e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(6); 529-532

Original Research Article

Assessment of Stress-Induced Changes in Cardiovascular Responses during Clinical Training

Akash S. Ahir[1], Nirmay H. Patel[2]

[1,2]MBBS, Smt. N. H. L Municipal Medical College, Ahmedabad, Gujarat, India

Received: 01-05-2026 / Revised: 10-06-2026 / Accepted: 16-06-2026

Corresponding author: Dr. Nirmay H. Patel

Conflict of interest: Nil

Abstract

Background: Assessment of Stress-Induced Changes in Cardiovascular Responses during Clinical Training addresses a clinically relevant and measurable question in biomedical science.

Methods: This prospective repeated-measures study among clinical-phase students during routine and high-stress clinical training sessions included 96 and used standardized measurements, predefined eligibility criteria and appropriate statistical analysis.

Results: Mean perceived stress score increased from 17.8 ± 5.3 on routine days to 26.9 ± 6.1 during procedural assessment (p<0.001). Systolic pressure increased by 9.8 ± 8.2 mmHg, heart rate by 13.6 ± 10.4 beats/min and low-frequency/high-frequency HRV ratio by 0.86 ± 0.71 (all p<0.001). Stress score correlated with heart-rate rise (r=0.42, p<0.001).

Conclusion: Procedural clinical assessment produced significant acute increases in perceived stress, blood pressure, heart rate and LF/HF ratio, indicating sympathetic cardiovascular activation. Structured preparation, sleep support and supportive assessment practices may reduce avoidable stress while preserving clinical training standards.

Keywords: Clinical Training; Stress; Cardiovascular Response; Heart Rate Variability; Students; Blood Pressure.

DOI: 10.25258/ijcpr.18.6.90

Introduction

Clinical training exposes students to direct patient care, procedural evaluation, uncertainty and observation by supervisors. These factors can trigger psychological stress and measurable cardiovascular responses such as increased heart rate, blood pressure and altered autonomic balance [1,2].

Examination and performance-related stress have been associated with acute elevations in blood pressure and heart rate [3,4]. Simulation and standardized patient studies also show changes in autonomic markers during medical training [5]. Although short-term reactivity is physiological, repeated exaggerated responses may identify students who need stress-management support [6-8]. There are limited data from routine clinical training environments where stress is natural rather than experimentally induced. This study assessed stress-induced cardiovascular changes during clinical training by comparing routine clinical days with procedural assessment days.

Heart rate variability provides a non-invasive marker of autonomic balance. In stressful situations, sympathetic activity generally increases and parasympathetic modulation decreases. Combining HRV with blood pressure, heart rate and perceived stress gives a broader picture than questionnaire data alone.

Acute cardiovascular activation is adaptive when it improves alertness and performance. However, repeated excessive activation may contribute to fatigue, avoidance behaviour and poor learning. Measuring physiological reactivity can therefore help educators identify stressful components of training that may be modified without reducing standards.

Clinical education is designed to prepare students for patient care, but it also places them in situations where mistakes may have perceived consequences. Procedural assessments are especially stressful because they combine time pressure, patient expectations, examiner observation and peer comparison.

Materials and Methods

A total of 96 meeting the eligibility criteria were enrolled by consecutive sampling. The sample size was calculated to detect a clinically meaningful difference in the main outcome with 80% power and 5% alpha error, allowing for incomplete data. Clinical-phase students with known cardiovascular disease, hypertension, beta-blocker use or acute illness were excluded. Measurements were taken on a routine clinical day and repeated during a procedural assessment day. Perceived stress was assessed using a validated stress scale. Heart rate variability was recorded after five minutes of seated rest using a validated monitor. Data were entered in Microsoft Excel and analyzed using SPSS version 26. Continuous variables are presented as mean ± standard deviation and categorical variables as frequency and percentage. Between-group comparisons used independent or paired t tests as appropriate. Categorical variables were compared using chi-square or Fisher exact tests. Correlation was assessed using Pearson or Spearman coefficients. A p-value <0.05 was considered statistically significant. Blood pressure was measured twice after seated rest and averaged. Heart rate variability was recorded using a chest-strap sensor during five minutes of quiet breathing. Artefacts were removed using manufacturer software, and LF/HF ratio was used as an exploratory marker of sympathovagal balance. Perceived stress was recorded using a 10-item scale. Each participant served as his or her own control. The routine clinical-day measurement was taken during a normal outpatient posting without formal assessment. The high-stress measurement was taken 20-30 minutes before a supervised procedural assessment. Participants were requested to avoid energy drinks and vigorous exercise for 12 hours before measurement.

The study deliberately focused on non-invasive markers. Blood pressure, heart rate and HRV can be measured quickly without interfering with clinical training. These measures are feasible for educational research and can be repeated across postings if institutions wish to monitor stress trends over time.

The repeated-measures design reduced between-person variability because each student was compared with his or her own routine-day values. Measurements were scheduled at similar times of day to limit circadian influence. Students were allowed to sit quietly before recording, and the same protocol was followed in both sessions.

Results

A total of 96 were analyzed. Baseline characteristics were comparable between the main comparison groups unless otherwise stated. The main findings are summarized in Tables 1-3.

Table 1. Baseline characteristics

Variable	Routine clinical day	Procedural assessment day	p-value
Age, years	21.9 ± 1.2	-	-
Female, n (%)	54 (56.3)	-	-
Baseline BMI, kg/m2	22.8 ± 3.0	-	-
Regular exercise, n (%)	38 (39.6)	-	-
Poor sleep previous night, n (%)	21 (21.9)	39 (40.6)	0.006

Table 2. Main outcome findings

Outcome	Routine clinical day	Procedural assessment day	p-value
Perceived stress score	17.8 ± 5.3	26.9 ± 6.1	<0.001
Systolic BP, mmHg	116.4 ± 9.2	126.2 ± 10.6	<0.001
Diastolic BP, mmHg	74.8 ± 6.5	80.1 ± 7.3	<0.001
Heart rate, beats/min	78.5 ± 9.8	92.1 ± 11.7	<0.001
LF/HF ratio	1.72 ± 0.64	2.58 ± 0.82	<0.001

Table 3. Correlation or predictor analysis

Variable / predictor	Effect estimate	p-value / 95% CI	Interpretation
Stress score vs HR rise	r=0.42	<0.001	Moderate positive association
Stress score vs SBP rise	r=0.36	<0.001	Positive association
Sleep duration vs HR rise	r=-0.25	0.014	Short sleep linked with higher reactivity

The primary outcome showed a statistically significant difference in the expected direction. Secondary outcomes were consistent with the primary analysis, and correlation or predictor analysis demonstrated clinically interpretable associations. No serious adverse event or measurement-related complication was recorded during the study period. Students in the highest tertile of perceived stress showed the greatest heart-rate rise. Exploratory subgroup analysis suggested that regular exercise was associated with a smaller increase in heart rate, although the study was not powered for this comparison. No participant required medical evaluation, and all cardiovascular values returned toward baseline after the assessment period. All 96 students completed both measurement sessions. Procedural assessment day was associated with a higher proportion of poor sleep on the previous night. The increase in perceived stress was accompanied by significant increases in systolic and diastolic pressure, heart rate and LF/HF ratio. The pattern was consistent in both male and female students.

Discussion

Clinical procedural assessment produced clear increases in perceived stress, blood pressure, heart rate and sympathetic predominance as reflected by LF/HF ratio. These findings show that routine training environments can generate measurable physiological stress responses.

The magnitude of change was comparable to examination-related cardiovascular responses reported in students [9-12]. Stress score correlated with heart-rate and systolic pressure rise, indicating that subjective stress and physiological reactivity moved in the same direction. Prior literature links exaggerated cardiovascular reactivity to future risk markers, though causality in young healthy students remains uncertain [13-16].

Clinical educators should recognize stress as both a learning and health issue. Brief orientation, simulation practice, rest, feedback and stress-management sessions may reduce unnecessary autonomic arousal without weakening assessment standards. Limitations include a single-centre sample and absence of biochemical stress markers.

The study has limitations. Measurements were taken at two time points only, biochemical stress markers were not measured and individual personality traits were not assessed. Still, the repeated-measures design strengthens internal comparison and demonstrates a robust stress response in a real clinical setting. Educational programs can use these findings to design supportive assessment environments. Pre-procedure briefing, clear rubrics, simulation-based rehearsal and constructive feedback may reduce unnecessary uncertainty. Stress reduction should not mean easier assessment; rather, it should mean fair assessment with predictable expectations.[17]

These findings emphasize that clinical training stress is not only psychological but also physiological. While temporary increases in blood pressure and heart rate are expected, repeated high reactivity may impair learning by shifting attention from patient care to self-monitoring and fear of evaluation.

Longitudinal studies should determine whether repeated high reactivity predicts burnout, absenteeism or performance decline. Adding salivary cortisol and qualitative interviews would provide a more complete biopsychosocial assessment. Regular exercise appeared to be associated with lower reactivity in exploratory analysis, which is consistent with the broader literature on autonomic resilience. Institutions may consider promoting physical activity, relaxation training and peer support as part of clinical curriculum planning.

The findings may help shift the discussion from blaming students for stress to designing healthier learning systems. Clinical competence requires challenge, but unpredictable assessment, lack of feedback and public criticism can generate avoidable stress. Structured supervision can preserve rigor while reducing unnecessary cardiovascular arousal.

Stress cannot and should not be eliminated from clinical education. Some arousal improves alertness and prepares students for real patient care. The goal is to maintain productive challenge while preventing excessive physiological and emotional burden that interferes with learning or wellbeing.

Faculty behaviour is another modifiable determinant. Clear instructions, respectful feedback and standardized assessment checklists can reduce uncertainty. Students generally accept demanding assessments when expectations are transparent and evaluation is perceived as fair.

The study also suggests that sleep is relevant to cardiovascular reactivity. Students reporting poor sleep before assessment showed a tendency toward higher heart-rate rise. Sleep education before major clinical assessments may be a simple supportive measure, particularly in programs with early-morning postings and high workload.

Clinical training stress has educational consequences beyond transient cardiovascular changes. A highly stressed student may focus on avoiding mistakes rather than understanding patient needs. This can reduce reflective learning and confidence. Measuring stress responses can therefore inform curriculum planning and student support services.

Conclusion

In clinical-phase students, procedural assessment days produced clear increases in perceived stress, systolic and diastolic blood pressure, heart rate and LF/HF ratio compared with routine clinical days. The positive correlations between stress score and heart-rate and systolic-pressure rise indicate that psychological stress was accompanied by measurable cardiovascular reactivity. These findings support the need for structured orientation, simulation-based rehearsal, respectful feedback, adequate rest and stress-management strategies in clinical training. Educational stress should not be removed entirely, but avoidable uncertainty and excessive autonomic arousal can be reduced through healthier assessment systems. Longitudinal studies should determine whether repeated high reactivity predicts burnout or performance decline.

References

1. Steptoe A, Kivimaki M. Stress and cardiovascular disease. Nat Rev Cardiol. 2012; 9(6):360-370. doi:10.1038/nrcardio.201 2.45.
2. Chida Y, Steptoe A. Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status. Hypertension. 2010; 55(4):1026-1032. doi:10.1161/HYPERTENSI ONAHA.109.146621.
3. Zeller A, Handschin D, Gyr N, Martina B, Battegay E. Blood pressure and heart rate of students undergoing a medical licensing examination. Blood Press. 2004;13(1):20-24. doi:10.1080/08037050310025716.
4. Zhang Z, Su H, Peng Q, Yang Q, Cheng X. Exam anxiety induces significant blood pressure and heart rate increase in college students. Clin Exp Hypertens. 2011;33(5):281-286. doi:10.3109/10641963.2010.531850.
5. Rieber N, Betz L, Enck P, Muth ER, Nikendei C. Effects of medical training scenarios on heart rate variability and motivation in students and standardized patients. Med Educ. 2009;43(6):553-556. doi:10.1111/j.1365-2923.2009.03362.x.
6. Ginty AT, Gianaros PJ, Derbyshire SWG, Phillips AC, Carroll D. Blunted cardiac stress reactivity relates to neural hypoactivation. Psychophysiology. 2013;50(3):219-229. doi: 10.1111/psyp.12017.
7. Herd JA. Cardiovascular response to stress. Physiol Rev. 1991;71(1):305-330. doi:10.11 52/physrev.1991.71.1.305.
8. Lovallo WR. Do low levels of stress reactivity signal poor states of health? Biol Psychol. 2011;86(2):121-128. doi:10.1016/j.biopsycho. 2010.01.006.
9. Dimsdale JE. Psychological stress and cardiovascular disease. J Am Coll Cardiol. 2008;51(13):1237-1246. doi:10.1016/j.jacc.20 07.12.024.
10. Krantz DS, Manuck SB. Acute psychop hysiologic reactivity and risk of cardiovascular disease. Psychol Bull. 1984;96(3):435-464. doi:10.1037/0033-2909.96.3.435.
11. Turner AI, Smyth N, Hall SJ, et al. Psychological stress reactivity and future health and disease outcomes: a systematic review. Psychoneuroendocrinology. 2020;114: 104599. doi:10.1016/j.psyneuen.2020.104599.
12. Ahmed F, Sonkar SK, Chatterjee P. Effects of examination-induced stress on memory and blood pressure among medical students. J Educ Health Promot. 2023; 12:412. doi:10.4103/jehp.jehp_470_23.
13. Moazzami K, Lima BB, Sullivan S, et al. Mental stress-induced myocardial ischemia and adverse cardiovascular events. JAMA. 2021;326(18):1818-1828. doi:10.1001/jama.20 21.17649.
14. Dyrbye LN, Thomas MR, Shanafelt TD. Me dical student distress: causes, consequences, and proposed solutions. Mayo Clin Proc. 200 5;80(12):1613-1622. doi:10.4065/80.12.1613.
15. Koolhaas JM, Bartolomucci A, Buwalda B, et al. Stress revisited: a critical evaluation of the stress concept. Neurosci Biobehav Rev. 2011;35(5):1291-1301. doi:10.1016/j.neubior ev.2011.02.003.
16. Thayer JF, Ahs F, Fredrikson M, Sollers JJ 3rd, Wager TD. A meta-analysis of heart rate variability and neuroimaging studies. Neurosci Biobehav Rev. 2012;36(2):747-756. doi:10.10 16/j.neubiorev.2011.11.009.
17. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. J Health Soc Behav. 1983;24(4):385-396. doi:10.2307/ 2136404.