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Effects of Motion Sickness on Encoding and Retrieval Performance and on Psychophysiological Responses

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Abstract

Background: Motion sickness has previously been found to deteriorate performance. In complex working environments, sustained ability to perform despite motion sickness is crucial. This study focuses on effects of motion sickness on encoding and retrieval of words. In addition, the temporal development of psychophysiological responses and their relationship with perceived motion sickness were investigated.

Methods: Forty healthy participants (20 male and 20 female, age 19-51) performed an encoding and retrieval task during exposure to an optokinetic drum and were compared with 20 controls (8 male and 12 female, age 21-47) not exposed to motion sickness. Measurements of heart rate, heart rate variability, skin conductance, blood volume pulse, respiration rate, and skin temperature were made throughout optokinetic drum exposure.

Results: Moderate levels of motion sickness did not affect the ability to encode or retrieve words. Perceived motion sickness was positively related to heart rate, blood volume pulse and skin temperature and negatively related to respiration rate.

Conclusions: The psychophysiological measurements did not show consistent patterns of sympathetic activation and parasympathetic withdrawal, as could be expected. Subjective reports of progressing symptoms are still likely to be the most reliable way of assessing motion sickness.

Keywords: Autonomic responses; Memory; Motion sickness; Human performance

Abbreviations: BVP: Blood Volume Pulse; CRT: Continuous Recognition Task; ECG: Electrocardiogram; HF: High Frequency; HR: Heart Rate; HRV: Heart Rate Variability; LF: Low Frequency; PPG: Photoplethysmography; Resp: Respiration Rate; RMSSD: Root Mean Square of Successive Differences; SCL: Skin Conductance Level; Temp: Skin Temperature

Introduction

Being able to perform under the influence of motion sickness is essential in operational and working environments where people are being exposed to real or apparent motion, e.g., on board ships and aircrafts or in simulators. Operators in high performance occupations need to devote significant cognitive attention to their tasks and when such tasks are executed in moving environments, either continuously or occasionally, cognitive performance in terms of memory capacity is indeed crucial. Research on how different cognitive abilities are affected by motion sickness is, however, sparse. Recent studies have reported decreased cognitive task performance [1,2] in motion sickness triggering environments. However, these studies did not specifically address the relationship between perceived motion sickness and cognitive performance. The impact of motion sickness on human performance has been studied with regards to psychomotor functions and over learned skills [3,4], and in novel situations requiring the use of short term memory [5,6]. Motion sickness impacted negatively on performing novel tasks and on verbal short term memory [6-9], whereas over learned skills often were managed despite being under the influence of motion sickness [3]. To our knowledge, no previous research has studied the effects of motion sickness on long term memory.

Motion sickness, being a state of perceived illness following exposure to motion or illusory motion [10], triggers an autonomic reaction that, if not stopped, will lead to emesis. It is a subjective sensation, similar to other subjective sensations such as pain or fatigue, and cannot be directly observed unless the extreme effects of the conditions are present, e.g., vomiting [11]. Identifying objectively measured predictors of motion sickness is tempting, since they could ultimately provide the possibility of early detection of developing motion sickness, and thus indicate when countermeasures are needed. In complex working environments, early detection would be valuable, especially if motion sickness is found to deteriorate multiple cognitive abilities.

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Several studies have tried to characterize motion sickness based on different psychophysiological responses [12-15]. There have also been attempts to identify predictors of motion sickness [16-19]. These studies have, however, not reached a consensus of which measurements provide the most comprehensive information, although it is mostly concluded that a general sympathetic nervous system activation with concurrent parasympathetic withdrawal is present [11,15,20]. The inconsistencies in previous studies can partly be explained by differences in study designs, since several different motion sickness triggering stimuli have been used and the assessment of psychophysiological responses varies between studies. The results may be different depending on whether the temporal development during exposure, integrative statistics (e.g., mean of entire test) or development against perceived motion sickness are studied.

The aim of this study was to investigate the effects of motion sickness on encoding and retrieval of words. The encoding and retrieval task used in the present study was novel to the participants and designed to measure long term memory performance. Based on the literature concerning performance of novel tasks [6,7], we hypothesized that motion sickness, triggered by an optokinetic drum, would have a negative impact on long term memory performance. Secondly, we explored the temporal development of different psychophysiological responses during exposure to an optokinetic drum and investigated the relationship between psychophysiological responses and self-rated motion sickness scores, to identify possible predictors of motion sickness.

Materials and Methods

Participants

Forty participants (20 male and 20 female, mean age 25 years, range: 19-51) were recruited for the optokinetic drum experiment. According to self-reports before the experiment, all participants were healthy and no one had previously been exposed to an optokinetic drum. In addition, a control group of 20 volunteers (8 male and 12 female, mean age 26 years, range: 21-47) carried out the performance task without being exposed to the optokinetic drum. The participants were recruited amongst employees at Linköping University Hospital and through e-mail advertisement to all the medical students at Linköping University. The study was carried out in accordance with the Declaration of Helsinki. After being presented to the aim of the study, the participants gave their informed consent and were aware of the possibility to withdraw at any time without any consequences. They were also fully informed about the confidentiality and usage of their data. According to the Swedish Act concerning the Ethical Review of Research Involving Humans (Swedish law: SFS 2003:460) the study was not eligible to be approved by a regional ethical committee. However, the study was approved according to the local ethical advisory board guidelines.

Procedure

The participants were instructed not to eat for 2-3 hours before start of the experiment. Intake of anti-motion sickness medications, anti-emetic medication, antihistamines or alcoholic beverages was not allowed within 24 hours prior to the experiment. This was checked for on a self-report basis. An initial questionnaire was completed before the experiment with questions regarding susceptibility to, and previous experiences of, motion sickness. Thereafter, equipment for psychophysiological measurements was fitted to the participant.

The experiment began with the first phase of the performance task. Thereafter, the participant entered the optokinetic drum and was instructed to keep his/her head still and eye gaze straight forward. The drum was 104 cm high and 120 cm in diameter, and the interior was covered with alternating 7.5 cm black and 7 cm white vertical stripes. During the exposure time, the drum rotated at a velocity of 10 rpm or 60°/s. The participant sat in the drum with eyes open for approximately five minutes before the motion sickness triggering exposure began as the drum started to rotate. Data acquisition began during this acclimatization period. The last minute before start of drum rotation was used as baseline data for the analyses.

Throughout the exposure period, the participant was asked to rate the degree of motion sickness every minute on the Borg CR10 scale [6]. When the participant first scored 2 or more on the Borg scale (corresponding to weak sensations of motion sickness) phase two of the performance task began and lasted for eight minutes. The drum exposure continued for a maximum of 25 minutes and the participant could abort whenever he/she wanted to.

After approximately 35 minutes had passed since the start of drum exposure, the last phase of the performance task was completed. Each participant also completed a questionnaire regarding their experience of the optokinetic drum exposure and the performance task.

Performance task

The cognitive test used in this study was a modified version of a word recognition test previously used by Levin et al. [9]. This continuous recognition task (CRT) consists of three consecutive phases. It starts with encoding of 48 familiar words before drum exposure, followed by encoding and retrieval of 96 words during drum rotation (48 new and 48 old words) and, finally, a retrieval phase with 98 words after drum exposure (48 new and 48 old words). Since the participants were required to look at the stripes while inside the optokinetic drum for the drum to have the desired effect, the second phase of the test had to be conducted with pre-recorded words instead of words shown on a computer screen. The purpose of using the CRT was to investigate the ability to encode and recognize words presented during motion sickness. A word shown for the first time was referred to as "new" and a repeated word was always regarded as "old". In the second and third phase, the participant responded "new" or "old" to each word. Retrieval was defined as a correct response to an "old" item and false alarm was defined as an incorrect answer to a "new" word. It was thus possible to compare the performance in four different conditions:

1. Retrieval during motion sickness (encoding before and retrieval during drum exposure).
2. No motion sickness condition (encoding before and retrieval after drum exposure).
3. Encoding and retrieval during motion sickness (both encoding and retrieval during drum exposure).
4. Encoding during motion sickness (encoding during and retrieval after drum exposure).

The control group performed the task with the same timing between phases, but without entering the optokinetic drum.

Psychophysiological recordings

Heart rate (HR), skin conductance level (SCL), blood volume pulse (BVP), respiration rate (Resp) and finger temperature (Temp) were collected using the digital real-time monitoring system MobileMe (Biosentient Inc), an 8-channel recording system with 14

bit resolution. HR (beats/min) was computed via R-peak detection of the electrocardiogram (ECG), which was measured via a standard lead II configuration. SCL (μS) was recorded on the volar surface of the medial phalanges of the left index and middle fingers. During SCL recordings, the potential across the electrodes was held constant at 0.5V. BVP measurements were made with a photoplethysmography (PPG) probe placed on the left ring finger. BVP (arbitrary unit, a.u.) was calculated as the relative amplitude (peak-to-trough difference) of the PPG signal. Respiration, measured as chest expansion, was recorded using a strain sensitive sensor strapped around the chest. Resp (breaths/min) was computed breath-to-breath from the respiration signal. Temp recordings (°C) were derived from a thermistor placed on the little finger of the left hand. SCL, BVP, Resp and Temp measures were sampled at 32 Hz, whereas ECG was sampled at 256 Hz. These measurements were recorded for approximately five minutes before the optokinetic drum started to rotate and throughout the exposure. For the statistical analyses, all measurements were averaged over 1 min intervals.

Heart rate variability (HRV) parameters were calculated from each 1 minute segment of the recorded R-peak intervals of the ECG using Kubios HRV Analysis Software (The Biomedical Signal Analysis Group, Department of Applied Physics, University of Kuopio, Finland) [21]. Firstly, the raw ECG and R-R interval data were inspected for noise or ectopic beats according to the recommendations by the Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology [22] and artifacts were edited using linear interpolation. Root mean square of successive differences (RMSSD) of normal R-R intervals was then calculated. Furthermore, one frequency-domain variable was calculated using Fast Fourier Transform and included the power spectra integrated over the high-frequency (HF, 0.15–1.0 Hz) band. Spectral power density was expressed in absolute units (ms^2).

Data analysis

Statistical analyses were performed using SPSS version 18 (SPSS Inc., Chicago, IL, USA). The limit for statistical significance was set at α=0.05.

Differences in successful retrievals between the four conditions of the performance task were analyzed by means of Friedman's test. The number of false alarms was compared between phase two and phase three with Wilcoxon's signed ranks test. Possible differences in performance between the exposure group and the control group were investigated using Mann-Whitney U-tests. Spearman rank correlations were calculated to further investigate the possible influence of motion sickness on performance. Correlations were calculated between mean Motion sickness scores from phase two and retrieval and between false alarm and mean Motion sickness scores from phase two.

The psychophysiological measurements were first analyzed using repeated measures MANOVA. Four time points; baseline and the first, middle and last minute of each participant's drum exposure were selected for this analysis. Main effects were tested for time (baseline, start, middle and stop) and abort (abort or endure) along with the interaction effect time × abort. Corrections according to the Greenhouse-Geisser procedure were performed whenever sphericity was violated. Effect sizes were calculated for significant results by partial eta squared (η^2), expressing the amount of variance explained in the dependent variable by the respective effect. Bonferroni corrected post hoc tests were performed when significant main effects were found.

Thereafter, the relationships between subjective ratings of motion sickness and psychophysiological measurements were investigated by calculating each participant's mean of each variable for each Borg scale rating. Since all participants did not rate at all levels of the Borg scale, these data were analyzed with linear mixed regression models. The fixed factor was motion sickness (Borg score 0 to 10) whereas participant (1 to 40) was applied as a random factor. Estimates of fixed effects were calculated for significant results.

Results

Fourteen participants (6 male, 8 female) aborted the drum exposure and 26 (14 male, 12 female) endured the entire 25-minute exposure period. The median exposure time for those who aborted was 16 minutes (range 5 to 22 minutes). In the initial questionnaire, the participants were asked whether they thought they would become motion sick in a situation where 50% of all people normally develop motion sickness. Fifteen participants answered "no" and 25 "yes". Comparing these results with the grouping of participants according to whether they chose to abort or not showed that 58% of the participants classified themselves in the right group. Regarding previous experiences of motion sickness, 14% reported that they often experience motion sickness, 40% sometimes and 31% seldom become motion sick. Only 5% reported that they very seldom or practically never experience motion sickness.

After completion of the experiment, the participants were asked to rate (from 1 to 7) the stressfulness of the experiment and the mental strain during the experiment. Median ratings were 3 points for stress and 4 points for mental strain, respectively. The symptoms triggered by the optokinetic drum were, by 85% of the participants, considered to be representative to symptoms usually perceived during motion sickness.

Performance

Five participants in the exposure group (two male and three female) never reached 2 on the Borg scale and were, hence, excluded from analyses of performance data. The motion sickness ratings from phase two of the CRT increased from 2.5 points (SD .9) in the beginning to 4.0 points (SD 1.9) at the end of the phase. The mean Borg rating for phase two was 3.3 points (SD 1.1). After phase three of the CRT and just before leaving, the mean Borg score had decreased to 0.8 points (SD 0.8).

There was no significant difference in success of retrieval between the four conditions (Table 1). Hence, the ability to encode and retrieve words was the same, regardless of whether the encoding, the retrieval, or both were carried out under the influence of motion sickness. There was a borderline significant difference between the exposure group and controls in the "no motion sickness condition", where the exposure group actually performed better. Further scrutinizing the data showed that this was due to a significantly lower performance of this condition compared with the other conditions in the control group.

The number of false alarms was significantly higher in phase three than in phase two (Table 2). A difference between the numbers of false alarms indicates a change in the participants' attention to "new" words between phase two and three. There were no significant differences in false alarms between the exposure group and the controls. The control group also reported a higher number of false alarms in phase three compared with phase two.

There were no significant correlations between level of motion sickness during phase two and encoding and retrieval performances.

Five participants aborted the drum exposure during phase two

		Retrieval during motion sickness	No motion sickness condition	Encoding and retrieval during motion sickness	Encoding during motion sickness	Difference between conditions
						p-value
Exposure group (n=35)	Correct retrievals Mean (SD)	20.4 (3.2)	20.1 (3.1)	20.1 (5.0)	20.4 (2.5)	.805
	Retrieval rate (%)	84.9	83.7	83.8	85.1	
Control group (n=20)	Correct retrievals Mean (SD)	19.0 (4.5)	17.2 (5.0)	21.0 (2.3)	20.7 (1.9)	.029
	Retrieval rate (%)	79.0	71.7	87.3	86.3	
Difference between groups	p-value	.337	.051	.805	.723	

Table 1: Retrieval measured as number of words correctly recalled as being "old", and retrieval rate, expressed as the conditional probability of a correct response. The control group was not exposed to motion sickness.

		Phase two	Phase three	Difference between phases
				p-value
Exposure group (n=35)	Number of false alarms Mean (SD)	5.1 (4.6)	6.7 (5.9)	.003
	False alarm rate (%)	10.6	14.0	
Control group (n=20)	Number of false alarms Mean (SD)	4.2 (3.8)	6.6 (4.5)	.027
	False alarm rate (%)	8.7	13.7	
Difference between groups	p-value	.555	.510	

Table 2: Number of false alarms, i.e. wrong answers to "new" words, and false alarm rate, expressed as the conditional probability of responding "old" to a novel word.

	Time			Abort			Time × Abort			
Variable	F(3,111)	p	η^2	F(1,37)	p	η^2	F(3,111)	p	η^2	ε
Motion sickness	137.5	<.001	.79	20.7	<.001	.36	16.2	<.001	.30	.672
HR	.7	.536		0.3	.596		1.9	.138		
SCL	2.7	.082		4.4	.043	.11	2.1	.135		.607
BVP	10.7	<.001	.22	5.3	.027	.13	2.4	.092		.762
Resp	6.1	.001	.14	0.1	.759		0.7	.540		
Temp	18.9	<.001	.34	4.8	.035	.12	0.0	.927		.520
lnRMSSD	8.6	<.001	.19	1.0	.317		0.4	.731		
lnHF power	5.9	.001	.14	1.2	.272		0.5	.715		

Table 3: Results from the repeated measures ANOVAs, showing the main effects of Time (Baseline, Start, Middle, Stop) and Abort (Abort or Endure) and their interaction effects. Effect sizes (η^2) are given for significant results and the Greenhouse-Geisser correction factor (ε) is indicated where applicable.

of the performance task, but continued to score motion sickness >2 throughout phase two. Excluding these participants from the analyses resulted in a significant difference between the exposure group and the control group in the "no motion sickness condition".

Psychophysiology

One participant had missing HRV data at the last time point and was therefore not included in the repeated measures MANOVA, leaving 25 participants that endured the optokinetic exposure and 14 that aborted in this analysis. The repeated measures MANOVA showed significant multivariate effects of time ($F(24,14)=19.7$, $p<.001$, $\eta^2=.97$) and abort ($F(8,30)=4.1$, $p=.002$, $\eta^2=.52$). The interaction effect time × abort was also significant ($F(24,14)=3.8$, $p=.006$, $\eta^2=.87$). Furthermore, the univariate tests showed that the time effects were significant for Motion sickness, BVP, Resp, Temp, lnRMSSD and lnHF (Table 3). Motion sickness scores increased significantly from each time point to the next (Figures 1a-1c). BVP exhibited a small but not significant decrease at the start of drum exposure and was then significantly higher than start at middle and stop (Figure 1d). Resp increased slightly at start and then decreased to a significantly lower level at stop compared with start (Figure 1e). Temp decreased significantly from baseline to start and then increased to significantly higher levels at middle and stop compared with baseline and start (Figure 1f). The time domain measurement of heart rate variability, RMSSD, decreased significantly to the middle of the exposure compared with baseline and start (Figure 2a). HF power showed a similar development over time but the only significant difference was between baseline and middle (Figure 2b).

The abort effect was significant for Motion sickness ratings, SCL, BVP and Temp. The estimated mean differences (95% CI) between the abort group and the endure group were; Motion sickness = 1.5 points (.8 – 2.1), SCL = 4.7 μS (.2 – 9.3), BVP = .6 a.u.(.1 – 1.1) and Temp = 2.4°C (.2 – 4.5). The interaction effect time × abort was only significant for Motion sickness ratings, showing a steeper increase in motion sickness ratings during the exposure in the abort group. The group division was based on exposure tolerance, whereas others have classified participants according to perceived motion sickness. Re-running the analyses with a group division based on a median split of Motion sickness scores at stop yielded similar results; the only difference being non-significant main group effects of BVP and Temp.

Analyses of the relationships between psychophysiology and motion sickness ratings showed that HR, BVP and Temp increased with increasing motion sickness, whereas Resp decreased with increasing motion sickness (Table 4).

Discussion

The present study did not find negative effects on encoding and retrieval during motion sickness triggered by an optokinetic drum. Sustained ability to encode, despite perceived motion sickness, seems to be feasible, at least under the conditions reported here. Furthermore, we found positive relationships between Motion sickness and HR, BVP and Temp whereas Resp was negatively related to perceived motion sickness. The HRV measurements RMSSD and HF power showed changes over time, but were not significantly related to motion sickness

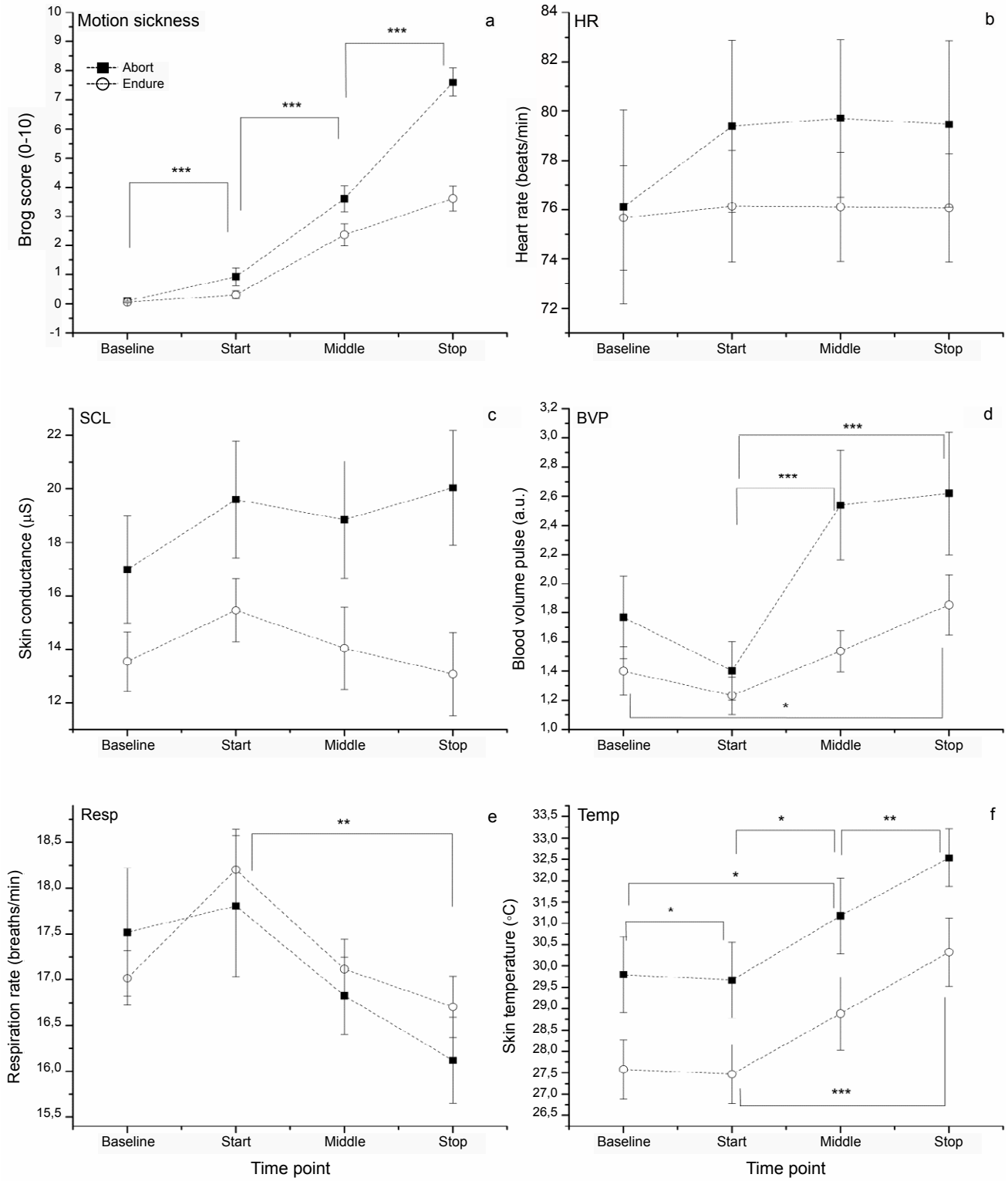

Figure 1: Mean (SEM) motion sickness ratings, HR, SCL, BVP, Resp, and Temp for the selected time points. Differences between time points, irrespective of group belonging, are indicated with asterisks. * $p < .05$, ** $p < .01$, *** $p \leq .001$.

scores. These results are, in some aspects, inconsistent with previous optokinetic drum studies [23,24] and studies involving other stimuli [14].

Performance

In our previous study regarding short term memory performance and motion sickness [6], we found that performance was negatively

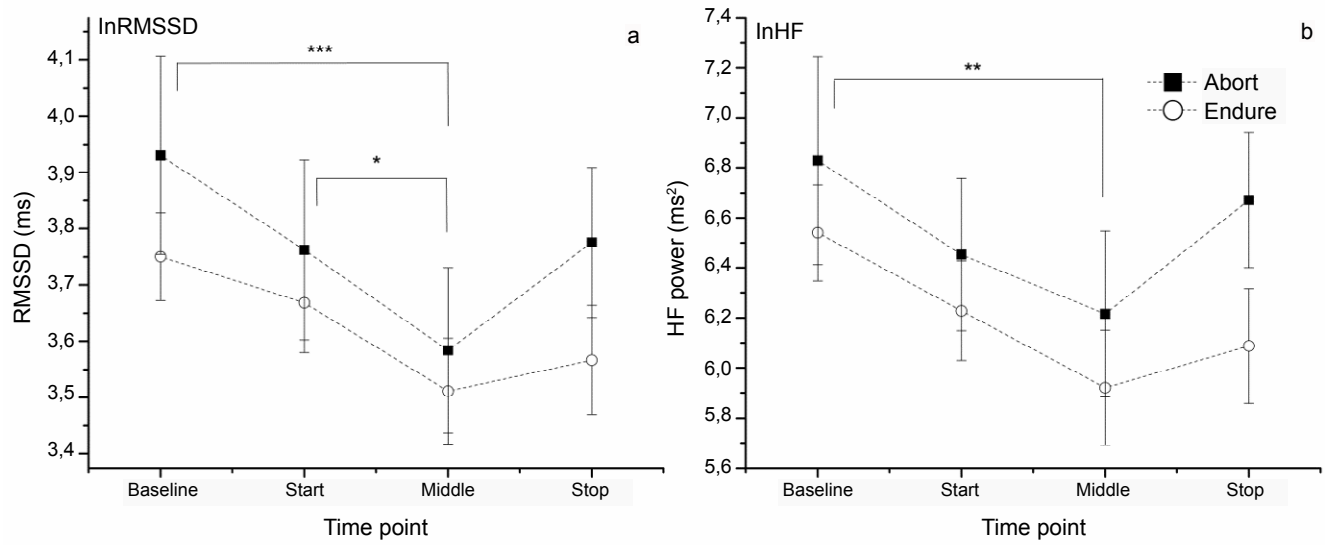

Figure 2: Mean (SEM) of the HRV measurements RMSSD and HF power for the selected time points. Differences between time points, irrespective of group belonging, are indicated with asterisks. * p < .05, ** p < .01, *** p ≤ .001.

Variable	F	p	Estimated effect
HR	6.9	.013	.47 (.11 - .83)
SCL	2.8	.104	
BVP	12.7	.001	.10 (.05 - .16)
Resp	23.5	<.001	-.26 (-.37 - -.15)
Temp	24.8	<.001	.37 (.22 - .52)
lnRMSSD	1.1	.309	
lnHF power	.001	.980	

Table 4: Results from the mixed model regression analyses. The estimated effect indicates the change in each variable with an increase in motion sickness rating.

affected only after reaching relatively high levels of motion sickness (mean Borg score 7.9, corresponding to "very strong"). Taken together, our results indicate that participants experiencing weak or moderate levels of motion sickness may still able to perform at their best. Results from studies of cognitive performance in relation to motion sickness have varied depending on the type of performance task used and whether motion sickness was visually or motion induced [2,5,8,25]. Speculatively, when motion sickness is induced by a stimulus that includes a motion component, it is plausible that performance could be mainly affected by the motion and not by the evolving motion sickness [1].

Motion sickness should not generally enhance performance [5]. The tendency towards a difference in performance between the control group and the exposure group, where the exposure group actually performed better, is therefore puzzling. This finding could, however, be explained by differences in the test procedures. Whereas the exposure group was seated inside the optokinetic drum most of the time between phase one and phase three, suggesting that such a bias could not fully explain the results. of the CRT, the control group stayed in a less controlled environment, possibly causing distraction from the task and thereby decreasing performance. Another explanation may be that people more frequently encountering motion sickness, may develop certain coping strategies to maintain performance [26]. These strategies could include focusing on other external stimuli, e.g., the spoken word in the present study, rather than on the motion sickness triggering stimulus, in order to suppress symptoms. However, only one out of seven of the participants reported frequent motion sickness experiences in the initial questionnaire

Although the present study found no effects on encoding and retrieval performance, there were significantly more false alarms reported in phase three than in phase two. This observation is probably due to the amount of words processed by then, rather than due to effects of motion sickness per se, since the control group showed a similar development. The fact that motion sickness ratings during phase three were at the same level as in phase one further strengthens this suggestion.

It could be argued that the participants may have been affected by motion sickness even during the final retrieval phase (referred to as phase three). However, the motion sickness ratings, according to the Borg scale, indicated that the participants felt little or no motion sickness as a result of their exposure, i.e., similar to the ratings before drum exposure. During drum rotation, the participants had to reach a level of "weak" motion sickness, i.e., Borg 2, before start of the CRT. This requirement was set in order to ensure that the participants actually were experiencing motion sickness when undertaking both the encoding and retrieval tasks in phase two.

Psychophysiology

The measurements of sympathetic nervous system activity showed ambiguous results. Based on the literature, HR, SCL, Resp, LF power and LF/HF ratio were expected to increase with time and/or with increasing motion sickness, whereas Temp and BVP were expected to decrease [12,14,24,27-32]. Particularly surprising was the decrease in respiration rate over time and with increasing motion sickness, which contradicts previous research where slow breathing has been shown to

ameliorate motion sickness [33,34]. However, Gianaros et al. [35] also found decreased respiration rate during optokinetic stimulation, but the decrease was not related to motion sickness severity.

The development of HR over time indicated increased heart rate in the abort group, although the differences between groups did not reach statistical significance. HR has been reported to increase during exposure to motion sickness triggering stimuli, especially among those reporting nausea [12,15,21,36]. However, it has been suggested that the temporal development of HR or mean HR over the entire exposure period does not necessarily reflect the progressive changes in sickness [15,24], thus emphasizing the need for correlational or regression analyses. The mixed regression analysis did show a positive relationship between HR and motion sickness ratings, which confirms previous findings [14,24,36,37].

In accordance with our results from a study using a motion platform [36], SCL showed a tendency to increase with motion sickness, although this effect was not significant. Similarly, LaCount et al. [38] found SCL to increase with increasing nausea during exposure to horizontally translating stripes. Hu et al. [15] found a significant correlation between SCL and perceived motion sickness and higher SCL among participants reporting nausea compared with non-nauseous participants in an optokinetic drum study. Contrary to our previous optokinetic drum study [6], SCL did not change significantly over time but did show a significant main group difference. The abort group had higher SCL even at baseline, possibly reflecting higher arousal at the start of the experiment due to expectations of emerging motions sickness from the trial.

The indicators of parasympathetic nervous activity, RMSSD and HF power, were expected to be lower during the exposure [15,27] and to decrease with increasing motion sickness [23,37]. The development from baseline to middle was as expected but the subsequent increase towards stop in both groups was somewhat surprising. There are, however, studies reporting no change in HF power during exposure to motion sickness stimulation [31], making these HRV measurements difficult to predict.

The psychophysiological responses seem to be more complex than simply sympathetic activation and parasympathetic withdrawal. The non-invasive measurements, including HRV, that reflect gross end-organ outputs do not seem to be specific enough to use as predictors of motion sickness. Inter-individual differences in these responses are large and even the intra-individual stability across multiple tests has been questioned [38]. More sophisticated methods that are able to separate the motion sickness responses from general arousal or stress responses are needed for prediction of motion sickness with sufficient reliability. Furthermore, susceptibility to motion sickness is contextually dependent [39] and possible differences in psychophysiological responses depending on the stimulus have to be taken into consideration.

Methodological considerations

Future studies should perform control group assessments in the exact same environment as the motion sickness cases, only excluding the motion sickness component, in order to increase comparability between groups. In addition, a cross-over design could be employed, testing the same participants with and without motion sickness. However, when repeatedly assessing memory performance, possible learning effects have to be taken into consideration.

The study design rendered different rotation times and different times between the CRT phases. Another possibility would have been to standardize the starting time of CRT phase two in the drum and perform a post-hoc splitting of the participants into a "sick" and "not sick" group according to their perceived level of motion sickness. Maybe this approach would have led to more significant results regarding the actual effect of motion sickness on performance. However, there would still be a problem with handling dropouts. The current design was chosen to ensure that the entire phase two of the CRT was performed under the influence of motion sickness.

A drawback of this study was the use of a modified performance test, making the validity of the test unclear. A difference in the present study compared with the Levin et al. study [9], was the use of different sensory modalities for encoding. Since the participants were required to look at the stripes while inside the optokinetic drum for the drum to have the desired effect, the second phase of the test had to be conducted with pre-recorded words, whereas the words in phase one and three were presented visually on a computer screen [40]. A possible explanation for the lack of effect of motion sickness on the participants' performances by auditory encoding being more effective is, however, not supported by Kintsch and Kozminski [41], who concluded that whether information was read or listened to did not affect the ability to retrieve it. Moreover, during performance under exceptional circumstances, expert approaches are commonly utilized. To understand the mechanism of these approaches, the traditional models of working memory involving temporary storage must include the long-term memory [41]. The acquired memory skills allow stimuli like the ones we used, regardless of modality, to be stored in the long-term memory and kept directly accessible by means of retrieval cues in short-term memory. Consequently, despite modifying the performance test without performing a criterion validity assessment, we do not assume that the results would have been different using the original test.

When conducting studies in a laboratory environment, well aware of motion sickness being contextually dependent [42,43], there is always the risk of inducing response behavior that is not representative for motion sickness in real environments. However, the participants stated that the optokinetic drum triggered symptoms similar to their previous experiences of motion sickness. The participants were not selectively screened for their susceptibility to motion sickness. It is possible that the lack of reported effect may be attributable to the fact that these individuals did not experience intense symptoms. Furthermore, future research should use more sophisticated methods to control for respiration in the HRV-analyses, rather than controlling for respiratory frequency in the statistical analyses. By minimizing the respiratory effects, any influence of motion sickness on HRV-parameters may become apparent.

Conclusions

Encoding and retrieval of words are not affected at moderate levels of motion sickness. Thus, sustained ability to encode despite perceived motion sickness seems to be feasible, at least under the conditions reported in the present study. How encoding and retrieval are affected by higher levels of motion sickness remains to be examined. The psychophysiological measurements did not show consistent patterns of sympathetic activation and parasympathetic withdrawal, as could be expected, and none of the investigated variables constitute a good candidate for prediction of motion sickness. Subjective reports of progressing symptoms are still likely to be the most reliable way of assessing motion sickness.

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References

1. Muth ER (2009) The challenge of uncoupled motion: duration of cognitive and physiological aftereffects. Hum Factors 51: 752-761.
2. Tal D, Gonen A, Wiener G, Bar R, Gil A, et al. (2012) Artificial horizon effects on motion sickness and performance. Otol Neurotol 33: 878-885.
3. Cowings PS, Toscano WB, DeRoshia C, Tauso R (2001) Effects of Command and Control Vehicle (C2V) operational environment on soldier health and performance. Hum Perf Extrem Environ 5: 66-91.
4. Kennedy RS, Moroney WF, Bale RM, Gregoire HG, Smith DG (1972) Motion sickness symptomatology and performance decrements occasioned by hurricane penetrations in C-121, C-130 and P-3 navy aircraft. Aerosp Med 43: 1235-1239.
5. Rolnick A, Gordon CR (1991) The Effects of Motion Induced Sickness on Military Performance, in Handbook of Military Psychology, G. Reuven and D.D. Mangelsdorff, Editors. John Wiley & Sons.
6. Dahlman J, Sjörs A, Lindström J, Ledin T, Falkmer T (2009) Performance and autonomic responses during motion sickness. Hum Factors 51: 56-66.
7. Muth ER, Walker AD, Fiorello M (2006) Effects of uncoupled motion on performance. Hum Factors 48: 600-607.
8. Paule MG, Chelonis JJ, Blake DJ, Dornhoffer JL (2004) Effects of drug countermeasures for space motion sickness on working memory in humans. Neurotoxicol Teratol 26: 825-837.
9. Levin B, Andersson J, Karlsson T (2007) Memory performance during G exposure as assessed by a word recognition task. Aviat Space Environ Med 78: 587-592.
10. Reason JT, Brand JJ (1975) Motion Sickness. New York, Academic press.
11. Muth ER (2006) Motion and space sickness: intestinal and autonomic correlates. Auton Neurosci 129: 58-66.
12. Cowings PS, Suter S, Toscano WB, Kamiya J, Naifeh K (1986) General autonomic components of motion sickness. Psychophysiology 23: 542-551.
13. Kim YY, Kim HJ, Kim EN, Ko HD, Kim HT (2005) Characteristic changes in the physiological components of cybersickness. Psychophysiology 42: 616-625.
14. Stout CS, Toscano WB, Cowings PS (1995) Reliability of psychophysiological responses across multiple motion sickness stimulation tests. J Vestib Res 5: 25-33.
15. Hu S, Grant WF, Stern RM, Koch KL (1991) Motion sickness severity and physiological correlates during repeated exposures to a rotating optokinetic drum. Aviat Space Environ Med 62: 308-314.
16. Smart LJ Jr, Stoffregen TA, Bardy BG (2002) Visually induced motion sickness predicted by postural instability. Hum Factors 44: 451-465.
17. Meissner K, Enck P, Muth ER, Kellermann S, Klosterhalfen S (2009) Cortisol levels predict motion sickness tolerance in women but not in men. Physiol Behav 97: 102-106.
18. Harm DL, Schlegel TT (2002) Predicting motion sickness during parabolic flight. Auton Neurosci 97: 116-121.
19. Gianaros PJ, Quigley KS, Mordkoff JT, Stern RM (2001) Gastric myoelectrical and autonomic cardiac reactivity to laboratory stressors. Psychophysiology 38: 642-652.
20. Stern RM (2002) The psychophysiology of nausea. Acta Biol Hung 53: 589-599.
21. Borg G, Borg E (2001) A New Generation of Scaling Methods: Level-anchored Ratio Scaling. Psychologica 28: 15-45.
22. Niskanen JP, Tarvainen MP, Ranta-Aho PO, Karjalainen PA (2004) Software for advanced HRV analysis. Comput Methods Programs Biomed 76: 73-81.
23. (1996) Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Circulation 93: 1043-1065.
24. Holmes SR, Griffin MJ (2001) Correlation Between Heart Rate and the Severity of Motion Sickness Caused by Optokinetic Stimulation. J Psychophysiol 15: 35-42.
25. Hu S (1990) Motion Sickness Adaption: Changes in Psychological and Physiological Variables. Doctoral thesis. College of Liberal Arts, Pennsylvania State University.
26. Bos JE, MacKinnon SN, Patterson A (2005) Motion sickness symptoms in a ship motion simulator: effects of inside, outside, and no view. Aviat Space Environ Med 76: 1111-1118.
27. Yardley L (1992) Motion sickness and perception: a reappraisal of the sensory conflict approach. Br J Psychol 83: 449-471.
28. Yokota Y, Aoki M, Mizuta K, Ito Y, Isu N (2005) Motion sickness susceptibility associated with visually induced postural instability and cardiac autonomic responses in healthy subjects. Acta Otolaryngol 125: 280-285.
29. Warwick-Evans LA, Church RE, Hancock C, Jochim D, Morris PH, et al. (1987) Electrodermal activity as an index of motion sickness. Aviat Space Environ Med 58: 417-423.
30. Wan H, Hu S, Wang J (2003) Correlation of phasic and tonic skin-conductance responses with severity of motion sickness induced by viewing an optokinetic rotating drum. Percept Mot Skills 97: 1051-1057.
31. Golding JF (1992) Phasic skin conductance activity and motion sickness. Aviat Space Environ Med 63: 165-171.
32. Ohyama S, Nishiike S, Watanabe H, Matsuoka K, Akizuki H, et al. (2007) Autonomic responses during motion sickness induced by virtual reality. Auris Nasus Larynx 34: 303-306.
33. Golding JF, Gresty MA (2005) Motion sickness. Curr Opin Neurol 18: 29-34.
34. Jokerst MD, Gatto M, Fazio R, Stern RM, Koch KL (1999) Slow deep breathing prevents the development of tachygastria and symptoms of motion sickness. Aviat Space Environ Med 70: 1189-1192.
35. Gianaros PJ, Quigley KS, Muth ER, Levine ME, Vasko RC Jr, et al. (2003) Relationship between temporal changes in cardiac parasympathetic activity and motion sickness severity. Psychophysiology 40: 39-44.
36. Himi N, Koga T, Nakamura E, Kobashi M, Yamane M, et al. (2004) Differences in autonomic responses between subjects with and without nausea while watching an irregularly oscillating video. Auton Neurosci 116: 46-53.
37. Dahlman J, Sjörs A, Ledin T, Falkmer T (2008) Could sound be used as a strategy for reducing symptoms of perceived motion sickness? J Neuroeng Rehabil 5: 35.
38. LaCount LT, Barbieri R, Park K, Kim J, Brown EN, et al. (2011) Static and dynamic autonomic response with increasing nausea perception. Aviat Space Environ Med 82: 424-433.
39. Graybiel A, Lackner JR (1980) Evaluation of the relationship between motion sickness symptomatology and blood pressure, heart rate, and body temperature. Aviat Space Environ Med 51: 211-214.
40. Lackner JR, Dizio P (2006) Space motion sickness. Exp Brain Res 175: 377-399.
41. Kintsch W, Kozminsky E (1977) Summarizing stories after reading and listening. J Educ Psychol 69: 491-499.
42. Ericsson KA, Kintsch W (1995) Long-term working memory. Psychol Rev 102: 211-245.
43. Benson AJ, Stott JRR (2006) Motion sickness, in Ernsting's Aviation medicine, D.J. Rainford and D.P. Gradwell, Editors. Hodder Arnold Ltd., London.