

The menstrual cycle and susceptibility to coriolis-induced sickness

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Abstract: Survey studies on motion sickness susceptibility suggest that females tend to report greater severity in illness and higher incidence of vomiting than males. Menstruation is said to be a contributing factor. A recent study suggested that females were least susceptible to seasickness during ovulation in a “round the world” yacht race. Sixteen subjects (18–36 years old) were exposed to Coriolis cross-coupling stimulation in the laboratory. They were tested once during permenstruation (Day 1–5), ovulation (Day 12–15) and premenstruation (Day 24–28), based on a normalized 28-day cycle, in a randomised design. Physiological measurements of motion sickness included forearm and calf cutaneous blood flow. Subjective evaluation of sickness symptoms was based on Graybiel’s diagnostic criteria and Golding’s rating method. Our results indicated that under controlled laboratory conditions, different phases of the menstrual cycle appear to have no influence on subjective symptoms of motion sickness or on cutaneous blood flow increase in the forearm and calf. The lack of commonality between the types and levels of hormones that are released during motion sickness and those that are involved in different menstrual phases appears to support our findings.

Keywords: Menstrual cycle, motion sickness, susceptibility, Coriolis

1. Introduction

A number of survey studies have suggested that females are more susceptible to seasickness than males [1,21,27,37]. Women rate themselves as more likely to suffer from motion sickness on all major forms of transport and in different motion situations, such as carnival devices and gymnastics [23]. Furthermore, women rate themselves as having greater vomiting incidence and more severe sickness [22]. A recent survey by Dobie et al. [6] concluded that greater motion susceptibility of females does not vary significantly with age and cannot be accounted for by differences in exposure to motion or physical activity. It was suggested that this difference in susceptibility may be attributable

to males being less inclined (or females more inclined) to admit illness. In contrast, Levy and Rapaport [24] reported that there was no difference in seasickness susceptibility between males and females participating in drug trials aboard large sailing yachts. Similarly, Turner [36] found no significant gender differences in motion sickness ratings or incidence of different symptoms during coach (tour bus) journeys. In animal studies, under controlled laboratory conditions, the incidence of emesis in male monkeys was essentially the same as that of the female monkeys [28]. The emetic response frequency was significantly greater in males and the emetic response latency was also significantly shorter which suggested that male monkeys may be more susceptible to motion sickness. With the exception of a few studies into gender differences in the susceptibility to visually induced sickness about the yaw axis [16,30] there are few laboratory investigations into gender differences in motion sickness susceptibility.

When greater female susceptibility is reported, it tends to be attributed to the influences of the female endocrine system. Specifically, menstruation has been

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reported as a possible contributing factor. However, this statement has never been substantiated conclusively. Schwab [33] described "... a nurse in the army medical corps who successfully crossed the Atlantic on a small vessel during rough weather without being ill, but who became nauseated and vomited in calm sea of the Mediterranean when her menstrual period began." (p. 636). More recently, during the 1996–97 "BT Global Challenge Yacht Race", Grunfeld and Gresty [15] reported that there was a slight increase in the number of sickness events reported during the premenstrual (Days 25–28) and permenstrual phases (Days 1–5) based on a normalized 28-day cycle. There were fewer complaints of sickness reported before and around the time of ovulation (Days 11–14). Grunfeld and Gresty's findings suggested that it might be important to classify each female's susceptibility to motion sickness according to her particular menstrual phase. This may be useful in situations where increasingly more women are being employed such as the military, offshore oilrigs, civilian aircraft and ocean liners. With the exception of Jones' study [17], which reported that menstruation has no influence upon nausea induced by Caloric irrigation following yaw rotation, there appears to be no published laboratory studies of motion sickness susceptibility in females over the different phases of their menstrual period. In Jones' study there was no attempt made to classify the non-menstrual group into different phases of the menstrual cycle. Therefore, the purpose of this study is to investigate whether the premenstrual, permenstrual and ovulatory phases of the menstrual cycle have an influence on the susceptibility of female subjects to motion sickness.

2. Materials and methods

2.1. Subjects

Sixteen healthy subjects between the ages of 18 and 36 participated in the study. Recruitment was from local universities and personnel from the laboratory. Approval for this study was obtained from the DCIEM (Defence and Civil Institute of Environmental Medicine) Human Ethics Committee. All subjects gave informed consent after obtaining medical approval to participate from a DCIEM physician. They had no known history of ophthalmologic, oculomotor or vestibular disorders, and they had no spontaneous nystagmus nor Romberg's sign with eyes opened or eyes closed. Subjects reported that they had no known

history of menstrual cycle abnormalities. All subjects were instructed to strictly abstain from alcohol, tobacco, and over the counter and prescribed medication for at least 24 hours prior to the experiment. None of the subjects had previous experience with Coriolis stimulation.

2.2. Apparatus

The motion device is a rotating platform (1.8 m in radius). It is driven by a motor of maximum torque (12.2 N-m) to produce a rotating force environment. The subject was positioned at the centre of rotation. Attached to the back of the seat is a motor-driven headrest, which was designed to assist the subjects to place their heads in a predetermined position. The headrest also served to guide the forward and downward movements of the head through an angle of 45° in the sagittal plane relative to the seated subject. All head movements were guided active head movements beginning with a head down movement from the natural head erect position.

2.3. Physiological measurements

Continuous cutaneous forearm and calf blood flow (BF) changes were monitored using the Perimed Periflux 5001 laser Doppler system in combination with two Perimed # 413 integrating probes (Perimed Inc., Stockholm, Sweden). With the subject seated and hands resting on a flat surface, palms facing down, the forearm integrating probe was positioned on the lateral surface of the left forearm in the region of largest circumference. The second integrating probe was positioned on the dorsal surface of the left calf, left of the midline in the region of largest circumference. Ambient temperature of the laboratory was maintained between 22 and 24°C and subjects were instructed to wear light-weight, loose fitting clothes. Subjects were instructed to relax and avoid active movement during the measurement period.

2.4. Symptom measurements

The procedure used to quantify the subjective severity of sickness before and after each trial was modified from Graybiel et al. [14] to include consideration of pallor, nausea and vomiting. During the exposure of Coriolis cross-coupling stimulation, the subjective rating scale by Golding and Kerguelen [13] was used. Subjects rated their degree of motion sickness after

each head movement on the following scale: 1 = No symptoms; 2 = Any symptoms, however slight; 3 = Mild symptoms, e.g. stomach awareness but no nausea; 4 = Mild nausea; 5 = Mild to moderate nausea; 6 = Moderate nausea but can continue; 7 = Moderate nausea, want to stop. Subjects memorized this scale before the commencement of the trial. They were informed that although the scale was ordinal, they did not have to follow the scale in the written sequence, but rather to pair symptoms they experienced at a particular instant with a specific level on the scale. The motor driven head-rest was stopped when the subject reported a rating of 7 or after 15 minutes, whichever came first.

2.5. For indication of anxiety level

The S-anxiety scale [34] was used as a sensitive indicator of change in transitory anxiety experienced by subjects. It was administered before and after each trial. This scale has been used extensively to assess the level of State anxiety induced by the stressful experimental procedure.

2.6. Design and procedure

A repeated measures factorial design was employed, each of the subjects was exposed once during her premenstrual (Days 25–28), permenstrual (Days 1–5) and ovulatory (Days 14–18) phase. The order of exposure was randomised. Some subjects started their first trial while in their ovulation phase while others started their first trial in their premenstrual or permenstrual phase. The distinction for the three phases was based on a normalised 28-day cycle with the first day of menstruation designated as Day 1. The mid-point of the cycle (typically day 14) was taken as the period of ovulation. Four subjects who were on oral contraceptives were also tested at the mid point of their cycles. Since not every subject has a 28-day cycle, the exact dates of the three menstrual phases of each subject were calculated according to the subject's history (past 2 months) of menstruation. Calculations for each subject were based on the subject's last menstrual cycle date in combination with the first day of the next period. In order to ensure that our calculation of the subject's menstrual phase was as accurate as possible, one of the investigators (RH) maintained frequent contact with the subjects to ensure that each motion sickness test was performed on the intended day within the specific menstrual phase of each subject. It has been shown that self observation (cyclic changes of cervical mucus, sensation of

wetness and lubrication of the vulva) allows a reliable detection of the time ovulation and correlate with thermic nadir day and the day or the period of echographic ovulation [29].

The motion stimulus started with the subjects rotating about the yaw axis at 120°/s. After 180 seconds of rotation, the subjects began moving their heads. Subjects pitched their heads forward and downward by 45° within 2 seconds. Next, they paused for 12 seconds before returning their heads to upright. After another 12 second pause, the sequence was repeated. This manoeuvre produces an unusual stimulation of the vestibular organs that elicits disorientation and motion sickness.

2.7. Statistical analysis

Prior to the analyses, artifacts from raw BF data during each trial were reviewed and excluded from further analyses. Artifacts constituted less than 1% of the recorded data. With the exception of the period of Coriolis stimulation during the Coriolis trials, the mean of the last 60 seconds of BF data during identical motion stimuli, in all trials, was used in the final analyses. The time for peak forearm and peak calf blood flow for each Coriolis trial were identified, and 30 seconds before and after each peak were also used in the analyses of BF. Data were analysed by repeated measures ANOVA using Statistica by Statsoft, with the significance level α set to ≤ 0.01 . All post hoc testing was completed using planned comparisons. P-values for factors with more than two levels were adjusted using Greenhouse-Geisser's epsilon correction factor.

3. Results

Of the 16 subjects, three did not complete their second and third trials due to the severity of sickness experienced. Two of these subjects were tested during their ovulation phase and one was tested during her premenstrual phase. Results of the subjective and objective evaluation of motion sickness on the remaining 13 subjects are described as follows.

3.1. Blood flow measurements

Forearm and calf blood flow records for each menstrual phase from one subject are displayed in Fig. 1. A 2 (blood flow measurement location) x 3 (menstrual phase) repeated measures ANOVA was per-

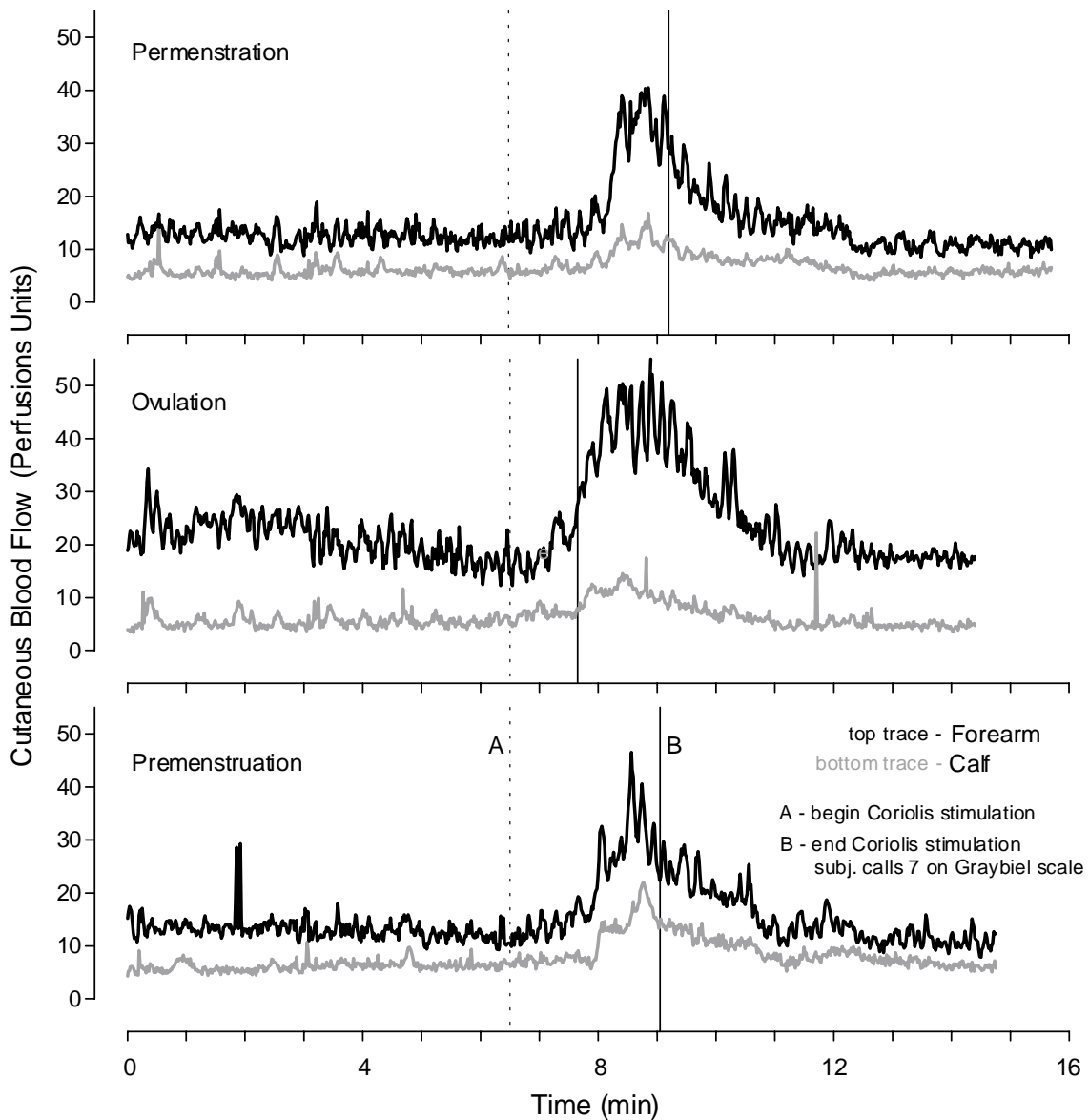


Fig. 1. Forearm and calf cutaneous blood flow during each menstrual phase as a function of time.

formed on the cutaneous forearm and calf blood flow (BF) data. There was a significant main effect for location ($F(1, 12) = 31.99, p \leq 0.01$). The magnitude of forearm blood flow as measured by perfusion unit is higher than that of calf blood flow. This finding is consistent with previous reports which state that forearm and calf cutaneous blood flow increases during motion sickness [5] and that head movements or yaw rotation alone did not result in blood flow changes. The main effect for location, illustrated in Fig. 2, reveals that forearm blood flow is significantly greater than calf blood flow in each of the three menstrual phases. This main

effect for location of blood flow (forearm versus calf) is maintained during head movements while stationary and during yaw rotation alone [5]. The different phases of the menstrual cycle did not have an effect on Coriolis induced BF increases ($F(2, 24) = 1.14, p = 0.34$).

3.2. Symptom measurements

Friedman non-parametric ANOVA of ranks was performed on each of the pre-Coriolis and post-Coriolis evaluations of state anxiety, the severity of motion sickness measured using the Graybiel Diagnostic Cri-

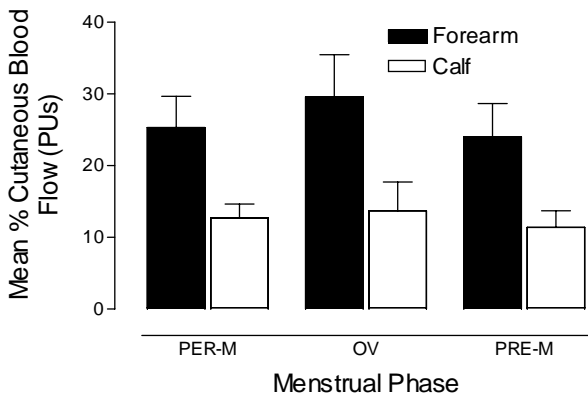


Fig. 2. Mean percentage of cutaneous blood flow 30sec before and after peak flow as a function of time (Error bars represent SEM).

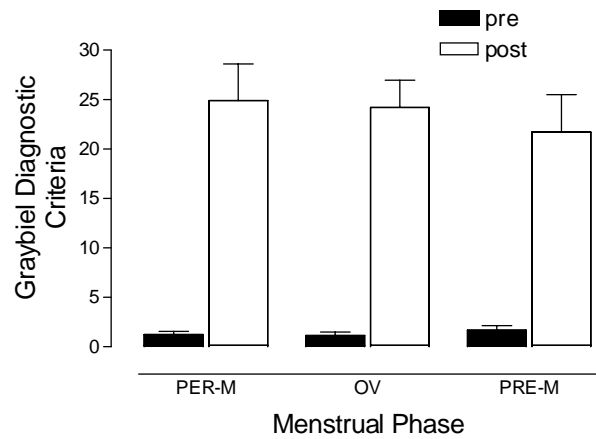


Fig. 4. Graybiel diagnostic criteria score before and after Coriolis stimulation (Error bars represent SEM).

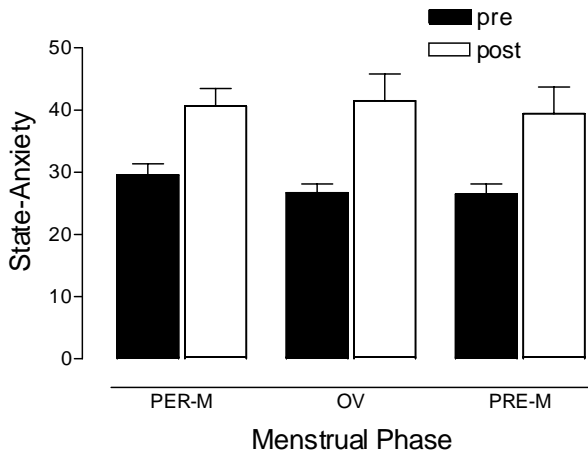


Fig. 3. State-Anxiety score before and after Coriolis stimulation (Error bars represent SEM).

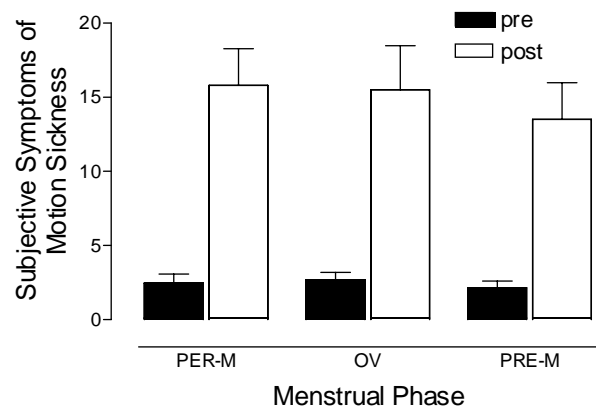


Fig. 5. Subjective symptoms of motion sickness score before and after Coriolis stimulation (Error bars represent SEM).

teria, and subjective evaluation of motions sickness symptoms. The results were similar for each analysis. Differences between pre-Coriolis and post-Coriolis evaluations were evident for state anxiety at $p < 0.001(x^2 = 31.60714, df = 13)$ Graybiel Diagnostic Criteria at $p < 0.001(x^2 = 49.91, df = 13)$ and subjective evaluation of motions sickness symptoms at $p < 0.001(x^2 = 42.54, df = 13)$. Using Wilcoxon signed ranked tests significant contrasts were found between pre-Coriolis and post-Coriolis evaluation in each phase for each symptom measurement ($p < 0.004$) with the exception of the pre-Coriolis and post-Coriolis state anxiety difference during the ovulation phase where $p = 0.015$. However, no significant contrast effects were observed within pre-Coriolis and within post-Coriolis evaluations that is, there were no significant difference across all three menstrual phases.

3.3. Head movements

Subject susceptibility as measured by the Coriolis test can be expressed in terms of total additional head movements made or the percentage change in head movements performed between experimental conditions. The utility of this approach in evaluating changes in motion sickness resistance or total Coriolis stress endured by the subject in reaching a predetermined level of symptomatology was shown by Miller and Graybiel [25,26] where Coriolis stress is directly proportional to the number of head movements made. A one way repeated measures ANOVA was performed on the number of head movements tolerated in each of the three menstrual phases. No significant effects were observed ($F(1, 24) = 1.54, p = 0.23, \text{power estimate is } 0.8$ at an α level of 0.05). We were unable to identify

any difference in the number of head movements tolerated among the three phases of the menstrual cycle.

4. Discussion

It should be noted that most of the studies that have investigated motion sickness susceptibility differences in males and females relied on either questionnaires (measurement of sickness during or immediately after stimulation) or past motion sickness history (measurement of susceptibility as a characteristic of an individual over past experiences). Although proportionately more females than males report motion sickness, whether this reported sex difference in susceptibility is based on physiological or psychological differences, or on a combination of the two, is unknown. It has been proposed that it is merely a reflection of a socialization process in which it is more acceptable for women to report illness, including motion sickness. Questionnaires and self-reports may themselves be subject to a sex bias. There is no evidence that women show a greater sensory response to nauseogenic motion stimulus [31] and similarly there is no reason to assume that women adapt less readily than men [32]. Among women, there is no physiological reason to believe that their vestibular system sensitivity is different between the three menstrual phases. However, endocrine changes may well affect the sensory and associated autonomic responses. It was reported that women are more likely to be sick during the time of menstruation [15,33] and that there was a reduction of motion sickness around the time of ovulation [15]. However, our results do not support the above findings. Both physiological and psychophysical measurement of the severity of sickness induced by Coriolis cross-coupling across the three menstrual phases did not reach significance. Two of the three subjects who withdrew from the study due to severity of sickness in their first trial were tested during their ovulation phase. The lack of effect of menstrual phases on sickness susceptibility remains the same when the four subjects who were taking oral contraceptives were removed from the analysis.

Is there a common denominator in endocrine response as a result of Coriolis-induced sickness and hormones that are released during different phases of the menstrual cycle? Our review of the literature fails to find a relation between hormones released during motion sickness and during the different phases of the menstrual cycle. It has been reported that stressful Coriolis-induced sickness in the laboratory leads to the

release of arginine vasopressin (AVP), cortisol (CORT), prolactin (PRL), growth hormone (GH), adrenocorticotrophic hormone (ACTH), norepinephrine (NE) and epinephrine (EPI) [8,18,38]. Among the hormones described above, the release of arginine vasopressin (AVP) was the earliest and most pronounced endocrine marker for motion sickness [8]. Antidiuresis has long been reported as a measure of motion sickness [35]. Specific and highly potent vasopressinergic antagonists were found to be effective in abolishing emesis and the development of significant symptomatology of motion sickness in the squirrel monkey [4]. The secretion of vasopressin varies during normal menstrual cycle with the highest plasma concentrations around the time of ovulation and the lowest at the onset of menstruation [11]. Based on this evidence it would appear that the susceptibility to motion sickness might be higher during ovulation; however, a survey conducted during a yacht race suggested that female sailors were least sensitive to seasickness during ovulation [15].

The ability of AVP to release ACTH is well known [3]. In considering the possibility that certain hormonal responses to stressful motion might serve an adaptive role, individual susceptibility to stressful Coriolis stimulation was found to correlate with normal resting levels of ACTH, and with the responsiveness of ACTH, epinephrine, and norepinephrine [19,20]. Individuals with low susceptibility were found to possess higher endogenous levels of ACTH. Similarly, subjects with low susceptibility reportedly display greater elevations of epinephrine and norepinephrine after exposure to provocative motion than do highly susceptible subjects. On the other hand, basal concentrations of ACTH [12] and norepinephrine [10] were found to be similar across all three menstrual cycle phases.

Similarly, there appears to be a lack of correlation between prolactin and growth hormone release during motion sickness and the cyclical release of these hormones during different menstrual phases. Prolactin and growth hormone were found to increase by 400% and 115% within motion sickness affected subjects and to a lesser degree 120% and 40% increases respectively in the control group [7]. The secretion of prolactin is minimal during permenstrual phase with maximum release during pre-ovulatory and an intermediate release during the premenstrual phase [2]. On the other hand, the mean serum Growth hormone concentration in late follicular phase women was higher than observed in early follicular phase women. The GH concentration for mid-luteal phase women was intermediate between but not statistically different from that observed in the early and late follicular phase women [9].

It is difficult to compare observations made under operational environments and experimental results from a controlled laboratory study due to the different stimuli involved. The fluctuations in sex hormones that occur during menstrual cycle have an effect on electrolyte and water metabolism that may trigger pathophysiological changes in the central nervous system. However, there is no relation between the different levels of hormones being released, especially AVP, during motion sickness and during different phases of the menstrual period. In summary, our study suggested that susceptibility to acute motion sickness is not influenced by different menstrual phases.

References

- [1] K. Abe, M. Amatori and S. Kajiyama, Genetical aspects of susceptibility to motion sickness and frost bite, *Human Heredity* **20** (1970), 507–516.
- [2] J. Buvat and M. Buvat-Herbaut, Changes in the Gonadotrophins, in the prolactin and in the sexual steroid levels throughout the normal menstrual cycle, *J Gynecol Obstet Biol Reprod (Paris)* **10** (1981), 99–108.
- [3] D.E. Carlson, A. Dornhorst, S.M. Self, A.G. Robinson and D.S. Gann, Vasopressin-dependent and independent control of the release of adrenocorticotropin, *Endocrinology* **7** (1980), 714–718.
- [4] B. Cheung, R.L. Kohl, K.E. Money and L.B. Kinter, Etiologic significance of arginine vasopressin in motion sickness, *J Clin Pharmacol* **34** (1994), 664–670.
- [5] B. Cheung and K. Hofer, Coriolis-induced cutaneous blood flow increase in the forearm and calf, *Brain Res Bulletin* **54** (2001), 609–618.
- [6] T. Dobie, D. McBride, T. Dobie Jr. and J. May, The effects of age and sex on susceptibility to motion sickness, *Aviat Space Environ Med* **72** (2001), 13–20.
- [7] C. Drummer, H. Stromeyer, R.L. Riepl, A. Konig, F. Strollo, R.E. Lang, H. Maass, L. Rocker and R. Gerzer, Hormonal changes after parabolic flight: implications on the development of motion sickness, *Aviat Space Environ Med* **61** (1990), 821–828.
- [8] T. Eversmann, M. Gottsman, E. Uhlich, G. Ulbrecht, K. Von Werder and P.C. Scriba, Increased secretion of growth hormone, prolactin, antidiuretic hormone, and cortisol induced by the stress of motion sickness, *Aviat Space Environ Med* **49** (1978), 53–57.
- [9] A.C. Faria, L.M. Berkenstein, R.A. Booth Jr, V.A. Vaccaro, C.M. Asplin, J.D. Veldhuis, M.O. Thorner and W.S. Evans, Pulsatile growth hormone release in normal women during the menstrual cycle, *Clin Endocrinol (Oxf)* **36** (1992), 591–596.
- [10] R.R. Freeman and R. Girges, Effects of menstrual cycle and race on peripheral vascular alpha-adrenergic responsiveness, *Hypertension* **35** (2000), 795–799.
- [11] M.L. Forsling, P. Stromberg and M. Akerlund, Effect of ovarian steroids on vasopressin secretion, *J Endocrinology* **95** (1982), 147–151.
- [12] E.A. Galliven, A. Singh, D. Michelson, S. Bna, W. Gold and P.A. Deuster, Hormonal and metabolic responses to exercise across time of day and menstrual cycle phase, *J App Physiol* **83** (1997), 1822–1831.
- [13] J.F. Golding and M. Kerguelen, Comparison of the nauseogenic potential of low frequency vertical versus horizontal linear oscillation, *Aviat Space Environ Med* **63** (1992), 491–497.
- [14] A. Graybiel, C.D. Wood, E.F. Miller and D.B. Cramer, Diagnostic criteria for grading the severity of acute motion sickness, *Aerosp Med* **39** (1968), 453–455.
- [15] E. Grunfeld and M.A. Gresty, Relationship between motion sickness, migraine and menstruation in crew members of a “round the world” yacht race, *Brain Res Bulletin* **47** (1998), 433–436.
- [16] M.D. Jakerst, M. Gatto, R. Fazio, P.J. Gianaros, R.M. Stern and K.L. Koch, Effects of gender of subjects and experimenter on susceptibility to motion sickness, *Aviat Space Environ Med* **70** (1999), 962–965.
- [17] M.H. Jones, Influence of menstruation upon nausea induced from the vestibule, *Amer. of Psychol* **58** (1945), 496–509.
- [18] R.L. Kohl, Endocrine correlates of susceptibility to motion sickness, *Aviat Space Environ Med* **56** (1985), 1158–1165.
- [19] R.L. Kohl, C. Leach, R.L. Homick and F.T. LaRochelle, Motion sickness susceptibility related to ACTH, ADH, and TSH, *Physiologist* **26** (1983), S117–118.
- [20] R.L. Kohl and S. MacDonald, New pharmacologic approaches to the prevention of space/motion sickness, *J Clin Pharmacol* **31** (1991), 934–946.
- [21] A. Lawther and M.J. Griffin, The motion of a ship at sea and the consequent motion sickness amongst passengers, *Ergonomics* **29** (1986), 535–552.
- [22] A. Lawther and M.J. Griffin, A survey of the occurrence of motion sickness amongst passengers at sea, *Aviat Space Environ Med* **59** (1988), 399–406.
- [23] J.M. Lentz and W.E. Collins, Motion sickness susceptibility and related behavioral characteristics in men and women, *Aviat Space Environ Med* **48** (1977), 316–322.
- [24] G.D. Levy and M.H. Rapaport, Transderm scopolamine efficacy related to time of application prior to the onset of motion, *Aviat Space Environ Med* **56** (1985), 591–593.
- [25] E.F.2d. Miller and A. Graybiel, A provocative test for grading susceptibility to motion sickness yielding a single numerical score, *Acta Otolaryngol Suppl* **274** (1970), 1–20.
- [26] E.F.2d. Miller and A. Graybiel, A comparison of five levels of motion sickness severity as the basis for grading susceptibility, *Aerosp Med* **45** (1974), 602–609.
- [27] J.H. Nieuwenhijzen, *Experimental investigation of seasickness*, University of Utrecht, 1958, A64890.
- [28] J.M. Ordy and K.R. Brizzee, Motion sickness in the squirrel monkey, *Aviat Space Environ Med* **51** (1980), 215–223.
- [29] G. Pagano, P. Geraci and M.C. Lucifora, Prediction and detection of ovulation relationship between women’s observations of cervical mucus changes, thermic and echographic of ovulation, *Acta Eur Fertil* **19**(5) (1988), 269–271.
- [30] A.H. Park and S. Hu, Gender differences in motion sickness history and susceptibility to optokinetic rotation-induced motion sickness, *Aviat Space Environ Med* **70** (1999), 1077–1080.
- [31] J.T. Reason, Relations between motion sickness susceptibility, the spiral after-effect and loudness estimation, *Br J Psychol* **59** (1968), 385–393.
- [32] J.T. Reason and J.J. Brand, *Motion sickness*, Academic Press London, New York, 1975.
- [33] R.S. Schwab, The nonlabyrinthine causes of motion sickness, *Jt Intern Record Med* **167** (1954), 631–637.
- [34] C.D. Spielberger, *Manual For The State-Trait Anxiety Inventory (Form Y)*, Consulting Psychologists Press, Inc. Palo Alto, C.A. 1983.

- [35] N.B.G. Taylor, J. Hunter and W.H. Johnson, Antidiuresis as a measurement of laboratory induced motion sickness, *Can J BiochemPhysiol* **35** (1957), 1017–1027.
- [36] M. Turner, *A survey of motion sickness in road coaches*, Proceedings of the United Kingdom informal group meeting on human response to vibration, the Army Personnel Research Establishment, Ministry of Defence, Farnborough, 20–22 Sept. 1993.
- [37] M. Turner and M.J. Griffin, Motion sickness incidence during a round-the-world yacht race, *Aviat Space Environ Med* **66** (1995), 849–856.
- [38] W. von Restrorff, F.G. Krammling, G.K. Stallia and K. von Werder, Hormonal responses to Coriolis stress, *Reprints from Aerospace Medical Association* **143** (1984).