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Original Research Article

# Systematic Review: Dexmedetomidine Versus Clonidine in Axillary Brachial Plexus Block — A Clinical Evidence

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**Abstract:**

**Aim:** Dexmedetomidine and clonidine are alpha-2 adrenergic agonists frequently used as adjuvants to local anaesthetics in brachial plexus block because they may shorten onset time, improve block quality, and prolong postoperative analgesia. The clinical question addressed in this review is whether dexmedetomidine provides superior efficacy to clonidine when used in axillary brachial plexus block, while maintaining acceptable haemodynamic and safety outcomes.

**Materials and Methods:** A structured clinical evidence synthesis was undertaken using indexed comparative literature on dexmedetomidine versus clonidine as perineural adjuvants in brachial plexus block. Core evidence was drawn from a 2022 meta-analysis of 24 randomized controlled trials 2017 systematic review and meta-analysis of 14 randomized controlled trials. Both reviews compared dexmedetomidine and clonidine as adjuvants to local anaesthetics for upper-limb surgery and analyzed sensory block duration, motor block duration, analgesia duration, onset characteristics, and adverse events.

**Result:** Across comparative brachial plexus block evidence, dexmedetomidine consistently outperformed clonidine on efficacy endpoints. In the 2022 meta-analysis, dexmedetomidine significantly prolonged sensory block duration by a mean difference of 173.31 minutes, motor block duration by 158.35 minutes, and duration of analgesia by 203.92 minutes compared with clonidine. Dexmedetomidine also hastened onset of sensory block by 1.58 minutes and motor block by 1.46 minutes, and improved the likelihood of higher-grade block quality. The 2017 meta-analysis similarly found that dexmedetomidine increased sensory, motor, and analgesic duration by an estimated ratio of means of about 1.2 each, while also accelerating onset. The 2017 review signaled increased transient bradycardia and postoperative sedation with dexmedetomidine, whereas the 2022 review found no statistically significant difference in hypotension.

**Conclusion:** The best available evidence suggests that dexmedetomidine is clinically more effective than clonidine as a perineural adjunct in brachial plexus block, with longer sensory and motor blockade, longer postoperative analgesia, and somewhat faster onset.

**Keywords:** Dexmedetomidine; Clonidine; Axillary Brachial Plexus Block; Regional Anaesthesia; Clinical Evidence Synthesis.

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## Introduction

Axillary brachial plexus block is a well-established regional anaesthetic technique for surgery below the shoulder, particularly for procedures on the forearm, wrist, and hand. It is widely valued because it can provide surgical anaesthesia, reduce opioid use, preserve spontaneous ventilation, and extend postoperative pain relief. The comparison between dexmedetomidine and clonidine is clinically relevant because both agents belong to the same pharmacologic class yet differ in receptor selectivity, potency, and possibly adverse-event profile. If dexmedetomidine offers clearly superior block characteristics, it may justify preference in practice despite greater concern over bradycardia or sedation.

This review was therefore designed as a focused clinical evidence synthesis on dexmedetomidine versus clonidine in axillary brachial plexus block. Because the direct axillary comparative evidence base is narrow, the review deliberately combines axillary-specific context with the strongest available comparative data from brachial plexus block meta-analyses. The objective is not only to summarize

whether dexmedetomidine is superior to clonidine, but also to clarify the level of certainty for an axillary-specific conclusion, identify the main efficacy and safety signals, and present the evidence in a format usable for academic and clinical decision-making.

## Materials & Method

This document is a narrative systematic review and clinical evidence synthesis prepared from published comparative literature rather than a de novo meta-analysis of individual patient data. The evidence base was identified from indexed sources retrieved through targeted searches for dexmedetomidine, clonidine, axillary brachial plexus block, randomized trials, and meta-analysis. Priority was given to high-level evidence, particularly systematic reviews and meta-analyses of randomized controlled trials. Two major sources formed the backbone of the synthesis.

The first was the 2022 Braz J Anesthesiol meta-analysis that searched the Cochrane Library, PubMed, PubMed Central, Scopus, LILACS, Google Scholar, trial registries, and bibliographies without language or time restrictions up to September 22, 2021. The second was the 2017 Anesthesia & Analgesia systematic review and meta-analysis that examined randomized studies comparing perineural dexmedetomidine with clonidine for upper-extremity surgery, predominantly in supraclavicular block.

Study selection within the principal 2022 review included randomized controlled trials, whether blinded or open-label, comparing dexmedetomidine and clonidine as adjuvants to local anaesthetics in brachial plexus blocks. Exclusion criteria in that review comprised neuraxial or intravenous administration of the alpha-2 agonists, autonomic nerve blocks, interfacial plane blocks, observational studies, case series, case reports, duplicate studies, retracted reports, and studies from predatory journals. Adult patients older than 18 years undergoing upper-limb surgery under brachial plexus block were eligible, while paediatric populations and patients receiving general anaesthesia in addition to the block were excluded. These criteria were considered appropriate for informing an evidence synthesis centered on peripheral brachial plexus blockade.

Outcome domains extracted for this review were aligned with the major published analyses. Primary efficacy outcomes included duration of sensory block, duration of motor block, and duration of analgesia. Secondary outcomes included onset of sensory block, onset of motor block, time to complete sensory block, time to complete motor block, quality of block, rescue analgesic consumption, sedation, and haemodynamic or other adverse events. In the 2022 meta-analysis, continuous outcomes were pooled as mean differences or standardized mean differences and dichotomous outcomes as risk ratios or risk differences using inverse-variance methods and a random-effects model. Heterogeneity was assessed using the I2 statistic, and meta-regression examined local anaesthetic type, block localization technique, and dose ratio as potential moderators. Certainty of evidence was graded using the GRADE framework.

No new statistical computation was performed for this manuscript, but published effect estimates were extracted and organized for clinical interpretation. The review prioritized internally valid comparative findings over isolated uncontrolled observations. Where the published reviews differed, the more comprehensive and recent meta-analysis was given greater weight for pooled efficacy estimates, while the earlier systematic review was used to enrich safety interpretation because it specifically reported increased transient bradycardia and sedation with dexmedetomidine. Limitations such as high heterogeneity, sparse adverse-event reporting, and indirectness to the axillary approach were explicitly incorporated into the final appraisal.

## Observation Tables

**Table 1: Core Characteristics of the Principal Evidence Base**

| Study | Design and scope | Population | Block approach represented | Main message |
|---|---|---|---|---|
| Bajpai et al., 2022 | Meta-analysis of 24 randomized controlled trials | 1,448 patients | All included studies were brachial plexus blocks, predominantly supraclavicular; authors noted limited generalizability to other approaches | Dexmedetomidine prolonged sensory and motor block and analgesia more than clonidine, with moderate-to-high certainty for major efficacy outcomes |
| El-Boghdadly et al., 2017 | Systematic review and meta-analysis of 14 randomized controlled trials | 868 patients | Single-injection supraclavicular brachial plexus block | Dexmedetomidine improved sensory, motor, and analgesic block characteristics versus clonidine, but increased transient bradycardia and sedation |

**Table 2: Pooled Efficacy Outcomes Favoring Dexmedetomidine**

| Outcome | Effect estimate | Interpretation |
|---|---|---|
| Duration of sensory block | Mean difference 173.31 minutes longer; 95% CI 138.02 to 208.59 | Clinically important prolongation of sensory blockade with dexmedetomidine |
| Duration of motor block | Mean difference 158.35 minutes longer; 95% CI 131.55 to 185.16 | Longer motor block may improve intraoperative density but may delay functional recovery |
| Duration of analgesia | Mean difference 203.92 minutes longer; 95% CI 169.25 to 238.58 | More prolonged postoperative pain relief with dexmedetomidine |
| Onset of sensory block | Mean difference -1.58 minutes; 95% CI -2.18 to -0.99 | Faster sensory onset with dexmedetomidine, though the absolute gain is modest |
| Onset of motor block | Mean difference -1.46 minutes; 95% CI -2.38 to -0.54 | Modestly faster motor onset with dexmedetomidine |
| Quality of block | Risk ratio 1.97; 95% CI 1.60 to 2.41 | Higher probability of excellent block quality with dexmedetomidine |

**Table 3: Safety Observations from Comparative Syntheses**

| Safety endpoint | Evidence summary | Clinical implication |
|---|---|---|
| Hypotension | No significant difference in the 2022 meta-analysis; RR 2.59, 95% CI 0.63 to 10.66, based on only two studies | Evidence is too sparse to rule in or rule out a true difference |
| Bradycardia | Increased transient bradycardia with dexmedetomidine in the 2017 review; odds ratio estimate 7.4, 99% CI 1.3 to 40.8 | Monitoring is warranted, especially in patients with limited cardiovascular reserve |
| Sedation | Increased postoperative sedation with dexmedetomidine in the 2017 review; odds ratio estimate 11.8, 99% CI 1.9 to 73.6 | Sedation may be desirable intraoperatively but problematic in ambulatory recovery settings |

**Table 4: Applicability of Current Evidence to Axillary Brachial Plexus Block**

| Domain | Strength for axillary question | Appraisal |
|---|---|---|
| Direct head-to-head axillary evidence | Low | The strongest comparative reviews were dominated by supraclavicular rather than axillary studies |
| Pharmacologic plausibility | Moderate | The peripheral adjuvant mechanisms of alpha-2 agonists should not be unique to a single brachial plexus level |
| Overall comparative efficacy signal | High for brachial plexus block overall | Across pooled randomized evidence, dexmedetomidine consistently performs better than clonidine on major efficacy endpoints |
| Confidence in axillary-specific recommendation | Moderate to low | Recommendation for axillary block must be presented as cautious extrapolation rather than definitive axillary-only proof |

## Result

The evidence synthesis shows a consistent benefit in favor of dexmedetomidine when compared with clonidine as a perineural adjuvant in brachial plexus block. In the 2022 meta-analysis, dexmedetomidine significantly prolonged sensory block by about 173 minutes, motor block by about 158 minutes, and analgesia by about 204 minutes. The 2017 systematic review and meta-analysis reached the same overall conclusion using a somewhat different statistical approach. Across 14 studies and 868 patients undergoing single-injection supraclavicular block, dexmedetomidine extended sensory block, motor block, and analgesic duration by a ratio-of-means estimate of approximately 1.2 relative to clonidine. It also shortened both sensory and motor onset times.

Block quality also favored dexmedetomidine. The pooled risk ratio of 1.97 for higher-grade block quality suggests that dexmedetomidine nearly doubled the probability of achieving an excellent block as defined by the contributing studies. Better block quality can translate into fewer intraoperative complaints, less need for supplemental analgesia, and reduced conversion pressure toward general anaesthesia. This endpoint is especially relevant in axillary block, where incomplete nerve coverage can occasionally reduce procedural reliability.

The pooled estimates for sensory duration, motor duration, analgesia duration, and onset outcomes showed very high I2 values in the larger review, often 97% to 99%. This indicates substantial between-study variability, likely arising from differences in local anaesthetic type, volume, adjunct dose, block localization technique, and

outcome definitions. Importantly, although heterogeneity affected the exact magnitude of benefit, it did not reverse the direction of benefit; dexmedetomidine remained superior across major efficacy outcomes. The GRADE assessment still rated the evidence for the three primary duration outcomes as high quality because the association was strong and consistent in direction.

The 2022 meta-analysis found no significant difference in hypotension, but the estimate was based on only two studies and had a wide confidence interval, which limits certainty. The 2017 review, however, reported an increased risk of transient bradycardia and postoperative sedation with dexmedetomidine. These findings suggest that the efficacy advantage of dexmedetomidine may be accompanied by a higher probability of predictable alpha-2 agonist-related adverse effects, particularly in sensitive patients or at higher doses.

**Statistical Analysis**

The statistical backbone of the principal evidence source was a random-effects meta-analysis using inverse-variance methods, which is appropriate when clinically diverse trials are expected to vary in their underlying true effects. Continuous outcomes such as sensory block duration, motor block duration, and analgesia duration were summarized as mean differences with 95% confidence intervals, while dichotomous outcomes such as block quality and hypotension were summarized using risk ratios. The use of random-effects modeling acknowledged that differences in local anaesthetic formulations, doses of dexmedetomidine and clonidine, and localization techniques could produce genuine variation in effect size across trials.

Block localization technique emerged as a significant predictor for sensory block duration, with ultrasound-guided and other methods likely affecting local anaesthetic spread and therefore block performance. This is statistically and clinically important for axillary block because technique-dependent differences may be especially relevant when individual terminal nerves are targeted under ultrasound guidance. GRADE certainty was rated high for sensory block duration, motor block duration, and analgesia duration, moderate for quality of block, and lower for several secondary outcomes because of sparse data and inconsistency. From an evidence-synthesis perspective, this means the strongest statistical confidence attaches to the major duration endpoints rather than to adverse events or minor secondary measures.

**Discussion**

The findings of the present study align with the contemporary evidence base suggesting that dexmedetomidine is a more effective alpha-2 adrenergic agonist adjuvant than clonidine when combined with local anaesthetics for brachial plexus block. The largest recent comparative meta-analysis by Bajpai et al. included 24 randomized trials with 1,448 patients and demonstrated that dexmedetomidine produced longer sensory block, longer motor block, longer duration of analgesia, and better block quality than clonidine, without a statistically significant difference in hypotension risk. When the present study is viewed against these pooled data, any observation of prolonged block duration, improved postoperative analgesia, or superior block quality with dexmedetomidine is not an isolated result but part of a reproducible pattern across varied brachial plexus techniques and local anaesthetic regimens.

A major strength in comparing the present study with the literature is that several references focus specifically on supraclavicular brachial plexus block, the setting in which dexmedetomidine and clonidine have most often been directly compared. El-Boghdadly et al. analysed 14 clinical studies including 868 patients and found that, compared with clonidine, perineural dexmedetomidine prolonged sensory block, motor block, and analgesia by about 20% while also hastening onset; however, it was associated with higher rates of transient bradycardia and sedation. Therefore, if the present study observed earlier onset and longer postoperative pain relief with dexmedetomidine, it closely parallels this focused meta-analytic evidence, whereas any increase in bradycardia or sedation should be interpreted as an expected pharmacodynamic trade-off rather than an unexpected complication.

The direct randomized study by Swami et al. offers one of the most clinically relevant comparators for the present work because it compared dexmedetomidine and clonidine head-to-head in supraclavicular block using similar perioperative outcomes. In that trial, the onset of sensory and motor block did not differ significantly between groups, but dexmedetomidine significantly prolonged analgesia, with rescue analgesia required at 456 ± 97 minutes versus 289 ± 62 minutes with clonidine, and produced a higher proportion of excellent-quality blocks. If the present study likewise found a greater duration of analgesia and better block quality with dexmedetomidine, it strongly corroborates Swami et al.; if it additionally demonstrated faster onset, then the difference may reflect variation in local anaesthetic concentration, dose of adjuvant, technique guidance, or sample characteristics rather than contradiction of the older study.

The comparative findings of the present study are also supported by smaller prospective work such as that reported by Jinjil et al., which examined dexmedetomidine and clonidine as adjuvants to

ropivacaine in supraclavicular brachial plexus block. Although individual small trials are inherently less definitive than systematic reviews, the aggregate direction of evidence from such studies has consistently favored dexmedetomidine for prolonging sensory and motor block and extending analgesia after upper-limb surgery, thereby reinforcing the internal validity of similar results in the present study. Thus, when the present study demonstrates superiority of dexmedetomidine over clonidine, it should be interpreted as concordant with both pooled evidence and the trajectory of multiple head-to-head randomized comparisons.

The mechanistic basis for the superiority observed in the present study is biologically plausible. Virtanen et al. showed that medetomidine, whose active dextrorotatory enantiomer is dexmedetomidine, is a highly potent, selective, and specific alpha-2 adrenoceptor agonist with markedly greater alpha-2 to alpha-1 selectivity than reference compounds such as clonidine. This higher receptor selectivity provides a rational explanation for why dexmedetomidine tends to produce more pronounced prolongation of sensory and motor blockade than clonidine in both individual trials and pooled analyses, and it supports interpreting the present study's comparative advantage of dexmedetomidine as pharmacologically grounded rather than merely empiric.

When the present study is compared with the broader literature evaluating dexmedetomidine against local anaesthetic alone, the magnitude and direction of benefit become even easier to contextualize. Hussain et al. synthesized 18 randomized controlled trials involving 1,092 patients and showed that dexmedetomidine hastened sensory onset by about 3.19 minutes, hastened motor onset by about 2.92 minutes, prolonged sensory block by 261 minutes, prolonged motor block by 201 minutes, and prolonged analgesia by 289 minutes, with reversible bradycardia occurring more often than in controls. Accordingly, if the present study found that dexmedetomidine outperformed clonidine, that result is consistent with the premise that dexmedetomidine is itself a powerful block-enhancing adjuvant; clonidine may also prolong analgesia, but the literature suggests its effect size is smaller.

The updated meta-analysis by Vorobeichik et al. further strengthens this interpretation because it focused specifically on brachial plexus nerve blocks and incorporated a larger evidence base than earlier reviews. Across 32 trials and 2,007 patients, dexmedetomidine prolonged sensory block by at least 57%, motor block by at least 58%, and analgesia by at least 63%, while also reducing postoperative oral morphine consumption and improving patient satisfaction; however, it increased the odds of bradycardia and hypotension. Therefore, if the present study demonstrated improved analgesia quality and longer block duration with dexmedetomidine, these findings are highly consistent with established quantitative evidence, while any haemodynamic slowing should be interpreted within the same known safety profile.

Abdallah and Brull's earlier systematic review is also important because it captures the stage at which evidence for perineural dexmedetomidine was still emerging. In that 2013 analysis of nine randomized trials, the authors found that dexmedetomidine prolonged motor block duration and time to first analgesic request, and suggested possible prolongation of sensory block in brachial plexus block, though statistical confidence and safety data were still limited at that time. Compared with that early evidence, the present study contributes to a more mature body of literature in which subsequent trials and meta-analyses have converted early signals into consistent comparative conclusions, especially against clonidine rather than placebo or local anaesthetic alone.

Safety findings from the present study should be discussed with equal care because the literature does not support a purely benefit-only interpretation. El-Boghdadly et al. found an increased odds of transient bradycardia and postoperative sedation with dexmedetomidine compared with clonidine in supraclavicular block, while Schnabel et al. reported that perineural dexmedetomidine increased postoperative analgesia by around 4.87 hours compared with local anaesthetic alone but was also associated with higher risks of bradycardia and hypotension, though side-effect evidence was of low certainty. Consequently, if the present study showed superior analgesia with acceptable but slightly greater haemodynamic depression in the dexmedetomidine group, such findings would mirror the central efficacy-safety balance already recognized in the meta-analytic literature.

The place of clonidine in this comparison remains clinically relevant because it is not an ineffective adjuvant; rather, it appears less potent than dexmedetomidine. Popping et al. demonstrated that clonidine, compared with local anaesthetic alone, prolonged postoperative analgesia by about 122 minutes, sensory block by 74 minutes, and motor block by 141 minutes, but it also increased hypotension, bradycardia, orthostatic events, and sedation. This is an important interpretive point for the present study: when dexmedetomidine proves superior to clonidine, the comparison is between two active adjuvants, meaning superiority of dexmedetomidine reflects incremental benefit over an already useful comparator rather than benefit over no adjuvant.

Older reviews such as Murphy et al. and textbook descriptions such as Fischer's discussion of brachial

plexus anaesthesia provide useful historical context for the present study. These earlier sources describe the rationale for using adjuncts to improve block quality and prolong analgesia in brachial plexus anaesthesia, but they arose before the present volume of trial-based evidence had clarified the relative ranking of alpha-2 agonists in routine practice. In that sense, the present study belongs to a later evidence phase in which the question is no longer whether adjuvants help at all, but which adjuvant offers the best balance of block prolongation, onset characteristics, and haemodynamic tolerability.

The pediatric caudal study by El-Hennawy et al. and the epidural comparison by Bajwa et al. are not directly comparable with adult brachial plexus block, yet they are relevant because they show the same directional pharmacologic relationship between dexmedetomidine and clonidine across regional anaesthesia settings. In those non-brachial techniques, dexmedetomidine also prolonged analgesia more effectively than clonidine, supporting the concept that its superior alpha-2 agonism translates into stronger neuraxial and peripheral block augmentation across procedures. Therefore, if the present study concludes that dexmedetomidine is the better adjuvant in brachial plexus block, this inference is supported not only by same-technique trials but also by wider regional anaesthesia experience.

Another noteworthy comparison concerns the consistency of dexmedetomidine's effect despite variability in local anaesthetics and block approaches across studies. Esmaoglu et al. showed that dexmedetomidine added to levobupivacaine prolonged axillary brachial plexus block, while Dai et al., Ping et al., and other meta-analyses summarized in later reviews similarly found improved onset and longer analgesia with dexmedetomidine across different local anaesthetic formulations and study designs. This suggests that, if the present study used ropivacaine or bupivacaine in supraclavicular block and still found dexmedetomidine superior to clonidine, its conclusions remain externally consistent with evidence generated in axillary and mixed brachial plexus populations.

The 2021 review by Vorobeichik et al. revisited the question of whether peripheral nerve blocks should routinely be augmented with perineural dexmedetomidine and reflects a more cautious modern interpretation. While endorsing meaningful block enhancement, later reviews emphasize that benefits must be weighed against dose-dependent haemodynamic effects and the clinical consequences of excessively prolonged motor block, particularly in ambulatory settings. Hence, the present study should not merely report dexmedetomidine as statistically superior to clonidine, but should also discuss whether the observed prolongation was clinically desirable, whether discharge or monitoring was affected, and whether the dose used achieved an appropriate efficacy-safety balance.

In comparing the present study with prior evidence, methodological differences also deserve attention. Meta-analyses such as Bajpai et al. reported very high heterogeneity for sensory block, motor block, and analgesia outcomes, and identified block positioning technique as a significant predictor of heterogeneity, indicating that ultrasound guidance, paresthesia techniques, nerve stimulation, drug dose, and local anaesthetic choice can alter the apparent magnitude of benefit. Therefore, if the effect size in the present study is larger or smaller than pooled estimates, that does not necessarily weaken its validity; instead, it may reflect context-specific procedural and pharmacologic factors that are well recognized in the literature.

Taken together, the present study appears to fit closely within the dominant evidence narrative that dexmedetomidine is superior to clonidine as an adjuvant in brachial plexus block, especially for prolonging sensory and motor blockade, extending postoperative analgesia, and improving block quality. At the same time, comparison with the cited references indicates that this superiority should be presented alongside careful acknowledgement of transient bradycardia, sedation, and possible hypotension, since these adverse effects recur across meta-analyses and define the practical limits of routine use. A balanced interpretation, therefore, is that the present study confirms dexmedetomidine as the more efficacious adjuvant, but its clinical adoption should remain dose-conscious, monitored, and individualized to surgical setting and patient haemodynamic reserve.

## Conclusion

The present systematic review indicates that dexmedetomidine is superior to clonidine as an adjuvant to local anaesthetics in brachial plexus block when the comparison is judged on the outcomes that matter most to perioperative practice: duration of sensory blockade, duration of motor blockade, duration of analgesia, and overall block quality. The magnitude of benefit is clinically relevant rather than merely statistically significant. The conclusion must be framed with methodological honesty.

In summary, dexmedetomidine should be regarded as the preferred alpha-2 agonist adjuvant over clonidine when the clinical aim is to maximize block duration, enhance analgesia, and improve block quality in brachial plexus anaesthesia. For axillary brachial plexus block, this preference is evidence-informed and clinically reasonable, but it should be

expressed with acknowledgment of indirectness. A balanced conclusion for clinical evidence synthesis is therefore as follows: dexmedetomidine is likely more effective than clonidine in axillary brachial plexus block, but the certainty of that axillary-specific claim is moderate to low because most comparative data arise from other brachial plexus approaches.

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