Original Article

Effect of Buprenorphine as an Adjuvant to Local Anaesthetics in Axillary Brachial Plexus Block: A Prospective Randomized Controlled Study

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Abstract

Background: Axillary brachial plexus block is one of the cornerstone methods of anaesthesia for the upper extremity, yet the limited duration of postoperative analgesia from conventional local anesthetics, prompt the need for promising adjuvant to provide prolonged analgesia. Buprenorphine has the potential to prolong peripheral nerve block analgesia through peripheral and central mechanisms. This study aimed to evaluate the effect of buprenorphine on postoperative analgesia and block characteristics in axillary brachial plexus block. **Material and Methods:** This prospective, randomised, double-blind controlled trial enrolled 60 ASA I-II patients aged 20-60 years undergoing forearm and hand surgery. Participants received axillary brachial plexus block using a two-needle perivascular technique with either: Group B (n=30) receiving lignocaine 2% with adrenaline 1:200000 (5 mg/kg) + bupivacaine 0.5% (1.5 mg/kg) and Buprenorphine 0.3 mg; or Group C (n=30) receiving the same local anaesthetic mixture without Buprenorphine and total volume being 35 ml in either group. The primary outcome of this study is postoperative analgesic duration. Secondary outcomes comprised sensory & motor block characteristics, sedation, hemodynamic parameters, and complications. **Results:** Demographic profiles were comparable between groups. Postoperative analgesia duration was markedly prolonged in Group B (26.87 ± 11.58 vs. 6.95 ± 1.17 hours; $p<0.001$) compared to Group C. Sensory block duration was also prolonged in Group B (319.5 ± 48.7 vs. 268.0 ± 36.5 minutes; $p<0.001$). Motor block onset was faster in Group B (2.54 ± 1.40 vs. 3.31 ± 1.33 minutes; $p=0.03$), without prolongation of total motor block duration. Mild sedation occurred in 30% of Group B patients, while hemodynamic stability was maintained in both groups. Complication rates were similar, with no serious adverse events. **Conclusion:** Addition of Buprenorphine 0.3 mg to local anaesthetics in axillary brachial plexus block significantly prolongs postoperative analgesia, increases sensory duration while accelerating motor onset without prolonging motor blockade, offering a safe and effective technique for enhanced recovery in upper limb surgery.

Keywords: Buprenorphine, axillary brachial plexus block, regional anaesthesia, postoperative pain relief, opioid adjuvant.

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Introduction

Surgeries in the orthopaedic field account for a large proportion of regional anaesthetic procedures. Regional anaesthesia is associated with greater benefits compared with general anaesthesia, such as maintaining airway reflexes, maintaining hemodynamic stability, and efficiently managing postoperative pain.[1] The axillary technique is preferable compared to other approaches to brachial plexus blockade, as it has a good safety profile, is reliable, and does not cause severe complications such as pneumothorax and phrenic nerve palsy with supraclavicular or interscalene techniques.[2] Although these have advantages, the conventional use of local anaesthetics such as lignocaine and bupivacaine has constraints in their clinical use due to finite duration and postoperative analgesic effects.[3] This fact has led to the exploration of different adjuvant agents such as opioids, 2-agonists, and steroids to extend analgesia and enhance the quality of blocks.[4] The rationale for opioid adjuvants is based on the discovery of peripheral opioid receptors on primary afferent nerve fibres and their axonal transport, suggesting that these have a direct peripheral analgesic effect without requiring central nervous system penetration.[5,6]

Thebaine derivative buprenorphine has distinct pharmacological properties that qualify it as a peripheral nerve blocker. Being a lipophilic partial μ-opioid receptor agonist with a dissociation half-life of about 166 minutes, it is 25-50 times stronger than morphine.[7] Its prolonged kinetics of receptor dissociation and

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high affinity for μ-receptors theoretically make it have long-lasting analgesic effects when applied perineurally.[8] Past studies have shown that buprenorphine, as a local anaesthetic, when combined with the supraclavicular and interscalene brachial plexus, provides longer analgesia after surgery.[9,10] Nevertheless, data on the use of the axillary approach are very limited, especially in a mixed surgical sample.

Although encouraging evidence existed,[11,12] in which buprenorphine (0.3 mg) showed positive results when added with supraclavicular and axillary blocks, differences in practice methods and population characteristics across studies require additional confirmation. Moreover, the biochemical process underlying buprenorphine's peripheral-acting mechanism is not fully explained by proposed pathways involving direct binding to neuronal receptors, transport along central axons to dorsal horn synapses, and low systemic absorption due to its high lipophilicity,[13] The present research aims to fill these gaps by assessing the effects of buprenorphine, specifically in the axillary approach, using a high-rigor method to determine block effects, analgesic activity, and safety profile.

We were interested in the hypothesis that the effect of adding buprenorphine 0.3 mg to a usual lignocaine-bupivacaine combination was to significantly increase postoperative analgesia without negatively influencing motor block characteristics or hemodynamic stability. This prospective randomised controlled trial aims to provide evidence-based guidance for prolonging analgesia with axillary brachial plexus blockade in clinical practice.

Materials and Methods

Study Design and Setting: This prospective, randomised, double-masked controlled study was conducted at a tertiary care centre in western India during 2009-2010.

Participant Selection: Sixty patients of either sex, aged 20-60 years, with American Society of Anaesthesiologists (ASA) physical status I-II, scheduled for elective or emergency orthopaedic surgeries of the forearm and hand, were enrolled. Exclusion criteria comprised: known hypersensitivity to local anaesthetics or opioids; coagulopathies or bleeding disorders; cardiovascular, respiratory, hepatic, or renal impairment; pregnancy; opioid dependence; epilepsy; and diabetes mellitus. A detailed pre-aesthetic evaluation, including history, physical examination, and baseline investigations (haemoglobin, blood glucose, renal function tests, bleeding time, clotting time), was performed. Patients >40 years or with indicated comorbidities underwent chest radiography and electrocardiography.

Randomization and Blinding: Computer-generated randomization allocated patients in a 1:1 ratio to either Group B (buprenorphine) or Group C (control). Allocation concealment was achieved using sequentially numbered, sealed opaque envelopes. Study drugs were prepared by an independent anaesthesiologist not involved in block performance, while independent observers, blinded to group assignment, conducted all post-block assessments.

Anaesthetic Technique: All patients received standard premedication with intravenous glycopyrrolate 0.2 mg, ranitidine 50 mg, and ondansetron 4mg. No sedatives or analgesics were administered preoperatively. Intravenous access was secured with a 20-gauge cannula, and lactated Ringer's solution infusion commenced. Baseline vital parameters (pulse rate, blood pressure, respiratory rate, SpO_2) were recorded.

Axillary Brachial Plexus Block Procedure: The two-needle perivascular technique described by Burnham was employed. Patients were positioned supine, with the arm abducted to 90° and the elbow flexed. After sterile preparation, axillary artery pulsations were palpated at the lateral border of the pectoralis major muscle. Using a 24-gauge, 1.5-inch needle, the first needle was inserted superior to the artery at a 30° angle until a characteristic 'click' indicated sheath penetration. A second needle was introduced inferior to the artery. After negative aspiration for blood, half the prepared solution was injected through each needle. A proximal arm tourniquet was inflated for 5 minutes post-injection to prevent caudal spread, followed by gentle massage to ensure uniform distribution.

Study Drug Formulation: Group B received: lignocaine hydrochloride 2% with adrenaline 1:200,000(5 mg/kg) + bupivacaine hydrochloride 0.5% (1.5 mg/kg) + buprenorphine hydrochloride 0.3 mg (1 ml) diluted with sterile water to a total volume of 35 ml. Group C received an identical mixture without buprenorphine, with sterile water, for a total of 35 ml.

Assessment Parameters:

Sensory Block: Evaluated every minute for 30 minutes using a 23-gauge hypodermic needle at territories corresponding to median (thenar eminence), ulnar (little finger), radial (lateral dorsum of hand), and musculocutaneous (lateral forearm) nerves. Grading: Grade 0 (sharp pain), Grade 1 (analgesia-dull sensation), Grade 2 (anaesthesia-no sensation). Onset time is defined as the time to Grade 1 in any nerve territory; peak effect is the time to Grade 2 in all territories; duration is the interval from Grade 2 onset to Grade 1 recovery.

Motor Block: Assessed using a 3-point scale: Grade 0 (normal power), Grade 1 (paresis, weak grip), Grade 2 (paralysis, inability to move fingers). The time from Grade 1 to Grade 2 to Grade 1 recovery was recorded.

Success Criteria: Full block achieved when it is clear that Grade 2 sensory and motor function has been achieved in all four nerve areas in 30 minutes. An incomplete block was considered to have 1 nerve territory deficiency, whereas failure was defined as more than 1 unaffected nerve after 30 minutes.

Sedation Score: This scale was assessed after 30 minutes; the scale ranged 0 (fully awake), 1 (drowsy, reacts to spoken instructions), 2 (sleepy, can be aroused on touch), 3 (unresponsive).

Hemodynamic Parameters: Pulse rate, blood pressure, respiratory rate, and SpO_2 were recorded pre-operatively, every 5 minutes for 15 minutes post-block, then every 15 minutes intra-operatively, and hourly for 6 hours post-operatively.

Postoperative Analgesia: Assessed using a 10-cm Visual Analogue Scale (VAS) hourly for 12 hours, then at 12, 24, 36, and 48 hours. Duration of analgesia is defined as the time from block injection to a VAS score ≥4. Rescue analgesia comprised intravascular aqueous diclofenac sodium 1.5 mg/kg. Total analgesic consumption over 48 hours was recorded.

Complications: Monitored for nausea, vomiting, bradycardia

(>20% pulse rate decrease), respiratory depression (SpO_2<90% or RR<10/min), local anaesthetic toxicity, hypersensitivity, arterial puncture, hematoma, urinary retention, and neurological sequelae.

Statistical Analysis: Based on pilot data assuming a mean difference of 6 hours in postoperative analgesia duration with a standard deviation of 8 hours, a sample size of 28 patients per group was required to achieve 80% power at a two-sided α level of 0.05. To account for possible dropouts, 30 patients were enrolled in each group. Data were analysed using SPSS version 16.0. Continuous variables expressed as mean ± standard deviation were compared using Student's unpaired t-test. Categorical variables were analysed using the chi-square test. p<0.05 considered statistically significant, p<0.001 highly significant.

Results

Patient Demographics and Baseline Characteristics: Demographic data revealed no inter-group differences [Table 1]. Mean age was 34.8±12.5 and 30.9±12.4 years in Groups B and C, respectively (p>0.05). Male predominance was observed (70% overall) with comparable sex distribution. Mean weight (58.0±4.8 vs. 59.1±3.2 kg) and ASA distribution (76.7% vs. 83.3% ASA I) were similar. Surgical procedures predominantly comprised open reduction internal fixation (56.7%) and closed reduction (40.0%), with a mean surgical duration of 84.7±23.1 vs. 78.0±24.4 minutes (p>0.05).

Block Characteristics: Sensory Blockade: Mean onset time was 3.29±1.68 minutes in Group B versus 4.05±1.51 minutes in Group C (p>0.05). Time to peak sensory effect was 11.47±6.87 vs. 12.56±3.81 minutes, respectively (p>0.05). However, total sensory block duration was significantly prolonged in Group B (319.5±48.7 vs. 268.0±36.5 minutes; p<0.001) [Table 2].

Motor Blockade: Group B demonstrated significantly faster motor onset (2.54±1.40 vs. 3.31±1.33 minutes; p=0.03). Time to peak motor block was comparable (9.87±4.39 vs. 10.75±3.23 minutes; p>0.05). Total motor block duration showed no significant difference (280.0±52.3 vs. 291.0±35.8 minutes; p>0.05) (Table 2).

Sedation and Hemodynamic Parameters: Sedation analysis at 30 minutes revealed mild sedation in Group B: 70% scored 0 (alert), 23.3% scored 1 (drowsy), and 6.7% scored 2 (sleepy but arousable). All Group C patients scored 0 (p<0.001). Hemodynamic parameters remained stable throughout the perioperative period without inter-group differences (p>0.05). No episodes of bradycardia, hypotension, or respiratory depression occurred in either group.

Postoperative Analgesia: The primary outcome of post-operative analgesia duration was significantly prolonged in Group B (26.87±11.58 vs. 6.95±1.17 hours; p<0.001) compared to Group C. Notably, in the post-block 48-hour study period, 13.3% of Group B patients were absolutely pain-free, while all (100%) Group C patients required rescue analgesia. Rescue analgesic consumption was markedly lower in Group B compared to Group C patients (p<0.001) [Table 3].

Complications: Minor complications included nausea and vomiting in one Group B patient each, managed with ondansetron. Inadvertent arterial puncture occurred in 4 Group B and 5 Group C patients, resolved with brief digital compression and needle repositioning without sequelae. No hematoma, local anaesthetic toxicity, neurological deficits, or urinary retention were observed.

Table 1: Demographic and Baseline Characteristics

Parameter	Group B (n=30)	Group C (n=30)	p-value
Age (years)	34.83 ± 12.45	30.93 ± 12.37	>0.05
Sex (Male: Female)	21:09	24:06	>0.05
Weight (kg)	58.03 ± 4.81	59.13 ± 3.19	>0.05
ASA Status I:II (%)	76.7:23.3	83.3:16.7	>0.05
Surgical Duration (min)	84.66 ± 23.12	78.00 ± 24.35	>0.05

Values expressed as mean ± SD or ratio. Group B received buprenorphine; Group C served as control.

Table 2: Block Characteristics

Parameter	Group B (n=30)	Group C (n=30)	p-value
Sensory Block			
Onset time (min)	3.29 ± 1.68	4.05 ± 1.51	>0.05
Peak effect (min)	11.47 ± 6.87	12.56 ± 3.81	>0.05
Total duration (min)	319.5 ± 48.73	268.0 ± 36.52	<0.001
Motor Block			
Onset time (min)	2.54 ± 1.40	3.31 ± 1.33	0.03
Peak effect (min)	9.87 ± 4.39	10.75 ± 3.23	>0.05
Total duration (min)	280.0 ± 52.28	291.0 ± 35.81	>0.05

Values expressed as mean ± SD. Bold indicates statistically significant difference.

Table 3: Postoperative Analgesia and Sedation

Parameter	Group B (n=30)	Group C (n=30)	p-value
Postoperative Analgesia			
Duration (hours)	26.87 ± 11.58	6.95 ± 1.17	<0.001
Pain Free (%)	13.3%	0%	<0.001
Rescue analgesia required (%)	86.7%	100%	<0.001
Rescue doses in 48h (median)	3 (2-4)	5 (4-6)	<0.001
Sedation Score at 30 min			
Score 0 (%)	70.0	100.0	

Score 1 (%)	23.3	0.0	<0.001
Score 2 (%)	6.7	0.0	

Values expressed as mean ± SD, percentage, or median (range). Bold refers to statistically significant difference.

Discussion

This randomised controlled trial indicates that buprenorphine 0.3 mg when added to a lignocaine-bupivacaine mixture provides an axillary brachial plexus block that demonstrates a significant extension of postoperative analgesia and sensory blockage, albeit without motor block extension and without hemodynamic instability. These results complement and widen current knowledge about the use of peripheral opioid adjuvants during regional anaesthesia. The most significant clinical outcome is the 3 times longer duration of postoperative analgesia (26.9 vs. 7.0 hours). This is in line with prior research on the use of buprenorphine via supraclavicular and interscalene methods, which reported 17.4-22.3 hours of analgesia,[11,12] and a median of 20 hours.[9] The better outcome in our research could be the more distal administration of the axillary method, which could have enabled more perineural opioid-receptor communication before diffusion into central neuraxial areas. The finding of a 13.3 percent complete analgesia after 48 hours indicated buprenorphine's long action, which can be explained by its high receptor affinity and slow dissociation rate.[7]

Several pathways are implicated in the mechanism by which buprenorphine causes its peripheral analgesic effect. Opioid receptor sites were initially discovered in the primary afferent fibres by Fields et al.[5] Still, secondary studies have verified the presence of μ-receptors in peripheral nerves and dorsal root ganglia.[6] Buprenorphine is a lipophilic compound that allows rapid perineural penetration through strong nerve sheaths and myelin, resulting in greater bioavailability in the peripheral nervous system and minimising absorption into the central nervous system.[13] There are also axonal transport mechanisms that could facilitate centripetal migration to dorsal synapses in the horns, leading to spinal-level analgesia with minimal supraspinal effects.[14] This peripheral-spinal synergy is probably the cause of such efficient analgesia with no respiratory depression. The synergistic effects of the analogous local anaesthetic effect are evident, as the buprenorphine group showed a significantly earlier onset of motor block (2.5 vs. 3.3 minutes). Stein,[6] suggested that opioids reduce neuronal conductance of sodium and potassium and have a direct membrane-stabilizing effect. This action, which resembles a local anaesthetic one, coupled with the so-called core and mantle pharmacokinetic principle, according to which motor fibres can be influenced sooner by somatotopic organization,[15] can enhance the induction of a paresis. Notably, there were no differences in the duration of total motor block, which provides a clinical benefit by allowing early patient mobilisation and preserving intraoperative surgical conditions, unlike dexmedetomidine or clonidine, which tend to prolong motor blockade.[16]

The mild sedation observed in 30 percent of buprenorphine patients, predominantly during the first 6 hours, was likely due to partial systemic absorption and central κ-receptor stimulation.[17] Nevertheless, the moderate level of sedation did not have a clinical implication, and all patients were arousable and cooperative. This is unlike epidural administration, in which greater central bioavailability results in greater sedation.[18] Absence of respiratory depression in the face of buprenorphine, although buprenorphine is a potent drug, confirms the peripherally acting nature of the drug when buprenorphine is used in a peripheral nerve block. The safety profile of this technique is supported by the fact that both groups remained hemodynamically stable. Buprenorphine also has a peripheral effect, unlike the centrally acting adjuvant, which causes bradycardia and hypotension with clonidine.[16] The few complications (nausea/vomiting 3.3%) associated with the axillary approach are a testament to the inherent safety and attention to technique. The rate of and puncture of the arteries is in accordance with the literature of the two-needle technique,[19] and no cases of hematomas were detected, which testifies to the safety of using fine-gauge needles and sufficient compression.

When compared to other opioid adjuvants, buprenorphine is superior. Morphine and fentanyl have not consistently provided reliable results regarding brachial plexus blockade, and some studies have yet to show analgesic effect.[20,21] The pharmacological properties of buprenorphine include high lipophilicity, a long duration of occupancy, and partial agonist effects, which make it a good fit for peripheral use. The selection of the dose of 0.3 mg was postulated by the work of Candido et al., who demonstrated good efficacy without dose-related effects.[12] However, future dose-response strategies would narrow this down.

Limitations of the study should be mentioned. The single-centre, specific surgical population (orthopaedic trauma) can limit generalisability to other surgical subtypes or patient populations. It can only apply to the extent that diabetic patients are not being considered in the research conducted in the region because of the issue of neuropathy. Even though our study sample provided sufficient power to yield the primary findings, additional multicenter studies would have increased external validity. We also did not measure plasma buprenorphine concentrations to assess systemic absorption, nor did we conduct long-term neurological follow-up beyond 48 hours. The future research must include pharmacokinetics and prolonged neurological monitoring. Clinical implications are high. Prolonged analgesia in ambulatory surgery reduces oral opioid use to decrease same-day discharge and is cost-effective and more effective at opioid stewardship.[22] It is also useful in resource-constrained environments where continuous catheter methods can be inconvenient. Moreover, the maintained motor function time allows for early initiation of physiotherapy, which is vital for positive functional recovery after hand and forearm surgeries.

Conclusion

The addition of buprenorphine 0.3 mg to lignocaine-bupivacaine in axillary brachial plexus block in this randomised controlled

trial was associated with longer analgesia during the postoperative phase, without extending the motor block, and with no significant adverse effects. These results imply that buprenorphine as an adjuvant in axillary blocks is potentially effective; however, more recent studies using ultrasound techniques should be conducted and then adopted on a larger scale.

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Conflicts of interest
There are no conflicts of interest.

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