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Review Article

Effect of Dexmedetomidine on Pulmonary Artery Pressure: A Systematic Review

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ABSTRACT

Background: Dexmedetomidine, a selective α2-adrenergic receptor agonist, is widely used for sedation and analgesia in critically ill pediatric patients. Its dose-dependent modulation of pre- and postsynaptic receptors induces sympatholysis and vascular effects. While systemic hemodynamic impacts are well-documented, its influence on pulmonary artery pressure (PAP) remains underexplored. This systematic review evaluates dexmedetomidine's effects on PAP.

Methods: This systematic review analyzes studies from databases including SID, IranMedex, Magiran, Google Scholar, Cochrane, Scopus, and Web of Science (2005–2024). Keywords such as "dexmedetomidine," "pulmonary artery pressure," and "pulmonary effects" identified cross-sectional studies assessing PAP changes. Fifteen high-quality articles met inclusion criteria.

Results: Dexmedetomidine's effects on PAP seem inconsistent. Animal studies have reported both increased PAP with intravenous administration and no significant changes. Paradoxically, some models demonstrated PAP reduction in hypertensive states via suppressed vascular contraction. Human studies have observed transient PAP elevation after bolus dosing, though loading doses have shown no sustained pulmonary vascular effects. Preoperative administration reduced pulmonary vascular resistance and mean arterial pressure. Secondary pulmonary outcomes included improved oxygenation and lung mechanics in restrictive lung disease, though benefits were not universal.

Conclusion: Dexmedetomidine exhibits variable PAP modulation, with evidence suggesting transient pressure spikes after bolus doses but neutral or beneficial effects in controlled administrations. Animal-human discrepancies highlight physiological differences, necessitating further clinical research. Beyond hemodynamics, dexmedetomidine may enhance oxygenation and ventilation-perfusion matching while mitigating pulmonary inflammation, though inconsistent oxygenation outcomes underscore context-dependent variability. These findings emphasize cautious dosing in pulmonary hypertension and identify gaps for future human trials to clarify its role in cardiopulmonary management.

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Introduction

Dexmedetomidine (Dex) is a potent α2-adrenergic receptor agonist, widely recognized for its sedative, anti-anxiety, pain-relieving, and sympatholytic effects. Initially approved in 1999 for short-term sedation in mechanically ventilated ICU patients, its use has since expanded across various clinical settings, including surgical procedures, procedural sedation, and pain management. Unlike traditional sedatives such as propofol or benzodiazepines, Dex induces a natural, sleep-like state by acting on the locus coeruleus, enabling easy patient arousal while maintaining effective sedation. Moreover, its analgesic properties significantly reduce postoperative opioid requirements, making it especially advantageous for patients at risk of respiratory depression, such as those undergoing bariatric surgery [1-3].

One of the significant advantages of Dex is its minimal impact on respiratory function, making it a safer option for ICU sedation and weaning patients from mechanical ventilation. In addition, it provides hemodynamic stability, although potential side effects such as bradycardia and hyper- or hypotension may occur, particularly with high doses or rapid infusion. These effects can be mitigated by employing slower infusion rates and adjusted loading doses. Dex has also been investigated for managing withdrawal from opioids, benzodiazepines, and cocaine, owing to its ability to reduce sympathetic activity without causing notable respiratory compromise. Furthermore, its neuroprotective properties may contribute to reducing cerebral ischemic injury by modulating neurotransmitter release and enhancing anti-apoptotic protein activity. However, further studies are required to confirm these neuroprotective effects [3].

Despite its numerous benefits, Dex does have limitations. One notable concern is its potential role in cancer progression, as some in vitro and in vivo studies suggest it may enhance tumor proliferation and migration by upregulating anti-apoptotic proteins. However, the clinical significance of these findings in oncological outcomes remains unclear.

Prolonged use of Dex beyond the FDA-approved 24-hour limit has been demonstrated to be safe in multiple studies, with some reporting its use for up to 30 days. With its unique pharmacological profile, Dex continues to expand its applications in clinical anesthesia, offering an effective alternative to traditional sedatives by providing sedation, analgesia, and hemodynamic control with minimal respiratory complications [4-5].

Chronic pulmonary hypertension (PH) is a multifaceted condition, distinct from systemic hypertension. In 2018, the Sixth World Symposium on Pulmonary Hypertension redefined PH as a mean pulmonary artery pressure (mPAP) exceeding 20 mm Hg. PH is categorized into three hemodynamic profiles: isolated precapillary PH, combined pre- and postcapillary PH, and isolated postcapillary PH, with definitive diagnosis requiring right heart catheterization. Advancements in understanding PH pathogenesis have led to the development of new therapies, contributing to improved survival rates. With rising obesity rates, an increasing number of PH patients may require surgical interventions. However, noncardiac surgery outcomes for this population remain less thoroughly understood. While echocardiography can estimate pulmonary arterial systolic pressure (PASP), accurate mPAP measurement necessitates right heart catheterization. Precapillary PH is characterized by a pulmonary vascular resistance (PVR) of ≥3.0 Wood units (WU) and normal left atrial pressure. In contrast, isolated postcapillary PH presents with elevated left atrial pressure and normal PVR. Combined pre- and postcapillary PH often correlates with heart failure [6-8].

Pulmonary arterial hypertension (PAH) can be categorized into various forms, such as idiopathic PAH, heritable PAH, and PAH induced by drug or toxin exposure. It is often linked to underlying conditions, including connective tissue disorders, HIV infection, portal hypertension, congenital heart abnormalities, and schistosomiasis. While some patients respond favorably to calcium channel blockers, others may exhibit features indicative of venous or capillary involvement.

PAH can also manifest as persistent pulmonary hypertension of the newborn (PPHN) and may arise secondary to left heart disease, which includes left ventricular systolic dysfunction (LVSD), left ventricular diastolic dysfunction (LVDD), or valvular heart disease (VHD). Various congenital and acquired conditions contribute to postcapillary pulmonary hypertension [9].

Additionally, pulmonary hypertension can result from lung diseases and/or hypoxia, encompassing obstructive and restrictive lung diseases as well as other pulmonary conditions with mixed restrictive/obstructive patterns. Hypoxia may occur independently of lung disease and may also be associated with developmental lung abnormalities. Chronic thromboembolic pulmonary hypertension (CTEPH) and other pulmonary arterial obstructions fall under this category.

Finally, pulmonary hypertension can arise from unclear multifactorial mechanisms, involving hematologic, systemic, or metabolic disorders, as well as complex congenital heart disease [8].

Although numerous studies have evaluated the effects of Dex on systemic blood pressure, there is limited information regarding its impact on PAP, particularly in critically ill pediatric patients following cardiothoracic surgery and cardiopulmonary bypass (CPB). This study

aims to comprehensively review the available literature and materials on this topic to draw a well-founded conclusion that may have been challenging to establish in the past.

Methods

In this systematic review, articles published between 2005 and 2024 in domestic and international journals were searched across databases including the Scientific Information Database (SID), Iran Medex, Magiran, UpToDate, Google Scholar, Cochrane, Scopus, and Web of Science. The keywords used included both Persian terms and their English equivalents for Dex, PAP, and pulmonary effects of Dex. All animal intervention, cross-sectional, and randomized clinical trial studies investigating the effects of Dex on PAP were included in the review.

To enhance validity and reliability, the articles were independently searched and their quality evaluated by two researchers. Initially, a list of titles and abstracts of all available articles was compiled and examined to identify and select relevant studies. Subsequently, the selected articles were incorporated into the research process without bias or external influence. Ultimately, 23 high-quality articles were included in the systematic review.

Results

The included animal and human intervention studies are summarized in (Table 1) [10-24].

In the animal studies group, diverse findings were reported regarding the effects of Dex on PAP. Some studies observed an increased PAP with intravenous Dex [15, 20]; a study found that whether Dex was administered via the peripheral vein or directly into the pulmonary artery, the outcomes remained consistent, with no elevation in mean PAP [17]. Additionally, one study suggested that Dex could mitigate PAP hypertension by inhibiting smooth muscle cell proliferation [18]. Finally, a study highlighted that adrenaline-induced porcine pulmonary artery vascular smooth muscle contraction was suppressed by Dex, leading to a reduction in PAP [10].

The human studies group yielded intriguing and conflicting findings. Some studies reported slight and transient elevations in PAP when Dex was administered in bolus doses [12]. However, Dex-loading doses, while capable of causing significant systemic vasoconstriction and hypertension, were found not to impact the pulmonary vasculature, even in pediatric patients with pulmonary hypertension, as noted in certain studies [11, 13, 16]. Oddly enough, some studies found that the pre-operative administration of dexmedetomidine attenuates the increase in pulmonary vascular resistance and decreases the need for administrating vasodilators, resulting in a lower MAP and MPAP [21-22, 24], while Zhang et al. found that using Dex will stabilize hemodynamics and lower PAP [23]. Additionally, there have been studies concluding that Dex is seemingly safe for use following congenital cardiac surgery [19, 25].

Several studies have investigated Dex's effects on ventilation. According to Xu et al., improving ventilation can help manage pulmonary hypertension and reduce PAP [26]. (Table 2) summarizes studies examining Dex's influence on ventilation [27-34]. Some studies found that Dex improves oxygenation by increasing the PaO_2/FiO_2 (P/F) ratio, enhancing lung compliance, reducing dead space, and lowering plateau pressure. It supports hypoxic pulmonary vasoconstriction (HPV), thereby optimizing ventilation/perfusion (V/Q) matching, while also mitigating oxidative stress and inflammation. Additionally, it may exert a bronchodilator effect. These factors collectively enhance lung mechanics and gas exchange, leading to moderate but significant improvements in oxygenation, particularly in morbidly obese patients with restrictive lung disease [27-28]. Another study highlighted Dex's protective effects on the lungs, including its ability to reduce inflammation and pulmonary damage through inhibiting pro-inflammatory cytokines, suppressing neutrophil infiltration, and preventing edema. By downregulating the TLR4/MyD88/MAPK pathway and reducing JNK and ERK1/2 activation, it alleviates oxidative stress and helps preserve lung function in animal models [29]. However, the need for further research remains, as some studies reported no improvement in oxygenation following Dex administration [30].

Table 1- Summary of animal and human intervention studies on the effect ofmedetomidine on pulmonary artery pressure

Author	Year	Design	Sample Size	Findings	Subjects
Sabine B R Kästner	2005	Comparative Study	6 Sheep	The injection of Dexmedetomidine (Dex) caused a temporary rise in mean pulmonary arterial pressure (MPAP) and pulmonary vascular resistance.	Animal

A. K. But	2006	Randomized Controlled Trial	32 Patients	Dex decreases effectively MAP and MPAP.	Human
S.B.R. Ka¨stner	2007	Animal intervention	7 Sheep	After the administration of Dexmedetomidine (Dex), there was a temporary two- to threefold rise in mean pulmonary arterial pressure (MPAP), pulmonary artery occlusion pressure (PAOP), and pulmonary capillary pressure (Pc).	Animal
Judith P. Lazol	2010	Prospective, observational, pilot study	22 Patients	The administration of Dexmedetomidine (Dex) following congenital cardiac surgery showed no association with an increase in pulmonary artery pressure.	Human
EH Jooste	2011	Randomized Controlled Trial	12 Patients	With bolus dosing, a temporary rise in wedge pulmonary arterial pressure (wPAP) was observed at one minute, returning to baseline within five minutes.	Human
Robert H. Friesen	2013	Comparative Study	21 Patients	In children, the pulmonary vasculature does not exhibit notable vasoconstriction when subjected to initial loading doses of Dexmedetomidine (Dex).	Human
J. Zhang	2013	Randomized Controlled Trial	40 patients	In patients receiving dexmedetomidine, hemodynamic stability was more effectively preserved, accompanied by a reduction in pulmonary artery pressure (PAP) following cardiopulmonary bypass.	Human
Unal Y	2014	Animal intervention	16 Pigs	Dex infusion did not elevate the mean PAP	Animal
S. Nishibe	2014	Retrospective Study	29 Patients	Dex infusion did not elevate the mean PAP	Human
Lidan Nong	2016	Prospective Observational Study	Peripheral human lung tissue from 62 patients	Dexmedetomidine (DEX) did not exert any observable impact on the tension of pulmonary arteries with intact endothelium.	Human +in vitro
H. Abdel-Hamid	2017	Randomized Controlled Trial	70 Patients	The perioperative administration of dexmedetomidine effectively lowers pulmonary artery systolic pressure (PASP) during both the operative and postoperative phases.	Human
Mami Chikuda	2019	In vitro	Endothelium-denuded porcine pulmonary arteries from pigs	Dexmedetomidine (Dex) inhibited the adrenaline-induced rise in contraction tension following the depletion of the calcium (Ca^{2+}) reservoir.	Animal
Muralidhar Kanchi	2020	Prospective interventional study	25 Patients	The administration of Dexmedetomidine (Dex) did not lead to any notable changes in pulmonary artery pressure (PAP) in children with congenital heart disease (CHD) and pulmonary hypertension (PH).	Human
B. Ghasemzadeh	2020	Randomized Controlled Trial	66 Patients	The application of dexmedetomidine during surgical procedures in children with pulmonary hypertension effectively lowers pulmonary artery systolic	Human

				pressure (PASP) both intraoperatively and postoperatively.	
Yohei Yamaguchi	2023	Preclinical study	In vivo: male rats in vitro: human tissue	Dexmedetomidine (Dex) alleviates pulmonary arterial hypertension (PAH) by suppressing the proliferation of pulmonary arterial smooth muscle cells.	Animal

Table 2- Summary of studies examining Dex's influence on ventilation

Author	Year	Design	Sample Size	Findings	Subjects
A. T. Nathan	2008	Case Report	1 Patient	Dexmedetomidine (Dex) provides arousable sedation and moderate analgesia, enhancing oxygenation while maintaining hemodynamic stability.	Human
H. M. Munro	2009	Case Report	1 Patient	The combination of dexmedetomidine (Dex) and ketamine proved highly effective for procedural sedation in a pediatric patient with pulmonary hypertension undergoing cardiac catheterization, with no observed hemodynamic or respiratory compromise. This underscores the potential for carefully selected drug combinations to enhance safety and efficacy in complex medical scenarios.	Human
Scott Kernan	2011	Prospective, randomized, double-blinded trial	19 Patients	Dexmedetomidine (Dex) does not negatively impact oxygenation during one-lung ventilation (OLV) in adults undergoing thoracic surgeries.	Human
Lili Jiang	2014	Animal intervention	Adult male Sprague-Dawley rats	Pre-treatment with dexmedetomidine (Dex) has the potential to mitigate pulmonary damage and suppress sterile inflammation caused by lung ischemia-reperfusion (I/R) injury.	Animal
Su Hyun Lee	2016	Randomized, double-blinded, placebo-controlled study	50 Patients	The administration of dexmedetomidine (Dex) may offer significant clinical benefits by enhancing oxygenation and improving lung mechanics in patients with moderate chronic obstructive pulmonary disease (COPD) undergoing lung cancer surgery.	Human
B. P. Das	2016	Case Report	3 Cases	The prolonged infusion of dexmedetomidine (Dex) was well-tolerated and exhibited a steady trend of enhancing oxygen saturation levels.	Human
Ahmed Hasanin	2018	Randomized Controlled Trial	42 Patients	The infusion of dexmedetomidine (Dex) led to a modest enhancement in both oxygenation and pulmonary mechanics.	Human
Seongsu Kim	2021	Randomized Controlled Trial	52 Patients	The perioperative infusion of dexmedetomidine (Dex) showed no significant improvement in oxygenation or lung mechanics, nor did it alleviate systemic inflammatory responses.	Human

Conclusion

It is important to address that studies on the effects of Dex on pulmonary artery pressure have yielded conflicting results. Evidence suggests that Dex either has no effect on PAP or may reduce it in cases with pulmonary hypertension. According to Chikada et al., Dex suppresses adrenaline-induced increases in contraction tension, which could imply a reduction in vasoconstriction-induced hypertension [10]. However, some studies found no significant changes in PAP following Dex administration [16-17, 19].

The administration route plays a key role, as studies indicate that bolus dosing causes a transient but notable increase in PAP, which subsides within approximately five minutes [12]. While certain studies have reported significant PAP elevation with Dex, they have been conducted on animal models rather than humans, highlighting the need for further research in this area [15, 20].

Among studies on dexmedetomidine, some findings, though tangential to this topic, provide valuable insights into its effects on the lungs and pulmonary system. These studies predominantly focus on two aspects: the impact on oxygenation and inflammatory factors. Dexmedetomidine has shown remarkable benefits, including improving oxygenation by increasing the PaO_2/FiO_2 (P/F) ratio, enhancing lung compliance, reducing dead space, and lowering plateau pressure. It also supports hypoxic pulmonary vasoconstriction (HPV), optimizes ventilation/perfusion (V/Q) matching, and mitigates oxidative stress and inflammation. Additionally, it may exhibit bronchodilatory effects, leading to better lung mechanics and reduced PAP in cases of PH [27-29]. Furthermore, dexmedetomidine protects the lungs by reducing inflammation and pulmonary damage, inhibiting pro-inflammatory cytokines, and preventing edema through pathways like TLR4/MyD88/MAPK modulation. However, some studies reported no improvement in oxygenation with dexmedetomidine [14]. This underscores the need for further research to clarify its role in pulmonary function and PH management [30].

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