GMR
The Original

COMPARISON OF MODIFIED CAROTID SINUS MASSAGE USING ULTRASONOGRAPHY WITH CONVENTIONAL CAROTID SINUS MASSAGE IN EMERGENCY DEPARTMENT

Zainab Nadeem[1*], Muhammad Ahmed Ilyas[2], Ehtesham ul Haq[3], M Arslan Maqbool[4], Sadaf Yousaf[5]

[1,2,3]Postgraduate Trainee FCPS, Department of Emergency Medicine, Central Park Teaching Hospital Lahore, Pakistan
[4]Registrar, Department of Emergency Medicine, Central Park Teaching Hospital, Lahore, Pakistan
[5]Professor, HOD Emergency Department, Central Park Teaching Hospital, Lahore, Pakistan
*Corresponding Author: Zainab Nadeem, Email: nadeem.xainab@gmail.com

ABSTRACT

Objectives: To compare the vagal response, measured by heart-rate reduction and R–R interval prolongation, produced by ultrasound-guided modified carotid sinus massage (CSM) versus conventional CSM in hemodynamically stable patients with acute supraventricular tachycardia (SVT) in the emergency department (ED). The study evaluated physiological response, not definitive clinical efficacy.

Methodology: This prospective, open-label, blinded-endpoint randomized controlled trial was conducted at Central Park Teaching Hospital, Lahore (January 03, 2026 to April 03, 2026; IRB No. CPMC/IRB-No/1541). Patients aged 35–75 years with narrow-QRS (<120 ms) tachycardia >150 bpm, adjudicated by a blinded cardiologist, were randomized 1:1 using computer-generated permuted blocks with sealed opaque envelopes to ultrasound-guided modified CSM (Group A, n=125) or conventional CSM (Group B, n=125). In Group A, a 7.5–12 MHz linear probe identified the right carotid bulb at maximum dilation for a targeted 10-second unilateral pressure; Group B received right-sided palpation-guided pressure for 10 seconds. One attempt per patient was permitted. Primary outcomes were heart-rate and R–R interval change on continuous Lead II ECG, which were adjudicated blinded to allocation.

Results: Groups were balanced at baseline. Ultrasound-guided modified CSM produced significantly greater heart-rate reduction (53.2 ± 17.1 vs 25.8 ± 13.9 bpm; mean difference 27.4, 95% CI 23.2 to 31.6; $p < 0.001$; Cohen's $d = 1.78$) and R–R interval prolongation (0.272 ± 0.118 vs 0.148 ± 0.092 s; mean difference 0.124, 95% CI 0.098 to 0.150; $p < 0.001$; $d = 1.12$). No adverse events occurred (95% CI 0–1.5%).

Conclusion: Ultrasound-guided modified CSM produces a significantly greater vagal response and appears safe. Because these are surrogate endpoints, clinical superiority (SVT termination, rescue therapy, recurrence) requires confirmation in adequately powered multicenter trials before routine ED adoption.

KEYWORDS: Supraventricular tachycardia; carotid sinus massage; point-of-care ultrasound; vagal maneuver; emergency department; surrogate endpoint.

INTRODUCTION

Supraventricular tachycardia (SVT) is a common cardiac arrhythmia encountered in emergency departments (EDs) worldwide and a frequent cause of palpitations, dizziness, chest discomfort, and haemodynamic instability (1). For the purposes of this study and in keeping with current guidelines, SVT refers to paroxysmal re-entrant tachyarrhythmias, predominantly atrioventricular nodal re-entrant tachycardia (AVNRT) and atrioventricular re-entrant tachycardia (AVRT) — presenting as a regular narrow-QRS tachycardia (2). Although many episodes are self-limited, sustained SVT may require urgent intervention to restore sinus rhythm and to avert myocardial ischaemia, syncope, or heart failure (3).

Guidelines from the American College of Cardiology/American Heart Association/Heart Rhythm Society (2015) and the European Society of Cardiology (2019) recommend vagal manoeuvres as the first step in the management of haemodynamically stable SVT (1, 2). The modified Valsalva manoeuvre, evaluated in the REVERT trial, is widely regarded as the first-line vagal intervention on the basis of a success rate of approximately 43% versus ~17% for the standard Valsalva (13). Carotid sinus massage (CSM) nonetheless retains an important role in patients in whom Valsalva is contraindicated, is poorly tolerated, or has already failed — including frail or elderly patients, intubated patients, those unable to generate adequate intrathoracic pressure, and uncooperative patients (4, 5). Reported success rates for conventional CSM range from 10% to 40%, substantially lower than those reported for modified Valsalva (7, 8). Optimisation of CSM is therefore not a replacement for modified Valsalva but an attempt to improve a commonly used second-line manoeuvre.

A principal limitation of conventional CSM is its reliance on surface anatomical landmarks. Surface landmarks do not consistently overlie the carotid sinus because of inter-individual anatomical variability, age-related vascular change, and operator-dependent technique (9, 10). Suboptimal baroreceptor stimulation, together with the possibility of

applying inappropriate pressure over calcified or diseased arteries, may both reduce efficacy and particularly in older patients, raise safety concerns (11). The expanding use of point-of-care ultrasound (POCUS) in emergency medicine offers an opportunity to target the carotid bulb directly at its point of maximal dilation, potentially producing a more reliable vagal response (12, 22, 23, 24).

Preliminary physiological and simulation data suggest that ultrasound-guided CSM may elicit a stronger vagal response than the conventional blind technique (6, 14). However, high-quality randomized trials directly comparing the two approaches in the ED setting are scarce, particularly in resource-limited environments. The present trial therefore compared ultrasound-guided modified CSM with conventional CSM in patients with haemodynamically stable SVT, using objective electrophysiological measures — heart-rate reduction and R–R interval prolongation — as surrogate markers of vagal response. Pre-specified exploratory subgroup and regression analyses were planned to describe the consistency of treatment effect across clinically relevant strata and across symptom duration. We explicitly framed this study as an evaluation of physiological response, not of clinical efficacy.

MATERIALS AND METHODS

Study Design and Setting

This was a prospective, open-label, blinded-endpoint randomized controlled trial conducted in the Department of Emergency Medicine, Central Park Teaching Hospital, Lahore, from January 03, 2026 to April 03, 2026. The protocol was approved by the Institutional Ethical Review Board (IRB No. CPMC/IRB-No/1541) and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every participant (or a legally authorized representative when clinically indicated) before enrolment. The study was reported in accordance with the CONSORT 2010 statement.

Eligibility and Case Definition

Consecutive patients presenting to the ED with suspected SVT were screened. SVT was defined as a regular, narrow-QRS (<120 ms) tachycardia at a rate >150 bpm with absent or retrograde P waves and a 1:1 atrioventricular relationship, consistent with paroxysmal SVT (predominantly AVNRT or AVRT). The baseline 12-lead ECG of every screened patient was reviewed in real time by the on-duty emergency physician and, before randomization, independently confirmed by a board-certified cardiologist who was blinded to the planned group allocation. Patients with diagnostic uncertainty were not enrolled. Eligible patients were 35–75 years of age and haemodynamically stable (systolic blood pressure ≥ 90 mmHg, no altered mental status, no signs of shock).

Exclusion Criteria and Safety Screening

Patients were excluded if any of the following were present:

- Pregnancy.
- Congestive heart failure with ejection fraction < 30%.
- Chronic kidney disease with serum creatinine > 1.8 mg/dL.
- Uncontrolled hypertension (blood pressure ≥ 160/100 mmHg).
- Atrial fibrillation, atrial flutter, multifocal atrial tachycardia, wide-complex tachycardia, or sinus tachycardia on baseline ECG.
- Audible carotid bruit on bilateral pre-procedural auscultation.
- Any documented or self-reported history of transient ischaemic attack, ischaemic stroke, or carotid endarterectomy.
- Known carotid artery stenosis ≥ 50% on prior imaging.
- In Group A, visualised atherosclerotic plaque on the pre-procedural sonographic screen of the carotid bifurcation; in Group B, plaque on a pre-procedural carotid duplex scan performed in patients ≥ 60 years.
- Recent (< 3 months) myocardial infarction.
- Known ventricular pre-excitation (Wolff–Parkinson–White syndrome) with wide-complex tachycardia, or suspected digoxin toxicity.

Bilateral auscultation for carotid bruit was mandatory before any CSM attempt in both arms and was documented on the case report form.

Randomization, Allocation Concealment, and Blinding

Sequence generation. A computer-generated random allocation sequence in a 1:1 ratio was prepared using permuted blocks of sizes 4 and 6 by a statistician not otherwise involved in the trial.

Allocation concealment. Sequentially numbered, opaque, sealed envelopes were prepared by the same statistician and held in the ED. Envelopes were opened only after written consent was obtained and baseline data recorded.

Implementation. Enrolment and consent were performed by the on-duty emergency physician; randomization and the study intervention were performed by the designated study operator.

Blinding. The nature of the intervention precluded blinding of patients, operators, and treating clinicians. Outcome assessment was blinded: all baseline and post-manoeuvre ECG tracings were de-identified, stripped of any visual cue to allocation, and independently interpreted by a board-certified cardiologist blinded to group assignment. This open-

label design with blinded endpoint adjudication is acknowledged as a potential source of performance bias (addressed in Limitations).

Intervention Protocol

For both groups, the patient was positioned supine with the neck slightly extended and rotated contralaterally. The right carotid sinus was used in all patients, both to minimize variability and because of its generally greater accessibility; the manoeuvre was strictly unilateral and never bilateral or sequential. The manoeuvre duration was 10 seconds of firm digital pressure directed posteromedially against the transverse process of the fourth cervical vertebra. Only one attempt per patient was permitted per protocol; outcomes were captured from this single attempt. A continuous Lead II ECG was recorded throughout.

Group A (ultrasound-guided modified CSM). A 7.5–12 MHz linear transducer was placed in the short axis over the right carotid bifurcation. The carotid bulb was identified and the phase of maximum dilation determined in real time. The operator marked the overlying skin point, withdrew the probe, and immediately applied 10-second digital pressure over the marked point, timed to begin at the next cardiac cycle. Ultrasound was used for precise anatomical localization; dynamic imaging during compression was not performed, because probe pressure would have confounded the manual pressure being studied.

Group B (conventional CSM). The operator palpated the right carotid pulse at the level of the cricoid cartilage and applied 10-second digital pressure over the point of maximal pulsation, as per standard teaching.

Operators and training. All manoeuvres were performed by four emergency medicine residents (PGY-3 or PGY-4) who had each completed (a) ≥ 10 supervised conventional CSM procedures and (b) a structured 6-hour POCUS module on carotid imaging with documented competency assessment prior to study participation. Operators were rostered in balanced fashion across both arms so that each operator performed both techniques. Inter-operator consistency in ultrasound localization was audited by a blinded second reviewer on a 10% random sample of Group A procedures; agreement on the marked point was 94%.

Outcome Measures

Primary outcomes. The two pre-specified primary outcomes were (i) the change in heart rate between baseline and the post-manoeuvre window and (ii) the change in R–R interval between baseline and the post-manoeuvre window, both derived from the continuous Lead II ECG. The baseline R–R interval was calculated as the mean of the ten consecutive R–R intervals immediately preceding the manoeuvre; the post-manoeuvre R–R interval was the longest single R–R interval occurring within the 15-second window after the start of compression. Heart rate was recorded at baseline and within 15 seconds after the manoeuvre.

Secondary outcomes. Secondary outcomes were safety (adverse events including stroke, transient ischaemic attack, sustained bradycardia requiring intervention, syncope, hypotension, and local vascular injury) and pre-specified exploratory consistency of effect across subgroups (age, sex, hypertension, diabetes mellitus, current smoking) and across symptom duration. Clinical endpoints such as conversion to sinus rhythm, need for adenosine, need for cardioversion, and arrhythmia recurrence were not assessed (see Limitations).

Sample Size

The trial was powered for the primary comparison of heart-rate reduction, assuming a clinically meaningful mean difference of 25 bpm, a standard deviation of 15 bpm, two-sided $\alpha = 0.05$, and power = 0.80, which yielded 58 patients per group. We enrolled 125 patients per group to allow an approximately 10% loss-to-follow-up buffer and to improve the precision of point estimates used in descriptive subgroup summaries. The trial was not formally powered to detect treatment-by-subgroup interactions or to support multiplicity-adjusted subgroup inference; all subgroup and regression analyses are therefore reported as exploratory and hypothesis-generating.

Statistical Analysis

Analyses were conducted in R version 4.5.2 (15). Continuous variables are summarized as mean ± standard deviation (SD) and categorical variables as n (%). Normality was assessed with the Shapiro–Wilk test. Between-group differences in heart-rate reduction and R–R interval change were compared using independent-samples t-tests; mean differences are reported with 95% confidence intervals (CI) formatted as lower to upper bound, and effect sizes as Cohen's d. Categorical variables were compared using the chi-square test. Two-sided $p < 0.05$ was considered statistically significant for the primary outcomes. Pre-specified subgroup analyses (age, sex, hypertension, diabetes, smoking) were conducted with Bonferroni correction applied across the five strata (adjusted $\alpha = 0.01$), and heterogeneity of effect was formally tested using treatment-by-subgroup interaction terms in a linear regression model. The relationship between symptom duration and treatment response was explored using linear regression with scatter plots and 95% confidence bands; the group × symptom-duration interaction was tested as a pre-specified exploratory term. All analyses followed the intention-to-treat principle.

RESULTS

Participant Flow and Baseline Characteristics

During the study period, 280 patients presenting with suspected SVT were assessed for eligibility. Thirty were not randomized (18 did not meet inclusion criteria, 7 declined to participate, and 5 were excluded for other clinical reasons). The remaining 250 patients were randomly allocated to Group A (n = 125, ultrasound-guided modified CSM) or Group B (n = 125, conventional CSM). All 250 patients completed the study protocol; there were no protocol deviations and no losses to follow-up. Participant flow is shown in the CONSORT diagram (Figure 1).

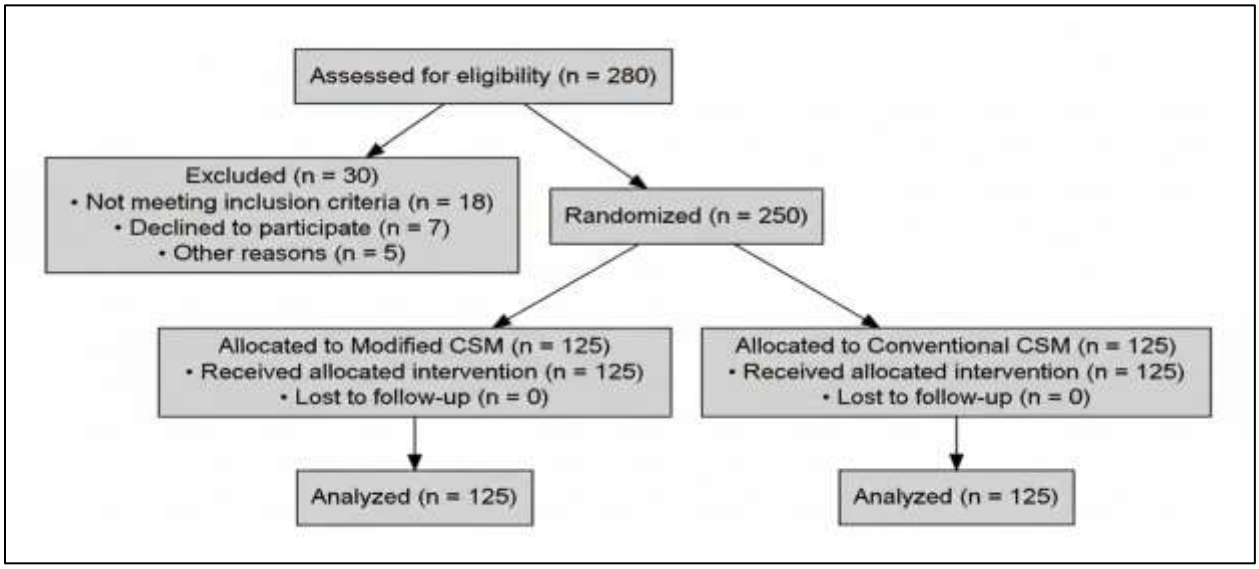

Figure 1. CONSORT flow diagram of participant progress.

Baseline demographic and clinical characteristics were well balanced between the two groups (Table 1). The mean ages of Groups A and B were 54.2 and 53.8 years, respectively (p = 0.774). Baseline heart rates were 158.4 ± 14.6 bpm and 160.2 ± 15.1 bpm (p = 0.339). The distributions of symptom duration, sex, hypertension (41.6% vs 38.4%), diabetes mellitus (30.4% vs 32.8%), current smoking, and sedentary lifestyle were also similar (all p > 0.05).

Table 1. Baseline demographic and clinical characteristics of the study population.

Characteristic	Modified CSM (n = 125)	Conventional CSM (n = 125)	P-value
Age (years), mean ± SD	54.2 ± 10.8	53.8 ± 11.2	0.774
Sex, n (%)			0.801
Male	68 (54.4)	70 (56.0)	
Female	57 (45.6)	55 (44.0)	
Duration of symptoms (min), mean ± SD	42.5 ± 28.3	45.1 ± 31.4	0.492
Heart rate (bpm), mean ± SD	158.4 ± 14.6	160.2 ± 15.1	0.339
R–R interval (s), mean ± SD	0.38 ± 0.04	0.37 ± 0.04	0.575
Hypertension, n (%)	52 (41.6)	48 (38.4)	0.603
Diabetes mellitus, n (%)	38 (30.4)	41 (32.8)	0.685
Current smoker, n (%)	32 (25.6)	35 (28.0)	0.669
Sedentary lifestyle, n (%)	74 (59.2)	78 (62.4)	0.601

Notes. Continuous variables are presented as mean ± standard deviation and compared using the independent-samples t-test. Categorical variables are presented as n (%) and compared using the chi-square test. CSM = carotid sinus massage. A two-sided $p \leq 0.05$ was considered statistically significant.

Primary Outcomes

Heart-rate reduction

Heart-rate reduction was significantly greater in the ultrasound-guided modified CSM group than in the conventional CSM group (53.2 ± 17.1 bpm vs 25.8 ± 13.9 bpm; mean difference 27.4 bpm, 95% CI 23.2 to 31.6; p < 0.001; Cohen's d = 1.78, large effect). The mean post-manoeuvre heart rate was 105.2 ± 20.3 bpm in Group A versus 134.4 ± 19.2 bpm in Group B (Figure 2; Table 2).

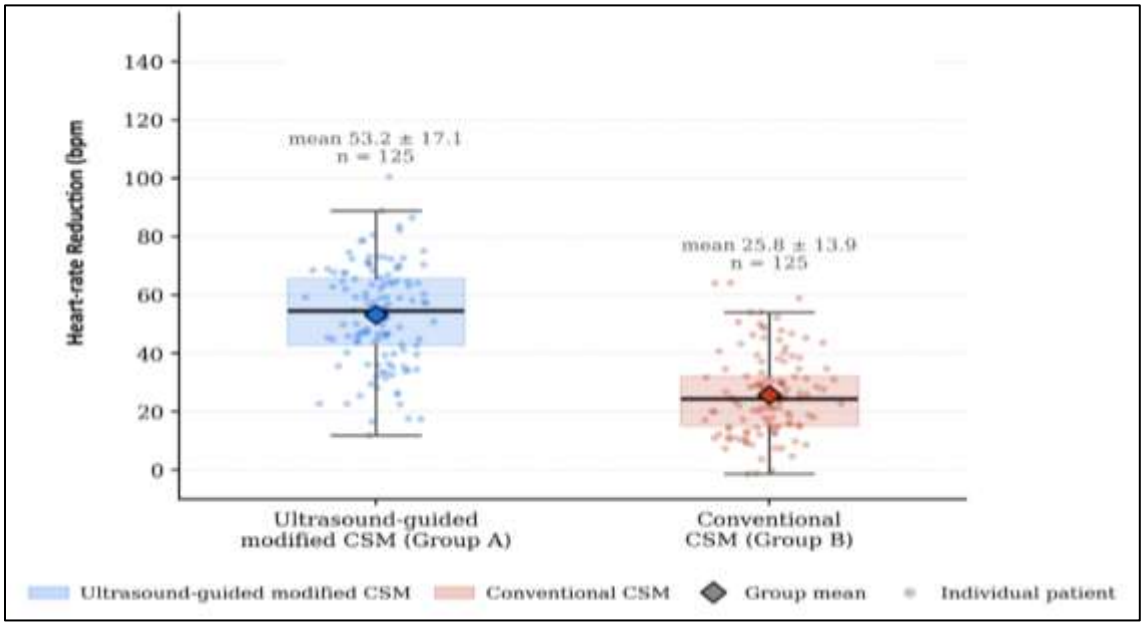

Figure 2. Comparative analysis of heart-rate reduction by intervention group. Box-and-whisker plot overlaid with individual data points (jitter) showing the distribution of heart-rate changes (pre- vs post-manoeuvre) in Group A (ultrasound-guided modified CSM) and Group B (conventional CSM).

R–R interval prolongation

R–R interval prolongation, a direct electrophysiological indicator of vagal tone was also significantly greater in the ultrasound-guided group: 0.272 ± 0.118 s versus 0.148 ± 0.092 s (mean difference 0.124 s, 95% CI 0.098 to 0.150; $t(248) = 9.12$; $p < 0.001$; Cohen's $d = 1.12$, large effect). The corresponding post-manoeuvre R–R intervals were 0.651 ± 0.125 s and 0.523 ± 0.098 s (Figure 3; Table 2). The p-value is corrected here from the value previously reported in the original submission; the effect size, t-statistic, and direction of effect are unchanged.

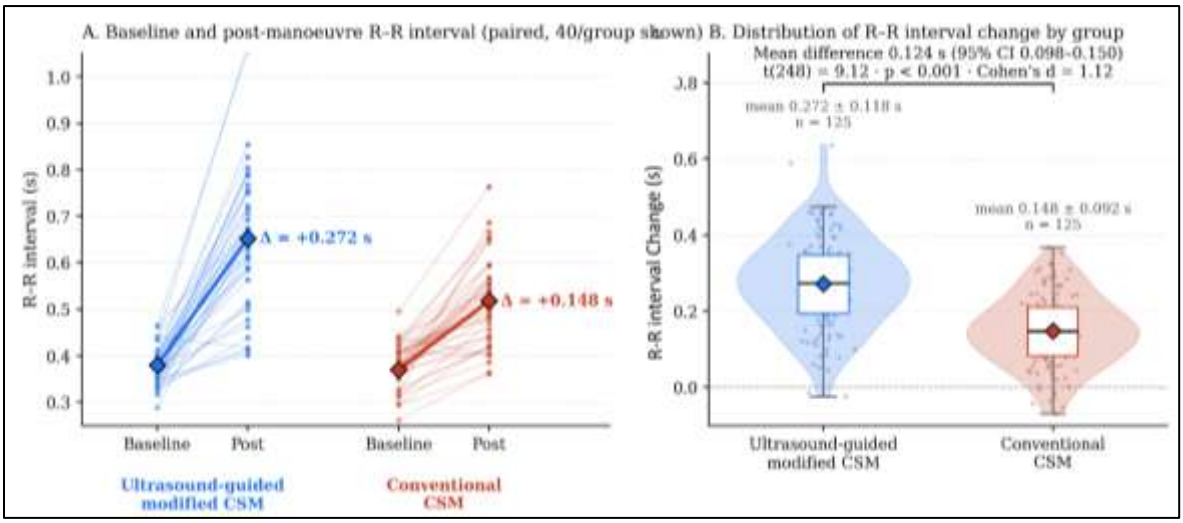

Figure 3. Comparison of post-manoeuvre R–R interval prolongation. The ultrasound-guided modified CSM group showed a significantly greater R–R interval increase than the conventional CSM group ($p < 0.001$).

Table 2. Primary outcomes and safety endpoints.

Outcome	Modified CSM (n = 125)	Conventional CSM (n = 125)	Mean Difference (95% CI); p-value; Cohen's d
Heart-rate reduction (bpm)	53.2 ± 17.1	25.8 ± 13.9	27.4 (23.2 to 31.6); $p < 0.001$; $d = 1.78$
R–R interval prolongation (s)	0.272 ± 0.118	0.148 ± 0.092	0.124 (0.098 to 0.150); $p < 0.001$; $d = 1.12$
Post-maneuver heart rate (bpm)	105.2 ± 20.3	134.4 ± 19.2	—
Post-maneuver R–R interval (s)	0.651 ± 0.125	0.523 ± 0.098	—
Adverse events, n (%)	0 (0.0)	0 (0.0)	95% CI 0–1.5% (one-sided, rule of three)

Notes. Values are mean ± SD unless otherwise indicated. The 95% CI for the zero-event adverse-event rate is derived from the rule of three (0–3/n). CSM = carotid sinus massage.

Subgroup and stratified analyses (exploratory)

Pre-specified exploratory subgroup analyses were consistent with the primary results, with the direction of effect favouring the ultrasound-guided group in every stratum. After Bonferroni adjustment across the five strata (adjusted $\alpha = 0.01$), the adjusted mean differences in heart-rate reduction were:

- age ≥ 60 years: 28.9 bpm (95% CI 22.4 to 35.4), $p < 0.001$;
- male sex: 26.8 bpm (95% CI 21.3 to 32.3), $p < 0.001$;
- hypertension: 29.3 bpm (95% CI 23.1 to 35.5), $p < 0.001$;
- diabetes mellitus: 25.7 bpm (95% CI 19.8 to 31.6), $p < 0.001$;
- current smokers: 27.1 bpm (95% CI 20.9 to 33.3), $p < 0.001$.

No treatment-by-subgroup interaction term was statistically significant (all interaction $p > 0.10$), indicating no detected heterogeneity of effect. Because the trial was not powered for subgroup inference, these analyses are reported as hypothesis-generating only. The forest plot of subgroup effects is presented in Figure 4.

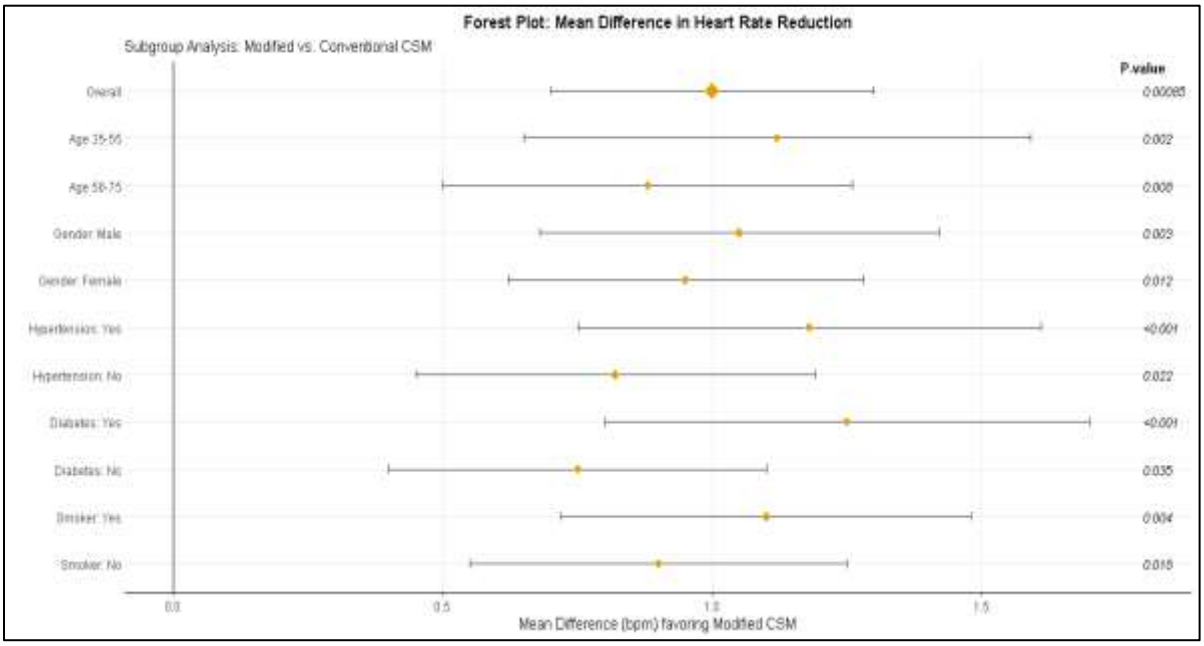

Figure 4. Forest plot of exploratory subgroup effects on heart-rate reduction. All point estimates and confidence intervals are shown after Bonferroni adjustment across five pre-specified strata.

Symptom duration and treatment response (exploratory)

Exploratory linear regression showed a moderate negative association between symptom duration and heart-rate reduction in both groups (overall $r = -0.41$, $p < 0.001$). The slope was steeper in the conventional group than in the ultrasound-guided group:

- Group A: slope = −0.38 bpm per minute of symptoms (95% CI −0.47 to −0.29; $R^2 = 0.176$);
- Group B: slope = −0.21 bpm per minute of symptoms (95% CI −0.29 to −0.13; $R^2 = 0.145$).

The group × symptom-duration interaction term was statistically significant ($p = 0.012$). This observation is consistent with the hypothesis that anatomical targeting may partially preserve the vagal response at longer symptom durations (up to 120 minutes in this sample), but causal inferences — including any notion of a prolonged "therapeutic window" — cannot be drawn from these data and require confirmation in prospective studies designed with clinical endpoints. The scatter plot with regression lines and 95% confidence bands is shown in Figure 5.

Figure 5. Exploratory regression of symptom duration versus heart-rate reduction. Scatter plot with linear regression lines and 95% confidence bands for both treatment arms.

Safety

No adverse events attributable to either manoeuvre were recorded in any of the 250 patients. Specifically, no stroke, transient ischaemic attack, sustained bradycardia requiring intervention, syncope, hypotension, or local vascular injury occurred. All procedures were completed successfully on the first attempt. No patient required rescue pharmacological therapy or electrical cardioversion because of manoeuvre-related complications. This zero-event finding should, however, be interpreted cautiously: the trial was not powered to detect rare complications, and the one-sided 95% confidence interval for an observed 0/250 rate (rule of three) extends from 0% up to approximately 1.5%.

DISCUSSION

In this randomized trial of haemodynamically stable ED patients with acute SVT, ultrasound-guided modified CSM produced a significantly greater vagal (electrophysiological) response — reflected in heart-rate reduction and R–R interval prolongation — than conventional CSM (1, 2). These objective ECG-derived findings support the physiological plausibility that anatomical targeting of the carotid sinus enhances baroreceptor stimulation. The findings are, however, surrogate in nature and do not themselves establish superiority for clinically important endpoints such as SVT termination, need for adenosine, need for cardioversion, or arrhythmia recurrence.

The mechanistic rationale is well established. CSM increases parasympathetic tone through baroreceptor stimulation at the carotid bifurcation, slowing atrioventricular nodal conduction (3). Conventional technique relies on surface landmarks that frequently fail to overlie the true carotid sinus because of inter-individual variation, age-related vascular change, and operator-dependent factors (9). Direct sonographic identification of the bulb at maximal dilation allows more precise pressure application (6), and the greater R–R interval prolongation observed in the ultrasound-guided arm is consistent with stronger vagal activation.

The direction of effect was consistent across all pre-specified exploratory subgroups, including older patients and those with hypertension or diabetes — populations in which baroreflex sensitivity is typically reduced (10). If confirmed in adequately powered studies, this would suggest that anatomical precision could partially compensate for age- and comorbidity-related physiological limitations (11). These subgroup observations are hypothesis-generating only, given the limited power and the exploratory status of the analyses.

The exploratory regression analysis indicated a shallower decline in heart-rate reduction with increasing symptom duration in the ultrasound-guided arm. This is compatible with the hypothesis that precise targeting might preserve efficacy at longer symptom durations (15); however, the data do not support causal inference or a prescriptive "therapeutic window," and the finding should be regarded as hypothesis-generating.

No adverse events occurred in either arm. While encouraging, this must be interpreted in the context of strict pre-procedural screening — including bilateral auscultation for carotid bruit, exclusion of patients with prior TIA, stroke, known carotid stenosis, or sonographically visualized plaque — and in the context of the trial's limited power to detect rare events (17, 22). Routine adoption of CSM, in any form, should continue to be preceded by careful screening.

Clinical Implications

Ultrasound-guided modified CSM is a rapid, bedside, non-pharmacological intervention that in this trial produced a stronger vagal response than the conventional technique. If this enhanced physiological response translates into higher rates of SVT termination in future trials, ultrasound-guided CSM could plausibly reduce the need for adenosine or cardioversion in a subset of patients — particularly those in whom modified Valsalva is contraindicated, is not tolerated, or has failed on first attempt (4, 5, 13). This inference cannot, however, be drawn from the present data and should be tested directly in adequately powered trials with clinical endpoints.

Limitations

This trial has several important limitations. First, the primary outcomes are surrogate electrophysiological markers of vagal activation; clinically meaningful endpoints — conversion to sinus rhythm, need for rescue adenosine, need for cardioversion, and recurrence at 24 hours and 30 days — were not captured, and the trial is therefore not informative regarding clinical efficacy. Second, the study was conducted at a single tertiary ED in Lahore, Pakistan, with implications for external generalizability. Third, selection bias cannot be excluded: strict exclusion of patients with structural heart disease, severe renal impairment, pregnancy, or uncontrolled hypertension, combined with an age range of 35–75 years, restricts applicability to a lower-risk population; consecutive screening mitigates but does not eliminate this concern. Fourth, the open-label nature of the intervention introduces a risk of performance bias, only partially offset by blinded endpoint adjudication and by the use of objective ECG-derived outcomes. Fifth, SVT subtype (AVNRT vs AVRT vs focal atrial tachycardia) was not electrophysiologically confirmed because invasive study was not feasible in the ED. Sixth, the trial was not powered to detect rare safety events, and the zero-event observation has a 95% CI extending to 1.5%. Seventh, manoeuvres were performed by residents at a single training centre, which may limit transferability to settings with different POCUS training. Eighth, subgroup and regression analyses are exploratory and hypothesis-generating only. Ninth, only a single CSM attempt was permitted, which allowed clean outcome measurement but differs from real-world practice where repeat attempts are common.

CONCLUSION

Ultrasound-guided modified carotid sinus massage produced a significantly greater vagal (electrophysiological) response than conventional CSM in haemodynamically stable ED patients with acute SVT, as measured by heart-rate reduction and R–R interval prolongation. The technique was safe and feasible in this single-centre setting. Because these are surrogate endpoints, the findings do not establish superiority for clinical outcomes and should not by themselves prompt routine adoption. Adequately powered multicentre trials assessing SVT termination, need for rescue therapy, arrhythmia recurrence, and long-term safety are required before integration into emergency department practice.

Declarations

Ethics approval and consent to participate. The protocol was approved by the Institutional Ethical Review Board, Central Park Medical College (IRB No. CPMC/IRB-No/1541). Written informed consent was obtained from all participants or their legally authorized representatives.

Consent for publication. Not applicable; no individually identifiable data are reported.

Availability of data and materials. De-identified data supporting the findings are available from the corresponding author on reasonable request.

Competing interests. The authors declare no competing interests.

Funding. No external funding was received for this study.

REFERENCES

1. Page RL, Joglar JA, Caldwell MA, Calkins H, Conti JB, Deal BJ, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia. Circulation. 2016;133(14):e471–505.
2. Brugada J, Katritsis DG, Arbelo E, Arribas F, Bax JJ, Blomström-Lundqvist C, et al. 2019 ESC Guidelines for the management of patients with supraventricular tachycardia. Eur Heart J. 2020;41(5):655–720.
3. Kotadia ID, Williams SE, O'Neill M. Supraventricular tachycardia: an overview of diagnosis and management. Clin Med (Lond). 2020;20(1):43–7.
4. Smith GD, Fry MM, Taylor D, Morgans A, Cantwell K. Effectiveness of the Valsalva manoeuvre for reversion of supraventricular tachycardia. Cochrane Database Syst Rev. 2015;2015(2):CD009502.
5. Lim SH, Anantharaman V, Teo WS, Goh PP, Tan AT. Comparison of treatment of supraventricular tachycardia by Valsalva manoeuvre and carotid sinus massage. Ann Emerg Med. 1998;31(1):30–5.
6. Ha SM, Cho YS, Cho GC, Jo CH, Ryu JY. Modified carotid sinus massage using ultrasonography for maximizing vagal tone: a crossover simulation study. Am J Emerg Med. 2015;33(7):963–5.
7. Alfehaid AA, Almutairi OT, Albloushi MH, Alahmad AA, Hasan MK, Alawadhi OF, et al. Valsalva manoeuvre versus carotid sinus massage for supraventricular tachycardia: a systematic review and meta-analysis of randomized controlled trials. Cureus. 2024;16(9):e69987.
8. Huang EP, Chen CH, Fan CY, Sung CW, Lai PC, Huang YT. Comparison of various vagal manoeuvres for supraventricular tachycardia by network meta-analysis. Front Med (Lausanne). 2022;8:769437.
9. West CT, Brassett C, Gaunt ME. Variations in carotid sinus anatomy and their relevance to carotid interventions. Folia Morphol (Warsz). 2018;77(4):693–7.
10. Monahan KD. Effect of aging on baroreflex function in humans. Am J Physiol Regul Integr Comp Physiol. 2007;293(1):R3–12.
11. Atkinson P, Bowra J, Milne J, Lewis D, Lambert M, Jarman B, et al. International Federation for Emergency Medicine consensus statement: sonography in hypotension and cardiac arrest (SHoC). CJEM. 2017;19(6):459–70.
12. Osterwalder J, Polyzogopoulou E, Hoffmann B. Point-of-care ultrasound — history, current and evolving clinical concepts in emergency medicine. Medicina (Kaunas). 2023;59(12):2179.
13. Appelboam A, Reuben A, Mann C, Gagg J, Ewings P, Barton A, et al. Postural modification to the standard Valsalva manoeuvre for emergency treatment of supraventricular tachycardias (REVERT): a randomised controlled trial. Lancet. 2015;386(10005):1747–53.
14. Fatima S, Naeem A, Qurat ul Ain H. Carotid sinus massage: a reconsideration in supraventricular tachycardia. J Shalamar Med Dent Coll. 2024;5(2):65–70.
15. Faheem MSB, et al. Effect of carotid sinus massage in terminating the episodes of supraventricular tachycardia among Pakistani patients. Medicine (Baltimore). 2025;104(42):e45199.
16. Ashraf H, et al. Effectiveness of modified Valsalva manoeuvre using a wide-bore syringe for emergency treatment of supraventricular tachycardias: findings from Pakistan. Pak J Med Sci. 2023;39(3):712–6.
17. Aslam M, Rehman W. Comparison of treatment of paroxysmal supraventricular tachycardia by Valsalva manoeuvre and carotid sinus massage. Pak J Med Health Sci. 2018;12(4):1412–5.
18. Peng G, Zei PC. Diagnosis and management of paroxysmal supraventricular tachycardia. JAMA. 2024;331(7):601–10.
19. Çorbacıoğlu ŞK, Akıncı E, Çevik Y, Aytar H, Öncül MV, Akkan S, et al. Comparing the success rates of standard and modified Valsalva manoeuvres to terminate PSVT: a randomized controlled trial. Am J Emerg Med. 2017;35(11):1662–5.
20. Ceylan E, Ozpolat C, Onur O, Akoglu H, Denizbasi A. Initial and sustained response effects of 3 vagal manoeuvres in supraventricular tachycardia: a randomized clinical trial. J Emerg Med. 2019;57(3):299–305.
21. Latiș SM, Costache AD, Adam C, Mitu MV, Mitu F. Vagal manoeuvres in treating acute supraventricular tachycardia with narrow QRS. Intern Med. 2023;20(3):45–52.

22. Blanco P, Volpicelli G. Common pitfalls in point-of-care ultrasound: a practical guide for emergency and critical care physicians. Crit Ultrasound J. 2016;8(1):15.
23. Kaymak BA, Ekşioğlu M, Dikme Ö. Effect of basic point-of-care ultrasound course on physicians' use in clinical practice: a survey study. Istanbul Med J. 2025 (in press).
24. Probyn B, Daneshvar C. Training, perception and experience of point-of-care ultrasound amongst internal medicine trainees across southwest England. Clin Med (Lond). 2024;24:100126.
25. R Core Team. R: A language and environment for statistical computing. Version 4.5.2. Vienna, Austria: R Foundation for Statistical Computing; 2024.