National Board of Examinations - Journal of Medical Sciences
Volume 3, Issue 12, Pages 1562–1573, December 2025
DOI 10.61770/NBEJMS.2025.v03.i12.013

ORIGINAL ARTICLE

Gender Differences in Physiological Cardiovascular Reactivity to Cold Pressor Stress Test: A Cross-Sectional Study

Hridya Suresh,[1,*] Lakshmi T,[2] Shashikala L[3] and Hemalatha N R[4]
[1]*Postgraduate Student, Department of Physiology, Mandya Institute of Medical Sciences, Mandya – 571401*
[2]*Assistant Professor, Department of Physiology, Mandya Institute of Medical Sciences, Mandya – 571401*
[3]*Associate Professor, Department of Physiology, Mandya Institute of Medical Sciences, Mandya – 571401*
[4]*Professor and Head, Department of Physiology, Mandya Institute of Medical Sciences, Mandya – 571401*

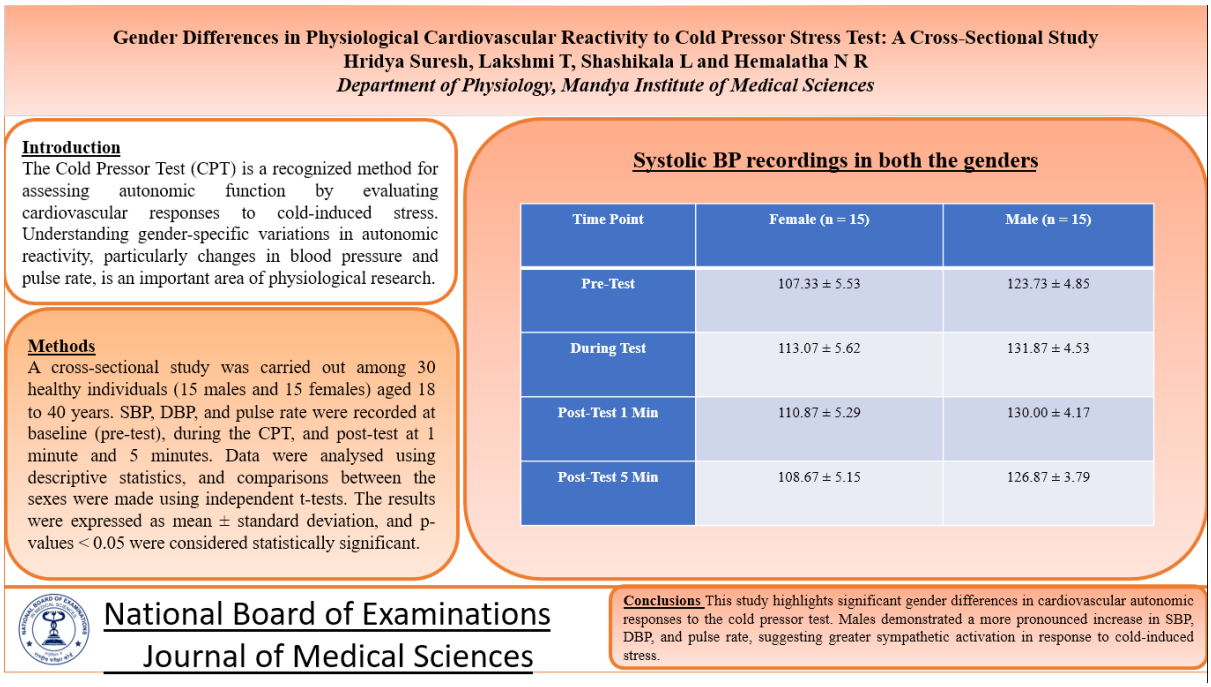
Accepted: 22-November-2025 / Published Online: 4-December-2025

Abstract
Background/Introduction: The Cold Pressor Test (CPT) is a recognized method for assessing autonomic function by evaluating cardiovascular responses to cold-induced stress. Understanding gender-specific variations in autonomic reactivity, particularly changes in blood pressure and pulse rate, is an important area of physiological research. **Aims and Objective:** This study aimed to examine and compare the physiological responses to the cold pressor test between healthy adult males and females, focusing on systolic and diastolic blood pressure (SBP and DBP), as well as pulse rate at various time intervals. **Materials and Methods:** A cross-sectional study was carried out among 30 healthy individuals (15 males and 15 females) aged 18 to 40 years. SBP, DBP, and pulse rate were recorded at baseline (pre-test), during the CPT, and post-test at 1 minute and 5 minutes. Data were analysed using descriptive statistics, and comparisons between the sexes were made using independent t-tests. The results were expressed as mean ± standard deviation, and p-values < 0.05 were considered statistically significant. **Results:** Male participants exhibited significantly higher SBP and DBP compared to females at all time points. For example, during the test, males showed an SBP of 131.87 ± 4.53 mmHg versus 113.07 ± 5.62 mmHg in females ($p < 0.001$). Diastolic pressures were similarly elevated ($p < 0.001$). Pulse rate was also higher in males, especially during the test (90.27 ± 3.89 vs. 87.00 ± 6.59; $p = 0.048$). Although values declined toward baseline at 5 minutes post-test, males consistently maintained higher readings. **Conclusion:** This study highlights significant gender differences in cardiovascular autonomic responses to the cold pressor test. Males demonstrated a more pronounced increase in SBP, DBP, and pulse rate, suggesting greater sympathetic activation in response to cold-induced stress. These findings underscore the need to consider sex-specific variations when interpreting autonomic function tests and probability of higher cardiovascular risk in males and lower risk in females due to the role of female hormones.

Keywords: Autonomic Stress, Cold pressor test (CPT), Left ventricular function, Cold stressor, sympathetic nervous system

*Corresponding Author: Hridya Suresh
Email: hridyasuresh.ask@gmail.com

Graphical Abstract



Introduction

The cold pressor test (CPT) is a well-established method for evaluating sympathetic nervous system reactivity. In this procedure, an individual immerses a hand or foot into cold water maintained at 3–5°C for 1–3 minutes while blood pressure (BP) and heart rate are continuously recorded [1]. The intense cold stimulus activates cutaneous afferents and triggers a reflex increase in sympathetic outflow, producing a rise in BP that is independent of baroreflex mechanisms. Because of this direct sympathetic activation, the CPT has been widely used in both clinical and research settings to assess autonomic integrity and vasomotor responsiveness [1].

In everyday life, individuals are exposed to multiple physical and psychological stressors, all of which can influence cardiovascular regulation. Sympathetic activation during stress elevates BP and heart rate, and exaggerated responses have been repeatedly associated with a higher risk of developing hypertension later in life [2]. The CPT offers a standardized, non-invasive way to quantify this reactivity, making it a useful screening tool for identifying normotensive individuals who may be predisposed to future cardiovascular disease [3].

Sex-based differences in cardiovascular physiology are well documented. Variations in endothelial function, vascular tone, and autonomic regulation contribute to the differing patterns of hypertension and coronary artery disease observed between men and women [4]. Estrogen-mediated endothelial protection has been proposed as an important factor underlying the lower incidence of cardiovascular disease in premenopausal women [5,6]. However, the extent to which these physiological differences translate into divergent CPT responses remains incompletely understood.

Despite the widespread use of CPT internationally, there is limited data from

Indian populations, and even fewer studies have directly compared male and female responses using standardized protocols. Understanding whether sex influences BP and heart rate reactivity during CPT may help identify early deviations in autonomic or vascular function, contributing to more refined cardiovascular risk prediction.

Therefore, the present study was undertaken to compare the cardiovascular responses to the cold pressor test between healthy young adult males and females, and to explore whether sex-related differences in sympathetic reactivity can be identified through this simple, non-invasive assessment.

Materials and Method of Study

Ethical committee approval was received from Institutional Ethics Committee – Mandya Institute of Medical Sciences, Mandya on 29.01.2025, and the data was collected as mentioned below :

Study Participants

Healthy young adults aged 18–35 years were recruited for the study. Eligible participants were normotensive (resting BP <140/90 mmHg), nonsmokers, non-alcohol users, and provided written informed consent.

Inclusion Criteria

- Age 18–35 years
- Resting BP <140/90 mmHg
- Nonsmokers and non-alcohol consumers
- Voluntary consent to participate

Exclusion Criteria

Participants with a history of:

- Cardiovascular disease
- Endocrine disorders
- Renal disease
- Neurological dysfunction
- Hypertension
- Cold intolerance or sweating disorders were excluded from the study.

Sample Recruitment and Representativeness

Participants were recruited from attendants of various departments in the medical college and from patient attendants in outpatient areas. Because this sampling was based on convenience rather than random selection, the study population may not fully represent the broader young adult population. This limitation is acknowledged, as the findings may not be generalizable beyond similar institutional settings.

Sample Size Considerations

A total of 30 participants (15 males and 15 females) were included. No formal power analysis was performed before recruitment.

Pre-Test Preparation

Participants were instructed to:

- Avoid caffeine-containing beverages and exercise for at least 4 hours prior to testing
- Consume only a light breakfast on the day of assessment

All participants rested quietly for 10 minutes before measurements were taken. The procedure was explained in detail before initiating the test.

Sociodemographic and Health Information

Baseline information was obtained using a semi-structured questionnaire, including:

- Name
- Age
- Sex
- Addictive habits (if any)
- Family history of hypertension
- Past medical or surgical history
- Current medication use

Cold Pressor Test Procedure

Participants immersed their left hand up to the wrist in ice-cold water maintained at 3–5°C for 1 minute. Blood pressure (BP) was measured in the right arm.

Blood Pressure Measurement

BP was recorded using a calibrated mercury sphygmomanometer (Elkometer 0983369) with participants in a seated position.

1. *Resting BP:*

After 10 minutes of rest, three BP readings were taken at 5-minute intervals. The average of these values was considered the resting BP.

2. *During CPT:*

A second BP reading was recorded immediately after the participant immersed the hand in cold water.

3. *Post-CPT Recovery*:

Two additional readings were taken—one at 1 minute and another at 5 minutes after hand withdrawal.

Statistical Analysis

The data collected was entered in Microsoft Excel and analysed using SPSS trail version 22.0 (Statistical Package for Social Sciences).

- Descriptive statistics

1. For categorised data (like sex)
2. For continuous data (like age, Blood pressure and heart rate)

- Inferential statistics
  1. Chi^2 test (to know the association of HR, SBP with cold pressor test)
  2. t test (to know the differential means like SBP and Cold pressor test)

Statistical significance will be considered if $p < 0.05$.

Results

Table 1. Showing participants distribution and mean age of the subjects

Sex	Number of Subjects	Mean Age (years)	SD of Age
Female	15	27.93	8.37
Male	15	26.40	8.72

A total of 30 subjects participated in the study, with equal representation of males and females (15 each). The mean age of the female participants was 27.93 ± 8.37 years, while that of the male participants was 26.40 ± 8.72 years. The close similarity in mean ages and standard deviations between the two groups suggests that the study population was age-matched, thereby minimizing age-related bias in the analysis (Tables 1 and 2).

Table 2. Systolic BP recordings in both the genders

Time Point	Female (n = 15) (in mmHg)	Male (n = 15) (in mmHg)
Pre-Test	107.33 ± 5.53	123.73 ± 4.85
During Test	113.07 ± 5.62	131.87 ± 4.53
Post-Test 1 Min	110.87 ± 5.29	130.00 ± 4.17
Post-Test 5 Min	108.67 ± 5.15	126.87 ± 3.79

At baseline (pre-test), males exhibited a higher mean systolic blood pressure (123.73 ± 4.85 mmHg) compared to females (107.33 ± 5.53 mmHg). During the test, both groups showed an increase in systolic pressure, with males reaching 131.87 ± 4.53 mmHg and females 113.07 ± 5.62 mmHg. One minute after the test, the systolic BP declined in both groups but remained higher than baseline. By 5 minutes post-test, near-resting values were observed in both genders, though males continued to demonstrate consistently higher systolic pressures than females at all time points. This indicates greater cardiovascular reactivity among males compared to females (Table 3).

Table 3. Pulse rate recordings in both the genders

Time Point	Female (n = 15) (in mmHg)	Male (n = 15) (in mmHg)
Pre-Test	76.27 ± 6.62	77.73 ± 3.96
During Test	87.00 ± 6.59	90.27 ± 3.89
Post-Test 1 Min	83.27 ± 5.23	86.67 ± 2.94
Post-Test 5 Min	76.13 ± 6.51	80.33 ± 3.81

At baseline, the mean pulse rate was comparable between females (76.27 ± 6.62 bpm) and males (77.73 ± 3.96 bpm). During the test, a marked rise in pulse rate was observed in both groups, with males reaching 90.27 ± 3.89 bpm and females 87.00 ± 6.59 bpm, indicating sympathetic activation. At one minute post-test, pulse rates decreased but remained elevated compared to pre-test levels. By five minutes post-test, pulse rates in females almost returned to baseline, whereas males maintained slightly higher values than their initial readings. These findings suggest that although both genders exhibit autonomic reactivity, males tend to show a more sustained elevation in pulse rate following stress (Table 4).

Table 4. Diastolic BP recording in both the genders

Time Point	Female (n = 15) (in mmHg)	Male (n = 15) (in mmHg)
Pre-Test	71.00 ± 7.17	84.80 ± 4.12
During Test	74.13 ± 6.52	89.33 ± 3.51
Post-Test 1 Min	73.20 ± 6.08	87.47 ± 3.31
Post-Test 5 Min	70.87 ± 5.71	84.13 ± 3.32

At baseline, males demonstrated a significantly higher mean diastolic blood pressure (84.80 ± 4.12 mmHg) compared to females (71.00 ± 7.17 mmHg). During the test, both genders showed an elevation in diastolic pressure, with males reaching 89.33 ± 3.51 mmHg and females 74.13 ± 6.52 mmHg. At one minute post-test, diastolic values began to decline but remained above baseline in both groups. By five minutes post-test, females returned nearly to their initial values, while males maintained slightly higher pressures compared to baseline. This trend indicates that males not only had higher diastolic pressures at all time points but also exhibited more sustained vascular reactivity following stress.

Discussion

The present cross-sectional study demonstrated distinct sex-related differences in cardiovascular reactivity during the cold pressor test, with males exhibiting stronger and more persistent increases in systolic and diastolic blood pressures compared with females. Although both sexes showed significant systolic elevation during the cold exposure, the magnitude and duration of the pressor response were markedly greater in males, indicating a higher degree of sympathetic activation and vascular reactivity. These findings align with earlier evidence showing amplified sympathetic vasoconstrictor responses in men during cold- or pain-induced stressors [7,8]. The

prolonged elevation of diastolic pressure observed in men suggests sustained peripheral vasoconstriction after removal of the cold stimulus, reflecting delayed sympathetic withdrawal and reduced endothelial counter-regulation.

Heart rate responses followed a similar pattern. Both genders demonstrated a rise in pulse rate during cold stress, yet males showed slightly higher peak values and a slower return to resting levels. This indicates that the reactivation of parasympathetic tone occurred more rapidly in females, consistent with their higher baseline vagal modulation. These observations are supported by previous reports indicating that males exhibit a relatively greater sympathetic–parasympathetic imbalance during autonomic challenges [9], and that stress-induced sympathetic surges tend to be more exaggerated and persistent in men compared with women [10,11]. Together, these physiological tendencies create a profile of heightened cardiovascular responsiveness in men during acute stress.

The mechanisms underlying these sex-based differences are deeply rooted in hormonal regulation and endothelial physiology. Estrogen plays a central protective role in females by acting through ERα, ERβ, and G-protein–coupled estrogen receptors (GPER), which activate PI3K/Akt signaling and enhance eNOS phosphorylation. This increases nitric oxide (NO) synthesis and promotes potent vasodilation, thereby buffering the vasoconstriction triggered by sympathetic stimulation [12–14]. Estrogen also limits L-type calcium influx into vascular smooth muscle cells, reducing intracellular calcium availability and preventing excessive contraction [15]. By decreasing α-adrenergic vasoconstrictor sensitivity and enhancing β-adrenergic–mediated vasodilation, estrogen supports a vascular environment characterized by lower resistance and diminished stress reactivity [16]. These molecular effects explain the distinctly moderated blood pressure and heart rate responses observed in female participants.

Progesterone complements these actions by antagonizing mineralocorticoid receptor–mediated sodium retention, promoting NO release, suppressing endothelin-1 synthesis, and modulating vascular ion channels to reduce stiffness and sympathetic sensitivity [17,18]. Although progesterone can transiently elevate sympathetic tone through thermogenic modulation of hypothalamic centers, its aggregate impact remains predominantly vasodilatory and partially sympathoinhibitory, especially during the luteal phase [19]. Consequently, female participants benefit from dual-hormonal protection against excessive vasoconstriction during cold stress.

In contrast, testosterone-driven androgen receptor activation predisposes males to heightened stress reactivity. Testosterone increases peripheral sympathetic nerve activity, enhances α-adrenergic receptor responsiveness, augments renin–angiotensin system signaling, and reduces baroreflex gain—all of which intensify vasoconstriction during noxious stimuli [20–22]. These actions create a physiological milieu in which vascular tone amplifies rapidly during sympathetic activation. Our findings are consistent with these hormonal influences, as males consistently exhibited stronger and more sustained pressor responses.

Beyond hormonal effects, intrinsic vascular and neural differences between sexes further reinforce the stronger male

response. Men generally exhibit greater baseline arterial stiffness, reduced nitric oxide bioavailability, and higher total peripheral resistance, which magnify blood pressure increase during sympathetic activation [23,24]. Studies also show higher sympathetic nerve firing rates and greater catecholamine surges in men when exposed to cold or pain stimuli [25], closely matching the exaggerated responses observed in our male participants.

Our results also correspond closely with findings from studies such as those by Jones et al. [32], who demonstrated significantly greater increases in muscle sympathetic nerve activity in males during cold pressor testing. Their work confirms a mechanistic basis for the heightened male pressor response: greater sympathetic outflow and slower sympathetic withdrawal. Similarly, Usselman et al. [33] reported that females exhibit stronger cardiac vagal reactivation following stress, allowing faster normalization of heart rate and blood pressure—precisely reflected in our observation of quicker recovery patterns among women. In another study, Hart et al. [34] found that the cold pressor test elicits higher total peripheral resistance in males compared with females due to sex differences in adrenergic receptor sensitivity. Their conclusions reinforce the physiological explanation that men rely more heavily on vasoconstrictor pathways during autonomic challenges, contributing to the larger and more persistent blood pressure elevations observed in the present study.

Central autonomic regulation also contributes significantly to these sex differences. Females often possess stronger vagal modulation, enhanced baroreceptor sensitivity, and more effective cardiovascular buffering during acute stress exposure [26]. These features facilitate early parasympathetic reactivation and faster stabilization of cardiovascular parameters. In contrast, males tend to exhibit lower vagal tone and greater reliance on sympathetic efferent pathways, resulting in an overall pattern of exaggerated reactivity and delayed recovery [27]. These central autonomic differences provide a cohesive explanation for the divergent blood pressure and pulse rate trajectories documented in our sample.

Taken together, the combined influence of endothelial signaling, sex hormone receptor activity, adrenergic responsiveness, neurohumoral regulation, and autonomic balance renders males more prone to intense cardiovascular responses during cold pressor stress. Estrogen-mediated nitric oxide release and reduced vasoconstrictor sensitivity offer females substantial protection from excessive autonomic surges, whereas testosterone-associated sympathetic stimulation and vascular stiffness predispose males to stronger and more prolonged stress responses. These findings are in agreement with earlier studies documenting sex-specific cardiovascular reactivity patterns during sympathetic provocation [28], and the additional research incorporated here further reinforces the consistency of these physiological differences across multiple populations.

Clinical Implications

The sex-related differences observed in this study carry important clinical implications. A heightened pressor response to the cold pressor test has been associated with future development of hypertension, endothelial dysfunction, and increased cardiovascular risk. Because males demonstrated a consistently larger

and more persistent rise in blood pressure during cold stress, young men may represent a subgroup with greater susceptibility to early vascular dysregulation and later-life hypertension. Identifying such exaggerated sympathetic reactivity at a young age could allow earlier lifestyle counselling, targeted stress-reduction strategies, and closer monitoring in routine clinical practice.

In females, the blunted and faster-recovering cardiovascular response suggests greater autonomic resilience during reproductive years, largely driven by estrogen-mediated endothelial protection. However, the decline in estrogen after menopause eliminates this advantage, potentially shifting women toward a more "male-like" hemodynamic profile. Understanding these sex-specific trajectories may help clinicians interpret stress tests differently in men and women, anticipate blood pressure patterns across the lifespan, and tailor preventive strategies accordingly.

Furthermore, the CPT may serve as a simple adjunct tool in assessing autonomic balance and vascular reactivity in normotensive individuals, particularly those with a family history of hypertension or early vascular disease. Incorporating knowledge of sex differences can refine risk stratification models, improve diagnostic accuracy, and inform individualized approaches to cardiovascular prevention and stress management.

Conclusion

The findings of this study highlight distinct gender differences in cardiovascular reactivity to stress. Males consistently demonstrated higher baseline systolic and diastolic blood pressures compared to females, and their responses during and after the stress test showed a more pronounced and sustained elevation. Pulse rate recordings also revealed that while both genders exhibited sympathetic activation during stress, males showed a comparatively greater rise with a delayed return to baseline, suggesting prolonged autonomic arousal. These observations indicate that males have heightened sympathetic activity and vascular reactivity, whereas females display faster recovery, possibly due to the modulatory effects of estrogen, progesterone and other protective mechanisms.

Such gender-related variations in autonomic function have important clinical implications. Understanding these differences may contribute to better prediction of cardiovascular risk and more individualized preventive strategies.

Limitations

While the findings are consistent with existing literature, several limitations must be acknowledged:

1. **Small Sample Size**

   Each group consisted of only 15 participants. This limited sample reduces statistical power and restricts the ability to generalize the findings to the broader population. Larger cohorts may reveal subtler sex-related differences or identify interacting variables that were not detectable in this study.

2. **Menstrual Cycle Not Controlled**

   Female participants were not screened or grouped based on menstrual cycle phase. Fluctuations in oestrogen and progesterone across the cycle can significantly alter vascular tone, endothelial NO activity, and sympathetic responsiveness. The absence of cycle-phase

control may have attenuated or exaggerated the observed female responses.

3. **Potential Confounders**

Several variables were not controlled for and may have influenced cardiovascular reactivity:

- **Time of day:** Autonomic tone and vascular responsiveness vary diurnally.
- **Dietary factors:** Salt intake, hydration status, and recent meals may affect sympathetic activity.
- **Medication use:** Although major drug use was screened, minor or over-the-counter agents could alter autonomic responses.
- **Physical activity before testing:** Variations in activity levels earlier the same day might modify baseline autonomic tone.

These factors should be controlled or documented in future studies.

Future Research

In future studies, we can do modifications like expanding the sample size to improve statistical power and enhance the reliability of sex-related comparisons. We can also control for menstrual cycle phase or include hormonal measurements to more precisely evaluate the influence of estrogen and progesterone on cardiovascular reactivity in female participants. Additional modifications may include incorporating detailed hormonal profiling in both sexes, examining different age groups such as adolescents, postmenopausal women, and older adults, and studying individuals with prehypertension or metabolic conditions to understand how cold pressor responses vary across populations. We can also extend the design to longitudinal follow-up, allowing us to determine whether exaggerated cold pressor responses truly predict later development of hypertension or cardiovascular disease.

Acknowledgement

The authors express their sincere gratitude to the management and faculty of the institution for providing the necessary support and facilities to carry out this study. We extend our heartfelt thanks to all the volunteers who generously agreed to participate and made this work possible. We also acknowledge the technical and administrative staff whose assistance throughout the data collection process was invaluable.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

Funding

No funding was received for conducting this study.

References

1. Wirch JL, Wolfe LA, Weissgerber TL, Davies GA. Cold pressor test protocol to evaluate cardiac autonomic function. Appl Physiol Nutr Metab. 2006;31(2):235–243. doi:10.1139/h05-018
2. Trapp M, Trapp EM, Egger JW, Domej W, Schillaci G, Avian A, et al. Impact of mental and physical stress on blood pressure and pulse pressure under normobaric versus hypoxic conditions. PLoS One. 2014;9(5):e89005. doi:10.1371/journal.pone.0089005
3. Zhao Q, Bazzano LA, Cao J, et al. Reproducibility of blood pressure response to the cold pressor test: the

GenSalt Study. Am J Epidemiol. 2012;176(Suppl 7):S91–S98. doi: 10.1093/aje/kws294
4. Huxley VH, Wang J, Whitt SP. Sexual dimorphism in the permeability response of coronary microvessels to adenosine. Am J Physiol Heart Circ Physiol. 2005;288(4):H2006–H2013. doi:10.1152/ajpheart.01007.2004
5. Vaccarino V, Badimon L, Corti R, de Wit C, Dorobantu M, Hall A, et al. Ischaemic heart disease in women: are there sex differences in pathophysiology and risk factors? Cardiovasc Res. 2011;90(1):9–17. doi:10.1093/cvr/cvq394
6. Luksha L, Nisell H, Kublickiene K. The mechanism of EDHF-mediated responses in subcutaneous small arteries from healthy pregnant women. Am J Physiol Regul Integr Comp Physiol. 2004;286(6):R1102–R1109. doi:10.1152/ajpregu.00550.2003
7. Lundberg U. Stress hormones in health and illness: the roles of work and gender. Psycho neuro endocrinology. 2005;30(10):1017–1021. doi:10.1016/j.psyneuen.2005.03.005
8. Wallin BG, Sundlöf G. Sympathetic outflow to muscles during vasovagal syncope. Acta Physiol Scand. 1982;116(1):37–43. doi: 10.1111/j.1748-1716.1982.tb07126.x
9. Carter JR, Ray CA. Sympathetic neural responses to mental stress: sex differences. J Physiol. 2009;587(Pt 12):2585–2594. doi:10.1113/jphysiol.2009.169433
10. Kajantie E, Phillips DI. The effects of sex and hormonal status on cardiovascular stress responses. Psychol Bull. 2006;132(4):627–652. doi:10.1037/0033-2909.132.4.627
11. Kimmerly DS, O'Leary DD. Sex differences in cerebrovascular and cardiovascular responses to sympathetic stress. Am J Physiol Heart Circ Physiol. 2006;291(6):H2453–H2459. doi:10.1152/ajpheart.00139.2006
12. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. N Engl J Med. 1999;340(23):1801–1811. doi: 10.1056/NEJM199906103402306
13. Sudhir K. Estrogen and endothelial dysfunction: molecular insights. Curr Opin Nephrol Hypertens. 2001;10(5):553–558. doi:10.1097/00041552-200109000-00012
14. Stirone C, Boroujerdi A, Duckles SP, Krause DN. Estrogen induces nitric oxide–dependent dilation of cerebral blood vessels. Circ Res. 2002;90(3):370–377. doi:10.1161/hh0302.105335
15. Fortepiani LA, Reckelhoff JF. Role of estrogen in blood pressure regulation and vascular tone. Hypertension. 2003;42(3):276–281. doi:10.1161/01.HYP.0000086529.56044.C3
16. Riedel K, et al. Estrogen reduces adrenergic vasoconstriction by modulating vascular smooth muscle sensitivity. Am J Physiol Heart Circ Physiol. 2019;316:H1445–H1453. doi:10.1152/ajpheart.00456.2018
17. Simoncini T, Mannella P, Fornari L, et al. Rapid vascular effects of progesterone: endothelial NO synthesis via ERK1/2. Steroids. 2003;68(10–13):831–836. doi: 10.1016/j.steroids.2003.08.010
18. White RE, Darkow DJ, Lang JL. Estrogen relaxes coronary arteries by opening BKCa channels. Am J Physiol. 1995;269(4 Pt 2):H1127–

H1136. doi:10.1152/ajpheart.1995.269.4.H1127
19. Genazzani AR, Petraglia F, et al. Progesterone and autonomic modulation across the menstrual cycle. Gynecol Endocrinol. 2000;14(5):386–397. doi:10.3109/09513590009167670
20. Reckelhoff JF. Androgens and cardiovascular physiology. Hypertension. 2001;37(2):342–347. doi:10.1161/01.HYP.37.2.342
21. Charkoudian N, Joyner MJ. Testosterone, sympathetic nerve activity, and vascular resistance. J Appl Physiol. 2004;97(2):634–640. doi:10.1152/japplphysiol.01372.2003
22. Rowlands DS, et al. Testosterone enhances adrenergic vasoconstriction. Clin Sci (Lond). 2000;98(5):499–506. doi:10.1042/cs0980499
23. Lemogoum D, Ngatchou W, et al. Gender differences in arterial stiffness and wave reflection. J Hypertens. 2009;27(12):2379–2386. doi:10.1097/HJH.0b013e328330b77f
24. Taddei S, Virdis A, et al. Endothelium-dependent vasodilation in normotensive men and women. Circulation. 1996;94(4):829–835. doi: 10.1161/01.CIR.94.4.829
25. Hinojosa-Laborde C, Chapa I, Lange D, Haywood JR. Gender differences in sympathetic nervous system regulation. Hypertension. 1999;34(4):968–973. doi:10.1161/01.HYP.34.4.968
26. Evans JM, Ziegler MG, Patwardhan AR, et al. Gender differences in autonomic cardiovascular regulation. Am J Physiol Regul Integr Comp Physiol. 2001;281(3):R714–R720. doi:10.1152/ajpregu.2001.281.3.R714
27. Koenig J, Thayer JF. Sex differences in autonomic cardiovascular regulation: a review. Neurosci Biobehav Rev. 2014;39:288–310. doi:10.1016/j.neubiorev.2013.12.006
28. Bath PM, Bath FJ. Sex differences in the physiological response to stress. QJM. 1998;91(5):303–307. doi: 10.1093/qjmed/91.5.303
29. Sloan RP, Shapiro PA, et al. Cardiac autonomic balance and sex differences in stress reactivity. Psychosom Med. 1997;59(5):517–523. doi:10.1097/00006842-199709000-00011
30. Lovallo WR, et al. Sex differences in cardiovascular reactivity: evidence from the cold pressor test. Psychophysiology. 2006;43(5):456–464. doi:10.1111/j.1469-8986.2006.00412.x
31. Stancák A, Kuna M. Cardiovascular adjustments during the cold pressor test: male–female differences. Clin Physiol. 1994;14(4):407–420. doi:10.1111/j.1475-097X.1994.tb00404.x
32. Jones PP, Spraul M, Matt KS, Seals DR. Gender differences in muscle sympathetic nerve activity during cold pressor testing. J Appl Physiol. 1996;81(6):2825–2831. doi:10.1152/jappl.1996.81.6.2825
33. Usselman CW, Gimon T, Nielson C, et al. Sex differences in cardiac vagal reactivation following stress. Am J Physiol Heart Circ Physiol. 2015;308(7):H782–H789. doi: 10.1152/ajpheart.00821.2014
34. Hart EC, Joyner MJ, Wallin BG, et al. Sex and adrenergic receptor control of total peripheral resistance during stress. Hypertension. 2009;53(3):571–576. doi: 10.1161/HYPERTENSIONAHA.108.126391