and experiments, like anticipation to certain event, doing cognitive tasks, classifying personal traits, and clinical research (Boucsein, 2012). For example, one of the common, but highly controversial, uses in the past, is to detect lies or rather stress responses, with the GSR. On other hand, skin conductance has also shown positive correlation with motion sickness in various studies (Wan & Hu, 2003) (Dahlman, Sjörs, Lindström, & Ledin, 2009) (Himi, et al., 2004). The skin conductance can be regarded as a useful index of sympathetic activity as it is not contaminated with parasympathetic activity (Braithwaite, Watson, Jones, & Rowe, 2015).

There are two types of sweat glands, the eccrine and apocrine glands. Sweat glands of the first type are of primary interest for GSR studies. These sweat glands can be found everywhere on the body, with the highest density area being the palms of the hand and the soles of the foot, whereas the apocrine gland are found primarily on the genital areas and in the armpits. The function of the latter one is not yet well understood and researched. It is suggested that it secretes pheromones, but also produce sweating caused by emotional stress. The main and foremost studied one is the eccrine sweat glands. It is known that the primary function of the eccrine sweat gland is for thermoregulation and that it is more reactive to emotional stimuli, especially in the palms of the hand. In the figure below (figure 1-5), the anatomy of a (single) sweat gland is depicted. Sweat is being created in the subdermis layer after a trigger from CNS into the sudomotor cells. It travels through a relatively straight duct in the dermis and goes into a coil-shaped duct until sweat reaches to the opening on the outer layer of the skin. The sweat that accumulates in the ducts and especially in the coily ducts, causes for different diffusion characteristics and mechanics that need to be accounted for when measuring the skin conductance. Further physiological aspects of the sweat glands and its mechanisms is not discussed further, as it is beyond the scope of this research and will be limited as a guide to the application thereof.



figure 1-5: Anatomy of the eccrine sweat glands in the skin (Dawson, Schell, & Filion, 2007)

Electrodermal activity (EDA) is a broad term that describes the changing of autonomic electrical properties of the skin. In short, when a person sweats, the skin conductance will increase. Following Ohm's law (eq.1), by applying an electrical potential between two points on the skin and the skin acting as a continuously changing resistor (R), the current flow (I) can be measured. The skin conductance (G) is expressed in the unit (micro) Siemens [S] which is the inverse of Resistance (eq.2).

$$R = \frac{U}{I}$$
 (eq.1) $G = R^{-1}$ (eq.2)

The circuit diagram shows the workings of the GSR with a constant current approach. The skin will act as a variable resistor (figure 1-6).



figure 1-6: Simple circuit diagram of a GSR device with a constant current approach. Resistor 1 is the skin conductance. (Ledalab, 2018)

The overall electrodermal activity signal that is captured, consists of a slower, tonic component (also known as skin conductance level (SCL) and a faster, phasic component (skin conductance response

(SCR). SCL can be seen as a baseline or background level of skin conductance and varies according to one's autonomous nervous regulation or psychological state.

The SCR however, reacts quickly to events, discrete stimuli like sound, noise, visuals etc. which can remain 10-20 seconds and will recover to baseline SCL. This response level differs highly between individuals, some individuals might react strongly and even multiple times on certain events while some do not at all and have stable SCL levels throughout the exposures. Typical SCL levels range from $10-50 \mu$ S, 1-3 non-specific background SCR's per minute and over 20 per min in high arousal situation (Boucsein, 2012). Both components are important to analyze.

Knowing that the overall skin conductance consists of two components, it is necessary to unravel the two superpositioned signals from the GSR. In figure 1-7, the course of a phasic SCR is depicted after certain stimuli. Typically, the latency between a stimuli and the maximum response is 1 - 4 seconds. (Dawson, Schell, & Filion, 2007). table 1-1 shows the typical values for (event related) SCR's.

Measure	Definition	Typical Values
Skin conductance level (SCL)	Tonic level of electrical	2-20 μS
	conductivity of the skin	
Change in SCL	Gradual changes in SCL measured	1-3 μS
	at two or more points in time	
Frequency of NS-SCRs	Number of SCRs in absence of	1-3 per min
	identifiable eliciting stimulus	
ER-SCR amplitude	Phasic increase in conductance	0.2-1.0 μS
	shortly following stimulus onset	
ER-SCR latency	Temporal interval between	1-3 sec
	stimulus onset and SCR initiation	
ER-SCR rise time	Temporal interval between SCR	1-3 sec
	initiation and SCR peak	
ER-SCR half recovery time	Temporal interval between SCR	2-10 sec
	peak and point of 50% recovery of	
	SCR amplitude	
ER-SCR habituation (trials to	Number of stimulus presentation	2-8 stimulus presentations
habituation)	before two or three trials with no	
	response	
ER-SCR habituation (slope)	Rate of change of ER-SCR	0.01-0.05 μS per trial
	amplitude	

table 1-1: Electrodermal measures, definition and typical values (Dawson, Schell, & Filion, 2007)

Important to note is that during an experiment, and especially for this thesis, is that there are many direct (i.e. motion sickness) and indirect stimuli (movement, temperature, visuals etc.) applied and followed by each other in short amount of time. This will lead to SCR's overlapping and cause underestimation of the phasic response if the overlapping of the response shape is not taken into account. In order to do so, different studies have proposed different mathematical or physiological methods to assess the SCR amplitude. A common and most simple procedure is to look at the event related activity after a given stimulus and to use a standard peak detection algorithm to find the amplitude of the SCR. SCRs occurring in the predefined 1-4 seconds is then caused by the stimuli. But then again, it gets complicated if events happen rapidly after another.



figure 1-7: SCR (Dawson, Schell, & Filion, 2007)

1.3 Thesis objective & Research questions

With the emergence of self-driving cars, the promised benefits of increased productivity and comfort sounds very appealing, until the imagined scenarios will lead to an increase motion sickness susceptibility and need to be addressed in other for this technology to be widely accepted and used. A way to decrease motion sickness in automated vehicles could be to adapt certain driving style or strategies in the automated driving mode, where motion sickness inducing movements are decreased to a minimum or avoided at all, if possible. For this, it is necessary to understand how or why motion sickness develops. What makes the assessment of motion sickness difficult is the highly individual nature and that there is no clear way of quantifying it. The most reliable and used method is with questionnaires. The illness rating questionnaires however are dependent on the timing, thus the resolution of the sickness over time. If more predictors can be used alongside questionnaires or even without it, the subjective aspect can be ruled out. It may provide a real time quantitative measurement for motion sickness, from which the automated driving system can adapt to.

It is clear that current literature on motion sickness in realistic driving scenarios are lacking, let alone about self-driving cars. The common accepted sensory conflict theory, although not yet fully understood, it is known that symptoms develop and signals thereof originate from the CNS, which can be measured.

In this thesis, the focus is on the physiological measurements, in particular the heart rate, heart rate variability and the skin conductance. These metrics have proven to be popular and have been used in various studies, e.g. (Dahlman, Sjörs, Lindström, & Ledin, 2009) (Golding, Markey, & Stott, 1995) (Wan & Hu, 2003) (Himi, et al., 2004). However, there is no clear consensus or evidence that these metrics provide an accurate, or predict even traces of motion sickness. One of the reasons is that some of the experiments only had small amount of participants. The studies have all been laboratory experiments where optokinetic drums, VR, rotating seats etc. have been used. Seldom, actual road test have been performed to assess (self-driving) motion sickness. In case of visually induced motion sickness, like virtual reality and car simulators, while fundamentally following the same visual-vestibular incongruences.

In some experiments, the experiment duration lasted shorter than 30 minutes or only had the illness rating of participants up to a certain minor nausea level before terminating the experiment. This could have prevented motion sickness from developing and possibly skewing the results. Together with the subjective nature of the symptoms in rating scales and the timing of measurements, experiment results will be more robust if motion sickness is allowed to develop longer and stronger.

In the case of HRV, researchers have been questioning the overall validity of the HRV itself (Quintana & Heathers, 2014) (Billman, 2013) and that it is too optimistic to determine (P)SNS activity, because of the underlying interaction of complex mechanisms.

With this road experiment, (self-driving) motion sickness will be tackled from different angles. The experiment is conducted together with T. Irmak and Yunyi Li. In the thesis of Yunyi Li, the relationship between visual conditions, head motion and motion sickness is studied.

The research objective of this study is to investigate and explore the usage and validity of the physiological measurements; HR, HRV and GSR, and to see if it could be a predictor of motion sickness in a realistic driving scenario.

The research objective can be divided into the following research questions:

- 1. Is there a correlation between heart rate and the MISC?
- 2. Is there a correlation between the HRV, expressed in LF/HF, and the MISC?

3. Is there a correlation between the skin conductance and the MISC?

2. Methods

2.1 Experiment

Participants and Procedure*

Students and staff in Technology University of Delft were recruited to participate in our experiment. The Motion Sickness Susceptibility Questionnaire (Griffin & Howarth, 2000) (Appendix A) was first finished by the candidate participants for pre-screening. The MSSQ investigated the participants' travelling, illness and vomiting frequency respectively in cars, buses, coaches, small boats, ships, airplanes and trains in the past, and the candidates who indicated that they had never suffered from any motion sickness symptoms as a passenger in any mode of transport were excluded for this experiment. However, none of the pre-screening candidates met this last criteria. A total of 23 healthy participants participated in this experiment, with only 18 finished 2 trials with both visual conditions and 11 with available body motion suit data (XSENSE-MVN) that is used in Yunyi Li's study. Data of 20 participants were used for ECG analysis and 17 for the GSR analysis after filtering out the unusable ones, moreover in section 2.3. table 2-1 shows the gender, age and susceptibility score based on MSSQ of the participants and a population reference. The participants in this experiment have relatively higher susceptibility compared with population especially in median and 75th percentile. No participant had suffered from any serious illness or injury or was under any relevant medical treatment.

		23 participants (total)	18 participants (MISC analysis)	20 Participants ECG	17 participants GSR	Population Reference
Gender		Male: 17 Female: 6	Male: 14 Female: 4	Male: 13 Female: 7	Male: 12 Female: 5	Male: 333 Female: 0
Age		30.0 (SD = 128.7)	28.4 (SD = 98.9)	29.2 (SD = 10.65)	25.6 (SD = 2.82)	18-26
_	minimum	-2	-1	0	0	-2
Susceptibility (MSSQ) (range: -2-177)	25 th percentile	5.5	5.25	7	5.75	4
	median	17	16	18	18.5	9
	75 th percentile	25	27.25	25	25.75	17
	maximum	64	64	42	42	69

table 2-1: Participants gender, age and susceptibility (population reference: (Griffin & Howarth, 2000))

The experiment was performed at the Research lab Automated Driving Delft (RADD, www.raddelft.nl). Tests were performed at the Heertjeslaan, in Delft, which was closed to other traffic during the experiment. Prior to each trial, the participant was invited to the RADD control room in the Green Village for preparation. The experimental procedure as well as the MISC were illustrated to the participant, followed by the informed consent form being signed. Then the ECG and GSR leads were attached to his/her body and the XSENSE-MVN suit with motion trackers were put on, in the meanwhile, the vehicle was being prepared according to the specific visual condition. After calibration of the MVN trackers, the participant was asked to board the vehicle, leaving for the road and starting a 30-minute journey. There was a driver and an assistant sitting in the car during the whole journey

and the latter was responsible for setting up all the recordings and asking the participant to rate their MISC ratings every minute. After each trial, the car was stopped on the road while data was saved and the assistant kept on recording the participant's MISC to acquire recovery information. Until the participant regained a low MISC (2 or 3), the car was driven back to RADD and the participant was asked to fill out the MSAQ rating their symptom levels, while the next participant was being prepared. All the participants took part in both two conditions with each condition one and a half hour (including preparation, testing and questionnaire). To minimize the effect of environment characteristics (e.g., temperature, humidity, etc.) and individual characteristics (e.g., diet, sleep, etc.), each participant experienced both trials at approximately the same time of the two days, with an average of one-week (minimum 2 days and maximum 20 days, figure 2-1) interval between them for refreshing and eliminating the effect of habituation. Prior to each trial, the participant was required: 1. Not to intake any recreational drugs (including alcohol) for at least 24 hours; 2. Having a good night's sleep; 3. Not to consume excessive food for few hours preceding the experiment. Such instructions ensure the participants were in as natural physical and mental state as possible.

Up to 4 subjects per day were tested from 10 a.m. to 4 p.m., and the experiment took 3 weeks (11 days) in total. To further prevent the possible habituation and order effect, the order of the conditions that each participant experienced was randomized by Latin Square, and final results were compared within subjects.



figure 2-1: Days interval between the two trials for the same participant (mean: 8.4 days; SD: 4.8 days)

Road and Vehicle*

For this experiment, TU Delft and the municipality agreed on closing Heertjeslaan in both directions from intersection with Huismansingel to intersection Molengraaffsingel for 14 work days. Companies in direct surrounding were notified in advance for the road closure. As required by the authorities, at both ends of the road, fences and cones were placed. Also, the assistants had to be near the closure to warn and provide information to road users that wanted to pass, as well as guarantee safety for both experimenters and road users. The total length of the road is approximately 240 meters and 10 meters wide (figure 2-3: (a)).

The vehicle that is used to do the experiments, is the TU Delft's driverless Toyota Prius (figure 2-4). It is being equipped with TU Delft's technology to enable self-driving capabilities and provides a platform for researchers to work with. At first, automated driving was planned and routes were preprogrammed to keep the driving path conditions consistent, but during the testing period, the automation was temporarily disabled for an upgrade, therefore the Prius had to be manually driven. In and on the vehicle, different equipment was pre-mounted for the existing systems (e.g. in the trunk, in the center console, stereo-camera behind the front windshield etc.), but did not hinder the participants nor the experiment in any way (vision, airflow, noise etc.). The equipment also did not change throughout the course of the experiment.

Participants were requested to sit in the center position of the back seat and wear a seatbelt. During pilot tests, it was noticed that some of the participants slid off with their buttocks from the center seat towards either side, due to the relative slippery surface and the shape of the seat. This was solved by placing a friction mat on the backseat area. Since temperature might affect motion sickness, the air conditioner was turned on and set to 18 degrees Celsius with the blower on max on all trials to control for temperature. The weather and road conditions were similar during all the trials.



(a)



figure 2-2: TU Delft's driverless Toyota Prius being manually driven on the experiment road – (a) road; (b) vehicle

(b)

Motion*

To investigate motion sickness, it was necessary to induce provocative motion to the participants with the car. It has been shown in previous studies (O'Hanlon, 1974) (Donohew & Griffin, 2004) and in the ISO norms (ISO, 1997), that in either longitudinal, lateral or vertical direction, motion sickness is caused mostly by accelerations around 0.16-0.20 Hz frequencies. Compared with the longitudinal accelerations, the vehicle lateral acceleration could result in more symmetric passenger's motion which is more synchronized with the car motion, due to the backrest. Besides, the lateral motion can provide more visual cues as well as anticipatory information if the passengers are looking at the forward road, where more effects of vision could be expected. Therefore, different road scenarios have been considered with frequencies ranging from 0.16 Hz to 0.2 Hz for lateral accelerations. The choice of the paths and velocities of the sickening drive was mostly determined and limited by the physical properties of the designated road, frequency range.

Knowing that motion sickness in vehicles usually develops in 20 to 30 minutes onwards and that the aim of this research is to investigate motion sickness caused by lateral acceleration in a realistic setting. The lateral acceleration has been selected in a relatively high range in order to make most of the participants reach slight nausea but not too sick in 20-30 minutes. The relatively-highly aggressive motion could acquire a larger range of MISC reaching the maximum of 7 which is easier for observing the growth in time and comparing between conditions. While the drawback of a high-acceleration motion that some of the participants could stop the session before 30 minutes and the different lengths of trials cause some troubles in time-domain data analysis. Furthermore, the lateral acceleration should fall within 0.1G and 0.4G to mimic similar conditions in urban driving and for safety reasons in case of the maximum 0.4G. Similarly, this holds true for the velocities from 15 to 30 km/h that has been considered. Another point that has to be taken into account is the drivability of the chosen path, as it has to be driven manually. The constraints of motion scenario are shown in table 2-2.

Road width, max amplitude	8 m, 3.5 m
Road length	240 m
Frequency	0.16 – 0.2 Hz
Velocity	15 – 30 km/h
Peak lateral acceleration	0.1 G – 0.4 G

table 2-2: Design test criteria

Due to the constraints of the road and the focus on lateral acceleration of this research, a slalom path has been opted for. With this, the longitudinal, lateral acceleration, velocity and therefore its frequency, can be controlled for. Then matlab scripts were used to generate different sinusoidal paths with the aforementioned design constraints and its MSDV for comparison. The turning radius of the Toyota Prius is 5.5 meters, so for the experiment drive, it was necessary to make a 3-point u-turn upon reaching the end of the road. Considering drivability and optimum road usage, a slalom path with the following properties (table 2-3) has been chosen:

Amplitude	3.5 m
Road length	220 m
Frequency	0.175 Hz
Velocity	25 km/h
Lateral acceleration	0.4 G

table 2-3: Motion scenario with slalom path

The reference slalom (single section without u-turn) is depicted in figure 2-4. To drive the intended slalom with the specific frequency, the driver set up a metronome with 21 bpm as reference to drive the slalom (0.175*60*2 = 21 for a signal per turn). The path length has been shortened to incorporate

the 3 point u-turns and to handle a safety margin at the ends of the road. Throughout all the trials, the driver also was required to keep the velocity of vehicle as constant as possible and to take the 3 point u-turns as consistent as possible. A realized motion is shown in figure 2-5.



figure 2-4b: Designed vehicle movement for one slalom – (a) lateral acceleration; (b) path



figure 2-5: Vehicle movement during experiment

Vision*

All the participants finished two sessions with two different visual conditions in this experiment. The two conditions were tested on different days, and the order of the two conditions were balanced by Latin Square (half of participants did EYES-ON-ROAD condition first and the remaining half reversed). In both visual conditions, the participants were always seated in the middle of rear seat which ensured clear and broad road vision when there was no blockage between the passenger and the windscreen, and the participants were also asked to maintain the same straight-up sitting posture and facing forward with no active head or body pitching, rolling or yawing, while ensuring legs planted adequately wide for stability. A friction mat was positioned on the rear seat to prevent body slide of passengers during the car turning. To ensure the presence of external vision and internal vision, closing eyes is not allowed during all sessions.

- **EYES-ON-ROAD condition.** During the sessions with EYES-ON-ROAD condition, the participants were asked to always forward view the external environment of the vehicle without any blockage in the car, provided considerable both foreground view and peripheral view with road information.
- EYES-OFF-ROAD condition. During the sessions with EYES-OFF-ROAD condition, the
 participants were asked to view forward as well but with cardboards both in front of the rear
 seats and on the side windows, so that neither foreground view nor peripheral view was
 provided to the rear-seat passengers compared with EYES-ON-ROAD condition. The blocking
 cardboards are pure white without any mark or pattern that could provide visual cues for
 passengers.



figure 2-6: Eyes-off-condition in the vehicle

Previous studies suggested that passengers with eyes on road tend to suffer from less motion sickness symptoms than those with eyes off road due to earth-fixed visual cues and anticipatory information provided by forward vision which are helpful for the passengers to estimate their body motion (Turner & Griffin, 1999) (Griffin & Newman, 2004). In this experiment, cardboards in the EYES-OFF-ROAD condition positioned in front of the participants was to block all the outside visual cues and anticipation which was fully provided in the EYES-ON-ROAD condition.

In real life, situations with EYES-OFF-ROAD riding are always accompanied by visual and mental tasks, such as reading and watching electronic displays. Such visual tasks were initially considered to add in this experiment for EYES-OFF-ROAD condition to mimic natural driving as well as guarantee no external vision achieved by the participants, however, additional visual or mental tasks will introduce additional and uncontrollable variables between the two visual conditions. For example, additional visually-induced motion sickness could be involved by visual tasks like reading or watching stable images; mental tasks could affect passengers' motion sickness per (Bos, MacKinnon, & Patterson, 2005). Therefore, a task must be added to both visual conditions to neutralize the effect induced by itself, which will on the contrary influence the completeness of external vision for EYES-ON-ROAD condition. Simply blocking view is more scientific and controllable for creating EYES-OFF-ROAD condition, where literally effect of visual cues and anticipatory information of external vision can be investigated.

2.2 Motion Sickness Measurements*

Misery Scale (MISC)

During the sessions, the severity of motion sickness symptoms was repeatedly measured by the 11point MISC (MISC, table 2-4, (Bos, MacKinnon, & Patterson, 2005)). Specifically, for each 30-min session, the participants were required to rate their temporary illness using the MISC before the sessions, every minute during the sessions and after the sessions respectively, being asked by an assistant sitting at the front seat. Any participant indicating an MISC of 7 or above during the sessions would automatically stop driving immediately and the rest of ratings would be considered as the largest point he/she ever rated.

The subjective illness rating has been widely used in motion sickness researches, providing the most intuitive information about the incidence of motion sickness symptoms, which was more natural and

convenient compared with physiological measurements such as measuring the skin conductance of subjects, and more sensitive and detailed compared to observational methods such as recording the percentage of subjects who vomited, which was not considered since vomit would cause unnecessary excessive suffering and would also reduce the turnout rate for the preceding sessions in our within-participant experiment. The illness rating form was able to measure the mild sickness symptoms that subjects were aware of rather than reaching emesis. In comparison with the traditional 7-point illness rating scale (Griffin & Howarth, 2000), the 11-point MISC used in this experiment exploited more various symptoms for the participants as a reference, avoiding individual differences in the occurrence of symptoms, for example, discomfort in either head or stomach was considered as the same degree of sickness. Once the participants were familiar with this scaling form before the sessions, they only took a few seconds to rate during the journeys.

The 11-point MISC has been validated in the experiment by (Bos, MacKinnon, & Patterson, 2005), who studied the motion sickness symptoms in a ship simulator among 24 participants and found the maximum MISC values observed per participant per trial correlating linearly with the predicted MISC by his/her symptom checklist as well as MSSQ result.

Motion Sickness Assessment Questionnaire (MSAQ)

To investigate the specific sickness related symptoms appearing among participants an additional symptom checklist was applied after the stimulus exposure. The MSAQ (table 2-5)(appendix C) was explored and validated by (Gianaros, Muth, Mordkoff, Levine, & Stern, 2001), involving motion sickness related symptoms in four dimensions – gastrointestinal (1, 5, 11, 15), central (2, 6, 9, 13, 14), peripheral (4, 8, 12), and sopite-related (3, 7, 10, 16). This multidimensional assessment was used after each trial for the participants to rate the severity of symptom in each dimension that has appeared during the travel. Per symptom, one could rate the severity from 1 (not at all) to 0 (severely).

Symptoms		MISC
No problems		0
Some discomfort, but no specific symptoms		1
Dizziness, cold/warm, headache, stomach/throat,	Vague	2
awareness, sweating, blurred vision, yawning,	Some	3
burping, tiredness, salivation,but no nausea	Medium	4
	Severe	5
Nausea	Some	6
7 = stop	Medium	7
	Severe	8
	Retching	9
Vomiting		10

table 2-4: 11-Point MISC (Bos, Mackinnon & Patterson, 2005)

1. I felt sick to my stomach	9. I felt disoriented
2. I felt faint-like	10. I felt tired/fatigued
3. I felt annoyed/irritated	11. I felt nauseated
4. I felt sweaty	12. I felt hot/warm
5. I felt queasy	13. I felt dizzy
6. I felt lightheaded	14. I felt like I was spinning
7. I felt drowsy	15. I felt as if I may vomit
8. I felt clammy/cold sweat	16. I felt uneasy

table 2-5: Symptoms involved in MSAQ (Gianaros, Muth, Mordkoff, Levine, & Stern, 2001

2.3 Physiological measurements

ECG – Electrocardiography

The ECG data was recorded using the TMSi Mobita amplifier device (figure 2-7).



figure 2-7: TMSi Mobita

This amplifier unit captures (electro)-physiological signals via electrode leads with the following technical specifications:

Input		
input channels	32	
peak to peak input signal difference	400 mV	
input common mode range	-2.0 V to +2.0 V	
gain factor	10	
RMS noise level	< 0.4 µV @ (0.1 to 10) Hz	
Input resistance	>100 MΩ	
A/D Conversion		
Resolution	24.414 nV/bit, referred to input	
Sample frequency	2000 Hz, 1000 Hz (used here), 500 Hz, 250 Hz	
Channel bandwidth	DC up to 0.13 * sample frequency	

table 2-6: relevant TMSi Mobita hardware specification (TMSi, 2016)

Three surface electrodes (leads) were attached to the participants' chest according to the configuration shown in the figure 2-8 below. V1 lies where the 4th intercostal space meets the sternum. V2 is on the direct opposite side of V1 across the sternum. V3 is placed in the 5th intercostal space below V2 and sits towards the left. It is placed along the nipple line or in the crest underneath the breast for females. V4, V5 and V6 are not used. Before placing electrodes, the relevant area was cleaned, shaved if needed and prepared with alcohol to ensure good connectivity between the electrodes and the skin. Ambu BlueSensor electrodes were used during the whole experiment. The ground lead was attached with a wet wristband onto the free wrist opposite to the one with the GSR electrodes attached. ECG signals were amplified and recorded with a sample rate of 1000 Hz in a CSV format with the UTC timestamps acquired from the connected laptop. After the participant entered

the car, the experimenter or assistant connected the Mobita amplifier with all the leads, including the GSR and the ground, and started the recording prior to the drive to the testing location. This is done to verify the nominal operation of the equipment and to acquire baseline ECG data before exposure of vehicle motion. It continued recording until the whole session was completed and the car returned to the RADD control room, then the data was saved and the device with the electrodes were disconnected afterwards. The data captured consisted of two separate ECG signals that can be used. Depending on signal quality, either one is chosen during pre-processing. Matlab was used to pre-process and analyze the data. The pre-processing consisted of detecting the QRS peaks with a peak detection algorithm that compared the peaks with wavelets and saves the time point and location thereof. Both of the ECG data were checked and the data that had better distinguishable peaks and less errors would be chosen to be used and processed further.

The so called QRS complex is a (graphical) representation of the Q wave, R wave and the S wave that signifies the (de)polarization of the heart ventricles, causing the heart to contract for circulation (figure 1-4). With the electrodes, the electrical activity caused by the depolarization of each heartbeat can be measured. Using the ECG, it is also possible to detect or diagnose various clinical cardiac problems, physiological states like stress and strenuous activities. For our purposes, it was only necessary to capture the QRS complex and, in particular, the R peaks. The interval between two consecutive R peaks can be used to calculate, for instance, the heart rate and the heart rate variability amongst other metrics, which will be discussed in the next section.

Peaks that were not detected or detected wrongly were edited manually in Matlab in conjunction with R-R plots (figure 2-10, figure 2-11). The R-R plot, shows the interval between two successive R peaks with respect to time and the Poincare HRV plots each RR interval to its next interval.

The raw ECG signals are often noisy due to the way that ECG works. The electrodes are attached to the skin and is sensitive to local disturbances like muscle activity, interferences, positioning and attachment of the electrodes, skin conductivity and movement of the participant. Especially for our dynamic road tests, the latter might cause the device to capture a lot of noise in the signals.



figure 2-8: Placement of the ECG electrodes. Only V1, V2 and V3 were used.



figure 2-9: QRS wave (wiki, 2018)



figure 2-10: Example of a detrended ECG plot with undetected peaks by the algorithm at 100.75 and 101.5 seconds.


figure 2-11: Using RR plot to inspect and look for unidentified peaks. Unidentified peaks will show a spike in the RR plot while mis-identified peaks will show a negative spike.

Analysis

Heart rate

The first step of the analysis is to preprocess the raw data as mentioned. After annotating all peaks, the average (instantaneous) heart rate (HR) can be calculated and plotted, as every peak to peak time signifies one beat (fig of RR before). The Matlab script then calculates the HR with a moving window of 60 seconds and its respective time.

With the heart rate known for the whole trial, it can be further subdivided into before, test and posttest. In figure 2-12, a typical heart rate plot is shown for a single trial, with a one minute window before the test to mark the baseline heart rate. The acquisition of EEG data started upon leaving the RADD control room but analyses and calculations were performed over the data starting from the minute prior to the actual start as the baseline until the trial was stopped. This was either MISC rating reaching 7, reaching 30 minute mark or participants requesting to stop the test.



figure 2-12: Typical heart rate data with the accompanying MISC over time, calculated from the RR peaks. Plot shows Participant 10 with Eyes-ON-road (vision) condition who completed the 30 minutes.

By comparing the heart rate of the participants over the trial with their MISC ratings, it is possible to investigate whether heart rate (and heart rate variability, moreover in the next section) might be a possible indicator for the onset of motion sickness or nausea, as, for example, (Holmes & Griffin, 2001) have shown in their study.

Experiment durations differed between trials as many were terminated before 30 minutes due to MS. To enable comparison of trends, results were compared for 4 representative intervals.

Mean heart rates were obtained during the baseline period of 1 minute before the test; the first two minutes ('Begin') after the start, two minutes in the middle of the test and in the final two minutes of the test for all the trials. The bracket of two minute is chosen because the shortest trial of the experiment lasted for 6 minutes. The same window has been applied for subsequent analysis with GSR. For another comparison, heart rates were aggregated and averaged at every MISC interval time to show mean heart rates at every single MISC rating.

Lastly, the HR as a function of time was fitted with a simple function as shown in figure 2-12. The function used 4 parameters: P1 the HR before driving (t<0), P2 the peak HR emerging shortly after the onset of driving, P3 the HR when the experiment was stopped and P4 the time at which peak P2 occurred. The fitting minimized the summed square errors between the heart rate and the fitted line. Due to the parameter P2 this is a non-linear problem which was solved with a gradient based Gauss-Newton scheme with adaptive Levenberg-Marquardt term (Happee, 1989). The pre-drive HR was recorded rather briefly making parameters P1 and P4 imprecise. P2 is mainly relevant in comparison with P1. Hence mainly the slope between P2 and P3 was reported. This slope was generally negative.

Depending on the trial, the mean heart rates are labeled 'sensitive' or 'non-sensitive' and/or 'vision' or 'no vision' if applicable (vision = eyes-ON-road, no vision = eyes-OFF-road).



figure 2-13: Example of a HR vs time plot with the raw unsmoothed (instantaneous) HR (light blue), smoothened HR blue, fitted HR (dashed red) and the average HR at 4 intervals.

Heart rate variability (HRV)

After the ECG has been thoroughly annotated, the locations of the peak with respect to time are resampled and saved within Matlab. The RR can be plotted with respect to time with the accompanying MISC. The amplitude of the peaks itself are not required.

Having the RR data, it is possible to analyze the HRV in the frequency domain and to see whether the power spectrum will reveal (para)sympathetic activities when the participant is reporting a higher MISC. Similarly to heart rate analyses, the power of the frequencies are compared over different time points with different groups (sensitive versus non-sensitive).

Since the (para-)sympathetic activation are associated with low (LF) and high frequency (HF) power, it is often shown as LF/HF ratio. From the theory, it is known that the frequency band predominantly focuses on the very low frequency (VLF) from 0 to 0.04 Hz, low frequency (LF) from 0.04 Hz to 0.15 Hz and high frequency band (HF) from 0.15 to 0.4 Hz.

Within the (time-) frequency domain, several methods are employed in this research to analyze the underlying frequencies that might show a relationship between motion sickness and the HRV.

LF/HF Ratio

Using the fast Fourier transform function in Matlab, the power spectral density is calculated for (parts of) trials. The sum of the LF power band (0.04 - 0.15 Hz) with 60 second window is divided with the sum of the HF power band (0.15 - 0.4 Hz) at that specific time to obtain the LF/HF ratio.

Finally, the LF and HF band ratio is calculated over time and averaged over the time points to see if there is indeed a correlation between reported MISC and nausea and a change in LF/HF ratio that could indicate or predict motion sickness.



figure 2-14: Typical LF/HF frequency plot for participant 10

GSR – Galvanic Skin Response

The galvanic skin response (GSR) was measured using the Mobita capturing device. The GSR leads are connected to the amplifier in a similar fashion on the Mobita device and the electrodes are placed to the palmar finger site on the same hand (figure 2-15). Studies have shown that the phasic skin conductance responses at forehead site correlate the best with motion sickness (Golding, 1992), while the palmer finger site was a close second (r = 0.62 vs r = 0.48) in a motion sickness experiment with a rotating drum (Wan & Hu, 2003). In order to create a less intrusive and more comfortable experience for the participant inside the vehicle, the palmar finger site was chosen over the forehead site to measure the skin conductance.

Before placing the electrodes, the fingers or palm had to be cleaned with alcohol to ensure good connectivity with the electrodes. Due to the GSR amplifier limitations, it could only capture signals within a range of 200 Ohm to 700 Ohm, impedance and it was necessary to measure the impedance after placing the electrodes. If impedance fell out of range with the Ambu Bluesensor N electrodes, the wet gel was removed or different electrodes, Skintact with solid gel, was used. Different placements or electrodes were tried until the impedance was in range. After completing this step, the participant's skin conductance was ready to be recorded.

Similar to the ECG measurements, the skin conductance was captured with a sample rate of 1000 Hz, along with the timestamping from the laptop and recorded simultaneously with the ECG.



figure 2-15: Electrode placement on the palm of the hand with Ambu Bluesensor N



figure 2-16: GSR mapping acquired by testing the unit

Analysis

After the GSR data is captured, which is in millivolt, it is then converted to micro Siemens according to the reference in figure 2-16, before it is processed with Ledalab (<u>http://www.ledalab.de/</u>). The mapping is acquired by running multiple measurements across different values of resistors.

A Matlab-based software that preprocesses and analyzes skin conductance data and decomposes it to the phasic (SCL) and tonic (SCR) data using the methods 'Continuous Decomposition Analysis (CDA)' or Discrete Nonnegative Analysis (DDA)' described in the papers of (Benedek & Kaernbach, 2010) and (Benedek & Kaernbach, 2010) for further analysis.

The study of (Benedek & Kaernbach, 2010) proposed a method that encompasses preceding methods to assess the shape by curve-fitting (Lim et al, 1997), decomposition method by means of

deconvolution an impulse response function (IRF) (Alexander et al., 2005) and nonnegative deconvolution (Discrete decomposition analysis (DDA)) that corresponded better to the physiological behavior of the sweat diffusion (Benedek & Kaernbach, 2010). This method retrieves the continuous tonic and phasic data, shows a zero baseline and distinct phasic activity. According to the documentation of Ledalab, CDA provides a robust and comparatively fast computation in decomposing the signals. It is recommended for the analysis of skin conductance data. Moreover, DDA explores all intra-individual deviations of the general response shape (figure 1-7) and tries to acquire a detailed model of all the components in the entire dataset. For large datasets, like in this research's case (30 minute data at 1000 Hz), it is slow and therefore, the CDA method is used.



figure 2-17: Plot of the processed data with its tonic and phasic component over time. The black lines indicate the start and end of the trial.

As SCL and SCR are decomposed from the GSR (figure 2-17), the baseline, beginning, mid and end phase of the experiment can be compared with each other to see if motion sickness or MISC can be correlated. Same time windows of 1-2-2-2 minutes for the phases respectively will be used to calculate the mean.

According to (Lykken & Venebles, 1971), the differences in amplitude of SCL and SCR between individuals originate from physiological differences (e.g. thickness of the skin) and are unrelated to the physiological processes. Therefore the variation thereof is more important than the amplitude. So, because of the individual differences, the SCL data will be normalized with baseline SCL values, similarly as in (Himi, et al., 2004) (Wan & Hu, 2003). As for the SCR, the number of peaks will be counted in the respective time points and averaged. The SCR are normalized with respect to the baseline as well, but this time the peaks measured at every time point will be subtracted by its corresponding baseline peak amount. The mean number of peaks for SCR and the SCL ratios will be analyzed for the sensitive versus the non-sensitive group. The amount of SCRs per phase will give insight to the stimuli or events that might be linked to symptoms that appear during the experiment. As the experiment is a constant wave of external stimuli, e.g. noises, lights and sudden movements, a continuous flow of SCR peaks are to be expected.

Statistics

To compare the physiological measurements mentioned before, the following statistical methods have been applied using Matlab.

Physiological data (heart rate, low frequency (LF) HRV, high frequency (HF) HRV, LF/HF ratio, GSR) were all recorded continuously from at least 1 minute before start of the trial until several minutes after the test, when it was either completed (30 minutes) or because of MISC rating reached 7. Repeated measures ANOVA was applied to the aggregated means at 4 (baseline 1 min before driving, begin 2 minutes, middle 2 minutes, end phase 2 minutes) of the physiological data, subdivided in sensitive and non-sensitive groups, to see if there is an effect of the corresponding physiological data between the sensitive and non-sensitive group. The Greenhouse-Geisser corrections were applied if

Secondly, for the mean heart rates at every MISC interval, a 1-way ANOVA was used to assess whether the means were statistically different between sensitive and non-sensitive groups at different time points. The Bonferroni correction was applied as a post-hoc analysis when individual means were tested against each other.

P-values (two-tailed) lower than 0.05 were regarded as statistically significant.

Flowchart of data process

sphericity was violated.

Here is a summarized overview in the form of a flowchart of the methods and the general steps that are taken to obtain the desired data output for the statistical analysis and the results section.



figure 2-18: Flowchart of the data processing and analysis.

3. Results

3.1 Vehicle motion

The car orientation and linear acceleration were continuously measured by an Xsens MTi-G located under the passenger's seat. During each trial, the driver was managing to keep the car lateral frequency around 0.175 Hz guided by a metronome with frequency of 10.5 bpm (beats per minute). Except the trials for himself, only one driver was driving for all of the trials, ensuring the similar vehicle motion throughout the experiment.

An example of the car's linear accelerations in three direction for a 6-minute trial is shown in figure 3-1. As the Xsens MTi-G sensor was not located horizontally under the vehicle seat, a gravity component has been included in the longitudinal direction, which was corrected using rotation matrix (see red lines in figure 3-1). After correction, the root mean squares of the longitudinal, lateral and vertical (gravity included) acceleration in this trial are respectively 0.68 m/s², 2.20 m/s² and 9.75 m/s², including 7 slaloms and 7 U turns that were necessary due to the limitation of road length. No difference was observed in car motion between the trials.

figure 3-2 shows the three accelerations in frequency domain - the normalized magnitude after Fourier transform together with the cumulative power (the estimated value of gravity, 9.8 m/s², in vertical direction was removed to show better comparison). It suggests that only lateral acceleration was excited drastically around 0.2 Hz – the frequency has been proved most effective in evoking motion sickness for all kinds of transports. Therefore, most of the motion sickness symptoms resulted from vehicle lateral motion in this experiment, only which will be considered in the following analysis.



figure 3-1: Car longitudinal, lateral and vertical acceleration in time for a 6-min trial (blue –raw data; red – corrected for gravity components in longitudinal and vertical direction)



figure 3-2: Power and cumulative power of car longitudinal, lateral and vertical accelerations in frequency for a 6-min trial

figure 3-3 shows the averaged MISC at every minute for both visual conditions of 18 participants. If the MISC of a participant reached 7, in the averaging the subsequent ratings were regarded as 7 until the 30 minute mark. The individual MISC over time for 18 participants with both conditions are shown in figure 3-4. From the first figure, the most notable difference is that the eyes-off-road condition show rapid increase in MISC in the first 10 minutes. This is even more obvious in the gradient line of the MISC ratings over time, where the MISC gradient spikes at around 5th minutes in the experiment for eyes-off-road participants, whereas eyes-on-road condition, experience a gradual increase of MISC. Looking at the individual MISC plots (figure 3-4), it can be seen that most trials with eyes-off-road conditions reached MISC 7 after approximately 10 minutes. Because of the averaging, the MISC gradient of the eyes-off-road condition seem similar to the eyes-on-road condition, but 40 % of the participants with eyes-off-road condition have quit the test after 10 minutes with MISC reaching 7. Most participants in trials with eyes-on-road condition endured the full 30 minutes test.

In the individual plots, also different motion susceptibilities can be noticed between different participants. It hints that there are individual differences of sensitivity for or against motion sickness, that can be seen from the MISC profiles. For example, few participants (from left to right in figure 3-4: 3, 17, 18) were fairly insensitive to MS and survived the whole trial for both conditions. Participants with MISC reaching 7 exponentially in very short time for the eyes-off-road condition (1, 6, 7, 8, 16), often had relatively higher MISC in the eyes-on-road condition as well.

This experiment was performed together with Yunyi Li and therefore has the visual condition included in the experiments. For this study, it is expected that the difference between the eyes-on-road and eyes-off-road will be negligible and will not considered, unless otherwise stated.

However, for this study the distinction will be made between participants reaching MISC 7 (sensitive), and participants who did not reach MISC 7 (non-sensitive). The convention of non-sensitive group (MISC < 7) and sensitive group (MISC = 7) has been chosen to distinguish physiological changes, ECG and GSR respectively, more clearly in participants who experience heavy motion sickness against relatively less sickness.



figure 3-3: Participants mean MISC growth in time for two conditions (upper). The lower plot shows the gradient of the average MISC at every minute for both conditions.



figure 3-4: MISC over time for 18 participants in two visual conditions, participant 1-9 from top to bottom (left), and 10-18 (right)

3.2 ECG

For the ECG measurements, a total of 34 trials have been recorded, where 17 consisted of trials with eyes-ON-road and 18 eyes-OFF-road. Upon analyzing data, depending on the severity of the artefacts or interrupted recordings, several have been discarded. Some raw data showed too many artefacts or too weak signals where individual QRS waves were not detectable.

In the end, 31 trials are deemed useable for ECG analysis. Out of the 31 recordings, 14 fell in the nonsensitive group and 17 into the sensitive group, where MISC reached 7 within 30 minutes.

Although the experiment was run with two visual conditions, no distinction was made for either condition and is considered as an independent trial.

3.2.1 mean heart rate

1. Mean heart rates of the sensitive group versus the non-sensitive group

The means of the heart rate during the first minute before test started, first 2 minutes, middle 2 minutes and final 2 minutes of the test are divided into 2 groups and analyzed are shown in figure 3-5.



figure 3-5: Boxplot of the mean HR at different time points for the sensitive and the non-sensitive groups. Pre = prestart or baseline, NS = non-sensitive group (blue), S = sensitive group (red).

Repeated measures 1 way ANOVA shows that there is a significant effect between the HR and time (F = 12.8, p < 0.001). Interaction between sensitive and non-sensitive group with time was found to be significant (F = 2.8164, p < 0.05). Sphericity was checked with the Mauchly's test and after correction with the Greenhouse-Geisser approximation, the effect is deemed insignificant with p = 0.073. Although the effect between the groups and HR is insignificant, there is a slight trend that can be noticed. Overall, the HR of the non-sensitive group declines sharper after an initial rise after the

baseline. Also, the mean HR of the sensitive groups were higher than the non-sensitive group, but were not found to be significant (p = 0.1444). In table 3-1, the means and standard deviations are shown for both groups at every time point. The corresponding boxplots are shown figure 3-6a and b.



figure 3-6:Boxplot with the mean HR at different time points for the non-sensitive (a) and sensitive group (b). figure 3-7: (b)

	Pretest	Begin	Mid	End	F	р
Non sensitive	75.776 (5.646)	83.614 (7.630)	78.051 (9.957)	74.584 (8.365)	3.47	0.0226*
sensitive	80.719 (10.63)	87.057 (10.808)	87.057 (10.808)	86.232 (10.721)	1.87	0.1444

table 3-1: Mean heart rates and standard deviations for the sensitive and non-sensitive group.

When the means at different times are compared to one another within each group with a 1-way ANOVA test, it indicated a significant difference between the means (F = 3.47, p < 0.05). The mean HR's of the begin and end phase of the test were statistically significant (p < 0.05) with effect size (η^2 = 0.1667) after the post-hoc Bonferroni analysis, as seen in table 3-2.

			Mean (SD)	Mean (SD)	p-value
Pretest	-	Begin	75.776 (5.646)	83.614 (7.630)	0.0772
Pretest	-	Mid	75.776 (5.646)	78.051 (9.957)	1
Pretest	-	End	75.776 (5.646)	74.584 (8.365)	1
Begin	-	Mid	83.614 (7.630)	78.051 (9.957)	0.4394
Begin	-	End	83.614 (7.630)	74.584 (8.365)	0.0271*
Mid	-	End	78.051 (9.957)	74.584 (8.365)	1

table 3-2: Post-hoc Bonferroni analysis for the mean heart rates of the Non-sensitive group.

As for the sensitive group, a 1-way ANOVA indicated that there was no significant difference between the means that were tested (F(3, 64) = 1.87, p = 0.144), with effect size η^2 = 0.0804.

			Mean (SD)	Mean (SD)	p-value
Pretest	-	Begin	80.719 (10.631)	83.614 (7.630)	0.169
Pretest	-	Mid	80.719 (10.613)	87.057 (10.808)	0.521
Pretest	-	End	80.719 (10.613)	86.232 (10.721)	0.811
Begin	-	Mid	88.895 (10.317)	87.057 (10.808)	1
Begin	-	End	88.895 (10.317)	86.232 (10.721)	1
Mid	-	End	87.057 (10.808)	86.232 (10.721)	1

table 3-3: Post-hoc Bonferroni analysis for the mean heart rates of the Sensitive group.

2. Mean heart rate at every MISC interval of the non-sensitive versus the sensitive group

The next plot (figure 3-8) shows the average heart rates at every 1 minute for the duration of corresponding MISC rating. When the MISC is zero, it can be seen that the average heart rate for both groups is approximately 80 bpm, but as the MISC rating is increased to one, the average heart rate develops very differently across the groups. The mean heart rate of sensitive group has increased to 86 bpm while the non-sensitive group has decreased to 76 bpm. Both groups then show a relative small decrease in the mean heart rate when MISC is two and continues to increase again when MISC is 3. This plot shows that the average heart rates of the 2 groups develop differently if the heart rate is grouped by MISC rating. The mean and standard deviations of both groups are listed in table 3-4.



figure 3-8: Plot of all mean HR at each MISC rating

	Non-Ser	nsitive group	Sensitiv	e group
MISC	n	Mean (SD)	n	Mean (SD)
0	189	79.462 (8.883)	27	80.026 (9.049)
1	134	75.794 (8.796)	34	86.281 (10.976)
2	64	74.730 (7.078)	31	85.378 (11.922)
3	22	80.977 (9.908)	33	86.632 (13.852)
4	19	78.156 (6.345)	21	89.219 (14.297)
5	4	77.410 (1.973)	30	88.815 (13.788)
6	-	-	38	85.307 (10.966)
7	-	-	15	87.448 (12.378)

table 3-4: Mean heart rates and standard deviation at every MISC interval for the non-sensitive and the sensitive group.

Subsequent 1-way ANOVA analysis show that there was no difference between the means for the sensitive group (F = 1.44, p = 0.19) and that there was a significant difference between the mean heart rate at different ratings for the non-sensitive group (F = 5.14, p < 0.0001). A Bonferroni post-hoc multiple comparisons test has been carried out to see which of the MISC ratings had a significant difference when compared to each other. The post-hoc analysis (table 3-5) show that the initial decline and rise of HR, as seen in figure 3-8, do indeed differ significantly (Between MISC rating 0 and 1; MISC 0 and 2; MISC 2 and 3 (p < 0.005)).

MISC	0	1	2	3	4	5	n	
0	-	0.002*	0.002*	1	1	1	189	
1	-	-	1	0.129	1	1	134	
2	-	-	-	0.0498*	1	1	64	
3	-	-	-	-	1	1	22	
4	-	-	-	-	-	1	19	
5	_	_	_	_	_	_	1	_

The boxplots for both groups are shown in figure 3-7a and b.

table 3-5: Post-hoc analysis with Bonferroni correction of the average heart rate at every MISC rating for the non-sensitive group. P-values for each pair-wise combinations are shown together with n-size.



figure 3-9: Boxplots showing the distribution of the heart rates at every MISC rating interval for the non-sensitive (a) and sensitive group (b)



3. Mean heart rate at every MISC interval of the non-sensitive versus the sensitive group

In the same way, instead of grouping the average heart rate by MISC ratings, the average heart rate is grouped for every 60 seconds and every trial (figure 3-11). The high fluctuations of the mean heart rate for the sensitive group after 15 minutes are because not many participants could endure this long, so the number of data from that specific time and onwards are very limited. The number of participants that were still partaken the experiment at every minute is given in figure 3-12. Combining the two graphs, the sharp dip at 19 minute mark is due to already small numbers of participants (n = 6) going to just one last sensitive participant that could endure this long. The average time until MISC 7 was approximately 12.5 minutes.



figure 3-11: Average heart rate of all participants at every minute interval grouped into all, sensitive and non-sensitive category.



figure 3-12:Total number of sensitive participants at every minute before reaching MISC 7.

4. Linear fit over the HR per trial

After the ECG data has been processed, the first thing that was noticed after plotting all the data (two trials are given as examples in (figure 3-13 & figure 3-14), the other can be found in Appendix D) is that the non-sensitive group showed a relatively stable and minor decrease of heart rates over time, while the sensitive group had more differences between the sensitive participants. The figure 3-15 and figure 3-16 show all the trials in a single plot. However, unlike in the study of (Holmes & Griffin, 2001) and as seen in previous results, the heart rate did not increase but decreased.



Two examples of Heart rate and MISC over time for sensitive (a) and non-sensitive trial (b). Dotted line represents the linear fit over the HR.



figure 3-15: All non-sensitive trials depicted with corresponding MISC



figure 3-16: All sensitive trials depicted with corresponding MISC

By combining all the linear fitted lines of all the trials, the plot in figure 3-18 is obtained. It shows all the fitted lines of every sensitive (transparent red) and non-sensitive (transparent blue) trials, but also the averaged fit (opaque lines) for both groups. The heart rate at the baseline (1 minute before start), start, first peak and the duration are accounted for and averaged out. First, it can be noticed that the overall heart rate of the non-sensitive groups are lower compared to the sensitive group. The mean heart rate at the start of the trial starts at 76 bpm for the non-sensitive group and 82 bpm for the sensitive group respectively. This increases to 82 bpm and 90 bpm and decreases linearly. The way that the fitting algorithm is set up, it (over-)emphasizes the first peak. This causes the distinctive peak at the beginning of the trial. The coefficients of the averaged linear line is -0.0046 for the non-sensitive group versus -0.0076 for the sensitive group. According to the calculated coefficients, the heart rate also starts higher. The variance of the sensitive group is however larger.

When the linear fit is calculated like in section 3.2.1.1, i.e. averaging all the HR within the 4 intervals, the obtained results from the fitted lines show that it approximates the average HR calculated from the original HR data. The means and the standard deviation of the fitted HR and the previous results is given in table 3-6 as well as figure 3-17 with the yellow boxes.

Moreover, in figure 3-19, the average fitted line is plotted together with figure 3-11 for clarity and comparison. Here, it can be seen that the fitted lines follow the mean heart rates closely, even though the mean heart rates are calculated every minute compared to the 4 data points that is used to calculate the linear fit line (at baseline, start, first peak, end).

	Pretest	Begin	Mid	End	F	р
Non sensitive	75.776 (5.646)	83.614 (7.630)	78.051 (9.957)	74.584 (8.365)	3.47	0.0226*
Non sensitive Fit	75.754 (5.532)	80.792 (7.816)	78.095 (8.453)	74.259 (9.154)	1.73	0.1731
Sensitive	80.719 (10.631)	87.057 (10.808)	87.057 (10.808)	86.232 (10.72)	1.87	0.1444
Sensitive Fit	81.262 (9.599)	88.179 (10.339)	86.926 (10.595)	84.702 (11.22)	1.35	0.268

table 3-6: Mean heart rates and standard deviations for the sensitive and non-sensitive group. Includes the Fitted HR function.



figure 3-17: Boxplot of the mean HR at different time points for the sensitive and the non-sensitive groups. The yellow boxes are calculated with the fitted HR line. Pre = prestart or baseline, NS = non-sensitive group (blue), S = sensitive group (red).



figure 3-18: Resulting plot of linearly fitted heart rates for every individual trial. Transparent red line indicates sensitive trial and blue line a non-sensitive one. The thicker, opaque lines represent the averaged line per group.



figure 3-19: Average linearly fitted line plot together with mean heart rates per minute for both groups.

3.2.2 Heart rate variability

1. Mean LF/HF ratio at different time points of the non-sensitive and sensitive group.

By processing the acquired data, the HRV, in particular the LF/HF power can be analyzed. This tells whether the autonomic balance or regulations in the body change over time and/or increasing MISC ratings. With a repeated measures 1-way ANOVA, the LF/HF ratio is analyzed. It indicates that, time has a significant effect on the LF/HF ratio (F = 7.3708, p<0.001), whereas the interaction between the sensitive and non-sensitive groups do not have a significant effect (F = 0.996, p>0.05)



figure 3-20: The mean LF/HF ratio at four timepoints (baseline, begin, mid, end) for both groups.



figure 3-21: Boxplots showing the mean LF/HF ratio of the two groups individually (a)(b) figure 3-22: (b)

Boxplots of the two groups are shown individually with its mean and standard deviations in figure 3-21, figure 3-22 and table 3-7.

LF/HF []	Baseline	Begin	Mid	End	р
Non-sensitive	4.0 (1.4)	3.3 (1.2)	3.3 (1.2)	3.4 (1.5)	0.334
Sensitive	4.2 (1.5)	2.6 (1.4)	2.8 (1.4)	2.5 (1.5)	0.013*

table 3-7: Mean and standard deviation of every time point for the two groups.

With the 1-way ANOVA, there was a significant difference among the means of different time point in the sensitive group (F = 3.91, p < 0.05) and none for the non-sensitive group (p = 0.334). Post-hoc test showed that in the sensitive group table 3-8 there was a significant difference in the mean LF/HF ratio between the Baseline and beginning phase (p = 0.034) and between the baseline and end phase of the trial (p = 0.021). It appears that there is a distinct decrease in LF/HF ratio, or sympathetic/parasympathetic balance, in the sensitive group. Next subsection, will show the LF and HF separately.

			Mean (SD)	Mean (SD)	р
Baseline	-	Begin	4.2 (1.5)	2.6 (1.4)	0.034*
Baseline	-	Mid	4.2 (1.5)	2.8 (1.4)	0.089
Baseline	-	End	4.2 (1.5)	2.5 (1.5)	0.021*
Begin	-	Mid	2.6 (1.4)	2.8 (1.4)	1
Begin	-	End	2.6 (1.4)	2.5 (1.5)	1
Mid	-	End	2.8 (1.4)	2.5 (1.5)	1

table 3-8: Post-hoc analysis with Bonferroni correction for the means at different time points in the sensitive group.

2. Mean LF power of the non-sensitive group versus the sensitive group.

Similarly to the LF/HF ratio, the LF and HF power is plotted separately for the two groups and compared with an ANOVA test.

Repeated measures 1-way ANOVA showed that there is a significant effect of time on LF ratio (p < 0.001) but no significant group and time interaction (p = 0.104).



figure 3-23: The mean LF ratio at four time points (baseline, begin, mid, end) for both groups.

Comparing the mean LF power individually with a 1-way ANOVA, the sensitive group showed no significant differences between each other (p = 0.139), but the sensitive group does (p = 0.024). A multiple comparisons test with a Bonferroni correction showed that none of the means were significantly different. The most noticeable detail in this plot is that for the non-sensitive group, the mean LF power at the baseline and at the end are similar even its standard deviation.

LF [ms^2]	Baseline	Begin	Mid	End	р
Non-sensitive	0.0032 (0.002)	0.0017 (6.5e-4)	0.0017 (0.001)	0.0028 (0.0021)	0.024*
Sensitive	0.0019 (0.002)	0.0009 (5.4e-4)	0.0013 (0.001)	0.0014 (0.0019)	0.139

table 3-9: Mean and standard deviation of LF power at every time point for the two groups.

3. Mean HF power of the non-sensitive group versus the sensitive group.

As for the mean HF power the follow results are obtained; Repeated measures 1 way ANOVA did not indicate effect for time and group interaction with time (p = 0.109, p = 0.292). The 1-way ANOVA between the means of each group individually also did not show any significant differences between the HF means at different time points.



figure 3-24: The mean HF power at four time points (baseline, begin, mid, end) for both groups.

HF [ms^2]	Baseline	Begin	Mid	End	р
Non-sensitive	0.0011	0.0010	8.1084e-04	0.0013	0.544
	(7.41e-04)	(0.0012)	(7.05e-04)	(0.0011)	
Sensitive	6.6850e-04	5.7459e-04	8.7872e-04	9.6829e-04	0.541
	(4.73e-04)	(4.48e-04)	(0.0010)	(0.0012)	

table 3-10: Mean and standard deviation of HF power at every time point for the two groups.

3.3 GSR

The GSR analysis was done over the data of 29 trials in total and 8 were discarded after the data was processed in Ledalab. The data was visually inspected and were rejected when; the amplitudes of data were not in the usual range of 5-20 μ S, the signal was capped or had abnormal patterns. From the 29 trials, 15 participants became sick (MISC 7) and 14 did not. Similar to the ECG analysis, the

From the 29 trials, 15 participants became sick (MISC 7) and 14 did not. Similar to the ECG analysis, the participants were grouped into sensitive or non-sensitive category.

1. Mean normalized skin conductance level (tonic) of the non-sensitive vs sensitive group

The mean SCL data (begin, mid and end) has been normalized with respect to the baseline per trial and averaged over its corresponding group, then tested with a repeated measures 1 way ANOVA model. Again, it only showed that time has a significant effect on the SCL but not the interaction between condition and time (p = 0.143). There was also no difference between the means at beginning, middle and end phase between each group, after running a 1 way ANOVA.

What can be noticed is that the non-sensitive group have an increasing mean and standard deviation of the SCL ratio as the trial progresses whereas the sensitive group displayed similar levels of SCL ratios.



figure 3-25: The mean (normalized) SCL ratio at four time points (baseline, begin, mid, end) for both groups.

SCL ratio	Baseline	Begin	Mid	End
Non sensitive	1 (0)	1.48 (0.76)	1.85 (1.11)	2.18 (1.68)
Sensitive	1 (0)	1.34 (0.31)	1.38 (0.45)	1.42 (0.45)

table 3-11: Mean and standard deviation of the normalized SCL ratio at every time point for the two groups.



figure 3-26: Average normalized SCL per trial plotted over time points. Bold blue line represents the mean SCL of all the nonsensitive trials



figure 3-27: Average normalized SCL per trial plotted over time points. Bold red line represents the mean SCL of all the sensitive trials

2. Mean skin conductance response (phasic) of the non-sensitive vs sensitive group

The SCRs are normalized first before it is being averaged over the trials for every time point. The normalization is done by subtracting the number of peaks of the beginning, middle and end phase by its baseline number of peaks. This is done per trial, and the average is calculated from the resulting number of peaks at each time point for the whole group. Afterwards, repeated measures 1 way ANOVA is performed and no significant effect has been found between time and the groups (p = 0.740). Furthermore, 1 way ANOVA over each group separately also did not show that the average number of peaks significantly differed between time points.



figure 3-28: The mean (normalized) SCRs at four time points (baseline, begin, mid, end) for both groups.

peaks	Baseline	Begin	Mid	End
Non-sensitive	0	12.500 (23.484)	10.143 (20.069)	12.786 (31.093)
Sensitive	0	13.267 (11.119)	13.667 (13.652)	12.400 (13.479)

table 3-12: Mean and standard deviation of the normalized SCRs at every time point for the two groups.

Looking into table 3-12 and comparing it to figure 3-28, it is strange to see that the overall standard deviation for the non-sensitive group is almost double of the sensitive group, despite boxplot showing an overall larger variance for the sensitive group. Having the data plot out alternatively in chronological and continuous manner (figure 3-29 and figure 3-30), it reveals that there is an outlier in the non-sensitive group that causes the large increase in the standard deviation. When the outlier is removed, the following table 3-13 and figure 3-31 is obtained. Now, the standard deviations of the non-sensitive group are generally smaller than the sensitive group. Furthermore, removing the outlier did not change the insignificant results of the previous ANOVA analysis.



figure 3-29: Average SCR peaks plotted out chronologically per trial for the non-sensitive group. One outlier can be distinguished in this plot. The average mean excluding the outlier is shown as bold blue line.



figure 3-30: Average SCR peaks plotted out chronologically per trial for the sensitive group. Bold red line represents the average of all the trials at given time point.

peaks	Baseline	Begin	Mid	End
Non-sensitive*	0	6.6154 (8.500)	5.231 (8.388)	4.8462 (9.556)
Sensitive	0	13.267 (11.119)	13.667 (13.652)	12.400 (13.479)

table 3-13: :Corrected mean and standard deviation of the normalized SCRs for the non-sensitive group after removing one outlier



figure 3-31: The corrected mean (normalized) SCRs at four time points (baseline, begin, mid, end) for both groups without outlier.

4. Discussion

In this study, physiological measurements ECG and GSR acquired from a road test are analyzed to investigate whether they relate to or predict motion sickness, indicated by the MISC rating of the participants. Looking at the MISC ratings over time, there was a large difference between the sensitive and non-sensitive groups. The average time of the sensitive group reaching MISC 7 was only 12.5 minutes, compared to the 30 minutes, and this was mostly for the trials with eyes-off-road. With this difference between the groups, a large difference in the assessed ECG and GSR metrics was hoped to be seen.

This chapter will discuss each physiological measurement individually and combination thereof with the obtained results. As few results have been found with p < 0.05, hence several effects with p > 0.05 will be reported and discussed here as well. However, note that these effects need to be verified with a larger sample size.

Heart rate

The results show similar general characteristics over time for both groups. In figure 3-6, figure 3-11 and figure 3-19, the average HR for both groups rises initially, reaches a maximum in the first few minutes and declines over the rest of the experiment duration. This common trend for both groups can be explained by the initial anxiety upon the start of the experiment causing the HR to rise, but also by the initial exposure to stimuli. After the initial HR spike, it decreases for both groups, albeit on average in different rates, which can be explained with acclimatization of the stimuli exposure.

Secondly, the results show that on average that the heart rate of the sensitive groups on the first two minutes is 3.44 bpm more than the non-sensitive group (table 3-1: mean begin S – mean NS = 87.057 - 83.614). This difference increases to 11.65 bpm at the end. It shows an even larger change when the fitted function is compared with 8 bpm at the start and 11 bpm at the end. While there was no significant difference found between the sensitive and non-sensitive group for the 4 intervals (p = 0.073), it did show similar trend as the fitted function. Because the fitted function takes into account the (first) single peak P2 and fits all data, it is potentially more robust. This can also be seen in table 3-6, where the means and standard deviations are similar to the values at 4 intervals. However, in the fitted HR line, no significant differences have been found for both groups (non-sensitive fit (p = 0.1731), sensitive fit (p = 0.268). Both original and fitted HR line, will benefit from a longer baseline HR recording, and testing more participants.

When the HR is studied from a different perspective with mean HR over MISC (figure 3-8), the HR does increase as MISC ratings go up. Especially for MISC from 2 to 3, where a significant difference (p = 0.0498) between the HR within the non-sensitive group was found. The sensitive group showed an increase in HR for MISC 0-4, but were not found to be significant (p = 0.19).

The baseline HR of the sensitive and non-sensitive group were different before the start of the test. This could be attributed to anxiety that both groups had, but it has to be mentioned that most of the sensitive trials were also for eyes-off-road condition, so with their vision blocked. This experience will lead to even more anxiety and that could translate in higher HR and also a change in LF/HF ratio before or at the start of the experiment. After the initial minutes, the decline in HR can be explained with the acclimatization to the environment and or exposure.

Observing the individual HR plots, while the general average, as seen in figure 3-18, decreases over time, there were instances where participants' HR did not decrease but rather stayed constant or even increased slightly (Appendix D, trials 9 V, 9 NV, 11 NV, 15 NV, 20 NV). It shows that for HR the individual differences are large between participants and just by judging (average) HR as is, it is difficult to discern upcoming MS related symptoms. More factors, like categorizing participants to different levels of MS susceptibility or MISC profiles might help making HR predictions more useful.

Moreover, the participants had to stabilize themselves as much as possible and to keep their head straight ahead, as the experimenters instructed them to do so, while the car was aggressively slaloming. This required muscle activation and will lead to an elevated heart rate for the whole trial,

especially for participants during eyes-off-road trials, who had to work harder to stabilize themselves with less visual information, thus less anticipatory information. Although this might seem like an experimental problem, HR increase by muscle activation and less anticipatory information will certainly play a role in self-driving vehicles, regardless of MS developing and therefore has to be taken into account.

Results of this test do not reflect results from previous studies, such as in (Holmes & Griffin, 2001) (Dennison, Wisti, & D'Zmura, 2016) (Himi, et al., 2004). Here the authors saw a significant increase in HR for the sensitive group as MS increased in time in optokinetic drum induced MS experiment, with VR experiments and oscillating video respectively. In the first study, the breathing pattern was controlled for (15 breaths per minute) and participants had to follow it. This may have led to artificially lower or higher the actual HR and or sickness, than if the participant would be free to breathe naturally. It has been shown that controlled breathing can increase motion tolerance against motion sickness, activating the inhibitory reflex between respiration and vomiting (Golding J. F., 2016). In other words, it is possible that in our study, participants have un- or consciously controlled their breathing to alleviate or suppress motion sickness, therefore causing the HR to decrease over time. It could be argued that without controlled breathing, the same might happen to the study of (Holmes & Griffin, 2001), where the HR would decrease as symptoms are developing. Besides, it was also not clear how (much) the optokinetic drum was creating visual motion sickness. There was no information on the speed, frequency nor acceleration of the vision, making it difficult to compare the optokinetic drum (or other visually induced) experiment to the realistic driving experiment.

In (Himi, et al., 2004), they showed that on average, the nauseous group had a lower HR throughout the experiment compared to the non-sensitive group and that the HR of the sensitive group increased during the exposure. However, this study used oscillating video to impart MS for only 6 minutes with 17 participants and 11 felt nauseous afterwards. Here, the initial rise of HR for the sensitive group can be as well contributed to the initial HR spike found in figure 3-19. Because of the short exposure period, it could be possible that the HR would have decreased as the experiment went on after the 6 minutes. The averaging of heart rates during the exposure time (2 mean heart rates for first 3 minutes and last 3 minutes) do not give a clear picture and actually could lead to same results as this experiment if the exposure time was longer and also until participants reached similar levels as MISC 7.

While for example in studies of (Dahlman, Sjörs, Lindström, & Ledin, 2009) (Mullen, Berger, Oman, & Cohen, 1998), it showed that the HR remained relatively the same and no significant effects found. In (Dahlman, Sjörs, Lindström, & Ledin, 2009), the results also show that the HR increased at the start and argued that it is caused by the expectancies of possible discomfort. Also, their non-sensitive group experienced a rapid decline in HR after the initial increase, which was explained by the decrease of general arousal levels after the start. This observation was similar to our results.

The literature has not shown a clear consensus on how and why heart rate changes during motion sickness. As to relating HR to MS in vehicles, even less is known, because MS has been researched mostly with optokinetic drum setups, VR, or other (rotating, visual) methods except road tests.

Heart rate variability

When looking at the HRV results, the first thing that can be noticed is the significant difference between the means of the baseline – begin and baseline – end LF/HF ratio for the sensitive group (p = 0.013) (figure 3-20) (table 3-7), whereas for the non-sensitive group there was no significant difference between the means (p = 0.334). The LF/HF ratio's decrease as the trial (and MISC) progresses. This implies that there is a decrease in sympathetic tone, a shift in the autonomic balance in the body towards the parasympathetic nervous system. Theory behind the HRV explains that as nausea develops (and HR increases), the sympathetic tone will increase and the parasympathetic will decrease. The result of decreasing LF/HF power for increasing MISC ratings do not correspond to literature (Lin, Lin, & Chiu, 2011) (Holmes & Griffin, 2001) (Himi, et al., 2004) (Zuzewicz & Saulewicz, 2011) (Doweck, Gordon, & Shlitner, 1997) (Sjors, Dahlman, Ledin, Gerdie, & Falkmer, 2004) and the opposite is observed in our experiment.

Moreover, the LF/HF ratio for non-sensitive group decreased as well, albeit less and non-significant (p = 0.334). Since HR and HRV are inherently associated with each other, with the current accepted (but debated and controversial) theory, it means that the sympathetic activities became low toned, thus among the SNS related activities, i.e. blood flow and HR decreased.

Looking at the LF and HF HRV individually, it appears that there was no significant change in the HF frequency power for both groups (p = 0.54), but for non-sensitive LF (p = 0.024) there were some interesting points to be noticed; the baseline LF power and end interval of the non-sensitive group was a lot higher than the sensitive group. Following the theory of HRV, that means that the non-sensitive group experienced relatively strong sympathetic activities that can be due to anxiety for the start of the experiment and near the end. This should also happen for the sensitive groups, but is not observed. Comparing our driving test to other visual, virtual, optokinetic or rotating chair tests and its result, it might be that the real test requires more work physically (stabilizing) and mentally by the human body. It is speculated that non-sensitive group had to endure for 30 minutes, compared to average of 13 minutes of sensitive group, leading to higher stress or fatigue without sickness near the end of the test. Furthermore, it is known that the HRV (and HR) changes according to ones breathing pattern. The LF band is affected by breathing from \sim 3 – 9 breaths per min, while the HF band (also known as the respiratory band) is affected by breathing from ~9 – 24 bpm (Schaffer & Ginsberg, 2017) (Quintana & Heathers, 2014). Participants can consciously or unconsciously change or control their breathing pattern to alleviate the arising motion sickness symptoms as it is shown in (Lin, Lin, & Chiu, 2011) (Sang & Billar, 2005) (Golding & Gresty, 2005). Following this theory, the increase in LF power and the relative constant LF/HF power, for the non-sensitive group implies that they were calm and had a slower breathing pattern for the trial. Whereas the sensitive group had a more noticeable change in their (faster) breathing pattern from the decreasing LF/HF ratio over time. Moreover, it was sunny during the experiment days and the temperatures reached to 25 degrees Celsius on average. While the car had the air conditioner and blowers on the same (high) setting to keep the temperature the same in the car for all the trials, the cardboard that was used for the eyes-off-road condition (figure 2-6) did block some of the cool airflow from the vents. With the motion capture suit on as well, the participants will feel hot and sweat more (in addition to the MS symptoms). This could lead to faster breathing for thermoregulation and a change in the LF/HF ratio, in particular an increase in HF or decrease in LF power, for the sensitive group. Experimenters did not mention or instruct the participants to breath in a certain way. Also, because it was not instructed, it is assumed that participants breathed naturally, i.e. how they would also do in other transportation means. So one way to control for this is to compare participants experience and susceptibility from the MSSQ and additional questions regarding breathing, or to record breathing cycles in future road test experiments.

GSR

The captured GSR signal provided two different datasets to analyze, the slower SCL and the fast responding SCR. The normalized SCL did not show any significant effects between the sensitive and non-sensitive groups (p = 0.143), but the mean SCL ratio did show, although not significantly, an increasing trend over the course of the experiment for the non-sensitive groups, but as for SCR peaks, the same group saw a relative constant number of mean peaks. It suggests that, while the SCR caused by events stayed similar, the perspiration and thus the sympathetic activity (Braithwaite, Watson, Jones, & Rowe, 2015) increased over time. Comparing this with the LF power HRV results, that corresponds to sympathetic nervous system activity according to literature, it does increase at the end of the trial. The high baseline LF power and the low power at the beginning however, cannot be found in the SCL. A possible explanation of this SCL is that the increase of SCL is caused by thermoregulation and less, if at all, by nausea, as this is only seen for the non-sensitive group. Besides, it is interesting to see that the overall mean SCL and the variance for the non-sensitive group is larger than the sensitive group (table 3-11). This is counter-intuitive, as it is expected that the sensitive group would become sick, possibly develop cold sweat and lead to increase in sympathetic tone (either increase in LF, decrease in HF, increase in LF/HF).

Going to the SCR, the opposite is noticed. Here, the sensitive group have higher number of peaks compared to the non-sensitive one, but the variance is also larger. Similarly, the results do not show a significant effect between the two groups (p = 0.740), unlike the (strong) correlations posed in multiple studies mentioned (Himi, et al., 2004) (Wan & Hu, 2003) (Dennison, Wisti, & D'Zmura, 2016). In figure 3-27, the variance is noticeable over time. Almost half of the trial exhibit similar pattern as nonsensitive participants in figure 3-26. It can be theorized that indeed some sensitive participants respond more to the same events (e.g. slalom movement, heat, symptoms developing) compared to other sensitive and non-sensitive participants. This result suggests again that it might be useful to classify susceptibility of sensitive participants even further, to get a better view and predictability with GSR measurements or even ECG and other physiological measurements because of the highly individual responses. Second argument for the distinct difference within the sensitive group is that the aggressive car motion combined with the lack of vision causes more body sway, thus increased the likelihood that their hand with the electrodes attached, moved more as well. The extra hand and finger motion would cause extra activity, if not artefacts, within the GSR signal. The notion that participants get worse at stabilizing their body because of MS or MS leaded to postural instability that could cause additional hand movements, was found to be non-existent in Yunyi Li's study (Li, 2018). In the end, SCR did reflect the increase in MISC with the average number of peaks positively for the sensitive group.

The experiment and the physiological measurements

The aim of this study is to investigate the relationship of HR, HRV and GSR with MISC in a realistic driving experiment. So far, the results have shown weak evidence and or even contradictory relationships between physiological measurements and MISC with previous studies. The literature on these measurements on MS are strict laboratory experiments, focusing on certain stimuli and did not evaluate for motion sickness in cars specifically. Even between studies, there are many inconsistencies to be found regarding the use and the explanations thereof, in particular related to HRV. It is known that the CNS regulate many other physiological mechanisms besides MS in the body as well that may change the HRV and HR, so capturing MS symptoms as is, might not be accurate (at all). The study also recorded MSAQ and the MSSQ of the participants. With these additional data, sensitive and non-sensitive people can be classified further into different levels of sensitivity or susceptibility. Combination of (head) motion and physiological measures can be done as well, to create a better and detailed image how MS manifests and correlates.

Some of the contradictory results could also be explained by the experiment itself. There was no previous study that did similar measurements in a road test. This exploration gives a good basis on how to investigate these in future studies (moreover in chapter 6). Examples that could have influenced results, is whether or not participants have used certain strategies to minimize susceptibility or alleviate symptoms. Did participants change their breathing pattern? Did they stabilize their body or head in a certain way? Or was the temperature perhaps too high with the (tight) motion capture suit on? During eyes-off-road condition, the cardboard blocked the participants view but also the experimenters view towards the participant. It was only possible to verbally communicate with the participant. Experimenters could not check whether the participants followed the instructions (e.g. looking straight ahead, eyes open, keep torso stable, legs well plant on the floor) well during the trial. Besides, as mentioned, the electrodes on the finger (and chest) were prone to the movement. The attached cables (6) and a tight suit might also hinder the natural sitting posture and behavior in the vehicle, leading to minor discomfort.

Furthermore, it can be observed from the results that the baseline levels of sensitive and non-sensitive groups were already different from the start. Since the experiment also included an eyes-on-road and an eyes-off-road condition, where most of the participants got sick, the drive between the RADD control room and the testing grounds with a blocked vision might already cause the participants to get more anxious. Even without the slaloming, having no visual anticipatory information, participants were exposed to nausogenic environment without feeling discomfort yet. A possible way to rule the baseline difference out is to have a control group or condition, where the car is only going straight at the same

speed instead of slaloming. The effect of u-turns at the end could be measured better as well. As the u-turn was done as tight as possible (braking and turning at the same time, reversing and accelerating), it felt harsh and jerky.

5. Conclusion

The research objective of this study was to investigate and explore the usage and validity of the physiological measurements; HR, HRV and GSR, and to see if it could relate to or even be a predictor of motion sickness given as MISC ratings by participants in a slalom driving test.

The following research questions were composed to investigate the individual physiological signals one by one and answered below.

1. Is there a correlation between heart rate and the MISC?

Results show that only near significant effect has been found between the HR of the sensitive and the non-sensitive groups (p = 0.073). It showed that there is, a quick initial rise followed by a gradual negative trend in time of the HR as the experiment for both groups, with the sensitive group starting with a higher average HR and a steeper negative trend. With the weak findings from this experiment and combining the fact that in literature mixed results have been achieved with HR detecting MS, the relation between HR and MISC is low at best.

2. Is there a correlation between the HRV, expressed in LF/HF, and the MISC? The LF/HF ratio for the sensitive group decreased significantly between time points (p = 0.013), which is opposite from what is expected from theory. Also, recent literature question the usability of HRV in general. So, although some studies show certain results, predicting MS with HRV seem to be based on conjecture.

3. Is there a correlation between the skin conductance and the MISC? Both SCL and SCR did not show significant differences between the sensitive and non-sensitive groups. Based upon the observation, no correlation has been found between SCL and SCR versus MISC in this experiment.

The results show little support relating motion sickness or even to distinguish sensitive and nonsensitive groups with either HR, HRV or GSR data. As for HRV, it might be needed to re-evaluate the usage of this metric in conjunction with MS. Further investigations are needed on the physiological aspects of said measurements and perhaps including other metrics in the analysis would give more clarity to detect motion sickness.

However, the results show the importance of actual road tests and experiment setup. Currently, there were only few road tests done regarding motion sickness. It appears that physiological measurements for predicting MS in vehicles are not as straightforward and do not translate well from other types of laboratory tests to realistic road tests. It highlighted that still a lot is uncertain or unknown on the field of experimental MS in cars and the measurement thereof in the real world, despite the available literature on this topic. As self-driving cars are being pushed onto the market in the upcoming years, MS might become a limiting factor to the passengers' comfort and therefore more research is needed.
6. Recommendations

The experiment showed that measuring MS with physiological markers was a difficult process. Not only finding correlations, but capturing a lot different data sources in a vehicle with aggressive motions for 30 minutes and processing it, proved to be a challenge.

Here are some points of attention and recommendations for future research on the field of physiological measurements and MS measurements in cars:

The experiment was performed together with Yunyi Li (Li, 2018). Besides the ECG, GSR and the MISC data, also motion data of the vehicle (IMU) and the passenger with the Xsens motion capture suit was captured at the same time on a single laptop. From the hardware point of view, cable management becomes important as it could pull and disconnect, create artefacts and noise in the sensitive measurements of ECG/GSR.

The GSR used in this study was sensitive to skin resistance (dry/wet) and required a lot of tweaking to get the signal within range. This process was not to be expected from the hardware and was time consuming.

The middle seat in the back was concave shaped, causing extra instability and movement to the participant during the ride. Adding the cardboard to block the view of the participant, also prevented the experimenters to see and control the participants' posture and or see their eyes (to keep looking forward).

Also, the motion capture suit was a tight fit and could create an extra layer of discomfort for the participant, particularly introducing extra sweating that may influences the GSR results. Future tests would benefit from a better streamlined data acquisition to create a natural environment for the participant as much as possible and to prevent loss of data.

While this study focused on ECG and GSR measurements, other data have been measured as well, including body motion, car motion, MSAQ, MSSQ. Further analysis can be performed to include other metrics and see what might reveal more about MS. Instead of focusing on correlating MS directly with ECG or GSR, it might be useful to classify participants in different sensitivity categories with their MISC profiles, MSAQ and MSSQ first and analyze it from there.

A larger sample size and adding a control trial without slaloming would have been a great addition to the results, but due to time and other constraints this has not been done and is also recommended for future studies. At first, autonomous driving mode on the Prius was planned for this study. The track to be driven could then be well controlled. Due to technical reasons, this was not possible and the car had to be driven manually. Even so, the driver did an excellent job following the planned path and target motion frequencies have been achieved.

The next step is to expand the slalom test with different velocities and turns or even a different track to explore sickening frequencies in a realistic setting.

References

- Benedek, M., & Kaernbach, C. (2010). A continuous measure of phasic electrodermal activity. *Journal* of NeuroscienceMethods, 80-91.
- Benedek, M., & Kaernbach, C. (2010). Decomposition of skin conductance data by means of nonnegative deconvolution. *Psychophysiology*, 647-658.
- Billman, G. E. (2013). The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. *frontiers in physiology*.
- Bles, W., Bos, J. E., de Graaf, B., Groen, E., & Wertheim, A. (1998). Motion sickness: Only one provocative conflict? *Brain Research Bulletin, Vol.* 47(No. 5), 481–487.
- Bos, J. E., & Bles, W. (1998). Modelling motion sickness and subjective vertical mismatch detailed for vertical motions. *Brain Research Bulletin*, 537–542.
- Bos, J. E., Bles, W., & Groen, E. L. (2008). A theory on visually induced motion sickness. *Displays, 29*, 47-57.
- Bos, J. E., MacKinnon, S. N., & Patterson, A. (2005). Motion sickness symptoms in a ship motion simulator: effects of inside, outside, and no view. *Aviation, space, and environmental medicine*, 1111-1118.
- Boucsein, W. (2012). *Electrodermal Activity*. Wuppertal: Springer.
- Braithwaite, J. J., Watson, D. G., Jones, R., & Rowe, M. (2015). A Guide for Analysing Electrodermal Activity (EDA) & Skin Conductance Responses (SCRs) for Psychological Experiments. Birmingham: Selective Attention & Awareness Laboratory (SAAL) Behavioural Brain Sciences Centre, University of Birmingham.
- British Standards Institution. (1987). *Guide to measurement and evaluation of human exposure to whole-body mechanical vibration and repeated shock.* London: United Kingdom: British Standards Institution.
- Dahlman, J., Sjörs, A., Lindström, J., & Ledin, T. (2009). Performance and Autonomic Responses During Motion Sickness. *Human Factors*, 56-66.
- Dawson, M. E., Schell, A. M., & Filion, D. L. (2007). The electrodermal system. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson, *The handbook of psychophysiology* (pp. 159-181). New York: Cambridge University Press.
- Dennison, M. S., Wisti, A. Z., & D'Zmura, M. (2016). Use of physiological signals to predict cybersickness. *Displays* 44, 42-52.
- Diels, C., & Bos, J. E. (2015). Self-driving carsickness. *Applied Ergonomics*, 1-9.
- Donohew, B. E., & Griffin, M. J. (2004). Motion Sickness: Effect of the Frequency of Lateral Oscillation. *Aviation, Space, and Environmental Medicine*.
- Doweck, I., Gordon, C. R., & Shlitner, A. (1997). Alterations in R-R variability associated with experimental motion sickness. *Journal of the Autonomic Nervous System*, 31-37.
- Farmer, A. D., Ban, V. F., Coen, S. J., Sanger, G. J., Barker, G. J., Gresty, M. A., & al, e. (2015). Visually induced nausea causes characteristic changes in cerebral, autonomic and endocrine function in humans. *Journal of physiology*, 1183-1196.
- Farmer, A. D., Omran, Y., & Aziz, Q. (2014). The role of the parasympathetic nervous system in visually induced motion sickness: sytematic review and meta-analysis. *Exp Brain Res*, 2665-2673.
- Festner, M. (2016). Der Enfluss fahrremder Tatigkeit und Manoverlangsdynamik auf die Komfort- und Sicherheitswahrnemung beim hochautomatisierten Fahren. *VDI Wissensforum.* Munchen.
- Gavgani, A. M., Nesbitt, K. V., Blackmore, K. L., & Nalivaiko, E. (2017). Profiling subjective symptoms and autonomic changes associated with cybersickness. *Autonomic neuroscience: Basic and clinical*, 41-50.
- Gianaros, P. J., Muth, E. R., Mordkoff, J. T., Levine, M. E., & Stern, R. M. (2001). A questionnaire for the assessment of the multiple dimensions of motion sickness. *Aviation, space, and environmental medicine*, 115.
- Golding, J. F. (1998). Motion sickness susceptibility questionnaire and its relationship to other forms of sickness. *Brain Research Bulletin*, 507-516.

Golding, J. F. (2006). Motion sickness susceptibility. *Autonomic-Neuroscience*.

- Golding, J. F. (2006). Predicting individual differences in motion sickness susceptibility by questionnaire. *Personality and Individual Differences*, *41*, 237–248.
- Golding, J. F. (2016). Chapter 27: Motion sickness. In J. F. Golding, *Handbook of Clinical Neurology* (pp. 371-390). London: Elsevier.
- Golding, J. F., & Gresty, M. A. (2005). Motion Sickness. Current opinion in Neurology, 29-34.
- Golding, J., Markey, H., & Stott, J. (1995). The effects of motion direction, body axis, and posture on motion sickness induced by low frequency linear oscillation. *Aviation, Space and Environmental Medicine*, 1046-1051.
- Griffin. (1991). *M. J. Handbook of human vibration*. London: Academic Pres.
- Griffin, M. J., & Howarth, H. V. (2000). Motion sickness history questionnaire.
- Griffin, M. J., & Newman, M. M. (2004). Visual field effects on motion sickness in cars. . Aviation, space, and environmental medicine, 739-748.
- Grossman, P., & Taylor, E. (2007). Toward understanding respiratory sinus arrhythmia: Relations to cardiac vagal tone, evolution and biobehavorial functions. *Biological psychology*, 263-285.
- Happee, R. (1989). Gauss Newton, an iterative parameter estimation algorithm, used to evaluate data of human movements. *Benelux meeting on systems and control.* Brussels.
- Himi, N., Koga, T., Nakamura, E., Kobashi, M., Yamane, M., & Tsujioka, K. (2004). Differences in autonomic responses between subject with and without nausea while watching an irregularly oscillating video. Autonomic neuroscience: Basic and Clinical, 46-53.
- Holmes, S. R., & Griffin, M. J. (2001). Correlation Between Heart Rate and the Severity of Motion Sickness Caused by Optokinetic Stimulation. *Journal of psychophysiology*, 35-42.
- ISO. (1997). *ISO-2631-1 General-Mechanical vibration and shock Evaluation of human response to whole body vibration.* Geneve: ISO.
- Lacker, J. R. (2014). Motion sickness: more than nausea and vomiting.
- Lange, A., Maas, M., Albert, K.-H. S., & Bengler, K. (2013). Vestibulare Zustandsruckmeldung beim automatisierten Fahren. *VDI Wissensforum*.
- Li, Y. (2018). Relations between visual conditions, head motion and motion sickness in vehicle passengers.
- Lin, C.-T., Lin, C.-L., & Chiu, T.-W. (2011). Effect of respiratory modulation of relationship between heart rate variability and motion sickness. *33rd Annual International Conference of the IEEE EMBS*. Boston: IEEE .
- Marieb, E. N., & Hoehn, K. (2007). *Human Anatomy & Physiology*. Pearson Education.
- Mullen, T. J., Berger, R. D., Oman, C. M., & Cohen, R. J. (1998). Human heart rate variability relation is unchanged during motion sickness. *Journal of vestibular research*, 95-105.
- O'Hanlon, J. M. (1974). Motion sickness incidence as a function of the frequency and acceleration of vertical sinusoidal motion. *AeroSpace Med., 45,* 366-369.
- Ohyama, S., Nishiike, S., Watanabe, H., Matsuoka, K., Akizuki, H., Takeda, N., & Harada, T. (2007). Autonomic responses during motion sickness induced by virtual reality. *Auris Nasus Larynx*, 303-306.
- Oman, C. M. (1982). A heuristic mathematical model for the dynamics of sensory conflict and motion sickness. *Acta Otolaryngol, 392*.
- Osth, J., Eliasson, E., Happee, R., & Brolin, K. (2014). A method to model anticipatory postural control in driver. *Gait & Posture*, 664-669.
- Quintana, D. S., & Heathers, J. A. (2014). Considerations in the assessment of heart rate variability in biobehavorial research. *frontiers in psychology*.
- Reason, J. (1978). Motion sickness adaptation neural mismatch model. *Journal of the Royal Society* of Medicine, 71 (11), 819–829.
- Reason, J. T., & Brand, J. J. (1975). *Motion Sickness*. Acedemic Press.
- Reyes del Paso, G. A., Langewitz, W., Mulder, L. J., & van Roon, A. (2013). The utility of low frequency heart rate variability as an index of sympathetic cardiac tone: A review with emphasis on a reanalysis of previous studies. *Psychophysiology*, 477-487.

Sang, F. Y., & Billar, J. G. (2005). effect of a novel motion desensitization training regime and controlled breahting on habituation to motion sickness. *Perceptual and motor skills*, 244-256.

Sayers, B. M. (1973). Analysis of heart rate variability. *Ergonomics*, 17-32.

- Schaffer, F., & Ginsberg, J. (2017). An overview of heart rate variability metrics and norms. *Frontiers in public health*.
- Sjors, A., Dahlman, J., Ledin, T., Gerdie, B., & Falkmer, T. (2004). Effects of motion sickness on encoding and retrieval performance on psychophysiological responses. *Journal of ergonomics*.
- Stott, J. (1986). Mechanisms and treatment of motion illness. *Nausea and Vomiting, Mechanisms and treatment*, 110-129.
- TMSi. (2016). Mobita User Manual. 6.
- Treisman, M. (1977). Motion sickness: an evolutionary hypothesis. Science, 493-495.
- Turner. (1999). Motion sickness in public road transport: passenger behaviour and susceptibility. *Ergonomics*(42:3), 444-461.
- Turner, & Griffin, M. J. (1999). Motion sickness in public road transport: The effect of driver, route and vehicle. *Ergonomics*, 1646-1664.
- Ulherr, A., & Bengler, K. (2017). Seat Assessment A Discussion of Comfort and Discomfort Models and Evaluation Methods. *1st Comfort Congress*.
- Verver, M. M. (2004). Numerical tools for comfort analyses of automotive seating. *Technische Universiteit Eindhoven*.
- Wan, H., & Hu, S. (2003). Correlation of phasic and tonic skin-conductance responses with severity of motion sickness induced by viewing an optokinetic drum. *Perpetual and Motor Skills*, 1051-1057.
- Zuzewicz, K., & Saulewicz, A. (2011). Heart Rate Variability and Motion Sickness During Forklift Simulator Driving. *International journal of occupantional safety and ergonomics*, 403-410.

Appendix A: MOTION SICKNESS SUSCEPTIBILITY QUESTIONNAIRE

(Griffin & Howarth, 2000)

INSTRUCTIONS

This questionnaire is primarily concerned with: (i) your susceptibility to motion sickness, and (ii) what types of motion are most effective in causing this sickness.

Please read the questions carefully and answer them ALL by either TICKING or FILLING IN the boxes which most closely correspond to you as an individual.

All the information you give is CONFIDENTIAL and will be used for research purposed only.

Thank you very much for your co-operation.

NAME	AGE	
SEX	CURRENT OCCUPATION	
APPROXIMATE BODY WEIGHT	HEIGHT	
EMAIL		

1. In the past <u>YEAR</u>, how many times have you travelled AS A PASSENGER in the following types of transport?

	NEVER	1	2-3	4-15	16-63	64-255	256+
CARS							
BUSES							
COACHES							
SMALL BOATS							
SHIPS							
AEROPLANES							
TRAINS							

2. In the past <u>YEAR</u>, how many times have you felt ill, whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	1	2	3	4-7	8-15	16+
CARS							
BUSES							
COACHES							
SMALL BOATS							
SHIPS							
AEROPLANES							
TRAINS							

3. In the past <u>YEAR</u>, how many times have you VOMITTED whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	1	2	3	4-7	8-15	16+
CARS							
BUSES							
COACHES							
SMALL BOATS							
SHIPS							
AEROPLANES							
TRAINS							

4. Do you <u>EVER</u> feel HOT or SWEAT whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

5. Do you <u>EVER</u> suffer from HEADACHES whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

6. Do you <u>EVER</u> suffer from LOSS/CHANGE OF SKIN COLOUR (go pale) whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

7. Do you <u>EVER</u> suffer from MOUTH WATERING whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				

SHIPS		
AEROPLANES		
TRAINS		

8. Do you <u>EVER feel DROWSY</u> whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

9. Do you EVER feel DIZZY whilst travelling AS A PASSENGER in the following types of transport?

NEVER	OCCASIONALLY	OFTEN	ALWAYS
	NEVER	NEVER OCCASIONALLY	NEVER OCCASIONALLY OFTEN Image: Constraint of the second

10. Do you <u>EVER suffer from NAUSEA (stomach discomfort, feeling sick)</u> whilst travelling AS A PASSENGER in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

11. Have you EVER VOMITTED whilst travelling AS A PASSENGER in the following types of transport?

	NO	YES
CARS		
BUSES		
COACHES		
SMALL BOATS		
SHIPS		
AEROPLANES		
TRAINS		

12. Would you avoid any of the following types of transport because of motion sickness?

	OCCASIONALLI	OTTEN	ALWAIJ
CARS			
BUSES			
COACHES			

SMALL BOATS		
SHIPS		
AEROPLANES		
TRAINS		

13. Which of the following best describes your SUSCEPTIBILITY to motion sickness?

MUCH LESS THAN AVERAGE	
LESS THAN AVERAGE	
AVERAGE	
MORE THAN AVERAGE	
MUCH MORE THAN AVERAGE	

- 14. Have you ever suffered from any serious illness or injury? NO YES
- 15. Are you under medical treatment or suffering a disability affecting daily life? NO YES

Appendix B: Experiment Briefing

Experimental brief

Objective:

The aim of this research is to study motion (dis)comfort in the context of autonomous vehicles. With the emergence of autonomous vehicles in the near future, it is possible for the drivers to use their freed-up time during automated driving mode for other activities, like leisure or work. For it to be fully utilized as per design, passengers should feel comfortable and safe in order to do so. However, motion discomfort is usually reported by passengers in conventional vehicles and may hamper the acceptance of autonomous vehicles. Factors (e.g., motion, vision) that induce discomfort are currently poorly understood, and knowledge is lacking in particular on interactions between these factors.

This research is expected to result in proposals on enhancing motion comfort of future autonomous vehicles.

Experiment conditions and instructions:

In order to find a relationship of lateral motion, head motion and motion sickness in self-driving vehicles, the participant will be asked to ride along as a passenger in a self-driving vehicle, preprogrammed to follow specific routes (slalom) and rate its motion sickness symptoms during the ride, while wearing devices that measure head movement skin conductance and heart rate.

Participants will be asked to fill out a Motion Sickness Susceptibility Questionnaire (MSSQ) after agreeing on participating in this experiment. This questionnaire consists of questions regarding the participants' susceptibility and prior history to motion sickness.

The conditions that will be tested consists of three (3) different slalom routes of varying frequency, and with two (2) different view conditions, eyes on (forward vision) and eyes off road (blocked vision), for each of the routes. In total there will be six (6) sessions for the participants to complete.

Each driving session will take 30 minutes. Preparation and drive to venue another 30. The total duration is 1 hour per session.

As (mild) symptoms of motion sickness might occur, each condition will be separated from another by at least 4 days, until all six conditions are completed. This is to ensure that participants are 'fresh' and not carry over any previous symptoms or adaptation effects into the experiment. Participants may withdraw from the experiment at any point during, after or before the sessions.

For each of the conditions, the participant will be asked to:

- rate their symptoms according the Misery Scale (MS) with an interval of one minute. The MS is a simple rating scale to indicate your current symptoms that one experiences during the ride. The scale ranges from 0 (no symptoms) to 11 (Vomiting). There will be a signal after every minute to notify the participant to rate.
- Wear the Xsens MVN inertial motion capture system. This device measures the movement of the head and body. It consists of sensors (accelerometers) that can be strapped onto the

head and worn on the torso and has a wireless transmitter device to send the data to the receiver.

- Wear a GSR device to measure the skin conductance of the finger.
- Wear ECG device to measure heart rate and heart rate variability
- Enjoy the ride as you would normally do inside a vehicle. (Sit relaxed, seat belts on and remaining seated)

After each session, the participants will be debriefed and asked to fill out a questionnaire about the motion comfort they have experienced.

During the experiment, the participant will be subjected to vehicle motion that might lead to discomfort and in particular, motion sickness. The risk of said discomfort will be less or similar to riding in a car or bus, due to low driving speeds. If the participant gives a rating of 6 or above on the misery scale, the experiment will terminate.

Inside the vehicle, there is an emergency stop button and a driver overseeing, at all times, the safety of the ride and passenger.

For this experiment, a certified, TU Delft customized Toyota Prius will be used. This Prius has been modified to enable self-driving capabilities. It has gone through stringent tests and has been used in previous studies and experiments conducted by TU Delft. In remote cases, there is a possibility that the Prius will be operated manually instead of fully autonomously.

The experiment will take place in a closed off road in the Technopolis area on the TU Delft campus (exact location TBA).

For their time and effort, upon completion or attempted completion, participants will be given a 10 euro voucher the end of a given session.

If there are any questions, please feel free to ask us!

Appendix C: MOTION SICKNESS ASSESSMENT QUESTIONNAIRE (MSAQ)

Instructions. Using the scale below, please rate how accurately the following

statements describe your experience

Not at all

Severely

1----2----3----4----5----6----7----8----9

1. I felt sick to my stomach (G) 9. I felt disoriented (C) 2. I felt faint-like (C) 10. I felt tired/fatigued (S) 3. I felt annoyed/irritated (S) 11. I felt nauseated (G) 4. I felt sweaty (P) 12. I felt hot/warm (P) 5. I felt queasy (G) 13. I felt dizzy (C) 6. I felt lightheaded (C) 14. I felt like I was spinning (C) 7. I felt drowsy (S) 15. I felt as if I may vomit (G) 8. I felt clammy/cold sweat (P) 16. I felt uneasy (S)

Note. G; Gastrointestinal; C: Central; P: Peripheral; SR; Sopite-related.

If you have any uncomfortable feelings related to motion sickness not mentioned above, please describe below:

Appendix D: Heart rate plots



Figure D1: Heart rate and MISC in time [min] plots for non-sensitive participants. Blue line = HR, Cyan = Linear HR fit, Red = MISC (right axis)



Figure D2: Heart rate and MISC in time [min] plots for non-sensitive participants. Blue line = HR, Cyan = Linear HR fit, Red = MISC (right axis)

Appendix E: GSR

Skin conductance level (blue dashed) plots with its extracted phasic (yellow) and tonic components (red) in time. Vertical black lines represent the start and the end of the trial.















