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Effects of Menstrual Cycle Phase on Resting Heart Rate in Healthy Women

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ABSTRACT

Teixeira ALS, Fernandes Júnior W, Moraes EM, Alves HB, Damasceno VO, Dias MRC. Effects of Menstrual Cycle Phase on Resting Heart Rate in Healthy Women. **JEPonline** 2012;15(4):47-54. The menstrual cycle (MC) is primarily responsible for changes in female physiology. The aim of this study was to assess the influence of different phases of the MC on resting heart rate (RHR) in healthy women with and without the use of oral contraceptive. Forty-four university women with age range from 18 to 35 yrs were divided into two groups. The Control Group (CG) who were oral contraceptive users (n=24) and the Experimental Group (EG) who were not contraceptive users (n=20). The participants made three visits to the laboratory; one in each phase of the cycle (follicular, ovulatory, and luteal). The RHR was evaluated during a 10-min period in the supine position, and was considered the lowest value recorded by the heart rate monitor. No significant differences ($P>0.05$) were obtained between the three phases of the MC within-groups and between-groups. The results suggest that the different phases of the MC did not alter the RHR in healthy university women independently of the use of oral contraceptive.

Key Words: Estrogen, Progesterone, Oral Contraceptive, Autonomic Nervous System

INTRODUCTION

The menstrual cycle (MC) has a cyclical nature that lasts approximately 28 days, and has been identified as a major factor in changes in female physiology (16). Changes in hormone concentrations secreted by the hypothalamus-pituitary-gonadal axis, particularly estrogen and progesterone, determine the three phases that constitute the MC: follicular, ovulatory, and luteal (37). The influence of the different phases of the MC has been studied on factors such as the risk of injury (5), flexibility (4), anaerobic power (35), muscle strength (25), sports performance (28), and cardiovascular mechanisms (2,13,20).

Heart rate (HR) is modulated by the autonomic nervous system through its sympathetic and vagal branches that secrete the hormones norepinephrine and acetylcholine, respectively. The HR control and response follows a well-defined pattern both at rest and during exercise (7,27,29). This autonomic control is an important indicator of health, because adults with autonomic dysfunction have higher all-cause and cardiovascular mortality rates (1,6,8,10,12,14,18,22-23,31,36). Resting heart rate (RHR) is frequently used during the evaluation of the functional condition of a person. The resting values determine have a direct relation to a person's intensity of aerobic training (19). In addition, the RHR is an independent predictor of mortality (10,12,18,31).

Intrinsic and extrinsic factors can modify the behavior of RHR, including body temperature (9,17). During the ovulatory phase of the MC, there is an increase in body temperature (15), which could change the values of RHR. In addition, the influence of oral contraceptive in this context requires further study. Thus, the purpose of this study was to assess the influence of different phases of the MC on RHR in healthy women with and without the use of oral contraceptive. It was hypothesized that RHR is greater during the ovulatory phase of the cycle.

METHODS

Subjects

A total of 44 healthy university women, age range from 18 to 35 years (23.4 ± 4.7 yrs) were enrolled. All participants reported they were knowledgeable about their MC, which occurred regularly between 25 and 40 days (35). Women who reported a disturbance of the endocrine system, the use of beta-blockers, who were smokers or who were ingesting substances that may interfere with the experimental procedures were excluded from this sample. The subjects were also asked to refrain from drinking coffee and alcohol and engaging in physical exercise 8 and 24 hrs, respectively, prior to the assessments. All evaluations were done in the morning. All subjects read and signed an informed consent form approved by the local institutional research committee.

Procedures

Anthropometry

The participants' body weight was assessed using a digital weighing scale (Fillizola®, Brazil). Height was determined using a stadiometer with mm precision (Sanny®, Brazil). Then, body density was estimated using the equation of Jackson et al. (19) through the sum of skinfolds (triceps, suprailiac, and thigh), and converted to fat percentage by the formula of Siri (32).

Menstrual Cycle Phases

The participants were divided into a Control Group (CG) who were oral contraceptive users (n=24) and an Experimental Group (EG) who were non-contraceptive users (n=20). To define the phase, in relation to the day of the cycle, the criterion proposed by Wojtys et al. (37) was used where three phases are considered (follicular, ovulatory, and luteal). The follicular phase is between the first and

the 9th day of the cycle. The ovulatory phase is between the 10th and the 14th day, and the luteal phase is from the 15th day until the end of the cycle.

Resting Heart Rate

The participants' RHR was identified as the lowest value recorded by the HR monitor (FS2, Polar Electro OY®, Finland) during 10-min in the supine position.

Statistical Analyses

All data are presented as mean \pm standard deviation (SD). The Komolgorov-Sminorf normality test and a homoscedasticity test (Levene`s test) were used to analyze the normal distribution of the data. All variables present a normal distribution and homoscedasticity. The independent *t*-test was used to verify the difference between the anthropometric variables between the groups. A two (CG and EG) by three (menstrual phases) ANOVA was used to analyze the difference between groups, followed by Tukey *post hoc* test when necessary. The significance level adopted was $P=0.05$ for all tests. The Statistical software version 6.0 (Statsoft, Inc., Tulsa, OK) was used in all analyses.

RESULTS

Table 1 shows the characteristics of the participants. No significant differences were found between the CG and the EG for all variables ($P>0.05$). In regards to RHR, no significant differences ($P>0.05$) were found between the groups in all menstrual phases (Table 2). In addition, there were no significant differences ($P>0.05$) among the three different phases of the cycle for both the CG and the EG (Figure 1).

Table 1. Descriptive Data for Each Group.

Variables	CG (n = 24)	EG (n = 20)	P
Age (yrs)	23.5 \pm 4.5	23.1 \pm 5.1	ns
Weight (kg)	55.9 \pm 6.9	61.0 \pm 12.5	ns
Height (cm)	162.4 \pm 5.5	163.2 \pm 4.7	ns
BMI (kg/m²)	21.1 \pm 1.5	22.8 \pm 3.7	ns
Fat (%)	28.8 \pm 4.3	32.1 \pm 7.4	ns

Values presents in mean \pm SD. BMI: Body Mass Index; CG: Control Group; EG: Experimental Group; ns: not significant.

DISCUSSION

The MC is characterized by rhythmic variation in the secretion of female hormones and corresponds to changes in the sexual organs and other physiological responses. The purpose of this study was to determine the influence of the MC on the RHR of healthy university students with and without the use of oral contraceptives. The hypothesis was that the RHR would be greater in the ovulatory phase but, contrary to this thinking, the findings showed that the MC did not change RHR.

Table 2. Values of the RHR (bpm) during the Menstrual Phases Between-Groups.

Phases	CG (n=24)	EG (n=20)	P
Follicular	69.9 ± 6.6	68.8 ± 10.3	ns
Ovulatory	71.2 ± 7.8	69.7 ± 8.7	ns
Luteal	71.6 ± 8.2	70.1 ± 6.9	ns

Values presents in mean ± SD. CG: Control Group; EG: Experimental Group; ns: not significant.

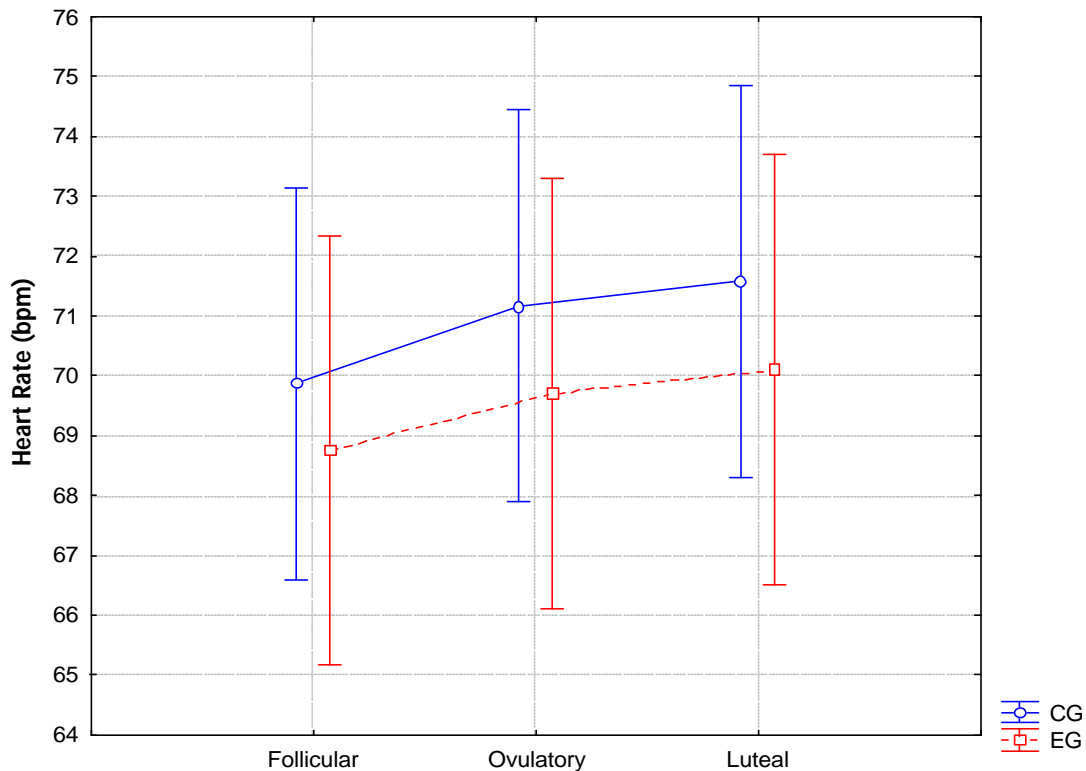


Figure 1. RHR values in the CG and the EG during the MC.

Effects of Oral Contraceptive on Cyclical Hormone Variations

The MC is divided into three different phases according to hormone variations. The follicular phase is characterized by low concentrations of female hormones. It is this phase that the development of the ovarian follicle occurs. The ovulatory phase is between the 10th and the 14th day with the peak of luteinizing (LH) and estrogen hormones. After ovulation, the luteal phase begins with particularly a high concentration of progesterone and, to a lesser extent, estrogen (16).

Progesterone plays an important role in making the uterus receptive to pregnancy. It also has the side effect of raising the woman's basal body temperature. Estrogen promotes the proliferation and growth of specific cells in the body responsible for the development of most female sexual characteristics. The use of oral contraceptives is a strategy often used by women to prevent pregnancy. The pills provide a constant estrogen dosage across the MC, thus attenuating the cyclical estrogen and

luteinizing hormone (LH; a hormone produced by the anterior pituitary gland) peak noted at ovulation (3).

Influence of Menstrual Cycle on Cardiac Autonomic Control

Previous studies have shown that the MC can modify the cardiac autonomic control. Sato et al. (30) observed through power spectral analysis of HR variability that sympathetic nervous activities are predominant in the luteal phase when compared to the follicular phase. However, it didn't change the RHR. In this context, Tanaka et al. (34) demonstrated that baroreflex control of HR is altered during the regular MC in healthy women, but it had no effect on RHR. These two studies are in agreement with our results.

In contrast, Leicht et al. (24) showed that higher values of RHR occur during the ovulatory phase of the cycle in comparison to the follicular and luteal phases. In their study, the authors found a significant correlation between peak estrogen levels and cardiac vagal activity. In addition, Bai et al. (2) demonstrated through HR variability that the high-frequency (HF) components decreased from the follicular phase to the luteal phase while the low-frequency (LF) components, the LF/HF ratio, and RHR increased. According to the authors, the follicular phase is characterized by enhanced vagal activity and the luteal phase is characterized by enhanced sympathetic activity.

This autonomic balance has a prognostic value because adults with autonomic dysfunction have higher all-cause and cardiovascular mortality rates (1,6,8,10,12,14,18,22-23,31,36). In addition, the RHR is an independent predictor of mortality (10,12,18,31). Interestingly, in accordance with the results of the present study, it appears that hormonal fluctuations resulting from the MC and the increase in body temperature at ovulation (15) are not sufficient to modify the autonomic balance represented by RHR.

Limitations, Practical Implications, and Future Investigations

It is important to note the limitation of this study. For example, the menstrual phases were based on self-report data from the participants. The estrogen/progesterone ratio in the urine (11,35), saliva (33) or blood (26) is the gold standard to identify the phases. However, due to operational difficulty and because the MC presents hormonal variations with well-defined standards, many studies are carried out without direct analysis of hormonal levels.

The findings in the present study indicate that the MC phases were not associated with RHR. The literature shows that high values of RHR increase the risk of mortality. Another practical application of RHR values is during a physical exercise prescription. The RHR is used in the HR reserve equation by Karvonen et al. (21). Therefore, any phenomenon that is likely to influence cardiovascular control is an important research question that should be elucidated.

Future research should be conducted with a more definitive determination of the menstrual phases, and the inclusion of other factors such as physical activity level and clinical status for future comparison of results and better clarification on this issue.

CONCLUSION

We conclude that the different phases of the MC do not modify the RHR in healthy university women independent of the use of oral contraceptives.

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REFERENCES

1. Ahmadi-Kashani M, Kessler DJ, Day J, Bunch TJ, Stolen KQ, Brown S, Sbaity S, Olshansky B. Heart rate predicts outcomes in an implantable cardioverter-defibrillator population. *Circulation*. 2009;120:2040-2045.
2. Bai X, Li J, Zhou L, Li X. Influence of the menstrual cycle on nonlinear properties of heart rate variability in young women. *Am J Physiol Heart Circ Physiol*. 2009;297:765-774.
3. Bell DR, Blackburn JT, Ondrak KS, Hackney AC, Hudson JD, Norcross MF, Padua DA. The effects of oral contraceptive use on muscle stiffness across the menstrual cycle. *Clin J Sport Med*. 2011;21:467-473.
4. Bell DR, Myrick MP, Blackburn JT, Shultz SJ, Guskiewicz KM, Padua DA. The effect of menstrual-cycle phase on hamstring extensibility and muscle stiffness. *J Sport Rehabil*. 2009;18:553-563.
5. Beynon BD, Johnson RJ, Braun S, Sargent M, Bernstein IM, Skelly JM, Vacek PM. The relationship between menstrual cycle phase and anterior cruciate ligament injury: A case-control study of recreational alpine skiers. *Am J Sports Med*. 2006;34:757-764.
6. Bigger Junior JT, Fleiss JL, Steinman RC, Rolnitzky LM, Kleiger RE, Rottman JN. Frequency domain measures of heart period variability and mortality after myocardial infarction. *Circulation*. 1992;85:164-171.
7. Boyett MR. 'And the beat goes on' The cardiac conduction system: The wiring system of the heart. *Exp Physiol*. 2009;94:1035-1049.
8. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MD. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med*. 1999;341:1351-1357.
9. Davies P, Maconochie I. The relationship between body temperature, heart rate and respiratory rate in children. *Emerg Med J*. 2009;26:641-643.
10. Engel G, Cho S, Ghayoumi A, Yamazaki T, Chun S, Fearon WF, Froelicher VF. Prognostic significance of PVCs and resting heart rate. *Ann Noninvasive Electrocardiol*. 2007;12:121-129.
11. Esformes JI, Norman F, Sigley J, Birch KM. The influence of menstrual cycle phase upon postexercise hypotension. *Med Sci Sports Exerc*. 2006;38:484-491.

12. Feldman D, Elton TS, Menachemi DM, Wexler RK. Heart rate control with adrenergic blockade: Clinical outcomes in cardiovascular medicine. **Vasc Health Risk Manag.** 2010;6:387-397.
13. Fu Q, VanGundy TB, Shibata S, Auchus RJ, Williams GH, Levine BD. Menstrual cycle affects renal-adrenal and hemodynamic responses during prolonged standing in the postural orthostatic tachycardia syndrome. **Hypertension.** 2010;56:82-90.
14. Galinier M, Pathak A, Fourcade J, Androdias C, Curnier D, Varnous S, Boveda S, Massabuau P, Fauvel M, Senard JM, Bounhoure JP. Depressed low frequency power of heart rate variability as an independent predictor of sudden death in chronic heart failure. **Eur Heart J.** 2000;21:475-482.
15. Garcia AMC, Lacerda MG, Fonseca IAT, Reis FM, Rodrigues LOC, Silami-Garcia E. Luteal phase of the menstrual cycle increases sweating rate during exercise. **Braz J Med Biol Res.** 2006;39:1255-1261.
16. Guyton AC, Hall JE. **Textbook of Medical Physiology.** 12th edition. Philadelphia: Saunders Company, 2011.
17. Hanna CM, Greenes DS. How much tachycardia in infants can be attributed to fever? **Ann Emerg Med.** 2004;43:699-705.
18. Hsia J, Larson JC, Ockene JK, Sarto GE, Allison MA, Hendrix SL, Robinson JG, LaCroix AZ, Manson JE. Resting heart rate as a low tech predictor of coronary events in women: Prospective cohort study. **BMJ.** 2009;338:577-580.
19. Jackson AS, Pollock ML, Ward A. Generalized equations for predicting body density of women. **Med Sci Sports Exerc.** 1980;12:175-181.
20. Karabag T, Hanci V, Aydin M, Dogan SM, Turan IO, Yildirim N, Gudul NE. Influence of menstrual cycle on P wave dispersion. **Int Heart J.** 2011;52:23-26.
21. Karvonen MJ, Kentala E, Mustala O. The effects of training on heart rate. A longitudinal study. **Ann Med Exp Biol Fenn.** 1957;35:307-315.
22. Lauer MS. Autonomic function and prognosis. **Cleve Clin J Med.** 2009;76:18-22.
23. Leeper NJ, Dewey FE, Ashley EA, Sandri M, Tan SY, Hadley D, Myers J, Froelicher V. Prognostic value of heart rate increase at onset exercise testing. **Circulation.** 2007;115:468-474.
24. Leicht AS, Hirning DA, Allen GD. Heart rate variability and endogenous sex hormones during the menstrual cycle in young women. **Exp Physiol.** 2003;88:441-446.
25. Loureiro S, Dias I, Sales D, Alessi I, Simão R, Fermino RC. Effect of different phases of the menstrual cycle on the performance of muscular strength in 10RM. **Rev Bras Med Esporte.** 2011;17:22-25.

26. Ludwig M, Klein HH, Diedrich K, Ortmann O. Serum leptin concentrations throughout the menstrual cycle. **Arch Gynecol Obstet.** 2000;263:99-101.
27. Oliveira TP, Ferreira RB, Mattos RA, Silva JP, Lima JRP. Influence of water intake on post-exercise heart rate variability recovery. **JEPonline.** 2011;14:97-105.
28. Oosthuysen T, Bosch AN. The effect of the menstrual cycle on exercise metabolism. Implications for exercise performance in eumenorrhoeic women. **Sports Med.** 2010;40:207-227.
29. Ricardo DR, Almeida MB, Franklin BA, Araújo CGS. Initial and final exercise heart rate transients. Influence of gender, aerobic fitness, and clinical status. **Chest.** 2005;127:318-327.
30. Sato N, Miyake S, Akatsu J, Kumashiro M. Power spectral analysis of heart rate variability in healthy young women during the normal menstrual cycle. **Psychosom Med.** 1995;57:331-335.
31. Seccareccia F, Pannozzo F, Dima F, Minoprio A, Menditto A, Lo Noce C, Giampaoli S. Heart rate as a predictor of mortality: The MATISS Project. **Am J Public Health.** 2001;91:1258-1263.
32. Siri WE. Body composition from fluid spaces and density: Analysis of methods. In Brozek J, Henschel A. **Techniques for measuring body composition.** Washington: National Academy of Science, 1961.
33. Stanford KI, Mickleborough TD, Ray S, Lindley MR, Koceja DM, Stager JM. Influence of menstrual cycle phase on pulmonary function in asthmatic athletes. **Eur J Appl Physiol.** 2006;96:703-710.
34. Tanaka M, Sato M, Umehara S, Nishikawa T. Influence of menstrual cycle on baroreflex control of heart rate: Comparison with male volunteers. **Am J Physiol Regul Integr Comp Physiol.** 2003;285:1091-1097.
35. Tsampoukos A, Peckham EA, James R, Nevill. Effect of menstrual cycle phase on sprinting performance. **Eur J Appl Physiol.** 2010;109:659-667.
36. Tsuji H, Larson MG, Venditti Junior FJ, Manders ES, Evans JC, Feldman CL, Levy D. Impact of reduced heart rate variability on risk for cardiac events. The Framingham heart study. **Circulation.** 1996;94:2850-2855.
37. Wojtys EM, Huston LJ, Lindenfeld TN, Hewett TE, Greenfield MLVH. Association between the menstrual cycle and anterior cruciate ligament injuries in female athletes. **Am J Sports Med.** 1998;26:614-619.

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