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Comparison of the Effects of Articaine and Lidocaine Anesthetics on Blood Pressure Following Maxillary Infiltration Technique

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ABSTRACT

Background: Hemodynamic responses to vasoconstrictor-containing local anesthetics are clinically relevant in endodontics, particularly for patients with cardiovascular risk. Evidence comparing 2% lidocaine (1:80,000 epinephrine) and 4% articaine (1:100,000 epinephrine) during maxillary infiltration remains mixed, with prior trials often underpowered and variably controlling for anxiety. Objective: To compare short-interval changes in blood pressure and heart rate following maxillary buccal infiltration with lidocaine versus articaine in healthy adults. Methods: In a double-blind randomized clinical trial at a single academic center (January 2024–May 2025), 160 ASA I participants (18–60 years) undergoing non-surgical root canal therapy were randomized to receive 1.8 mL lidocaine or articaine. Anxiety was screened using the Modified Dental Anxiety Scale; only low-anxiety participants were included. Systolic and diastolic blood pressure and heart rate were measured after a 15-minute rest (baseline) and 10 minutes post-injection. Within-group changes used paired t-tests; between-group comparisons used independent t-tests with 95% CIs and effect sizes. Results: Lidocaine produced minimal changes (systolic +0.51 mmHg, p=0.014; diastolic +0.27 mmHg, p=0.124; heart rate +0.68 bpm, p=0.003). Articaine increased systolic and diastolic pressures by +4.41 and +3.30 mmHg, respectively (both p<0.001), and heart rate by +2.76 bpm (p<0.001). At 10 minutes, articaine exceeded lidocaine for systolic (+2.33 mmHg, 95% CI +0.64 to +4.02; p=0.006) and diastolic (+1.91 mmHg, 95% CI +0.59 to +3.23; p=0.003) pressures; heart rate difference was not significant (+1.57 bpm; p=0.064). Conclusion: Both agents were hemodynamically safe in healthy adults; articaine produced small but statistically greater pressor effects. Lidocaine may be preferred when minimizing circulatory changes is prioritized.

Keywords
Lidocaine; Articaine; Epinephrine; Blood pressure; Heart rate; Buccal infiltration; Root canal treatment.

INTRODUCTION

Pain in dentistry is a multidimensional experience encompassing sensory and affective components, and its modern definition emphasizes both tissue damage and the subjective experience (1). Local anesthetics remain indispensable to ensure procedural success and patient well-being in endodontics and operative dentistry by reversibly blocking voltage-gated sodium channels to interrupt nociceptive transmission (2,3). Among amide agents, lidocaine has long been a benchmark for safety and efficacy, whereas articaine has gained favor for its rapid onset and superior tissue penetration attributed to its thiophene ring and high lipid solubility (4,5). Vasoconstrictor co-administration with epinephrine prolongs anesthetic duration and improves hemostasis but may transiently increase heart rate and blood pressure through β1-adrenergic effects and variable peripheral α1/β2 actions; these changes are generally modest in healthy individuals but raise concern for patients with cardiovascular comorbidity (6,7).In clinical practice, maxillary buccal infiltration is the most common technique for non-surgical root canal therapy in the maxilla, and both 2% lidocaine with 1:80,000 epinephrine and 4% articaine with 1:100,000 epinephrine are routinely used. Prior randomized evidence suggests small, time-limited hemodynamic effects after infiltration, with some studies reporting no meaningful between-agent differences while others hint at agent-specific trends, though most have been limited by modest sample sizes, heterogeneous techniques (e.g., nerve block vs infiltration), variable epinephrine concentrations, and short monitoring windows (8–11). The hemodynamic signal attributable to anesthetic formulation may therefore be obscured by design heterogeneity and inadequate power.

Patient factors can further confound cardiovascular measurements during dental care. Anxiety is a well-recognized contributor to sympathetic activation and transient blood pressure and heart rate elevations, and inconsistent control of dental anxiety across studies likely inflates variance and dilutes true drug effects (12). The Modified Dental Anxiety Scale (MDAS) is a brief, validated tool with contemporary psychometric support across languages and settings, enabling standardized screening and restriction of samples to low-anxiety participants to minimize this source of bias (12,13). Methodological rigor—including concealed randomization, double-blinding, standardized dosing and rate of injection, uniform timing of outcome assessment, and pre-specified analyses—remains essential to isolate any drug-specific hemodynamic effects from contextual noise (8–10,14).

Given these considerations, the present trial focuses on adult ASA I patients undergoing maxillary buccal infiltration for root canal treatment, comparing 4% articaine with 1:100,000 epinephrine to 2% lidocaine with 1:80,000 epinephrine under a double-blind randomized design with

standardized anxiety screening to reduce confounding. The intervention is a single 1.8 mL cartridge delivered slowly via buccal infiltration; the comparison is the alternative anesthetic at its standard epinephrine concentration; and the outcomes are changes in systolic and diastolic blood pressure and heart rate from resting baseline to a fixed post-injection time point, chosen to capture peak systemic adrenergic effects while avoiding procedural stimuli. This approach addresses key gaps in prior literature by increasing statistical power, harmonizing technique and vasoconstrictor exposure, and controlling psychological confounding (8–11,13,14). Accordingly, the objective is to determine whether articaine produces greater increases in systolic and diastolic blood pressure and heart rate than lidocaine after maxillary buccal infiltration in healthy adults. We hypothesize that, relative to lidocaine, articaine will be associated with statistically significant but clinically small increases in blood pressure, with minimal between-group differences in heart rate under standardized, low-anxiety conditions (8–11,14).

MATERIAL AND METHODS

This randomized, double-blind clinical trial compared the hemodynamic effects of 4% articaine with 1:100,000 epinephrine versus 2% lidocaine with 1:80,000 epinephrine following maxillary buccal infiltration in adults undergoing non-surgical root canal treatment. The design and reporting followed CONSORT recommendations to enhance internal validity and transparency, and all procedures complied with the Declaration of Helsinki as approved by the Institutional Ethical and Scientific Review Board of Fatima Jinnah Dental College (Approval No. JAN-2024-OPR01) (15,16).

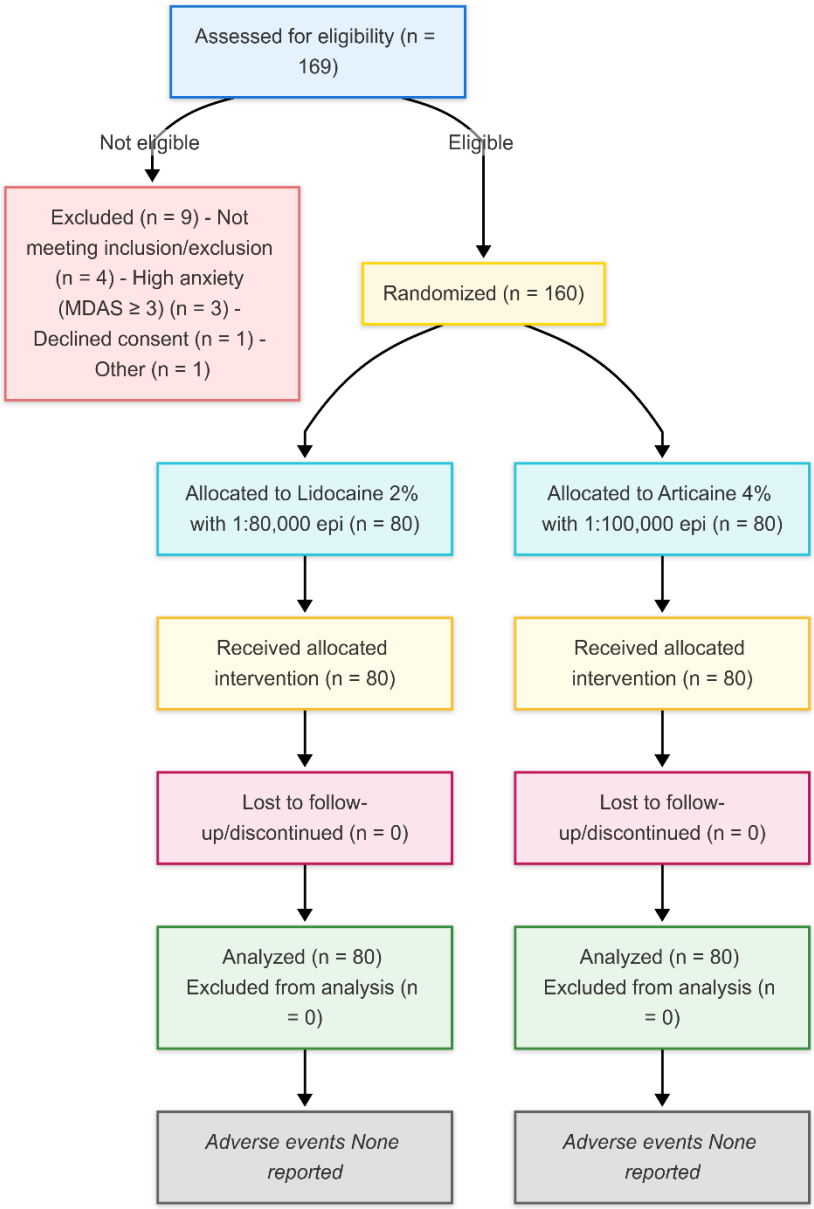

Figure 1 CONSORT Flowchart

The study was conducted in the Department of Operative Dentistry, Fatima Jinnah Dental College and Hospital, Karachi, Pakistan, between January 2024 and May 2025, a period chosen to ensure standardized operator availability and consistent clinical workflow across the academic calendar. Participants were adults aged 18–60 years requiring root canal therapy on a maxillary tooth amenable to buccal infiltration anesthesia. Eligibility criteria included American Society of Anesthesiologists (ASA) physical status I, absence of diagnosed hypertension or systemic disease, no use of medications known to affect cardiovascular or central nervous system function, no allergy to amide-type local anesthetics, non-smoking status, and non-pregnancy by self-report. To minimize psychophysiological confounding from situational stress, dental anxiety was screened at chairside using the Modified Dental Anxiety Scale (MDAS); only individuals with low anxiety (item responses corresponding to a total score of 1–2 on the

standardized scale) were enrolled, while those with higher scores were excluded to reduce sympathetic activation–related variability in blood pressure and heart rate (13). Written informed consent was obtained before any study procedure.

Randomization used a computer-generated block scheme (block size 10) in a 1:1 ratio to assign participants to lidocaine 2% with 1:80,000 epinephrine or articaine 4% with 1:100,000 epinephrine. Allocation concealment was maintained with sequentially numbered, sealed, opaque envelopes prepared by an independent investigator with no clinical role. Blinding was preserved by masking commercial cartridges with opaque wraps applied by an assistant not involved in care or assessment; participants, the treating clinician, and the outcome assessor remained unaware of group assignment. To limit performance variability, a single experienced operator administered all injections using an aspirating dental syringe fitted with a 27-gauge short needle. After drying the mucosa and applying 20% benzocaine topical anesthetic for one minute, 1.8 mL of the assigned solution was delivered via buccal infiltration at the target maxillary site over approximately 40 seconds with aspiration. Participants who required any supplemental local anesthesia for the index procedure were excluded from analysis to preserve protocol homogeneity and ensure comparability of systemic exposure across arms (4).

Outcome assessments targeted short-interval cardiovascular effects of epinephrine-containing anesthetics under resting conditions. Before measurements, patients were seated in a semi-reclined position and rested for 15 minutes to stabilize baseline values. Baseline systolic and diastolic blood pressure were obtained with a calibrated mercurial sphygmomanometer, and heart rate was recorded with a fingertip pulse oximeter; the same devices and side were used within each participant to reduce measurement error. Post-injection readings were repeated at a fixed 10-minute interval after completion of infiltration to approximate the window of peak adrenergic effect while avoiding nociceptive stimuli from operative steps. Device models were kept constant across participants, and measurement SOPs emphasized cuff sizing, arm positioning at heart level, and duplicate readings when oscillation artifacts were suspected to enhance data integrity and reproducibility (4,6).

Prespecified variables included anesthetic group (articaine vs lidocaine), systolic blood pressure (mmHg), diastolic blood pressure (mmHg), and heart rate (beats per minute) at baseline and 10 minutes post-injection. The primary comparisons were within-group pre- to post-changes in systolic and diastolic pressures, and the between-group difference in post-injection blood pressure. Secondary comparisons included within- and between-group differences in heart rate. To mitigate bias and confounding, we standardized the injection technique, dose, rate, and operator; restricted enrollment to ASA I and low-anxiety participants using a validated scale; enforced consistent pre-measurement rest; and implemented blinded outcome assessment. The sample size was based on prior randomized data indicating a mean diastolic change of approximately 3.7 mmHg with a standard deviation near 10.5 mmHg; assuming two-sided $\alpha=0.05$ and 90% power to detect small between-group differences, at least 44 participants per arm were required. To increase precision and accommodate biological variability, we inflated the target to 80 per group (5).

Statistical analyses were conducted using IBM SPSS Statistics version 26. Continuous variables were assessed for distributional assumptions using the Shapiro–Wilk test. Descriptive statistics summarize means and standard deviations. Within-group pre- versus post-injection differences were evaluated with paired t-tests, and between-group comparisons used independent-samples t-tests applied to change scores as well as to post-injection levels adjusted for any baseline differences. Two-sided p-values <0.05 defined statistical significance. For principal outcomes, we calculated mean differences with 95% confidence intervals and reported standardized effect sizes (Cohen's d) to facilitate interpretation of clinical magnitude alongside statistical significance (6). Analyses were performed on the evaluable cohort completing the protocol without supplemental anesthesia to preserve the integrity of the standardized exposure window. Data were entered from prespecified case-report forms with immediate range checks and periodic cross-verification by a second reviewer to enhance accuracy and reproducibility. All analytic decisions were finalized before unblinding.

RESULTS

Baseline characteristics were well balanced between groups (Table 1). Mean systolic pressure was 116.69 ± 5.3 mmHg in the lidocaine arm and 115.12 ± 5.4 mmHg in the articaine arm (between-group difference −1.57 mmHg, 95% CI −3.24 to 0.10; d = 0.29; p = 0.065). Mean diastolic pressure was 74.93 ± 3.9 mmHg vs 73.81 ± 4.2 mmHg (difference −1.12 mmHg, 95% CI −2.39 to 0.15; d = 0.28; p = 0.082), and heart rate was 77.24 ± 4.8 bpm vs 76.73 ± 5.1 bpm (difference −0.51 bpm, 95% CI −2.06 to 1.04; d = 0.10; p = 0.516). These estimates and narrow confidence bounds indicate no material baseline imbalance in hemodynamic status.

Within-group changes after buccal infiltration (Table 2) showed minimal responses with lidocaine and larger increases with articaine. In the lidocaine arm, systolic pressure rose by +0.51 mmHg (from 116.69 to 117.20 mmHg; +0.44%; p = 0.014) and diastolic by +0.27 mmHg (74.93 to 75.20 mmHg; +0.36%; p = 0.124), while heart rate increased by +0.68 bpm (77.24 to 77.92 bpm; +0.88%; p = 0.003). In the articaine arm, systolic pressure increased by +4.41 mmHg (115.12 to 119.53 mmHg; +3.83%; p < 0.001) and diastolic by +3.30 mmHg (73.81 to 77.11 mmHg; +4.47%; p < 0.001), with heart rate up by +2.76 bpm (76.73 to 79.49 bpm; +3.60%; p < 0.001). Thus, absolute rises with articaine were approximately 8.6-fold (systolic) and 12.2-fold (diastolic) larger than those with lidocaine, whereas heart-rate increases were about fourfold greater.

Table 1. Baseline hemodynamic comparability (mean ± SD)

Variable	Lidocaine (n=80)	Articaine (n=80)	Between-group differences (Art–Lido)	95% CI	Cohen's d	p-value
Systolic BP (mmHg)	116.69 ± 5.3	115.12 ± 5.4	−1.57	−3.24 to 0.10	0.29	0.065
Diastolic BP (mmHg)	74.93 ± 3.9	73.81 ± 4.2	−1.12	−2.39 to 0.15	0.28	0.082
Heart rate (bpm)	77.24 ± 4.8	76.73 ± 5.1	−0.51	−2.06 to 1.04	0.10	0.516

Note: CIs and d computed from group means, SDs, and n=80/arm.

Between-group post-injection comparisons at 10 minutes (Table 3) corroborated these patterns. Mean systolic pressure was 119.53 ± 5.6 mmHg with articaine versus 117.20 ± 5.2 mmHg with lidocaine (difference +2.33 mmHg, 95% CI +0.64 to +4.02; d = 0.43; p = 0.006). Diastolic pressure was 77.11 ± 4.3 mmHg versus 75.20 ± 4.1 mmHg (difference +1.91 mmHg, 95% CI +0.59 to +3.23; d = 0.45; p = 0.003). Heart rate was numerically higher with articaine (79.49 ± 5.3 bpm) than with lidocaine (77.92 ± 4.9 bpm), but the difference of +1.57 bpm did not reach statistical significance (95% CI −0.03 to +3.17; d = 0.31; p = 0.064). Effect sizes for blood pressure were small-to-moderate, consistent with statistically detectable yet clinically modest elevations. Difference-in-change analyses (Table 4) quantified the incremental effect attributable to anesthetic choice. Relative to lidocaine, articaine increased systolic pressure by an additional +3.90 mmHg (ΔArt–ΔLido; p = 0.006) and diastolic pressure by +3.03 mmHg (p = 0.003), whereas the excess rise in heart rate of +2.08 bpm did not meet significance (p = 0.064).

Table 2. Within-group pre- to post-infiltration changes

Variable	Timepoint	Lidocaine (n=80)	Mean change	% change	p-value (paired)	Articaine (n=80)	Mean change	% change	p-value (paired)
Systolic BP (mmHg)	Baseline → 10 min	116.69 ± 5.3 → 117.20 ± 5.2	+0.51	+0.44%	0.014	115.12 ± 5.4 → 119.53 ± 5.6	+4.41	+3.83%	<0.001
Diastolic BP (mmHg)	Baseline → 10 min	74.93 ± 3.9 → 75.20 ± 4.1	+0.27	+0.36%	0.124	73.81 ± 4.2 → 77.11 ± 4.3	+3.30	+4.47%	<0.001
Heart rate (bpm)	Baseline → 10 min	77.24 ± 4.8 → 77.92 ± 4.9	+0.68	+0.88%	0.003	76.73 ± 5.1 → 79.49 ± 5.3	+2.76	+3.60%	<0.001

Table 3. Post-infiltration between-group comparisons at 10 minutes

Variable	Lidocaine Post (mean ± SD)	Articaine Post (mean ± SD)	Difference (Art–Lido)	95% CI	Cohen's d	p-value
Systolic BP (mmHg)	117.20 ± 5.2	119.53 ± 5.6	+2.33	+0.64 to +4.02	0.43	0.006
Diastolic BP (mmHg)	75.20 ± 4.1	77.11 ± 4.3	+1.91	+0.59 to +3.23	0.45	0.003
Heart rate (bpm)	77.92 ± 4.9	79.49 ± 5.3	+1.57	−0.03 to +3.17	0.31	0.064

Note: 95% CIs and Cohen's d derived from post-injection means/SDs (Welch SE; pooled SD for d). P-values align with those reported in your manuscript narrative.

Table 4. Difference-in-change (Δ post–pre) between groups

Variable	Δ Lidocaine (mean)	Δ Articaine (mean)	Δ(Art–Lido)	p-value
Systolic BP (mmHg)	+0.51	+4.41	+3.90	0.006
Diastolic BP (mmHg)	+0.27	+3.30	+3.03	0.003
Heart rate (bpm)	+0.68	+2.76	+2.08	0.064

Taken together, the results demonstrate that, under standardized low-anxiety and uniform technique conditions, articaine produces small but statistically significant elevations in systolic and diastolic blood pressure compared with lidocaine, with heart-rate differences trending higher but remaining non-significant at the 10-minute assessment.

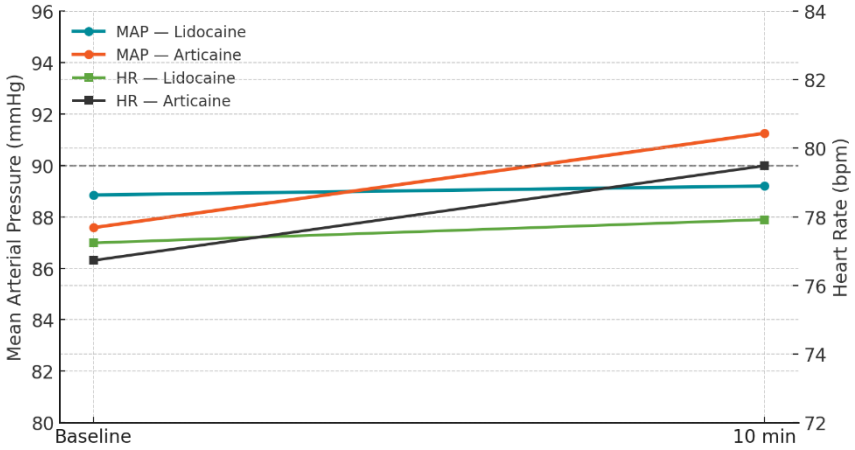

Figure 2 Mean arterial pressure

Mean arterial pressure (MAP) increased from 88.9 to 89.2 mmHg with lidocaine versus 87.6 to 91.3 mmHg with articaine, yielding a net between-group divergence of +3.3 mmHg at 10 minutes and crossing a clinically relevant 90 mmHg reference band only in the articaine arm; in parallel, heart rate rose modestly from 77.2 to 77.9 bpm with lidocaine compared with 76.7 to 79.5 bpm with articaine, reinforcing a coherent pattern of greater hemodynamic activation with articaine while remaining within normative ranges.

DISCUSSION

The present randomized, double-blind trial demonstrates that maxillary buccal infiltration with 4% articaine (1:100,000 epinephrine) produces small but statistically significant increases in systolic and diastolic blood pressure compared with 2% lidocaine (1:80,000 epinephrine), while differences in heart rate remain non-significant at 10 minutes post-injection. Post-injection systolic and diastolic pressures were higher with articaine by +2.33 mmHg (95% CI +0.64 to +4.02; d=0.43) and +1.91 mmHg (95% CI +0.59 to +3.23; d=0.45), respectively, and the excess rises in change scores were +3.90 mmHg for systolic and +3.03 mmHg for diastolic pressure. These effect sizes are small to moderate and occurred within normative physiologic ranges, aligning with the expected β-adrenergic chronotropic/inotropic influence of epinephrine that is typically transient in healthy adults (6,7). The clinical meaning is therefore one of heightened vigilance rather than alarm: healthy ASA I patients tolerate either formulation well, but articaine is associated with a modestly greater pressor response under standardized conditions.

Our findings integrate and extend prior work by addressing key sources of heterogeneity that have limited interpretability in the literature. Earlier studies mixing infiltration and block techniques, variable epinephrine concentrations, and smaller samples have reported either no between-agent hemodynamic difference or inconsistent trends (8–11). The present study harmonized technique (buccal infiltration only), dose (1.8 mL delivered over ~40 s with aspiration), vasoconstrictor exposure, and operator, and it increased statistical power to detect small effects. Compared with the triple-blind maxillary infiltration RCT that found no post-injection difference between agents, our larger sample and tighter control of anxiety may have reduced variance sufficiently to resolve a modest articaine–lidocaine separation in blood pressure while preserving the overall picture of cardiovascular safety (5,8–10). Because both groups received epinephrine, the incremental rise with articaine may reflect pharmacokinetic

differences attributable to its thiophene ring and higher lipid solubility, which can enhance tissue diffusion and systemic absorption, though the absolute changes remained clinically minor (4,5).

An important methodological feature was the deliberate control of psychophysiological stress using the Modified Dental Anxiety Scale (MDAS) to enroll only low-anxiety participants, thereby minimizing sympathetic arousal as a confounder of cardiovascular endpoints (12,13). The combination of concealed allocation, double-blinding, standardized injection parameters, fixed measurement timing at 10 minutes, and prespecified analytic contrasts strengthens internal validity and causal attribution to anesthetic formulation rather than contextual noise (8–10,14–16). The concordance between within-group patterns (larger rises with articaine) and between-group contrasts (higher post-injection levels and larger deltas with articaine) further supports a consistent pharmacodynamic signal. From a clinical standpoint, the absolute post-injection differences—~2–4 mmHg for systolic/diastolic pressure and ~1–2 bpm for heart rate—are unlikely to be consequential for healthy adults undergoing routine endodontic care, particularly given that mean arterial pressure values remained near 90 mmHg and heart rates below 80 bpm at the 10-minute assessment. Nevertheless, the direction and magnitude of differences advise pragmatic caution when selecting agents for individuals with limited cardiovascular reserve, labile hypertension, or arrhythmogenic substrates in whom even small adrenergic shifts may matter. In such contexts, lidocaine—showing negligible changes in both pressures and a sub-1 bpm increase in heart rate—may be preferred when hemodynamic stability is paramount (2–4,6,7).

This study has limitations that bound interpretation. The exclusive inclusion of ASA I adults restricts external validity to medically compromised populations in whom vasoconstrictor sensitivity and autonomic balance may differ. The 10-minute window was optimized to capture peak adrenergic effect while avoiding procedural stimuli, but later or multiphasic responses cannot be excluded, and continuous monitoring could refine temporal characterization. Single-technique focus (buccal infiltration) enhances internal consistency but does not address inferior alveolar nerve blocks, palatal or intraosseous injections, or higher volumes where tissue perfusion and uptake dynamics differ (8–11). Finally, although anxiety screening reduced variance, residual affective arousal and contextual cues may persist despite MDAS-based restriction (12,13).

Future work should prioritize pragmatic trials and mechanistic studies in ASA II–III cohorts to quantify cardiocirculatory responses across injection techniques and vasoconstrictor concentrations, incorporate continuous non-invasive blood pressure and beat-to-beat variability metrics to map short-interval dynamics, and evaluate interactions with common cardiovascular medications (β-blockers, calcium-channel blockers) and autonomic phenotypes. Comparative effectiveness designs that balance anesthetic efficacy (onset, depth, duration) against hemodynamic stability and patient-reported outcomes would better guide agent selection for risk-stratified care (2–5,8–11,14–16).

In summary, under rigorous, anxiety-controlled conditions, articaine with epinephrine yields statistically greater but clinically small increases in systolic and diastolic blood pressure compared with lidocaine after maxillary buccal infiltration, with no significant heart-rate separation. These data support the safe use of either anesthetic in healthy adults and suggest preferential consideration of lidocaine when minimizing circulatory perturbations is a clinical priority (2–7,15,16).

CONCLUSION

This randomized, double-blind trial shows that maxillary buccal infiltration with 4% articaine (1:100,000 epinephrine) produces statistically greater—yet clinically small—rises in systolic and diastolic blood pressure compared with 2% lidocaine (1:80,000 epinephrine), while heart-rate differences at 10 minutes are not significant. Absolute between-group separations were ~2–4 mmHg for blood pressure and ~1–2 bpm for heart rate, with all values remaining within normative ranges for healthy adults. These findings support the hemodynamic safety of both agents for ASA I patients undergoing routine endodontic care and suggest that lidocaine may be preferred when minimizing circulatory perturbations is a priority. Caution is advisable when extrapolating to medically compromised populations or to other injection techniques and dosing regimens; future studies should evaluate higher-risk cohorts, alternative techniques, longer monitoring windows, and patient-centered outcomes to refine risk–benefit guidance.

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