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Dexmedetomidine as an Anesthetic Adjunct in Off-Pump Coronary Artery Bypass Grafting: A Case Report

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Abstract

This case report describes the implementation of a dexmedetomidine (Precedexâ) infusion in a patient undergoing off-pump coronary artery bypass grafting (OPCABG). The focus of this case report is to analyze the impact dexmedetomidine has on perioperative opioid consumption, postoperative recovery, and hemodynamic effects that result. The addition of a dexmedetomidine infusion to the anesthetic plan of care in patients undergoing OPCABG is supported by current literature. Dexmedetomidine has many advantageous effects including analgesia, improvement in hemodynamic stability, and offers cardiac protection.[1] The end result may lead to decreased narcotic requirements which may enable earlier extubation times and an overall decreased length of stay in the ICU.[1,2,3] Intraoperative hemodynamic instability resulting from fluctuations in plasma levels of norepinephrine and epinephrine due to surgical stimulation are attenuated by dexmedetomidine due to the reduction in sympathetic output as a result of alpha-2 agonism.[4] Heart rate, mean arterial pressure, and systemic vascular resistance will be lower intraoperatively and postoperatively as a result.[4,5] Incidence rates of arrythmias occurring perioperatively such as atrial fibrillation, premature atrial contractions, premature ventricular contractions, and heart blocks are decreased with the use of dexmedetomidine.[4,6] Dexmedetomidine is shown to be a safe and useful anesthetic adjunct in patients undergoing OPCABG.

Dexmedetomidine as an Anesthetic Adjunct in Off-Pump Coronary Artery Bypass Grafting

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Introduction

- Dexmedetomidine is an alpha-2 agonist that produces sedative, analgesic, anxiolytic, and sympatholytic effects.[7]
- Dexmedetomidine was developed for sedation in the ICU, but its desirable effects have led to usage in the OR.
- Off-pump coronary artery bypass grafting (OPCABG) has advantages over on-pump cardiac bypass and is associated with fewer postoperative complications such as systemic inflammation, myocardial injury, and cerebral injury.[9]
- OPCABG results in increased sympathetic discharge due to surgical stimulation which can lead to tachycardia, hypertension, and increased myocardial oxygen demand.[8]
- The implementation of dexmedetomidine in the anesthetic plan for OPCABG may be beneficial in improving outcomes by decreasing perioperative opioid consumption and attenuating the effects of increased sympathetic discharge.

Case Report

Preanesthetic Evaluation

- A 52-year-old male, 97 kg, 68 inches presented for an OPCABG.
- Medical history: hypertension, hyperlipidemia, multivessel CAD, NSTEMI, CHF, ESRD, DM II, 20 pack year smoking history. ASA 4.
- Surgical history: cataracts surgery, Permcath, right knee scope
- Diagnostic testing: EKG- NSR with LVH, Echo- EF 55%
- Labs: WNL except BUN 31 mg/dl and Cr 5.5 mg/dl
- Preoperative vitals: BP 197/99, HR 72, RR 18, SpO_2 100%, temp 36.8°C
- Premedication: midazolam 2 mg IV

Intraoperative management

- ASA standard monitoring, radial arterial line placed prior to induction.
- Induction agents: sufentanil 20 mcg, lidocaine 100 mg, propofol 150 mg, vecuronium 8 mg
- 7.5 mm ETT placed at 21 cm using direct laryngoscopy
- Left IJ venous introducer and pulmonary artery catheter placed under sterile conditions.
- Anesthetic maintenance: sevoflurane, intermittent sufentanil boluses, vecuronium, and dexmedetomidine infusion.
- Hemodynamic support: norepinephrine infusion required after sternotomy due to SBP in 80s-90s.
- Dexmedetomidine infusion started after grafting was complete at 0.2 mcg/kg/hr.

Emergence/Postoperative Care

- After sternal wires in place, patient had 4/4 twitches on TOF and was given neostigmine 3 mg and glycopyrrolate 0.6 mg for NMB reversal.
- Hypoventilated to build end tidal carbon dioxide
- Norepinephrine infusion discontinued
- Once spontaneous respirations resumed and patient followed commands, patient was extubated on OR table.
- Dexmedetomidine infusion continued at 0.2 mcg/kg/hr during transfer to CVICU.

Follow Up

- Pt remained on dexmedetomidine infusion in CVICU.
- No vasopressor support required or postoperative arrythmias.
- Narcotic usage for 1st 24 hours: morphine 6 mg IV, 4 tabs of hydrocodone/acetaminophen 5/325.
- Ambulatory on POD 1 and transferred out of CVICU on POD 2.

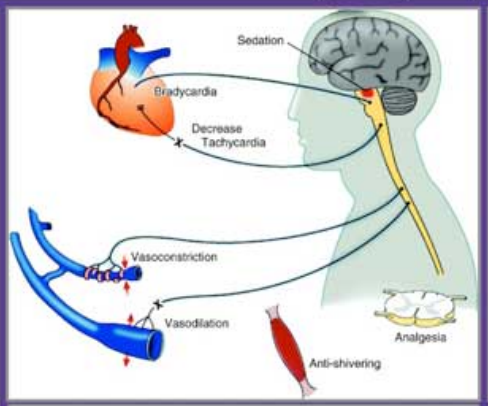

Figure 1: Dexmedetomidine's target sites include the brainstem and spinal cord for sedation and analgesia. The heart and vasculature are the target sites for hemodynamic altering effects.[10]

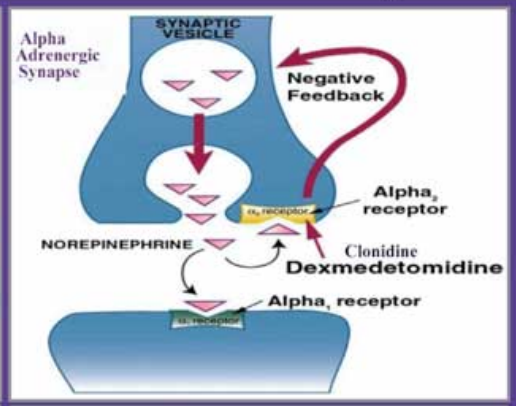

Figure 2: Cellular mechanism of action of alpha-2 agonists utilizing negative feedback to decrease release of norepinephrine.[10]

Pharmacokinetics and Pharmacodynamics of Dexmedetomidine[7]

Pharmacokinetics

- Absorption/Routes: Intravenous, intranasal, oral, transmucosal.
- Distribution: Highly protein bound (94%). Increased volume of distribution (Vd) in patients with hypoalbuminemia.
- Metabolism: Hepatic biotransformation through direct N-glucuronidation and hydroxylation by cytochrome P450 enzymes. High hepatic extraction ratio (0.7).
- Elimination: Renal excretion (95%). Elimination half-life of 2-3 hours.
- Context- sensitive half-time of 4 minutes following a 10-minute infusion, 250 minutes following an 8-hour infusion.
- High individual variability of clearance and Vd for patients with end-organ damage, unstable hemodynamics, and decreased cardiac output.

Pharmacodynamics

- Sedation: Alpha-2 receptors in the locus coeruleus inhibit the release of norepinephrine (NE) which induces a state of natural sleep.
- Analgesia: Mediated by central and spinal cord receptors which hyperpolarize interneurons leading to a reduction in the release of nociceptive neurotransmitters such as glutamate and substance P.
- Cardiovascular: Produces a biphasic hemodynamic response resulting in decreased blood pressure at lower concentrations and hypertension at higher concentrations or after a bolus. Presynaptic alpha-2 receptors inhibit the release of catecholamines when activated leading to a decrease in heart rate and vasodilation.
- Respiratory: Minimal respiratory depression with preservation of respiratory drive to increased CO_2 when administered alone. Increased risk of respiratory depression when administered with opioids or sedative agents.

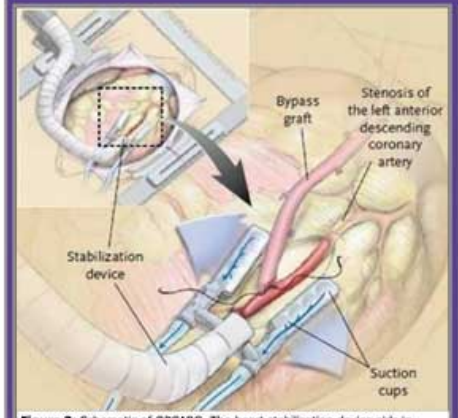

Figure 3: Schematic of OPCABG. The heart stabilization device aids in positioning the heart.[9]

Literature Search

- Databases searched: PubMed, Medline Complete, EMBASE.
- Keywords used: Dexmedetomidine, Precedex®, off- pump coronary artery bypass, CABG, anesthesia, open heart surgery, cardiac surgery.
- Total of 21 relevant articles retrieved and 10 used in this case report.

Case Critique

- Dexmedetomidine infusion was started too late intraoperatively
- The infusion should have been started after induction
- Appropriate follow up in the CVICU when the infusion was discontinued

Recommendations for Future Research

- Further research is needed to determine the ideal time to start infusion for OPCABG.
- More research is necessary regarding dosing postoperatively
- Proper duration to continue the infusion in the postoperative period after extubation.

Discussion

Opioid Requirements and Postoperative Recovery

- Dexmedetomidine provides a synergistic effect on opioids which may decrease the opioid requirement needed to obtain surgical analgesia.[3]
- Overall opioid consumption is decreased when dexmedetomidine infusion is used during OPCABG.[2,3]
- Decreased opioid administration leads to faster extubation times.
- Faster extubation times will aid in decreased length of stay in ICU and overall hospital stay.[2]
- If patient had to remain intubated, then dexmedetomidine is the ideal infusion for sedation compared to propofol due to the preservation of respiratory drive and easier assessment of neurological status.[1]

Hemodynamic Effects

- Sympatholytic effects can be beneficial due to the surge of catecholamines released due to surgical stimulation.
- Decreasing circulating catecholamines leads to a lower heart rate, MAP, and SVR which will decrease myocardial work and prolong diastolic perfusion.[5]
- Intraoperative hemodynamic disorders such as tachycardia and hypertension that can lead to myocardial ischemia are attenuated with dexmedetomidine.[4]
- Dexmedetomidine decreases the incidence rate of arrythmias such as PACs, PVCs, VT, and AFIB both intraoperatively and postoperatively for OPCABG.[4,6]
- Administration of hemodynamic altering drugs such as nitroglycerine, lidocaine, and esmolol were used less in patients receiving Precedex.[4]
- Dosing includes optional 1 mcg/kg loading dose over 10 minutes, then starting infusion at 0.5 mcg/kg/hr after induction and titrating to desired effect in the range of 0.2-0.7 mcg/kg/hr.[2,3,7]

Conclusions

- Dexmedetomidine is an alpha- 2 agonist that provides multiple benefits when used for OPCABG.
- Benefits include the potential to decrease overall opioid consumption, enhance postoperative recovery, attenuate the sympathetic response, improve myocardial oxygenation, and prevent arrythmias.[1-9]
- Anesthetic plan for OPCABG should include a multimodal approach with the use of volatile anesthetics, opioids, neuromuscular blockers, and alpha- 2 agonists such as dexmedetomidine.
- Two most evident side effects to monitor for clinically are hypotension and bradycardia.

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