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Research Article

Transthoracic Coronary Sinus Blood Flow Monitoring and PC6 Transcutaneous Electrical Acupoint Stimulation in Elderly Hip Fracture Surgery: A Three-phase Randomised Trial

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Abstract

Background Perioperative myocardial injury in elderly hip fracture patients is usually silent and arises mainly from oxygen supply–demand mismatch. Transthoracic echocardiographic coronary sinus blood flow (CSBF) has been proposed as a non-invasive window on coronary perfusion, but its obtainability in this frail population is unknown. This trial assessed whether a definitive trial of PC6 transcutaneous electrical acupoint stimulation (TEAS) using such monitoring is feasible. *Methods* Three-phase, single-centre, sham-controlled randomised pilot and feasibility trial (June 2023–May 2025). Phase I (n = 30) assessed CSBF obtainability and acute responses to bilateral PC6 TEAS; Phase II (n = 60) explored stimulation parameters in a 2 × 2 factorial (2 vs. 5 Hz; 15 vs. 25 min); Phase III (n = 40) applied the leading combination under standardised total intravenous anaesthesia. Primary outcomes were feasibility outcomes: CSBF obtainability, predictors of measurement failure, recruitment, retention and safety. Clinical measures were secondary and exploratory, and progression criteria were pre-specified. The study was not powered to detect efficacy effects. *Results* Adequate CSBF spectra were obtainable in 64% of patients (55/85, Phases I–II). Chronic obstructive pulmonary disease (adjusted odds ratio 3.45), body mass index above 25 kg/m^2 (2.87) and thoracic kyphosis (3.21) independently predicted failure; obtainability reached 90.5% when none was present but fell to 56.8% intraoperatively. Recruitment averaged 4.4 patients/month, no patient was lost to follow-up, and no adverse event was attributable to TEAS. Exploratory estimates: coronary sinus velocity–time integral rose 20.8% (95% CI 11.2–30.4) without change in mean arterial pressure; rate-pressure product remained below 12,000 throughout in the TEAS arm, with a pooled between-group separation of 1759 (95% CI 876–2642) at peak-stress time points. *Discussion* A definitive trial is feasible, but not with transthoracic CSBF as its monitoring backbone: the technique fails in approximately a third of patients, and selectively in those at highest cardiovascular risk. We recommend proceeding with an amended protocol that substitutes pulse-wave analysis. The haemodynamic signals reported here are hypothesis-generating. *Trial registration Chinese Clinical Trial Registry (https://www.chictr.org.cn), ChiCTR2600126845; registered 17 June 2026, retrospectively registered. Recruitment ran from June 2023 to May 2025, so registration followed completion of recruitment.*

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Keywords

Pilot Study, Transcutaneous Electrical Acupoint Stimulation, PC6, Coronary Sinus Blood Flow, Rate-pressure Product, Hip Fracture, Elderly

1. Introduction

Hip fracture in the elderly carries substantial perioperative cardiovascular risk, and perioperative myocardial infarction is a recognised and prognostically important complication in this population [1]. The typical patient is over 80 years old, carries two or three chronic diseases, and has limited physiological reserve to absorb the haemodynamic insult of surgery and anaesthesia.

Perioperative cardiac risk in this population is largely silent. In hip fracture cohorts, myocardial injury after non-cardiac surgery (MINS) is identified predominantly by routine troponin surveillance rather than by symptoms, and its occurrence is associated with worse postoperative outcome [2, 3].

The pathophysiology differs from that seen in the catheterisation laboratory. Optical coherence tomography studies in perioperative myocardial infarction have shown coronary thrombus in only about 13% of cases versus 67% in non-operative MI [4], and the 2021 American Heart Association Scientific Statement [5] and 2022 European Society of Cardiology guidelines [6] conclude that the vast majority of perioperative myocardial injury arises from oxygen supply–demand mismatch. The 2024 AHA/ACC/multisociety guideline assigns preoperative cardiac troponin only a Class 2b (Level B-NR) recommendation, below the Class 2a assigned to natriuretic peptides [7]. Two large randomised trials have further shown that intraoperative blood-pressure targeting alone does not reduce myocardial injury: POISE-3 BP demonstrated that a 20-mmHg separation in mean arterial pressure target did not change MINS incidence [8], and IMPROVE-multi reinforced this null result with individualised MAP targeting [9]. Together, these findings indicate that haemodynamic optimisation and myocardial protection are distinct constructs.

Rate-pressure product (RPP), the arithmetic product of systolic blood pressure and heart rate, was originally validated against directly measured coronary sinus myocardial oxygen consumption [10] and confirmed under surgical anaesthesia [11], and it retains prognostic relevance in the elderly hip fracture population: elevated perioperative RPP has been shown to predict postoperative delirium through an IL-6-mediated pathway in exactly this cohort [12]. RPP nevertheless ignores wall stress and can underestimate myocardial oxygen consumption in dilated or hypertrophied ventricles [13], a caveat we return to in the Discussion.

Transcutaneous electrical acupoint stimulation (TEAS) at PC6 has accumulated evidence as a perioperative cardioprotective intervention. Li and colleagues randomised 122 elderly coronary disease patients undergoing non-cardiac surgery to TEAS at PC6 plus PC4 or sham and observed reductions in postoperative high-sensitivity troponin T on days 1, 3, and 5 with heart-rate-variability shifts indicating attenuated sympathetic activation [14]; Wang and colleagues randomised 388 patients (204 completing full follow-up) and reported that a single pre-procedural electroacupuncture session reduced peri-PCI myocardial injury and two-year major adverse cardiovascular events (MACE) [15]; and Feng and colleagues randomised 232 stable ischaemic heart disease patients and reported a 12-month MACE rate of 8.4% with TEAS versus 19.3% with control ($p = 0.021$) [16]. The classical PC6 antiemetic effect remains the best-established human PC6 indication [17].

Coronary sinus blood flow (CSBF), which drains approximately three-quarters of total myocardial venous return, has been proposed as a non-invasive window on global coronary perfusion. Transthoracic echocardiographic (TTE) measurement of CSBF has been validated in coronary artery disease patients [18, 19], but published success rates for obtaining adequate Doppler spectra vary widely, and the technique has not been systematically evaluated in the elderly hip fracture population.

Before a definitive trial could be designed, two uncertainties had to be resolved: how often transthoracic CSBF measurement actually succeeds in elderly hip fracture patients, and which patients are unsuitable for it. It was equally unclear whether stimulation parameters for perioperative PC6 TEAS could be established in this population, and no estimate of recruitment, retention or tolerability was available. We therefore conducted a three-phase randomised pilot and feasibility trial whose primary objectives were feasibility objectives. The trial is reported in accordance with the CONSORT 2010 extension for randomised pilot and feasibility trials [20] (Additional file 1) and the intervention is described using the TIDieR checklist (Additional file 2). The study was not designed or powered to test the effectiveness of TEAS, and no efficacy claim is made.

1.1. Objectives

The primary (feasibility) objectives were to determine: (i) the proportion of patients in whom adequate transthoracic CSBF Doppler spectra could be obtained, and the patient characteristics predicting failure; (ii) the achievable recruitment rate and retention; and (iii) the tolerability and safety of preoperative PC6 TEAS in frail geriatric skin. A fourth objective was to identify a candidate stimulation frequency and duration to carry into

a definitive trial. Secondary, exploratory objectives were to obtain provisional estimates of the direction and variability of coronary sinus velocity–time integral (CS-VTI), cardiac output and rate-pressure product (RPP) responses, to inform, but not to determine, the design of a future trial. These objectives differ from those of the planned definitive trial, whose primary endpoint will be a troponin-defined measure of myocardial injury.

1.2. Key Messages Regarding Feasibility

What uncertainties existed regarding feasibility?

1) Could adequate transthoracic coronary sinus blood flow (CSBF) Doppler spectra be obtained in elderly hip fracture patients, and in what proportion?
2) Which patient characteristics predict measurement failure?
3) Could recruitment and retention at a single orthopaedic centre support a definitive trial?
4) Would preoperative PC6 TEAS be tolerated in frail geriatric skin?
5) Which stimulation frequency and duration should be carried forward?

What are the key feasibility findings?

1) Adequate CSBF spectra were obtainable in only 64% of patients, falling to 56.8% intraoperatively.
2) Measurement failed selectively in patients with chronic obstructive pulmonary disease, body mass index above 25 kg/m^2 or thoracic kyphosis, the phenotypes at highest perioperative cardiovascular risk.
3) Recruitment (4.4 patients/month), retention (no losses to follow-up) and safety (no attributable adverse events) were all acceptable.
4) 2 Hz for 25 min was selected as the candidate stimulation combination.

What are the implications of the feasibility findings for the design of the main study?

1) A definitive trial should proceed, but must abandon transthoracic CSBF as a generalised monitoring tool and substitute radial arterial tonometry with pulse-wave analysis.
2) Cardiac biomarkers, a covered sham device to blind the anaesthesiologist, and multicentre recruitment must be added.
3) The haemodynamic estimates reported here are too imprecise to power that trial; a conservative, externally derived effect size should be used instead.

2. Methods

2.1. Design

This randomised, sham-controlled, three-phase pilot and feasibility trial, with prospective data collection, was conducted at No. 1 Orthopedics Hospital of Chengdu between June 2023 and May 2025. Ethics approval, informed consent and the circumstances of trial registration are described under Ethical considerations; the pre-specified progression criteria are given under Study measures.

2.2. Ethical Considerations

The study was approved by the Ethics Committee of No. 1 Orthopedics Hospital of Chengdu (approval number 2022-02-03) and was conducted in accordance with the 2013 revision of the Declaration of Helsinki. Written informed consent was obtained from every participant before any study-specific procedure. Before the first participant was enrolled, the protocol, the primary feasibility outcomes, the secondary outcomes and the progression criteria were approved by the institutional ethics committee and were filed by the principal investigator with the National Medical Research Registration and Filing System, the record-filing system operated by the National Health Commission. The investigating team took that filing to constitute trial registration. It does not: the system is a national regulatory filing, is not publicly searchable, and is not a WHO primary registry, so it does not satisfy the requirement for prospective public registration. Because the principal investigator had completed only that filing, and this was not communicated within the wider team, the absence of a public registry entry went unnoticed until the present report was being prepared three years later. Registration with the Chinese Clinical Trial Registry was then completed on 17 June 2026 (ChiCTR2600126845). Since recruitment ran from June 2023 to May 2025, that registration is retrospective and post-dates completion of recruitment, and the registry record is flagged accordingly. None of the outcomes, the progression criteria or the analysis plan was altered after enrolment began; the ethics-approved protocol and the pre-enrolment filing record are available to the editors on request so that this can be verified. The ethics committee did not permit research-only venepuncture in this frail cohort, which is why cardiac biomarkers were not measured.

2.3. Sample

We recruited patients aged 70 years or older scheduled for elective hip fracture surgery (femoral neck or intertrochanteric fractures) with ASA class II–V, BMI 18–30 kg/m^2, and Mini-Mental State Examination score ≥ 28. We excluded patients with uncontrolled hypertension (SBP ≥ 160 or DBP ≥ 100 mmHg), diabetes with glucose ≥ 15.0 mmol/L, polytrauma, psychiatric illness or substance abuse, severe hepatorenal disease, pulmonary hypertension, heart failure, haemodynamically significant shunt lesions, skin breakdown at the acupoint sites, or implanted cardiac devices.

2.4. Study Procedure

2.4.1. Randomisation and Blinding

A research coordinator not involved in patient care generated the allocation sequence in SPSS 22.0 and sealed assignments in

opaque, sequentially numbered envelopes. Patients were informed in advance that stimulation might feel very faint or imperceptible, preserving patient blinding. The sonographer and statistician remained blinded throughout. The clinician delivering the TEAS was necessarily aware of allocation but took no part in data collection or analysis. The attending anaesthesiologist was unblinded.

2.4.2. Intervention and Sham Comparator

Stimulation was delivered with a Huatuo SDZ-V electronic acupuncture device (Suzhou Medical Appliance Factory). Bilateral PC6 acupoints were located 5–6 cm proximal to the wrist crease between the palmaris longus and flexor carpi radialis tendons. After skin preparation, 4×4 cm self-adhesive electrodes were applied. The device operated in dense–disperse wave mode; current was titrated from zero until the patient reported a comfortable sensation of numbness and distension (de qi), typically at 6–9 mA. Sham patients received identical electrode placement with the display switched on but no current delivered. Draping was identical in both arms.

2.4.3. Three-phase Structure

Phase I (n = 30). Fifteen patients received TEAS (2 Hz, 20 min) and 15 sham. Echocardiography (Mindray M9, P4-2s phased-array probe) was performed by a single sonographer before (T0) and within 5 min of stimulation completion (T1). Coronary sinus imaging used a fixed sequence of three acoustic windows; a measurement was judged adequate when Doppler interrogation angle was < 60 °and the sample volume lay within 1 cm of the coronary sinus ostium. CS-VTI was averaged over three consecutive cardiac cycles at quiet respiration; failures were logged with reason.

Phase II (n = 60). A 2 ×2 factorial design varied frequency (2 vs. 5 Hz) and duration (15 vs. 25 min), with 15 patients per cell. Measurements followed Phase I. We report Phase II strictly as cell-level descriptive statistics with 95% confidence intervals; per-cell evaluable samples of 9–10 patients are too small to support factorial hypothesis testing [20-22].

Phase III (n = 40). The most promising Phase II parameter combination (2 Hz, 25 min) was tested under standardised anaesthesia. Every patient first received an ultrasound-guided suprainguinal fascia iliaca block with 30 mL 0.33% ropivacaine. After preoxygenation, anaesthesia was induced with midazolam 0.02–0.03 mg/kg, sufentanil 0.2–0.3 μg/kg, etomidate 0.2–0.3 mg/kg, and rocuronium 0.5–0.6 mg/kg. Total intravenous anaesthesia used propofol 4–8 mg/kg/h and remifentanil 0.05–0.15 μg/kg/min, targeting BIS 40–60, to avoid the confounding cardiac-preconditioning effect of volatile agents [23]. Ventilation was volume-controlled (VT 6–8 mL/kg predicted body weight, PEEP 5 cmH2O, FiO2 0.4–0.6). Crystalloid ran at 3–5 mL/kg/h. Hypotension was pre-specified as MAP < 65 mmHg for ≥ 2 min [24, 25]; the first-line vasopressor was phenylephrine 50 μg (fixed dose), repeated to three boluses; the second-line agent was ephedrine 6 mg. Vasopressor doses were converted to phenylephrine equivalents (ephedrine 10 mg = phenylephrine 100 μg). The TEAS group received bilateral PC6 stimulation at 2 Hz for 25 min in the preoperative holding area, finishing 10–15 min before transfer. Controls received sham.

2.5. Study Measures

2.5.1. Primary Feasibility Measures

The primary outcomes were feasibility outcomes: (i) CSBF obtainability, defined a priori as the proportion of attempted examinations yielding a Doppler interrogation angle below 60 ° with the sample volume within 1 cm of the coronary sinus ostium and a reproducible trace over three consecutive cardiac cycles; (ii) patient-level predictors of measurement failure; (iii) recruitment rate, expressed as patients randomised per month; (iv) retention, expressed as the proportion of randomised patients providing analysable primary-outcome data; and (v) safety and tolerability, assessed by structured 24-hour inspection of the electrode sites and by recording any adverse event attributable to the procedure.

2.5.2. Secondary and Exploratory Clinical Measures

Outcomes. The Phase III primary endpoint was rate-pressure product (RPP = SBP × HR) at five time points: B1 (room entry), B2 (post-block, pre-induction), B3 (5 min post-intubation), B4 (skin incision), and B5 (5 min post-extubation). Secondary outcomes were total phenylephrine-equivalent vasopressor dose, hypotensive-episode count, time-weighted MAP < 65 mmHg exposure, 24-hour visual analogue scale pain scores, and a 30-day composite cardiovascular event count (death, non-fatal MI, new heart failure, new atrial fibrillation, or cardiovascular rehospitalisation). All clinical measures were secondary and exploratory. As recommended for pilot trials [20-22], they are reported as estimates with 95% confidence intervals to characterise direction and variability, and are not intended to establish effectiveness.

2.5.3. Progression Criteria

Progression criteria for a definitive trial were pre-specified: (i) CSBF obtainability of at least 60%; (ii) recruitment of at least four patients per month; (iii) absence of serious adverse events attributable to TEAS; and (iv) a haemodynamic effect-size estimate whose lower 95% confidence bound excluded zero. Criteria (i)–(iii) were treated as decisive. Criterion (iv) was pre-specified as a supportive, exploratory indicator only; consistent with current guidance we did not treat an efficacy-based threshold as determinative of progression, and we report it here for transparency rather than as a stop–go rule. A traffic-light interpretation was applied: all decisive criteria met, proceed; one unmet, proceed with protocol amendment; two or more unmet, do not proceed.

2.6. Sample Size

Following Whitehead and colleagues [21] and the Eldridge et al. framework [20], we enrolled 15 per group in Phases I–II and 20 per group in Phase III, sufficient to estimate variance and effect-size point estimates for a definitive trial. Phase II per-cell samples of 9–10 evaluable patients were pre-specified as sufficient for parameter-exploration ranking but too small for factorial hypothesis testing [22].

2.7. Data Analysis

Normality was checked with Shapiro–Wilk tests. A two-sided p value below 0.05 was prospectively determined to indicate significance for the primary endpoint. Continuous variables are summarised as mean ± SD or median (interquartile range), with between-group differences as point estimates and 95% confidence intervals calculated by Welch's method; differences in proportions used the Newcombe hybrid-score method [22]. Phase II is reported as cell-level descriptive statistics with pairwise Cohen's d and 95% CIs, each derived from the sample contributing that estimate (the CSBF-evaluable subgroup for ΔCS-VTI and ΔCO, all randomised patients for ΔHR and ΔRPP); factorial ANOVA p values were omitted to avoid Type I error and the "winner's curse" in underpowered designs [20, 22]. Phase III used repeated-measures ANOVA with Greenhouse–Geisser correction, the group × time interaction being the key inferential test. Degrees of freedom in Additional file 3 (Table S2) are uncorrected while p values incorporate the correction. Sensitivity analyses for unblinded pressor administration compared the proportion receiving any vasopressor and time-weighted exposure to MAP below 65 mmHg [26]. Primary and secondary endpoints were pre-specified. No multiplicity adjustment was made: under a pilot estimation framework, secondary results are effect-size estimates with their uncertainty, and intervals including zero are described as directional. Analyses used SPSS 22.0 (IBM Corp., Armonk, NY).

No formal power calculation was performed for any clinical measure, and the study is not powered to detect differences in them. Hypothesis testing on clinical measures is reported only where pre-specified, is secondary to the feasibility results, and should be interpreted with corresponding caution [20, 22]. Feasibility outcomes are summarised descriptively as proportions with 95% confidence intervals.

3. Results

3.1. Participant Flow, Recruitment and Retention

The feasibility outcomes are summarised against the pre-specified progression criteria in Table 1. Participant flow is reported first for Phase I, followed by the pooled figures for all three phases. Of 42 patients screened for Phase I, 30 met eligibility and were randomised (Figure 1). One TEAS patient withdrew (electrode-adhesive skin irritation) and one control was excluded (unscheduled atenolol dose), leaving 14 patients per group, well matched demographically (Table 2): mean age 76.2 ± 5.0 years, 61% female, hypertension in 61%, diabetes in 32%.

Table 1. *Feasibility outcomes assessed against pre-specified progression criteria.*

Feasibility outcome	Pre-specified progression criterion	Observed value	Criterion met
CSBF obtainability, Phases I–II	≥ 60% of attempted examinations	64.7% (55/85; 95% CI 54.1–74.1)	Yes
CSBF obtainability, intraoperative (Phase III)	No separate threshold pre-specified	56.8% (21/37)	Below overall threshold
Recruitment rate	≥ 4 patients randomised per month	4.4 patients/month (130 over 24 months)	Yes
Retention	No formal threshold; reported descriptively	0 lost to follow-up; 122/130 analysable	Yes
Safety and tolerability	No serious adverse event attributable to TEAS	0 attributable serious adverse events	Yes
Candidate parameter identified	Rank ordering across four cells	2 Hz for 25 min	Yes
Haemodynamic effect estimate (supportive only)	Lower 95% confidence bound excluding zero	ΔRPP met; ΔCS-VTI met marginally	Partially

The first five rows are the primary feasibility outcomes. The final row is an exploratory, supportive indicator and was not treated as a stop–go rule. Under the pre-specified traffic-light interpretation, the overall result corresponds to "proceed with protocol amendment". CI confidence interval, CSBF coronary sinus blood flow, CS-VTI coronary sinus velocity–time integral, RPP rate-pressure product, TEAS transcutaneous electrical acupoint stimulation.

Across the three phases, 130 patients were randomised over a 24-month enrolment window, a mean recruitment rate of 4.4 patients per month, exceeding the pre-specified threshold of four per month (Table 1). Retention was complete: no patient was lost to follow-up. Eight patients were excluded after randomisation: two in Phase I (one electrode-adhesive skin irritation, one unscheduled beta-blocker dose), three in Phase II (protocol deviations) and three in Phase III (one surgical plan change, one equipment failure and one midazolam overdose). This left 122 patients contributing analysable data (28 in Phase I, 57 in Phase II and 37 in Phase III). Exclusions after randomisation should be distinguished from loss to follow-up, of which there was none. Participant flow is shown in Figure 1 and Phase I baseline characteristics, which were well matched between arms, in Table 2.

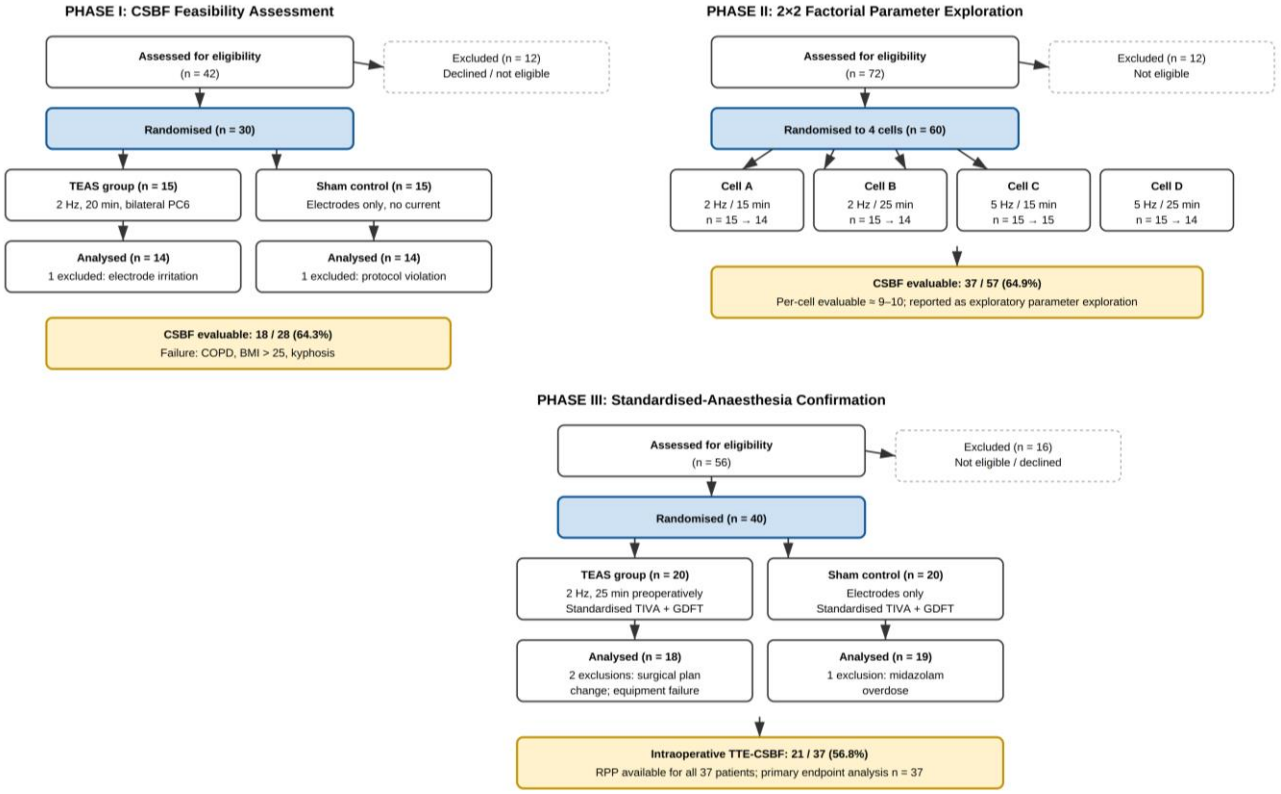


Figure 1. *CONSORT flow diagram for the three-phase randomised pilot and feasibility trial.*

Table 2. *Baseline characteristics of Phase I participants.*

Variable	TEAS (n = 14)	Control (n = 14)	t / χ^2	p
Age, years	76.4 ±4.8	75.9 ±5.2	0.264	0.793
Female, n (%)	9 (64.3)	8 (57.1)	0.155	0.694
Body mass index, kg/m^2	23.6 ±3.1	24.1 ±2.8	−0.454	0.651
ASA class II/III/IV, n	4/8/2	5/7/2	0.273	0.872
Hypertension, n (%)	9 (64.3)	8 (57.1)	0.155	0.694
Diabetes, n (%)	4 (28.6)	5 (35.7)	0.175	0.677
COPD, n (%)	2 (14.3)	3 (21.4)	0.239	0.626
Coronary artery disease, n (%)	3 (21.4)	2 (14.3)	0.239	0.626
Baseline heart rate, beats/min	74.2 ±9.8	73.5 ±10.4	0.185	0.854
Baseline mean arterial pressure, mmHg	91.3 ±12.1	89.7 ±11.6	0.359	0.724

Variable	TEAS (n = 14)	Control (n = 14)	t / χ^2	p
Baseline rate-pressure product	10,085 ±1623	9946 ±1578	0.229	0.824
Femoral neck/intertrochanteric, n	8/6	7/7	0.143	0.710

Values are mean ±SD or count (%). ASA American Society of Anesthesiologists physical status, COPD chronic obstructive pulmonary disease.

3.2. Primary Feasibility Outcome: CSBF Obtainability and Predictors of Failure

Adequate CSBF Doppler spectra were obtainable in 18 of 28 evaluable patients in Phase I (64.3%) and 37 of 57 in Phase II (64.9%), giving a pooled obtainability of 55 of 85 attempted examinations (64.7%, 95% CI 54.1–74.1%). This exceeded the pre-specified 60% threshold, but only marginally. Intraoperatively in Phase III, obtainability fell to 21 of 37 (56.8%), below the threshold.

Multivariable logistic regression identified three independent predictors of measurement failure: chronic obstructive pulmonary disease (adjusted odds ratio 3.45, 95% CI 1.12–10.64), body mass index above 25 kg/m^2 (2.87, 95% CI 1.04–7.93) and thoracic kyphosis (3.21, 95% CI 0.98–10.52) (Table 3). Among the 42 patients with none of these characteristics, obtainability was 90.5% (38/42). The technique thus fails selectively in patients with the least cardiopulmonary reserve: the very patients in whom perioperative supply–demand monitoring would be most valuable.

Table 3. *Independent predictors of transthoracic coronary sinus blood flow measurement failure.*

Predictor	Prevalence, n (%)	Adjusted OR (95% CI)	p
COPD	15/85 (17.6)	3.45 (1.12–10.64)	0.031
BMI > 25 kg/m^2	28/85 (32.9)	2.87 (1.04–7.93)	0.042
Thoracic kyphosis	12/85 (14.1)	3.21 (0.98–10.52)	0.054
None of the above	42/85 (49.4)	Reference	—

Multivariable logistic regression on the pooled Phase I–II cohort in whom measurement was attempted (n = 85). Measurement was obtainable in 90.5% (38/42) of patients with none of the three predictors. BMI body mass index, CI confidence interval, COPD chronic obstructive pulmonary disease, OR odds ratio.

3.3. Primary Feasibility Outcome: Safety and Tolerability

No adverse event was attributable to the TEAS procedure in any phase. Structured 24-hour visual assessment of the electrode application sites revealed no skin tears, persistent erythema, electrode-edge micro-blistering or thermal injury under dense–disperse stimulation at 6–9 mA, with the single exception of one Phase I participant withdrawn for adhesive contact dermatitis. Tolerability in frail geriatric skin was therefore judged acceptable.

3.4. Candidate Parameter Selection (Phase II)

Fifty-seven of 60 enrolled patients were analysed (three protocol deviations); adequate CSBF spectra were obtained in 37 (64.9%), leaving 9–10 evaluable patients per cell. Consistent with the pilot-trial framework [20-22], Phase II is reported as cell-level descriptive statistics with 95% CIs; factorial ANOVA p-values were omitted. Cell-level mean ΔCS-VTI estimates were 12.4% (95% CI 6.4–18.4%) at 2 Hz/15 min, 20.3% (95% CI 13.4–27.2%) at 2 Hz/25 min, 14.1% (95% CI 7.6–20.6%) at 5 Hz/15 min, and 17.8% (95% CI 10.4–25.2%) at 5 Hz/25 min. The rank ordering for ΔRPP placed 2 Hz/25 min first (−9.7%; 95% CI −13.2 to −6.2%), followed by 5 Hz/25 min (−7.2%), 2 Hz/15 min (−5.8%), and 5 Hz/15 min (−4.3%). The between-cell Cohen's d comparing 2 Hz/25 min with the 2 Hz/15 min reference was 0.94 (95% CI 0.01–1.91) for ΔCS-VTI, estimated in the 9–10 CSBF-evaluable patients per cell, and 0.82 (95% CI 0.07–1.55) for ΔRPP, estimated in all 14 randomised patients per cell. The lower confidence bound for ΔCS-VTI lies immediately above zero, so this effect size is compatible with a negligible true difference. These outputs identify 2 Hz/25 min as the leading candidate parameter combination for confirmatory testing; they do not constitute statistical confirmation of parameter superiority. Cell-level means and between-cell effect sizes for all four endpoints are summarised in Table 4 and displayed in Figure 2.

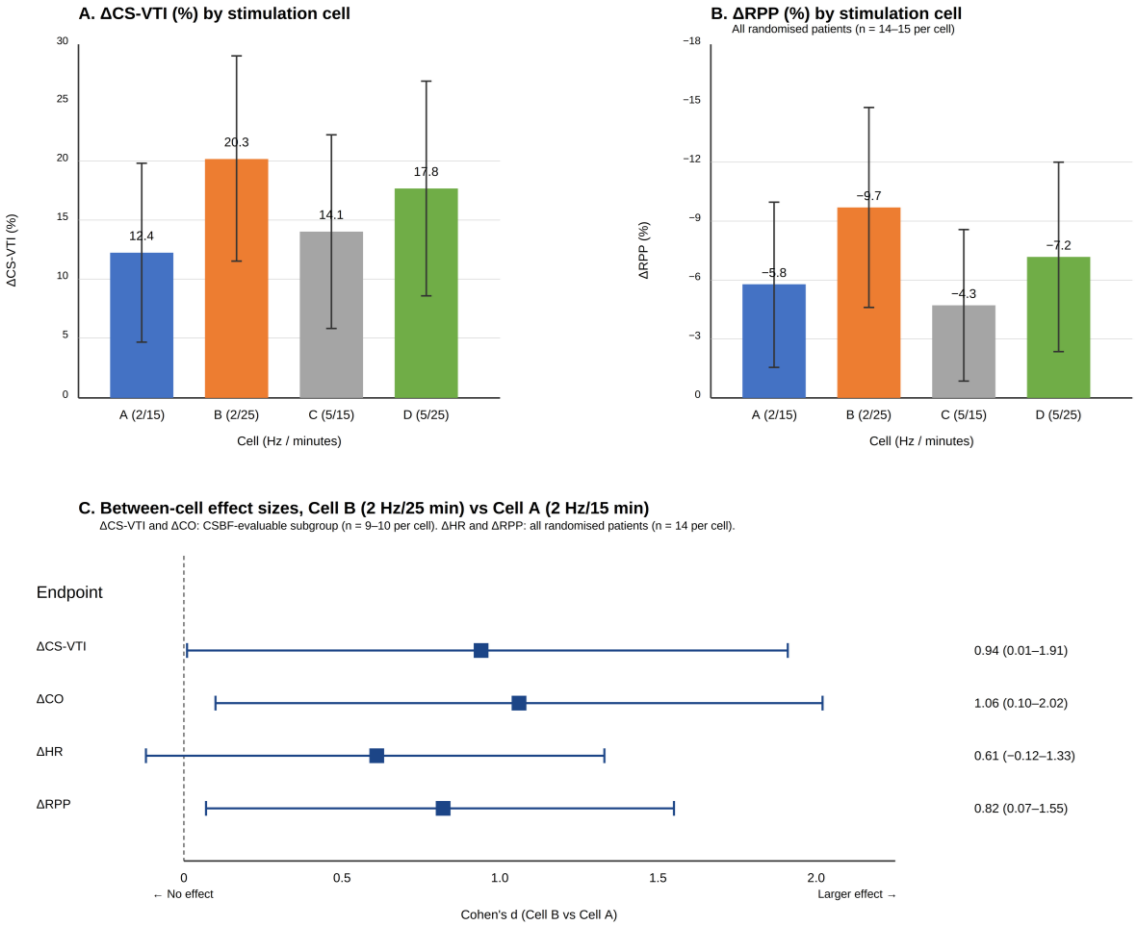


Figure 2. *Exploratory parameter-exploration signals in Phase II. (a) ΔCS-VTI (%) by stimulation cell, CSBF-evaluable subgroup (n = 9–10 per cell); (b) ΔRPP (%) by stimulation cell, all randomised patients (n = 14–15 per cell); (c) between-cell Cohen's d with 95% confidence intervals comparing Cell B (2 Hz/25 min) with Cell A (2 Hz/15 min). Bars and points show mean ± SD.*

Table 4. *Exploratory parameter-exploration estimates, Phase II (mean ± SD percentage change from T0 to T1).*

Measure	Cell A 2 Hz/15 min	Cell B 2 Hz/25 min	Cell C 5 Hz/15 min	Cell D 5 Hz/25 min	Cohen's d, B vs A (95% CI)
ΔCS-VTI, % [a]	+12.4 ± 7.6	+20.3 ± 8.7	+14.1 ± 8.2	+17.8 ± 9.1	0.94 (0.01 to 1.91)
ΔCardiac output, % [a]	+8.7 ± 5.3	+15.2 ± 6.8	+9.4 ± 5.9	+13.1 ± 6.4	1.06 (0.10 to 2.02)
ΔHeart rate, %	−2.1 ± 3.4	−4.3 ± 3.8	−1.8 ± 3.1	−2.9 ± 3.5	0.61 (−0.12 to 1.33)
ΔRate-pressure product, %	−5.8 ± 4.2	−9.7 ± 5.1	−4.3 ± 4.5	−7.2 ± 4.8	0.82 (0.07 to 1.55)

[a] ΔCS-VTI and Δcardiac output were calculated only in the subgroup in whom measurement was obtainable (n = 9–10 per cell); Δheart rate and Δrate-pressure product in all randomised patients (n = 14–15 per cell). Per-cell samples of 9–10 patients cannot support factorial hypothesis testing, so no p values are reported. Cell B was selected as the candidate parameter combination for the definitive trial by rank ordering, not by statistical confirmation. CI confidence interval, CS-VTI coronary sinus velocity–time integral

3.5. Exploratory Clinical Measures

In Phase I, among the 18 patients with obtainable spectra, CS-VTI rose from 10.6 ± 3.8 to 12.8 ± 4.1 cm in the TEAS arm (mean increase 20.8%, 95% CI 11.2–30.4%) while the sham arm drifted from 10.4 ± 3.5 to 10.9 ± 3.6 cm (4.8%, 95% CI −2.1 to 11.7%); the between-group difference in change was 16.0 percentage points (95% CI 4.8–27.2). Cardiac output rose

14.5% (95% CI 4.5–24.5%) in the TEAS arm only. Mean arterial pressure did not change in either arm, so the rise in CS-VTI occurred under unchanged coronary perfusion pressure (Table 5). These are small-sample estimates from the subgroup in whom measurement succeeded and are hypothesis-generating only.

Table 5. *Exploratory echocardiographic measures before (T0) and after (T1) preoperative stimulation, Phase I patients in whom measurement was obtainable (n = 9 per group).*

Parameter	TEAS T0	TEAS T1	Control T0	Control T1	Between-group difference in change (95% CI)
CS-VTI, cm	10.6 ± 3.8	12.8 ± 4.1	10.4 ± 3.5	10.9 ± 3.6	+1.7 (+0.4 to +3.0)
Cardiac output, L/min	4.21 ± 0.82	4.82 ± 0.91	4.15 ± 0.79	4.31 ± 0.85	+0.45 (+0.02 to +0.88)
IVC respiratory variation, %	38.2 ± 12.4	35.6 ± 11.8	37.8 ± 11.9	37.1 ± 12.1	−1.9 (−12.2 to +8.4)
Heart rate, beats/min	74.2 ± 9.8	71.5 ± 8.6	73.5 ± 10.4	73.1 ± 10.1	−2.3 (−9.6 to +5.0)
Mean arterial pressure, mmHg	91.3 ± 12.1	88.7 ± 10.4	89.7 ± 11.6	90.2 ± 12.3	−3.1 (−13.6 to +7.4)

Values are mean ± SD. Differences are point estimates with 95% CIs of the change score (T1 − T0), TEAS minus control. These are exploratory estimates from an unpowered pilot and from the subgroup in whom measurement succeeded. CI confidence interval, CS-VTI coronary sinus velocity–time integral, IVC inferior vena cava.

Three of 40 patients were excluded (two TEAS: surgical plan change, equipment failure; one control: midazolam overdose), leaving 18 TEAS and 19 control patients. Baseline demographics and intraoperative surgical/anaesthetic profiles were comparable (Additional file 3, Table S1), supporting the interpretation that observed haemodynamic differences reflect the TEAS intervention rather than confounders.

Repeated-measures ANOVA yielded a significant group × time interaction for RPP ($F = 4.23$, $p = 0.008$, Greenhouse–Geisser corrected). The full model is given in Additional file 3 (Table S2). At baseline (B1) and pre-induction (B2), the groups were indistinguishable. Divergence emerged at the three peak-stress time points (Table 6): the TEAS group's RPP never crossed 12,000, while controls exceeded that threshold at intubation, incision, and extubation. The full perioperative trajectories are shown in Figure 3, with the underlying values in Table 6.

Table 6. *Exploratory perioperative haemodynamic measures and rate-pressure product, Phase III (TEAS n = 18; control n = 19).*

Time point	TEAS heart rate	Control heart rate	TEAS SBP	Control SBP	TEAS RPP	Control RPP	Difference (95% CI)
B1 preoperative	73.8 ± 9.2	74.6 ± 10.1	136 ± 18	138 ± 19	10,037 ± 1854	10,285 ± 2012	+248 (−1008 to +1504)
B2 pre-induction	70.2 ± 8.4	72.8 ± 9.6	132 ± 16	135 ± 17	9267 ± 1672	9828 ± 1789	+561 (−541 to +1663)
B3 post-intubation	72.4 ± 8.8	79.2 ± 10.3	141 ± 19	153 ± 21	10,245 ± 1876	12,134 ± 2043	+1889 (+736 to +3042)
B4 incision	68.6 ± 7.9	76.4 ± 9.8	141 ± 17	149 ± 20	9687 ± 1654	11,423 ± 1897	+1736 (+604 to +2868)
B5 post-extubation	74.8 ± 9.4	82.6 ± 11.2	145 ± 20	152 ± 22	10,876 ± 1932	12,567 ± 2156	+1691 (+378 to +3004)

Values are mean ± SD. Heart rate in beats/min; SBP and rate-pressure product as stated. Rate-pressure product = heart rate × SBP. Difference is control minus TEAS. Repeated-measures analysis of variance gave a group × time interaction of $F = 4.23$, $p = 0.008$ (Greenhouse–Geisser corrected; Mauchly $W = 0.416$, $p = 0.012$); degrees of freedom are reported uncorrected in Additional file 3. This is an exploratory outcome in a trial not powered to test it. CI confidence interval, RPP rate-pressure product, SBP systolic blood pressure.

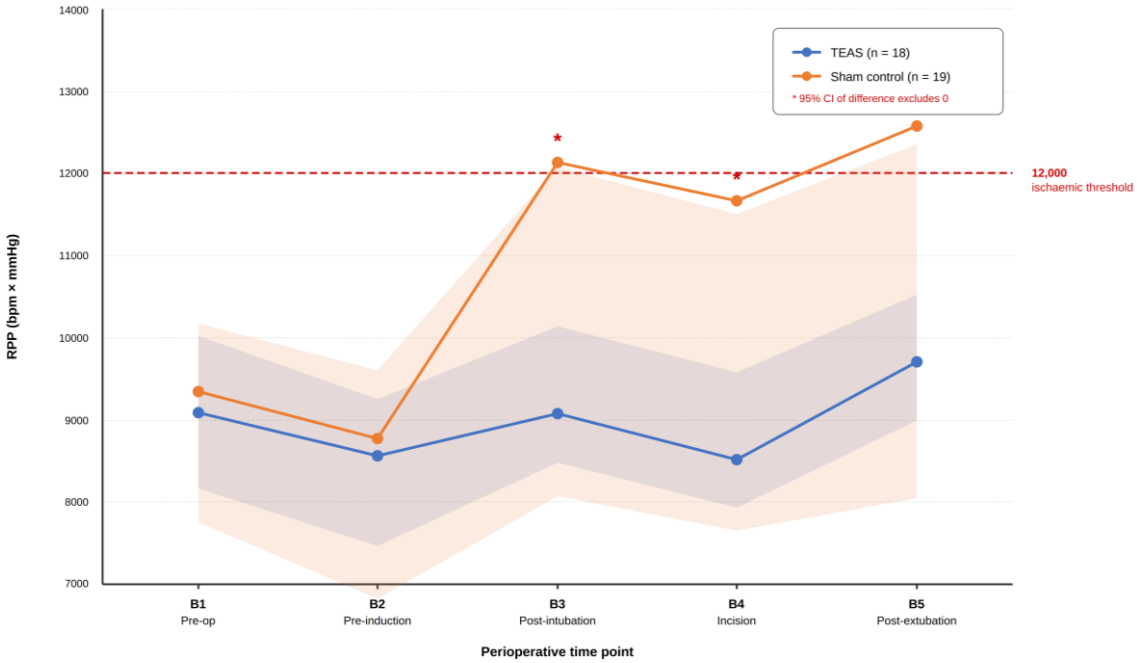


Figure 3. *Perioperative rate-pressure product (RPP) trajectories in Phase III. TEAS (n = 18) versus sham control (n = 19). Points are group means ± SD at the five pre-specified perioperative time points (B1–B5; see Methods). The dashed line marks an RPP of 12,000; asterisks denote time points at which the 95% confidence interval of the between-group difference excludes zero.*

Mean phenylephrine-equivalent dose was 243 ± 187 µg in TEAS versus 412 ± 234 µg in controls (difference −169 µg, 95% CI −306 to −32). The proportion of patients receiving any vasopressor decision was 28% versus 58% (risk difference −30.1 percentage points, 95% CI −54.5 to +1.5 by the Newcombe hybrid-score method; χ^2 p = 0.065). This confidence interval includes zero, so the vasopressor difference is a directional signal rather than a demonstrated effect (Additional file 3, Table S1). Critically, time-weighted MAP < 65 mmHg exposure, a measure independent of pressor administration, was also lower in the TEAS arm (2.1 vs. 4.8 min; difference −2.7 min, 95% CI −4.3 to −1.1), providing reassurance that the observed difference is not solely attributable to differential clinician behaviour. Twenty-four-hour VAS pain scores were similar (3.2 ± 1.4 vs. 3.6 ± 1.5).

At 30 days, cardiovascular event counts were 1/18 versus 4/19 (risk difference −15.5 percentage points, 95% CI −36.1 to +5.1); this is reported as a point estimate without efficacy inference. No adverse events were attributable to the TEAS procedure in any phase. Structured 24-hour visual assessment of the electrode application sites revealed no skin tears, persistent erythema, electrode-edge micro-blistering, or thermal injury under dense–disperse stimulation at 6–9 mA, apart from the single Phase I patient withdrawn for adhesive contact dermatitis.

Subgroup consistency was examined for RPP differences averaged across B3–B5 by COPD status, BMI category, ASA class, and age stratum (Figure 4C). Point estimates favoured TEAS in every subgroup and all interaction terms were non-significant (COPD interaction p = 0.412, BMI p = 0.618, ASA p = 0.583, age p = 0.691). With as few as 6 patients in the COPD subset, these exploratory analyses are heavily underpowered and should be interpreted with caution. Individual paired responses and the subgroup forest plot are shown in Figure 4. Panels (a) and (b) display only patients in whom measurement was obtainable, a subset that excludes those with chronic obstructive pulmonary disease, body mass index above 25 kg/m^2 or thoracic kyphosis.

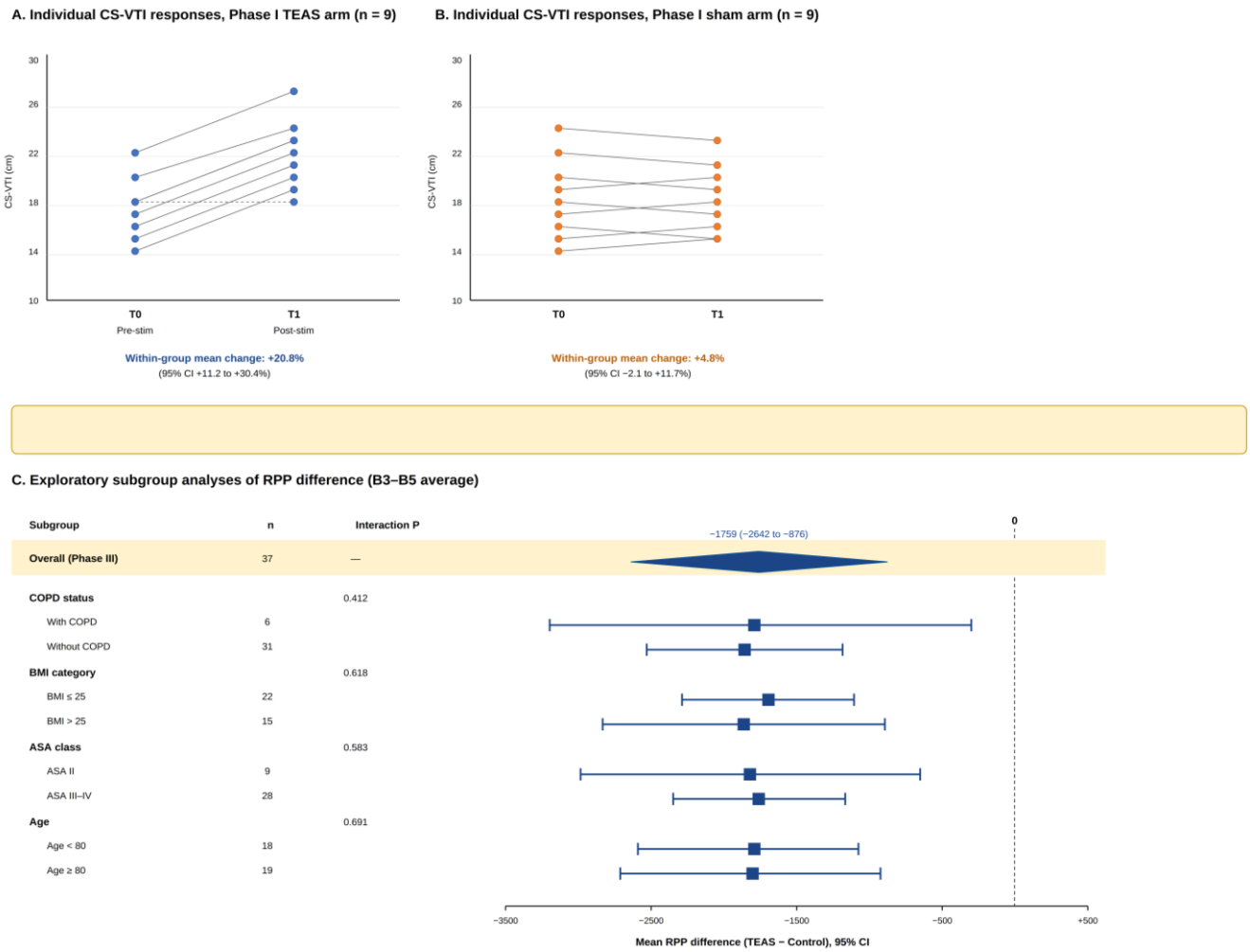


Figure 4. *Individual patient data and exploratory subgroup analyses. (a) Paired CS-VTI responses in the Phase I TEAS arm (n = 9); (b) paired CS-VTI responses in the Phase I sham arm (n = 9); (c) forest plot of between-group RPP differences averaged across B3–B5 by pre-specified subgroups, showing the TEAS − control mean difference with 95% confidence interval (negative values favour TEAS).*

3.6. Progression Criteria

Three of the four pre-specified progression criteria were met. CSBF obtainability of 64.7% exceeded the 60% threshold overall, although intraoperative obtainability of 56.8% did not; recruitment of 4.4 patients per month exceeded the threshold of four; and no serious adverse event was attributable to TEAS. The supportive exploratory criterion (a haemodynamic effect-size estimate whose lower confidence bound excluded zero) was met for RPP but only marginally for CS-VTI. Under the pre-specified traffic-light rule this corresponds to "proceed with protocol amendment" rather than "proceed unchanged".

4. Discussion

This three-phase pilot addressed whether a definitive trial of preoperative PC6 TEAS in elderly hip fracture surgery would be feasible, and how it should be designed. The answer is a qualified one. Recruitment, retention and safety were all acceptable, but the monitoring method we had intended to use was not.

4.1. The Central Feasibility Finding

The TTE feasibility data are the most clinically actionable finding of this pilot. The three predictors of measurement failure (COPD, BMI > 25 kg/m^2 and thoracic kyphosis) identify the very patients in whom perioperative supply–demand monitoring would matter most. The technique fails in approximately a third of the target population and reaches 90.5% success only after all three high-risk phenotypes are excluded. Transthoracic coronary sinus assessment is therefore unsuitable as a generalised intraoperative monitoring tool in elderly hip fracture patients, because it performs least well in those at highest risk. Exclusion of non-evaluable patients from the CSBF analysis also introduces a “healthy-survivor” selection bias that constrains inference about the magnitude of the TEAS effect on coronary flow.

Transoesophageal echocardiography (TEE) was not used

because the measurements had to be repeated in awake, unsedated patients in the preoperative holding area. TEE requires oesophageal intubation, topical pharyngeal anaesthesia and usually intravenous sedation, and carries risks of oropharyngeal and oesophageal injury and of sedation-related respiratory and haemodynamic depression that are greater in frail older adults [27]. Sedation would also have confounded both the TEAS intervention and the haemodynamic outcomes it was intended to influence. The comparison with TEE should in any case be interpreted with care. In an early head-to-head study, TEE identified the coronary sinus in all 40 patients examined but obtained a pulsed-Doppler flow-velocity recording in only 25 (63%), compared with none by TTE [28]. TEE equipment has improved since then, but the result shows that identifying the coronary sinus and measuring its flow are distinct problems, and that a better acoustic window does not by itself guarantee a usable Doppler signal.

4.2. Implications for the Design of the Definitive Trial

These feasibility data carry four design implications. Transthoracic CSBF should be abandoned as a generalised monitoring backbone and replaced by radial arterial tonometry with pulse-wave analysis, which retains feasibility across the full spectrum of phenotypes; CSBF may be retained in a small intensively-monitored mechanistic sub-cohort restricted to patients without the three failure predictors. Cardiac biomarkers must also be incorporated, which requires an ethics framework permitting research-only venepuncture and, in practice, a partner institution with routine perioperative troponin surveillance. The definitive trial must in addition blind the anaesthesiologist, which requires a covered active/sham device and automated log-stamped closed-loop infusion. Finally, 2 Hz for 25 min should be carried forward as the candidate parameter combination, recognising that it was selected by rank ordering rather than by statistical confirmation.

Sample size for the definitive trial should not be based on the effect estimates reported here. Per-cell evaluable samples of 9–10 patients produce effect-size estimates prone to upward random inflation, and the lower confidence bounds of our estimates descend close to zero. We propose powering the definitive trial against a conservative, externally anchored standardised effect size of 0.45, giving approximately 80 patients per arm and a total enrolment target of 240 allowing for attrition.

4.3. Interpreting the Exploratory Haemodynamic Signals

MAP did not change between T0 and T1 in either arm (TEAS 91.3 ± 12.1 → 88.7 ± 10.4 mmHg, p = 0.127; control 89.7 ± 11.6 → 90.2 ± 12.3 mmHg, p = 0.781), yet CS-VTI rose 20.8% (95% CI 11.2–30.4%) with TEAS. Since coronary flow is the quotient of perfusion pressure and vascular resistance, a rise under unchanged pressure implies reduced coronary vascular resistance, consistent with direct resistance-arteriole vasodilation rather than passive flow reduction from sympathetic withdrawal, and concordant with reports of acupoint-stimulation-induced vasodilation [29]. On 18 evaluable patients in a single phase, this remains a hypothesis for the confirmatory trial to test, ideally by coronary flow reserve under controlled perfusion pressure. It generalises only to the "healthy-survivor" subset without chronic obstructive pulmonary disease, body mass index above 25 kg/m^2, or thoracic kyphosis.

Under standardised anaesthesia, preoperative PC6 TEAS at 2 Hz for 25 min was associated with RPP values below the 12,000 oxygen-demand threshold throughout the perioperative period, with smaller phenylephrine doses and fewer hypotensive episodes. These are markers of reduction in a crude haemodynamic surrogate of myocardial work; they are not markers of myocardial injury and not direct measurements of oxygen supply–demand balance. POISE-3 [8] and IMPROVE-multi [9] together show that improving intraoperative macro-haemodynamics does not necessarily reduce perioperative myocardial injury; perioperative MINS is dominated by supply–demand mismatch at the myocyte level driven by inflammation, hypercoagulability, neuroendocrine surge, and microvascular dysfunction, which brachial MAP and rate-pressure products only partially capture [5, 30]. Our trial shows only that TEAS is a candidate intervention worth testing against troponin-defined endpoints in the confirmatory trial; the present results constitute hypothesis generation, not hypothesis confirmation.

Two institutional realities shaped this choice: our ethics committee classified research-specific venepuncture as a non-clinically-indicated procedure in this frail cohort, and our orthopaedic centre does not routinely measure perioperative troponin in asymptomatic patients. This aligns with the 2024 AHA/ACC guideline, which states only that preoperative troponin measurement "may be reasonable" (Class 2b, Level B-NR) [7], though the 2022 ESC guideline is more directive, assigning high-sensitivity troponin a Class I (Level B) recommendation in exactly our population [6]. Because MINS is defined by ischaemic troponin elevation [5], our study cannot diagnose it. POISE-3 BP showed that a 20-mmHg separation in mean arterial pressure did not change MINS incidence [8], confirming that haemodynamic optimisation and myocardial protection are distinct outcomes. All findings are therefore framed as haemodynamic optimisation. The confirmatory trial will measure high-sensitivity troponin T at baseline, 24, and 48 hours as a co-primary endpoint.

4.4. Methodological Cautions

The attending anaesthesiologist was unblinded, so haemodynamic differences could in principle reflect biased pressor administration. Following the template of INPRESS, POISE-

3, IMPROVE-multi, REGAIN, STRIDE, and RAGA [8, 9, 24, 31-33], we applied three pre-specified controls: a hard numeric trigger (MAP below 65 mmHg for at least 2 min), a standardised first-line agent and dose (phenylephrine 50 μg), and pre-specified sensitivity analyses. Time-weighted exposure to MAP below 65 mmHg, which is mathematically independent of pressor decisions, was lower with TEAS (2.1 vs. 4.8 min, 95% CI −4.3 to −1.1), as was total phenylephrine-equivalent dose (95% CI −306 to −32). The vasopressor proportion (28% vs. 58%) is directionally concordant but its interval includes zero. This consistency argues against a purely artefactual origin without eliminating the bias: anaesthetic doses, fluid volumes, and BIS were comparable, but we did not log time-stamped drug concentrations or fluid boluses and so could not perform causal-mediation analysis [34].

Myocardial oxygen consumption is governed less by the product of pressure and rate than by left ventricular wall stress, which depends on intracavitary pressure, chamber radius, and wall thickness [35, 36]. With age the aorta stiffens, so pressure-wave reflections return earlier in systole and magnify late-systolic wall stress [37, 38]. Gobel and colleagues cautioned that extrapolating RPP to hearts with altered geometry or compliance requires care [10], and Kal and Westerhof showed that RPP does not reliably track myocardial oxygen consumption under altered loading [13]. RPP probably underestimates true demand in our cohort, and a value below 12,000 does not exclude subendocardial mismatch. The between-group separation should therefore be read as a hypothesis-generating signal of attenuated sympathetic activation, not as evidence of preserved myocardial energetics. Wall-stress-aware indices, particularly the subendocardial viability ratio [39], are physiologically more appropriate; the confirmatory trial will use radial arterial tonometry as its primary reference index.

This trial measured haemodynamic surrogates only. Plasma catecholamines and cortisol were not assayed, and high-fidelity heart-rate variability was not recorded because our monitors stored 1-second trend data rather than the raw ECG required for frequency-domain analysis [40]. The causal chain we can support is therefore narrow: preoperative PC6 stimulation was followed by five measured changes: lower RPP throughout, higher coronary sinus velocity–time integral under unchanged mean arterial pressure, lower vasopressor dose, less hypotension exposure, and preserved cardiac output. These are linked to the intervention only by temporal association. Sympathoadrenal attenuation is the single hypothesised mediator, and it was not measured. Plausibility rests on peripheral neurophysiology: anxiolytic effects of acupoint stimulation [29], opioid-mediated analgesia [41], and heart-rate-variability shifts [14, 42]. We make no molecular claims; the PROKR2 vagal–adrenal pathway described in mice [43] is far removed from human surface stimulation and has been questioned anatomically [44] and mechanistically [45].

4.5. Relation to the Wider Evidence Base

With POISE-3 [8], IMPROVE-multi [9], and related null trials of supply-side blood-pressure targeting, a low-cost, non-invasive, demand-side intervention that reduces RPP is a different approach worth pursuing in a definitive trial. Our confirmatory design will be a three-arm multicentre trial stratified across propofol TIVA, sevoflurane-based volatile anaesthesia, and spinal anaesthesia with sedation, powered to detect a TEAS × anaesthetic-technique interaction; will deploy a covered active/sham TEAS device following the STRIDE precedent [32]; will use automated log-stamped closed-loop target-controlled infusion; and will model temporal trajectories of anaesthetic concentration, fluid administration, and vasopressor delivery as time-varying mediators in a controlled-direct-effect framework [34]. We will power against a conservative pre-specified standardised effect size of d = 0.45 (below the lower bound of our pilot RPP CI), giving approximately 80 per arm and a total enrolment target of 240 with a 33% attrition buffer.

4.6. Limitations

Several limitations deserve note. (1) The trial was registered retrospectively, and the registration (17 June 2026) postdates the end of recruitment (May 2025). It is also the most consequential limitation of the report. The cause was a failure of communication within our own team: the principal investigator had filed the study with the national record-filing system before enrolment and the team took that to be registration, so the absence of a public registry entry was not noticed for three years. That explains the omission but does not repair it. Public prospective registration exists to deter selective outcome reporting, to make ongoing trials discoverable and to allow readers to compare reported outcomes against those specified in advance; a registration entered after the data were complete cannot serve any of these functions. We can show that the outcomes and progression criteria were pre-specified: they appear in the ethics-approved protocol and in the pre-enrolment national record-filing, and they are unchanged in this report. That documentation is institutional rather than public, and readers are entitled to weigh it accordingly. Accordingly, we present the feasibility findings as the primary output of this work and the haemodynamic estimates as exploratory, and we recommend that the definitive trial be registered publicly and prospectively before its first participant is enrolled. (2) Performance bias from differential intraoperative titration or fluid timing cannot be entirely excluded; the attending anaesthesiologist was unblinded, and although pre-specified sensitivity analyses argue against a pure administration artefact, time-stamped anaesthetic concentrations and fluid-bolus timestamps were not logged. (3) Single centre with small per-group samples (15–20) limits generalisability and power; this is appropriate for a pilot [20] but not for efficacy inference. (4) TTE-CSBF feasibility of ~64% introduced selection bias toward less comorbid patients for coronary flow endpoints. (5)

Phase II per-cell n ≈ 9–10 permits only qualitative dose-ordering. (6) No troponin, BNP, catecholamines, or cortisol were measured, owing to institutional constraints. (7) Inflammatory markers were not measured. (8) RPP is a crude peripheral surrogate for myocardial oxygen consumption in stiff arteriosclerotic elderly hearts; SEVR via arterial tonometry, planned as the primary reference in the confirmatory trial, is physiologically more appropriate.

5. Conclusion

A definitive randomised trial of preoperative PC6 TEAS in elderly hip fracture surgery is feasible with respect to recruitment, retention and safety, but should not proceed on the protocol as piloted. The decisive feasibility finding is negative: transthoracic coronary sinus blood flow measurement is obtainable in only about two-thirds of these patients and fails selectively in those with chronic obstructive pulmonary disease, raised body mass index or thoracic kyphosis, the very patients in whom perioperative supply–demand monitoring matters most. We therefore recommend proceeding to a definitive multicentre trial that is registered publicly and prospectively before enrolment and follows an amended protocol: pulse-wave-analysis-derived monitoring in place of transthoracic CSBF, troponin-defined myocardial injury as the primary endpoint, a covered sham device to blind the anaesthesiologist, and 2 Hz for 25 min as the candidate stimulation parameters. The haemodynamic differences observed in this pilot are exploratory, imprecise and not evidence of clinical benefit; they justify a confirmatory trial but must not be used to power it.

6. Recommendations

We recommend that a definitive multicentre trial be undertaken with the following amendments: pulse-wave-analysis-derived monitoring in place of transthoracic CSBF; troponin-defined myocardial injury as the primary endpoint; a covered active/sham TEAS device to blind the attending anaesthesiologist; and 2 Hz for 25 min as the candidate stimulation parameters. The definitive trial should be registered publicly and prospectively before enrolment, and should be powered against a conservative, externally anchored standardised effect size rather than the pilot estimates reported here.

Abbreviations

ASA	American Society of Anesthesiologists
BIS	Bispectral Index
BMI	Body Mass Index
BNP	B-type Natriuretic Peptide
CO	Cardiac Output
COPD	Chronic Obstructive Pulmonary Disease
CSBF	Coronary Sinus Blood Flow
CS-VTI	coronary Sinus Velocity–Time Integral
ECG	Electrocardiogram
FiO2	Fraction of Inspired Oxygen
HR	Heart Rate
MACE	Major Adverse Cardiovascular Events
MAP	Mean Arterial Pressure
MINS	Myocardial Injury After Non-Cardiac Surgery
MMSE	Mini-Mental State Examination
PEEP	Positive End-Expiratory Pressure
RPP	Rate-Pressure Product
SBP	Systolic Blood Pressure
TEAS	Transcutaneous Electrical Acupoint Stimulation
TEE	Transoesophageal Echocardiography
TTE	Transthoracic Echocardiography
VAS	Visual Analogue Scale
VT	Tidal Volume

Supplementary Material

The supplementary material can be accessed at https://doi.org/10.11648/j.ijacm.20261402.18

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Hong Xiang: Investigation, Visualization, Writing – review & editing

Shasha Zhu: Validation, Visualization, Writing – review & editing

Yukai Zhou: Conceptualization, Funding acquisition, Investigation, Methodology, Supervision, Writing – original draft

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Data Availability Statement

The datasets generated and analysed during the current study are not publicly available because the ethics approval

did not include provision for public data deposition, but are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest.

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