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# A Case Series on the Utility of Dexmedetomidine for the Immediate Treatment of Postoperative Muscle Spasm

Van N. Trinh*, Joseph E. Villaluz

Department of Anesthesiology, Kaweah Health Medical Center, Visalia, CA, USA

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***Correspondence:**
*Dr. Van N. Trinh, Department of Anesthesiology, Kaweah Health Medical Center, Visalia, CA, USA;
Email: vtrinh@kaweahhealth.org



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## Abstract

Postoperative muscle spasm is a common complaint in the post-anesthesia care unit. Management of postoperative muscle spasm remains a major challenge as first-line anti-spasmodic agents are not without risk. Due to the adverse effects of sedation and risk for respiratory depression and pulmonary aspiration, conventional anti-spasmodic agents may not be an ideal choice for patients who are recovering from anesthesia. In this case series, we present three patients who underwent nonemergent surgeries with subsequent postoperative cervical muscle spasm that went unresolved with conventional PACU pain medications. Below, we demonstrate the potential utility of dexmedetomidine for management of postoperative muscle spasm. This is most notable when oral anti-spasmodic formulations are contraindicated in sedated patients and intravenous skeletal muscle relaxants are inaccessible in the PACU. After receiving incremental boluses of dexmedetomidine, the patients demonstrated immediate improvement in their cervical range of motion and their cervical muscle spasms were markedly reduced. Dexmedetomidine's unique qualities, such as its quick onset, intravenous route of administration, and minimal effect on respiratory physiology, make it an ideal choice for management of postoperative muscle spasm.

## Background

Postoperative muscle spasm is a common source of pain in the post-anesthesia care unit (PACU). Postoperative muscle spasm is commonly seen in patients who have undergone abdominal, cervical and lumbar related surgeries[1,2]. Treatment of muscle spasm remains a major challenge in the PACU as first-line anti-spasmodic agents are not without risk.

For example, the use of centrally acting skeletal muscle relaxants such as cyclobenzaprine, metaxalone, carisoprodol and methocarbamol is limited due to their adverse effects. These side effects include sedation, dizziness, drowsiness and blurry vision[3]. Unlike other skeletal muscle relaxants, methocarbamol can be administered intravenously. Although methocarbamol's intravenous formulation makes it an ideal agent in the PACU, its efficacy in the management of muscle spasm is questionable. A systematic review conducted by Chou and colleagues assessed the evidence for the comparative efficacy of skeletal muscle relaxants. The review found both inconsistent data and insufficient evidence to determine its relative efficacy and safety for treatment of musculoskeletal conditions[4].

Benzodiazepines, such as diazepam, are well-known for their management of spasticity associated with upper motor neuron disorders and as an adjunct therapy for muscle spasms[5].

Similarly to methocarbamol, diazepam is available in parenteral formulation. Nonetheless, its utility in the PACU is uncommon due to its adverse effect of respiratory depression especially in patients with severe respiratory insufficiency and obstructive sleep apnea. The concomitant use of diazepam with opiate agonists may cause profound sedation, respiratory depression, and death[5]. Importantly, administration of diazepam to older adults with already increased sensitivity to benzodiazepines and slower drug metabolism increases their risk for cognitive impairment, delirium, falls, and fractures[6].

Tizanidine, a centrally acting alpha-2 adrenergic agonist is also recognized for its efficacious treatment for myofascial pain syndrome[7]. However, similarly to the centrally acting skeletal muscle relaxants, tizanidine shares a common side effect of somnolence and drowsiness. Furthermore, its oral formulation restricts its use in sedated patients. The increased risk of aspiration along with the slow onset of action and requirement for titration over two to four weeks for optimal dosage, limits its efficacy in patients who require immediate relief from postoperative muscle spasm[8].

An ideal agent for management of postoperative muscle spasm requires the qualities of quick onset, convenient intravenous route of administration, and minimal effect on respiratory physiology. Because dexmedetomidine shares a similar mechanism of action to tizanidine, both being centrally alpha-2 adrenergic agonists, we have utilized dexmedetomidine with the goal of therapeutic efficacy in postoperative muscle spasm. In the case series, we present three cases where dexmedetomidine was administered and titrated to effect in order to treat patients with severe cervical muscle spasm after their procedures.

## Case Series

Consent for journal publication was obtained for patient in Case 1. No consents were obtained for patients in Case 2 and 3 since the patients were deceased and lost to follow up, respectively. Attempts were made to contact legal representatives for several months without success. Discussion of these patients' experiences were sufficiently anonymized to preclude identification of the subjects.

In this case series, all three patients underwent nonemergent procedures. The patients were premedicated with midazolam for anxiety prior to surgery. In the operating room, standard ASA monitors were applied and preoxygenation was conducted for at least five minutes. The patients received general anesthesia with standard induction with some variability, such as the addition of ketamine in Case 2 and use of succinylcholine in Case 3. Mask ventilation along with endotracheal intubation were easily performed. Anesthesia was maintained with sevoflurane in Case 1 and continuous intravenous infusion of propofol (50mcg/kg/min) and remifentanil (0.15mcg/kg/min) in Case 3. Emergence from anesthesia and extubation were executed uneventfully with the exception of Case 1 in which the patient experienced a sugammadex-induced hypersensitivity response. The patients were hemodynamically stable and transported to PACU with supplemental oxygen.

In the PACU, the patients complained of severe postoperative muscle spasm in the form of cervical muscle spasm or torticollis that was unresolved with opioid administration. Oral and IV skeletal muscle relaxants were not given at the time due to their delayed onset of action, lack of immediate availability in our PACU, and increase risk of aspiration especially in sedated patients. The case presentations below describe the unique use of dexmedetomidine and how it may be an ideal choice in the management of muscle spasm in patients who are recovering from anesthesia.

### Case 1

A 46-year-old male (106.6kg, BMI 34.0kg/m$^2$) with a past medical history of anxiety, depression, post-traumatic stress disorder, chronic neck pain, obstructive sleep apnea, and cervical spinal fusion presented for an elective right shoulder arthroscopy.

Prior to surgery, the patient received an interscalene nerve block. He underwent general anesthesia and was induced with the following medications: 100mcg of fentanyl, 60mg of lidocaine 2%, 200mg of propofol, and 60mg of rocuronium. The patient was intubated successfully with indirect video laryngoscope. Patient was securely placed in beach chair position. Anesthesia was maintained with sevoflurane. Surgery was uneventful. The patient was extubated awake with temporal presentation of bronchospasm after sugammadex administration.

Approximately 20 minutes in the PACU, the patient endorsed severe muscle spasm and pain with limited cervical mobility despite the administration of fentanyl 100mcg and acetaminophen 1000mg. Dexmedetomidine was titrated to effect with incremental boluses of 8mcg IV every one to two minutes for a total of 100mcg. Within 20 minutes of dexmedetomidine administration, the patient demonstrated marked improvement with cervical range of motion and his pain score decreased to zero. The patient remained arousable and hemodynamically stable throughout the drug administration. Vital signs were within normal range with blood pressure approximately 140/100, heart rate in the 70s, and pulse oximetry reading above 95% on room air. Shortly after, the patient was discharged home and denied any residual cervical muscle spasm.

The patient was followed up three months later via telephone and E-mail where he expressed, "I have been on

opioids and benzodiazepines to manage this [neck pain] for ten years…however, nothing compared to this drug you gave me. It lasted into the night, and never had so much relief"[9].

### Case 2

An 80-year-old female (53kg, BMI 31.6kg/m$^2$) with left anterior shoulder dislocation secondary to a fall, was scheduled for a closed shoulder reduction after multiple failed reduction attempts in the emergency department. Her past medical history consisted of dementia, hypertension, and hyperlipidemia.

In the operating room, the patient underwent general anesthesia and was induced with the following medications: 100mcg of fentanyl, 50mg of ketamine, 10mg of propofol, and 30mg of rocuronium. Patient was intubated successfully with direct laryngoscopy. Attempts to reduce the shoulder for at least five minutes were unsuccessful as the humeral head was non-mobile and chronically scarred to its current location. For this reason, further reduction attempts were aborted. The patient was subsequently reversed with 400mg of sugammadex, extubated without complications, and transported to PACU.

After ten minutes in the PACU, the patient endorsed left upper extremity contracture along with severe torticollis and spasm of the sternocleidomastoid muscle. These symptoms were both visualized and palpated by nursing and physician staff. Dexmedetomidine was used as an immediate alternative to treat her muscle spasm. Unlike Case 1, dexmedetomidine was titrated to effect with smaller boluses of 4mcg every one minute for a total of 20mcg with the understanding that elderly patients are more sensitive to anesthetics and susceptible to unwanted hemodynamic variations. Within five minutes of the administration of dexmedetomidine, the patient was no longer grimacing and moaning in pain. She showed instant improvement in cervical range of motion, neck suppleness, and relaxation of sternocleidomastoid muscle. On physical exam, the previously taut muscle band felt supple comparable to that of the contralateral side. The patient remained hemodynamically stable throughout her recovery in the PACU. Vital signs were within normal range with blood pressure at 114/50, heart rate in the 60s, and pulse oximetry at 100% on two liters nasal cannula. After meeting the Aldrete's discharge criteria, the patient was transported to the medical floor for close observation.

### Case 3

A 61-year-old female (50kg, BMI 16.5kg/m$^2$) with multiple comorbidities including Takotsubo cardiomyopathy with normal ejection fraction, hypertension, daily marijuana use, smoking, systemic lupus erythematous, and anxiety was scheduled for an anterior cervical decompression and fusion at cervical 4-7 levels.

The patient underwent general anesthesia and was induced with the following medications: 200mcg of fentanyl, 100mg of lidocaine 2%, 100mg of propofol, and 100mg of succinylcholine. Unlike Cases 1 and 2, succinylcholine was utilized during the induction of this case as monitoring for motor evoked potentials was required. The patient was intubated using fiberoptic bronchoscope to minimize cervical manipulation during intubation. Anesthesia was maintained with propofol and remifentanil infusion to avoid interference with monitoring sensory evoked potentials. Emergence and extubation were uneventful.

Approximately 30 minutes in the PACU, the patient was tearful as a result of neck pain and muscle spasm. On examination, the patient had severe cervical muscle spasm and restricted cervical range of motion that was markedly decreased from preoperative exam. Dexmedetomidine was easily accessed in our PACU and was administered in 10mcg boluses every one minute for a total of 50mcg. The patient's pain improved from a pain score of six to zero within seven minutes of starting drug administration. She remained alert and oriented, hemodynamically stable, and did not require additional pain medication. Vital signs were within normal range with blood pressure at 106/53, heart rate in the 80s, and pulse oximetry reading 98% on room air. Afterwards, she was transferred to the medical floor without further complaint of cervical muscle spasm or pain.

## Discussion

Management of postoperative muscle spasm remains a challenge in the PACU due to the adverse effects of sedation, risk of aspiration, and slow onset of action surrounding first-line anti-spasmodic agents. In the case series, we recognized the positive effects of dexmedetomidine,

**Table 1:** Data on the pain score, hemodynamics, and total consumption of fentanyl pre- and 30 minutes post- administration of dexmedetomidine.

| Cases | Pain score | | Blood pressure | | MAP[1] | | Heart rate | | Total opioid consumption in PACU (fentanyl) | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post | Pre | Post | Pre | Post |
| 1 | 10 | 0 | 139/93 | 141/95 | 106 | 104 | 101 | 78 | 100mcg | 0 |
| 2 | *N/A | *N/A | 176/61 | 114/42 | 75 | 71 | 75 | 64 | 0 | 0 |
| 3 | 6 | 0 | 167/80 | 106/53 | 109 | 73 | 108 | 86 | 0 | 0 |

*Not applicable
[1]Mean arterial pressure

specifically its ability to reduce muscle spasm with minimal effect on respiration and ability to keep the patient arousable in a dose dependent manner[10].

Though limited, some literature has discussed the potential benefit of dexmedetomidine for the management of muscle spasm in patients with tetanus. For example, Girgin et al. described how dexmedetomidine infusion was used to treat six patients with tetanus over seven days. Though it did not completely abolish muscle spasms and rigidity, it managed to reduce the need for other sedatives and muscle relaxants[11]. A case report by Miya and others favorably suggests the concomitant use of dexmedetomidine and propofol to suppress muscle spasms of patients with severe tetanus without the additional use of benzodiazepines and muscle relaxants[12]. Furthermore, Talukdar and colleagues were the first to use dexmedetomidine in a pediatric patient with systemic tetanus and proposed its potential effects to significantly reduce muscle spasms[13].

The mechanism of dexmedetomidine's role in reducing muscle spasm remains unknown. Rank et al. have explored the promising effect of modulating adrenergic receptors to reduce muscle spasm in rats after chronic spinal injury. This study claimed that alpha-2 receptor agonists most likely produce their antispastic action primarily by inhibiting afferent transmission to motor neurons and secondarily by hyperpolarizing motor neurons, making motor neurons less likely to be activated during muscle spasm[14]. In a different study, dexmedetomidine was found to be superior to propofol in the control of paroxysmal sympathetic hyperactivity, specifically, shortening the average remission time and frequency of paroxysmal hypermyotonia[15]. The mechanism is mostly by dexmedetomidine's ability to stimulate both presynaptic and postsynaptic membrane alpha-2 receptors which inhibits norepinephrine release and neuronal excitability[15]. A case report by Fujino et al. has also described the potential mechanism of dexmedetomidine in management of a patient with progressive encephalomyelitis with rigidity and myoclonus (PERM). They suggested that dexmedetomidine ameliorated PERM-associated symptoms by decreasing noradrenergic neuronal activity which resulted in the attenuation of antibody-mediated disinhibited hyperexcitability of motor neurons[16].

Despite the effective treatment observed in the three patients, there are limitations to our case series. First, there is no set standard dosage for dexmedetomidine administration causing its effect to be subjected only to the anesthesiologist's discretion. Second, we understand that there is a possibility that the observed effect may not have been due to dexmedetomidine alone. It could have resulted from the synergistic effects between dexmedetomidine and other pain adjuvants, such as fentanyl, remifentanil, ketamine, or acetaminophen. Although the exact pharmacokinetic and pharmacodynamic interactions remain unknown, synergy has been observed when dexmedetomidine is combined with paracetamol and opioid analgesics. A study conducted by Hadipourzadeh et al. showed that adding paracetamol to dexmedetomidine in a continuous infusion pump can provide better analgesia, reduce the need for opioids, and reduce the duration of intubation after cardiac surgery[17]. According to Weerink and colleagues, the addition of remifentanil to dexmedetomidine sedation reduced the intensity of painful stimuli without compromising the subjects' ability to arouse to graded stimuli, such as trapezius squeeze and laryngoscopy[10]. The synergistic antinociceptive interaction between dexmedetomidine and pain adjuvants such as ketamine, morphine, and fentanyl have also been described in acute pain models and at the spinal levels in rodents[18,19]. Despite literature supporting dexmedetomidine's synergy with other medications in regards to analgesia, there have yet to be robust studies in regards to dexmedetomidine's synergy resulting in muscle spasm relief.

In conclusion, we identify a potentially positive response to dexmedetomidine and its role to reduce and treat muscle spasm in the PACU setting. Unlike previous studies where dexmedetomidine was administered as an infusion to treat muscle tetany, we interestingly showed how bolused dosing of dexmedetomidine can be used in the PACU setting to reduce pain and muscle spasm. As described in our previous case report on dexmedetomidine, its unique qualities of quick onset, intravenous route of administration, titratability, and minimal effect on respiratory physiology make it an ideal choice for immediate treatment of postoperative muscle spasm[9]. As shown in Table 1, dexmedetomidine can be safely titrated in the PACU. In our limited case series, our patients remained hemodynamically stable without need for intervention for bradycardia, hypotension, or respiratory support. As a result, further prospective and larger studies to investigate dexmedetomidine as a viable treatment for postoperative muscle spasm are needed. Investigations into the mechanism, proper dosage, efficacy of bolused titration versus continuous infusion, and safety profile as a sole agent will help solidify dexmedetomidine as a tool in the armamentarium of PACU treatments for muscle spasm.

## Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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