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Studying the Efficacy of Different Dexmedetomidine Doses in Producing Controlled Hypotension during Functional Endoscopic Sinus Surgery

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I. Introduction

Functional endoscopic sinus surgery is the primary approach used today for the surgical treatment of chronic sinusitis and sinonasal polyps. It is a minimally invasive procedure and is commonly performed under controlled hypotensive anesthesia.[2] Induced or Controlled hypotension is a technique in which the arterial blood pressure is decreased in a predictable and deliberate manner. The intent of deliberate hypotension is to reduce bleeding that facilitate surgery and to decrease the need of blood transfused.

In FESS surgeries, impaired visibility of the operative field due to excessive bleeding which leads to various complications[1]. So a dry operative field is necessary for a more definitive removal of the polyp (lesion) which leads less damage to vital structures and thereby tissue infection is also minimized.[1] Controlled hypotension is a technique used to limit intraoperative blood loss and provide the best possible field for surgery.[1]

Controlled hypotension is anesthetic method provides an enhanced illumination and visualization has dramatically improved surgical dissection. Several pharmaceuticals have been used successfully to produce controlled hypotension during general anesthesia for example inhalational anesthetics, direct vasodilators (sodium nitroprusside and nitroglycerin), beta adrenergic antagonists (propranolol and esmolol), alpha adrenergic agonists (clonidine and dexmedetomidine), calcium channel blockers, prostaglandin E1 (alprostadil), adenosine[2] and l-receptors agonists (remifentanil) .

AIMS AND OBJECTIVES
AIM OF THE STUDY
AIM OF THE STUDY
The aim of the study to find the efficacy of different dexmeditomedine regimens in bringing controlled hypotension in FESS surgeries.

OBJECTIVES
To study the efficacy of dexmeditomedine in bringing effective hypotension for FESS surgery.
To determine the effective surgical field quality of the drug.
To study the hemodynamic parameters both intraoperative and in postoperative period.
To evaluate the need of other hypotensive agents in-spite dexmeditomedine for providing hypotension during surgery.
To study any untoward side-effects and complications.
To study postoperative sedation score and postoperative complications as hemodynamic instability, desaturation or bleeding from surgical field.

FUNCTIONAL ENDOSCOPIC SINUS SURGERY

Controlled hypotension is widely being used as a means of limiting intraoperative blood losses or limiting the need of blood transfusions, it is also used to achieve a clear operative field which is needed for successful middle ear microsurgery and FESS.[4]

Intraoperative hemorrhage would decrease the visibility of the surgical field during FESS procedure and it may land on to risk of vascular, orbital and procedural failure as well as intracranial complications[5]. Therefore, it is of prime importance to the surgeon and the anesthetist to minimize surgical bleeding and get a quite clear field for the surgery[5].

Because of the extensive vascular supply of the nose and paranasal sinuses and the impact of the pathophysiological changes due to the disease in the patient, bleeding would be difficult to manage surgically.

Among various reasons Capillary bleeding could be the most serious problem of note during the procedure, barring any inadvertent trauma to the feeding arterial and venous vessel. Likely bleeding from the capillaries can be greatly minimized by reducing the patient's mean arterial pressure and with use of local vasoconstriction agents.[5]

METHODS TO DECREASE HAEMORRHAGE

POSITIONING The reverse Trendelenburg is 15° head up position. That allows for venous decongestion from upper part of the body and increased venous pooling in the lower limb and thereby decreases the hemorrhage.[5]

INJECTED AND TOPICAL LOCAL ANESTHETICS AND VASOCONSTRICTORS

These might work to decrease postoperative pain, reduce blood loss and mucosal congestion. Commonly used vasoconstrictors include cocaine, epinephrine, and phenylephrine.[5]

MAINTANANCE OF DEPTH OF ANESTHESIA Creating the adequate depth of anesthesia would play a vital role to minimize the surgical bleeding. During a light anesthetic plane the patient might be coughing or straining which would lead to, an increase in intrathoracic pressure which impair venous drainage from the head and might increase surgical bleeding. The muscle relaxants would effectively prevent above mentioned incidences during the procedure. Intermittent positive pressure ventilation to be adjusted as that the airway pressures should be kept at a minimum level, by properly adjusting the intermittent positive pressure ventilation. Avoid positive end expiratory pressure to prevent higher intra thoracic pressure[5].

CHOICE OF ANESTHETIC AGENT

Volatile anesthetic agents can cause vasodilatation by decreasing systemic vascular resistance. Because of increased tissue perfusion due to volatile, it may provoke surgical bleeding. Induction agent propofol does not just decreases the systolic blood pressure by reducing systemic vascular resistance but it also effectively blunts the sympathetic response to endotracheal tube insertion and various surgical stimulation. Propofol can reduce blood flow through the ethmoidal and the supraorbital artery to the ethmoid, sphenoid and frontal sinuses and improves surgical visibility.[5]

CONTROLLED HYPOTENSION[10]

Controlled hypotension is defined as a reduction of the systolic blood pressure to 80–90mm Hg, a reduction of mean arterial pressure (MAP) to 50–65mm Hg or a 30% reduction of baseline MAP.[10]

From recent past, Controlled hypotension has been acknowledged and practiced widely to reduce surgical bleeding and thereby gives a quality bloodless surgical field. It has been practicing in maxillofacial surgery (mandibular osteotomy, facial repair), endoscopic sinus or middle ear microsurgery, spinal surgery and other neurosurgery (aneurysm), major orthopedic surgery (hip or knee replacement, spinal), prostatectomy, cardiovascular surgery and liver transplant surgery.[10]

Pharmacological agents are of vital to bring controlled hypotension. They can be used as a sole agent or might be used an adjunctive to reduce other dosage requirements and their adverse effects. Agents used successfully alone include inhalation anesthetics, sodium nitroprusside, nitroglycerin, trimethaphan camsilate, alprostadil (prostaglandin E1), adenosine, remifentanyl, and agents used in spinal anesthesia. Agents that can be used alone or in combination include calcium channel antagonists (e.g. nicardipine), β-adrenoceptor antagonists (β-blockers) [e.g. propranolol, esmolol] and fenoldopam. Agents that are mainly used adjunctively include ACE inhibitors and clonidine.[10]

The main side effects of these drugs are due to hypotension which impairs the perfusion of vital organs, potent hypotensive agents would lead to their own side effects based on their concentration that could be treated with available potent vasopressors. So use this drugs with extra caution to limit the major risks.[10]

The natural hypotensive effect of current available anesthetic drug can be used to administer hypotensive anesthesia. An ideal hypotensive agent should be easy to administer, should have a short action time, hypotensive effect has to wear off quickly as soon as the drug is discontinued, should have a fast elimination, gives very less or nil toxic metabolites, zero effects on vital organs, and should have a predictable dose-dependent effect. Inhalation agents (isoflurane, sevoflurane) have dual effect of providing hypnotic and hypotensive agents at clinical concentrations, and can be used alone or with other agents to limit tachycardia and rebound hypertension. Higher concentrations is needed for a significant reduction in vascular resistance to control intra-op bleeding when they are used alone.[10]

The reliable efficacy and ease-of-use to toxicity ratio predict at clinical concentrations will predict the probability of an anesthetic agent can be used alone for bringing hypotensive anesthesia without the need for potent hypotensive agents. The current available satisfactory technique is a combination treatment of remifentanyl either with propofol or an inhalation agent (isoflurane, desflurane or sevoflurane) at clinical concentrations. The literatures quoted the above because of their safety profile and ease of use.[10]

DEXMEDETOMIDINE[7]

The newer α2-adrenergic agonist Dexmedetomidine was introduced in the market in 1999 for sedating patients on mechanical ventilation those who are admitted in intensive care units, Later it was approved by FDA for ICU sedation. After a fairly long period of use in the market, FDA expanded its use to sedate non-intubated patients during surgical and non-surgical procedures in 2008. [7]

But in India the drug was approved in 2009 for the above mentioned purpose by the Central drug standard control organization. More upcoming clinical studies have been exploring this newer drug in anesthetic practices for well-balanced anesthesia. In addition this drug use in perioperative area is widely expanding. This drug produces conscious sedation, analgesia and anxiolysis the main components of balanced anesthesia.[7]

α2 ADRENERGIC RECEPTORS

Adrenergic receptors are of two types alpha and beta receptors. These receptors have their inherent physiological functions due to their variable potency to natural and synthetic catecholamines.[7] These α2-receptors are located over pre-synaptic site which affects the neurotransmitter release.[7] This contributes for their major effect through regulating adenosine (ATP) and noradrenaline flow through a unique negative feedback mechanism. [7]

These subclass of alpha receptors are found at post-synaptic and extrasynaptic areas. [7] There are three different α2 receptors had been postulated by pharmacologic studies. Neither clonidine nor dexmedetomidine found to be totally selective to particular type of the α2-AR subtypes, perhaps dexmedetomidine has a greater α2A-AR and α2C-AR affinity than its congener.[7]

ADRENERGIC RECEPTOR SITES

Central nervous system, Liver, pancreas, eye, kidney, platelet and in peripheral nervous system these receptors are located.[7] The response to α2-receptors stimulation will vary depend on their site of location. Three different α2 receptors are α2A, α2β and α2C which are found in different sites all over the body.[7] α2A & α2C are of central nervous system specific, physiologic activation of these receptors results in sedation, analgesia and sympatholytic effects.[7]

α2B receptors are predominantly found on vascular smooth muscles exerts vasoconstriction.[7] α2 receptors are belong to Guanine nucleotide (G Proteins) coupled receptor family. This second messenger system on activation inhibits adenylate cyclase and in turn reduces the formation of 3,5 cyclic adenosine monophosphate (CAMP). Due to low level of CAMP hyperpolarisation of cell membrane results due to efflux of potassium. [7] As a result of hyperpolarization calcium ions entering into nerve terminal is inhibited and further suppresses neuronal firing results in inhibitory action an secretion of neurotransmitters.[7]

CHEMICAL STRUCTURE OF DEXMEDETOMIDINE[7]

Dexmedetomidine is the dextro-rotatory S-enantiomer of medetomidine and the chemical name is S-4 [1-(2,3-dimethylphen ethyl)]-3H imidazole. [7]

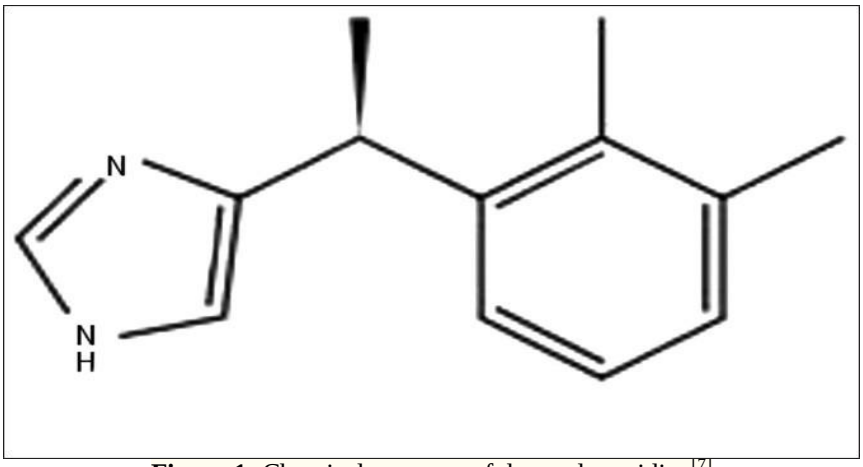


Figure 1: Chemical structure of dexmedetomidine[7]

MECHANISM OF ACTION[7]

This imidazole compound exerts more specific effect over α2 receptor especially for 2A subtype of α2 receptor, makes this drug as a more effective sedative and analgesic producing agent compared to its congener clonidine.[7]

The specificity of dexmedetomidine for α2 receptors over clonidine is 8 times more, ratio of α2:α1 for dexmedetomidine is 1620:1 and the ratio for clonidine is 220:1.[7]

The predominant clinical effect of dexmedetomidine is principally due to its action in the brain stem. It has been found that dexmedetomidine stimulates α2 receptors of spinal cord, which in turn inhibits nociceptive neurons firing locus coerulus, nucleus in brain which is designed to monitor vigilance.[7]

The substantia gelatinosa of dorsal born of the special cord has α2receptors, when stimulated suppresses the neuronal firing which then reduces the nociceptive neurotransmitters release, substance P is important.[7] This unique mechanism of action differs dexmedetomidine from other currently available sedative drugs like clonidine.[7]

α2A adrenergic receptor stimulation in brain and spinal cord most likely produces hypotension, bradycardia, sedation and analgesia. Dexmedetomidine can cause decreased salivation, decreased intestinal secretion, decreased bowel motility and vasopressor effects due to its stimulation of α2 receptors in other areas.[7] It might suppress renin and insulin release, increases GFR, increases secretion of sodium and water and decreases intra ocular pressure.[7]

Analgesic effect of dexmedetomidine is most likely due to presynaptic activation of α2 receptors which further decreases release of nor-epinepherine and then stops pain signals propagation.[7] Postsynaptic activation as sympatholytic effects which are located in CNS.[7] It has not been fully studied regarding how exactly the nociceptive action is mediated. It is still controversial, because apart from both spinal and supraspinal activation for anti-nociceptive effects, evidences are showing peripheral α2 - receptors stimulation might also have nociceptive signals inhibitory effect, the other postulate for its analgesic effect it inhibits neuronal firing and its propagation along the nerve terminal.[7]

PHARMACOKINETICS

ABSORPTION AND DISTRIBUTION

The recommended infusion dose range for Dexmeditomidine is of 0.2 to 0.8 μg/kg/hr where it has linear pharmacokinetics upto 24hrs. The drug has half-life of distribution around 6 min because of its rapid distribution, the elimination half -life lasts for 2hrs. [7]

The steady state volume of distribution is 118L.[7] It has constant protein binding capacity of average 94%. The context sensitive half- life varies from 4 min after a 10min infusion to 240min after 8 hrs of infusion. This drug exhibits very poor oral availability due to its very high first pass metabolism. [7]

This drug when administered sublingually has a bioavailability of 84%, because of its higher bioavailability via this route makes to use this drug for sedation and premedication among pediatric population. [7]

METABOLISM AND EXCRETION[7]

It is near completely metabolized by cytochrome-p-450 and N- glucuronidation to inactive metabolites.[7] Most of its inactive metabolites are passed in urine and the remaining in feces. Due to hepatic metabolism, drug should be used judiciously in hepatic failure patients. [7]

THE BIPHASIC BP RESPONSE[7]

It shows a short hypertensive phase due its action over α2B-AR and subsequent hypotension phase due to its α2A-AR stimulation.[7] Several records had shown that this drug brings bradycardia and sinus arrest which could be managed successfully with anticholinergic agents. This effects were seen predominently in patients with high vagal tone.[7]

PHARMACODYNAMICS

CENTRAL NERVOUS SYSTEM[7]

The neuroprotective effect exhibited by this drug through reducing cerebral blood flow and its oxygen consumption.[7] Studies had revealed it reduces the circulating and brain catecholamines regarding their effect on intracranial pressure, cognitive performance, peri-ischemic effects to nervous system has been exploring.[7]

RESPIRATORY EFFECTS[7]

Various recent studies evidenced that this drug doesn't suppress much of respiratory mechanics even at higher doses both in surgical and non-surgical procedures including ICU patients.[7]

ENDOCRINE AND RENAL EFFECTS[7]

Sympatholytic effect seen with this drug which is mediated by its action through peripheral pre-synaptic α2-AR receptor.[7] As its most likely etomidate both exhibits imidazole compound in its structure despite Dexmeditomidinie doesn't inhibit steriodogenesis unlike the other.[7]

ADVERSE EFFECTS[7]

Dexmedetomidine produces hypertension, hypotension, Nausea, Vomiting, dry mouth, bradycardia, atrial fibrillation, pleural effusion, atelectasis, hyperglycemia, hypocalcemia and pulmonary edema. [7] It can show a transient hypertension due to its α2β-AR vasoconstriction after rapid intravenous administration (If loading dose of 1μg/kg/hr administered in less than 10 min) [7].

Due to its α2-AR action, it might show hypotension and bradycardia as adverse effects. It's classified as category 'C' Pregnancy risk so it should be handled cautiously during pregnancy[7]

CLINICAL APPLICATIONS OF DEXMEDITOMIDINE[7]

PREMEDICATION

Dexmeditomidine can be used as premedicant particularly in stressed individuals due to its excellent α2 agonist action.[7] Previous studies had showed that at a close of 0.3 to 0.6 μg/kg iv should be given 20 minutes before surgery would minimize its side effects[7]

ATTENUATING THE STRESS RESPONSE

Because of its sympatholytic effect, it can be used to reduce the hemodynamic stress response during intubation and extubation. More-ever, It accentuates the effects of other agents which are co-administered with dexmeditomidine hence requirement of overall anesthetic agents will reduce. [7]

The main limiting factor with Dexmeditomidine for its pharmacological use is hypotension and bradycardia. [7] Unlike several drugs Dexmeditomidine has zero effect with respiration, which makes this drug can be continued even during extubation process[7]

AS AN ADJUVANT[7]

Dexmeditomidine increases duration of regional block both sensory and motor blockade used with local anesthetics.[7] It's rapidly absorbed into CSF due to lipophilic property and binds to α2 receptors in the spinal cord and thereby produces analgesia. [7] For efficient result dose of 1μg/kg with local anesthetics is used. It enhances the local anesthetic effect in every form of regional anesthesia and peripheral nerve block techniques when used.[7]

INTENSIVE CARE SEDATION[7]

It's gaining popularity in intensive care management due to its unique property of conscious sedation. So that patients would be in a clam, cooperative and awake. [7] This drug doesn't affect patients respiration, so it can be given safely even to critically ill patient in those respiratory mechanisms are compromised [7].

Several reports documented that dexmeditomidine reduces the total ICU bill amount because it hastens weaning the patient from ventilator support. [7] This conscious sedation brings a natural way of sleeping, thereby it hastens patient recovery. [7] When compared to other sedative agents which are commonly practiced in ICU dexmeditomidine offer several benefits with low risk. [7]

Table:2 Comparison of DT other ICU sedatives[7]

Effects	Dexmedetomidine	Benzodiazepines	Propofol	Opioids	Haloperidol
Sedation	√	√	√	√	√
Analgesia	√			√	
Alleviation of anxiety	√	√			
Cooperative sedation	√				
Facilitation of ventilation during weaning	√				
No respiratory depression	√				√
Control of delirium	√				√
Organ protection	√		√		
Control of stress response	√				
Antishivering agent	√				
Mimicking of natural sleep	√				

Based on data from Pandharipande *et al.*[36]

PROCEDURAL SEDATION

This drug produces an excellent alternative for the short procedure like elective awake fiberoptic intubation, tonsillectomy, colonscopy, shockwave lithotripsy, transesophageal echocardiography and pediatric MRI. [7] Bolus dose for sedation is 1μg/kg followed by 0.2μg/kg as maintenance dose. The onset will be within 2 minutes and its peak action, within 15 minutes. [7]

CONTROLLED HYPERTENSION
Currently the scope of Dexmeditomedine widens and it produces excellent bloodless surgical field by producing controlled hypotension through its sympatholytic effect. [7] Functional endoscopy sinus surgery, spinal surgeries like idiopathic scoliosis can be done safely and effortlessly. [7]

ANALGESIA
It might be used to relieve neuropathic pain due to its α2 agoinst action in spinal cord reduces the pain producing substance (Substance - P) transmission and has opioid sparing effect. [7]

CARDIAC SURGERY
The drug has shown myocardial protective effect by reducing the extent of ischemic injury mainly due to its maintenance of stable hemodynamic profile during cardiac surgery. [7] The popularity has been increasing in mitral valve replacement to reduce pulmonary vascular resistance. [7]

NEUROSURGERY
It prevents the sudden rise in ICP during neurosurgical and anesthetic procedures which provides a stable hemodynamic picture. [7] By reducing the neurocognitive impairment such as delirium and agitation, it permits early postoperative patient evaluation. [7] The drug itself produces neuroprotective effects, mechanism are poorly understood. It doesn't affect neurological monitoring and has been used in awake craniotomies. [7]

OBSTETRICS
Due to its opioid sparing analgesic effect and providing the stable hemodynamic profile, the drug has been expanded into managing obstetric patients. [7]

PEDIATRICS
It has been used as an off label drug for sedating and analgesic effects in pediatric patients in intensive care unit, radiological imaging studies like MRI and computed tomography. [7]

INTRAVENOUS REGIONAL ANESTHESIA
The use of Dexmedetomidine in IVRA at the dose of 1 μg/kg has been increasing recently, it prolongs the analgesia duration without much side effects. [7]

II. Review Of Literature

Neamat I. Abel Rahman et al[2] studied and compared the ability of dexmedetomidine in two different regimens to produce controlled hypotension for FESS in adults. They had given bolus dexmedetomidine 1μg/kg iv over 10min for all the patients. For group Dex-0.4, patients received dexmedetomidine infusion as 0.4μg/kg/h, group Dex-0.8 received dexmedetomidine infusion as 0.8μg/kg/h and group Dex-P patients received saline infusion .They concluded that the intraoperative mean arterial pressure was maintained within target range throughout the surgery in group Dex-P and group Dex-0.8. In group Dex-0.4, the MAP showed fluctuations at different time intervals. No nitroglycerin infusion was needed in group Dex-0.8. Fromme et al. bleeding score was lower in Dex-0.8 group and higher in group Dex-0.4. The differences are statistically significant ($P < 0.05$).[2] However The number of patients experienced hypotension and requirement of the ephedrine doses due to higher doses of dexmeditomidine were higher. Sedation level was higher in patients who received dexmedetomidine at either doses.

Boezaart AP, Merwe J, Coetzee A et al [1] studied the appropriate surgical conditions for functional endoscopic sinus surgery (FESS) using either sodium nitroprusside (SNP) or esmolol as controlled hypotensive agents under general anesthesia in a group of 20 adult patients. Concluded that surgical conditions were poor with (ACS = 3.63 ± 0.22) in mild SNP-induced hypotension group, But in esmolol group, ideal surgical conditions (ACS = 2.94 ± 0.34) recorded at MABP > 65 mmHg. But they achieved the superior surgical conditions not merely through the pharmaceutical agents but the effects were contributed by increased venous drainage due to the reverse trendelenburg position.

Degoute CS, Ray MJ et al [4] induced controlled hypotension in children using remifentanil combined with sevoflurane in fourty children to reduce middle ear blood flow (MEBF) which is measured by laser-Doppler, and the quality of satisfactory operative field. Both groups received $1\mu g \cdot kg^{-1}$ remifentanil iv infusion. Then either group followed a continuous infusion of 0.2 to 0.5 μg/ $kg^{-1}min^{-1}$ nitroprusside iv and alfentanyl *iv*. Even though nitroprusside induces increase in $PaCO_2$ and its complications were not studied properly. Remifentanyl had post infusion sedation effects especially in pediatric population.

Richa F, Yazigi A, Sleilaty Get al [5] compared dexmedetomidine with remifentanyl an ultra-short-acting opioid to obtain a better exposure during tympanoplasty in 24 adults through controlled hypotension.

Mean arterial pressure and heart rate were significantly lower in the remifentanyl group ($P = 0.03$). Surgical field exposure condition ($P = 0.039$) and surgeons satisfaction scores ($P = 0.039$) were significant after remifentanil compared with dexmedetomidine. Even though the precious dose specification of two agents, their side-effects both intra operatively and postoperatively and the need of other vaso-dilatory agents was not been documented.

Vineela Ganapathi et al [8] studied the effect of controlled hypotension with dexmedetomidine studied in 60 patients, which were recruited and randomized into two groups to compare nitroglycerine (0.5-10 µg/kg/min) with dexmedetomidine (loading dose of 1 µg/kg over 10 min followed by continous infusion rate of 0.2- 0.7 µg/kg/hr) for reducing blood loss and to obtain satisfactory surgical field during controlled hypotensive anesthesia in adults. They concluded that the target mean arterial pressure was achieved in both groups with the blood loss significantly less in dexmed group. Even though insight into whether hypotensive episodes truly requiring vasoactive agents and the post-operative level of sedation was limited.

Gurbet A, Turker G et al [9] studied the infusion of dexmedetomidine intra operatively to reduce perioperative analgesic requirements in 25 adult patients. Group D received a loading dose of dexmedetomidine 1 $\mu g \cdot kg^{-1}$ *iv* during induction of anesthesia and a continuous infusion at a rate of 0.5 $\mu g \cdot kg^{-1} \cdot hr^{-1}$ till the end. Group P received a volume-matched bolus and placebo infusion. Concluded that Continuous *iv* dexmedetomidine during abdominal surgery provides effective postoperative analgesia and significantly reduces postoperative morphine requirements during abdominal surgery. The pain and sedation scores observed were almost similar between the groups throughout the entire period of observation.

DK bharathwaj and Kamath SS et al [11] compared the hypotensive effect of dexmedetomidine with propofol based anesthesia in functional endoscopic sinus surgery. Patients were induced with 2 mg/kg of propofol in titrated doses followed by rocuronium 0.8 mg/kg for facilitating endotracheal intubation. They observed that both drugs were able to reduce heart rate significantly from baseline. With group dexmedetomidine reported lower heart rate (63.93 ± 3.362) when compared with group B (70.52 ± 2.589). Using Fromme and Boezaart et al score, surgeons graded the quality of the surgical field which were similar in the two groups ($p = 0.094$, $p > 0.05$).

Tarek Shams and Ragaa El-Masry et al [12] did a comparative study in fourty patients of ASA I and II posted for FESS to induce controlled hypotension with dexmedetomidine and esmolol. Esmolol loading dose 1mg/kg was given over one min followed by 0.4-0.8 mg/kg/h infusion as maintanence dose then maintained with sevoflurane 2%-4%. Eventhough the emergence time, cortisol level and surgical field quality were acceptable in both the groups, analgesic request for esmolol group were inconclusive and further analgesic demand was not sorted out.

Aliakbar Eghbal, Mohammad Khalili et al [13] to compare the effect of labetalol and dexmedetomidine for inducing hypotension to reduce intraoperative blood loss and a better surgical field conditions in FESS surgery was conducted in a group of 100 candidates. Dexmedetomidine(1µg/kg) as loading dose given over for a period of 10 min and consequently 0.4–0.8µg/kg/h given as the maintanance dose. Other group received labetalol (0.25 mg/kg/IV) given for 10min intravenously as the loading dose and then 1–2 mg/min/IV was administered as the maintanance dose. The mean of arterial blood pressure, heart rate and arterial oxygen saturation were noted. The extubation and recovery time was longer in dexmedetomidine group than labetalol group. Labetalol group experienced few complications too which were managed judiciously.

Esmail Moshiri and Bijan Yazdi et al [14] compared the effects of propofol and dexmedetomidine on controlled hypotension and bleeding during functional endoscopic sinus surgery in a group of 100 patients. For propofol group, a dose of propofol 50–150 µg/kg/min was infused. Hemodynamic parameters were measured and the intraoperative bleeding was evaluated. The mean time for extubation in Dexmed group was 87.64 min and it was 81.44min in Group Propofol which was not significant between two groups ($P = 0.094$). Intra-op bleeding score in Group D was 1.14 ± 0.70 and for Group P it was 1.24 ± 0.74, no significant difference between the two groups in terms of bleeding score ($P = 0.490$) noted. Even though the analgesic requirements either during intra-operative and post-operative period were undocumented for both groups. Heart rate measurements were significantly lower in Group P (propofol) than dexmed group.

Randa Ali Shoukrya and Ahmed El-Sayed Mahmoudb[15] compared the NTG infusion of 3–5µg/kg/min or Magnesium sulphate 30mg/kg, administered as a slow intravenous bolus and 10mg/kg/h by continuous infusion during the operation, to provide controlled hypotension in a group of 50. Heart rate values are significantly higher in the NTG group compared with the magnesium group. The longer extubation time and recovery time was reported in comparison with the NTG group.

III. Methodology

This is a comparative prospective randomized control study done in Coimbatore medical college and hospital, Coimbatore Tamilnadu in the Department of Anesthesiology, operation theatre and the postoperative ICU or postoperative ward in patients undergoing elective functional endoscopic sinus surgeries over a period of one year.

STUDY PERIOD : January 2018 to January 2019
STUDY DESIGN : A comparative prospective randomized control study
STUDY SUBJECTS : Sample size of 15 patients in each group (n=45)

INCLUSION CRITERIA

Age between 18 to 50 years.
ASA Grade I and II
Willing to give informed consent.

EXCLUSION CRITERIA

Age less than 18 and more than 50 years
ASA III and IV
Patient's refusal
History of allergies to particular drugs (local anesthetic, antihistamines)

METHOD OF RANDOMIZATION

Sealed envelope method

MATERIALS REQUIRED

- Dexmedetomidine ampoule
- 2% Lignocaine without preservative (Xylocard)
- 2% Lignocaine with adrenaline
- Infusion pump
- Intravenous catheter (18G) and adult infusion set
- Intravenous fluids for infusion
- Sterile gloves and drapes
- Macintosh laryngoscope instruments set
- Anesthesia Machine
- Monitors
- Emergency drugs
- Syringes
- Ephedrine ampoule and nitroglycerine ampoule.
- Distilled water
- Anesthestic drugs, vaporizers.
- Anesthesia monitoring chart.
- Emergency equipments
- Ambu bag
- Bougie
- Appropriate Size Endotracheal tube
- Appropriate oral and nasal airway and masks
- Good working Suction Apparatus

INTERVENTION DETAILS

After detailed pre-operative assessment, patient was shifted to the operation theatre and their vital parameters including Pulse rate(PR), systolic blood pressure(SBP), diastolic blood pressure(DBP), mean arterial blood pressure(MAP), arterial oxygen saturation (SPO2) were recorded before induction.

Before inducing for general anesthesia the participants were randomly allocated into three groups, everyone had received bolus dose of dexmedetomidine 1µg/kg iv infusion over 10-20mins. Then patients were induced with fentanyl 2µg/kg, propofol 2µg/kg and non- depolarizing muscle relaxant atracurium 0.5mg/kg.

- Group Dex-0.2, in which patients received bolus dose of dexmedetomidine 1µg/kg iv followed by continous iv infusion of 0.2µg/kg/hr.
- Group Dex-0.4, in which patients received bolus dose of 1 µg/kg iv and continous iv infusion of 0.4 µg/kg/hr dexmedetomidine

- Group Dex-C, Patients recieved bolus dose of dexmedetomidine and saline infusion.

PRE-ANESTHETIC WORK UP

- Written Consent form signed by the patient and a witness obtained for the study.
- Patient's condition and hemodynamic status recorded preoperatively.

LABORATORY INVESTIGATIONS

- Complete blood count
- Random blood sugar (Fasting and postprandial blood sugar if necessary)
- Renal function test
- Chest X-ray
- Electrocardiogram

ANESTHETIC TECHNIQUES

After verifying and satisfied with pre-anesthetic check out procedures as mentioned above, dexmedetomidine 1µg/kg was loaded with normal saline and connected to infusion pump set for infusion. Infusion was started in a well secured and working intravenous cannulation line which had put earlier. Infusion rate was set a level so that it has to be infused within 20 minutes with meticulous monitoring of the vitals.

Then slowly induce the patient with fentanyl 2µg/kg slow iv and propofol 2µg/kg iv was given slowly until the patient loses eye lash reflex or patient would not be communicative. Paralyze the patient with non depolarising agents, Atracurium 0.5mg/kg. After obtaining the ideal intubation condition, patient was intubated with appropriate size endotracheal tubes and was secured after confirming the air entry on both sides. Meanwhile the ongoing dexmed infusion was monitored.

Maintained the plane of anesthesia with non-depolarizing muscle relaxant, inhalation anesthetics like sevoflurane and intravenous agents like fentanyl, propofol. The target MAP is 55–65 mmHg, if not achieved by the infused study drug, nitroglycerin infusion was added in a titrating manner started with 0.1µg/kg/min and increased gradually till the target MAP is reached.

During the intra-op and post op period patient pulse rate (PR), systolic blood pressure (SAP), diastolic blood pressure (DBP) and mean arterial blood pressure (MAP), oxygen saturation (SPO2) was monitored and recorded. The surgical field quality was assessed periodically using Fromme et al. bleeding score adapted by Boezaart et al and recorded.

After extubation patient's sedation was assessed and they were shifted to recovery room to monitor the vitals, recovery from sedation, bleeding from surgical site and adverse effects due to drugs and to the procedure.

Group Dex-0.4

Dexmedetomidine bolus dose of 1 µg/kg iv infused via infusion pump loaded previously with normal saline, appropriate to patients weight. Infused over a period of 20 min then intubated with appropriate tracheal tubes and secured it with plaster. Then continous iv infusion of dexmedetomidine 0.4µg/kg/h was started and the infusion started time was recorded along with vitals for every 10 minutes till the surgery ends.

The target MAP is 55–65 mmHg were achieved with nitroglycerin infusion added in a titrating manner started with 0.1µg/kg/min and increased gradually till the target MAP was reached if not maintained with dexmedetomidine. Undesirable Hypotension was corrected with vasoconstrictors like ephedrine 30mg iv in titrated doses.

FROMME ET AL BLEEDING SCORE

The surgical field quality can be assessed by using Fromme et al. bleeding score adapted by Boezaart et al. Assessment of blood loss obtained from quantity of blood clots collected in the suction canister and visual assessment of surgical field using a 6 point grading system mentioned below.

Grade 0 = no bleeding

Grade 1 = slight bleeding so blood evacuation not necessary

Grade 2: slight bleeding so sometimes blood has to be evacuated

Grade 3 = low bleeding so blood has to be often evacuated and operative field is visible for some seconds after evacuation

Grade 4 = average bleeding so blood has to be often evacuated, and operative field is visible only right after evacuation and

Grade 5 = high bleeding so constant blood evacuation is needed, sometimes bleeding exceeds evacuation and surgery is hardly possible.

ASSESSMENT OF SEDATION

Assessment of sedation was done using Ramsay sedation score after extubation.

- score of 2 to 3 - anxiolysis
- score of 4 to 5 - moderate sedation
- score of 6 - deep sedation
- score of 7 to 8 - general anesthesia.

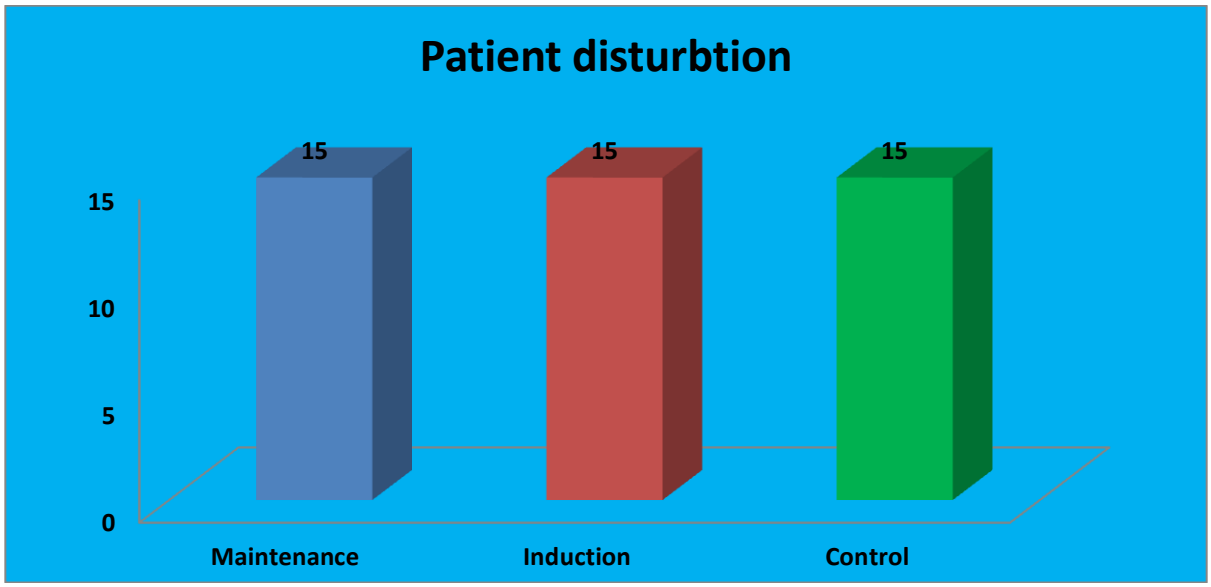
STATISTICAL ANALYSIS

The collected data was reviewed in specially designed sheet and analyzed by SPSS version (statistical package for social sciences) statistics software 23.0Version. Data descriptive statistics frequency analysis was used to describe descriptive data and percentage analysis was used for categorical variables. For continuous variables mean and standard deviation were used. Independent t-test was used for finding the significant difference between bivariate samples in Independent Groups and to find significant difference in categorical data. A probability value of ≤0.05 was considered to be significant.

IV. Results

Table 1: Distribution of the patients in the study

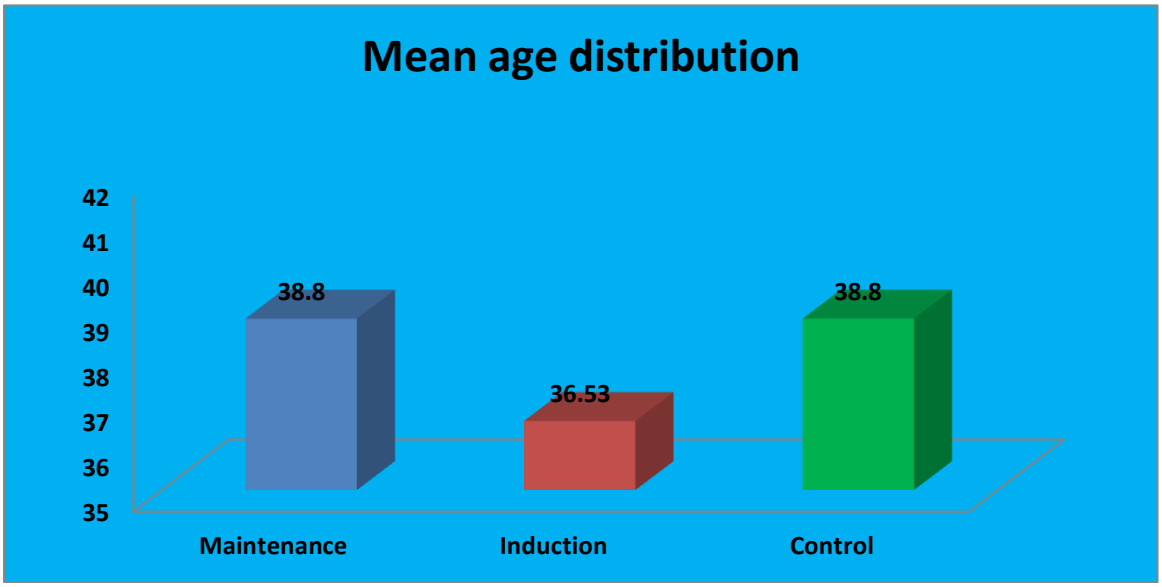
	Frequency	Percent
DEX - 0.4	15	33.3
DEX - 0.2	15	33.3
DEX-C	15	33.3
Total	45	100.0



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 2: Mean Distribution of the patients

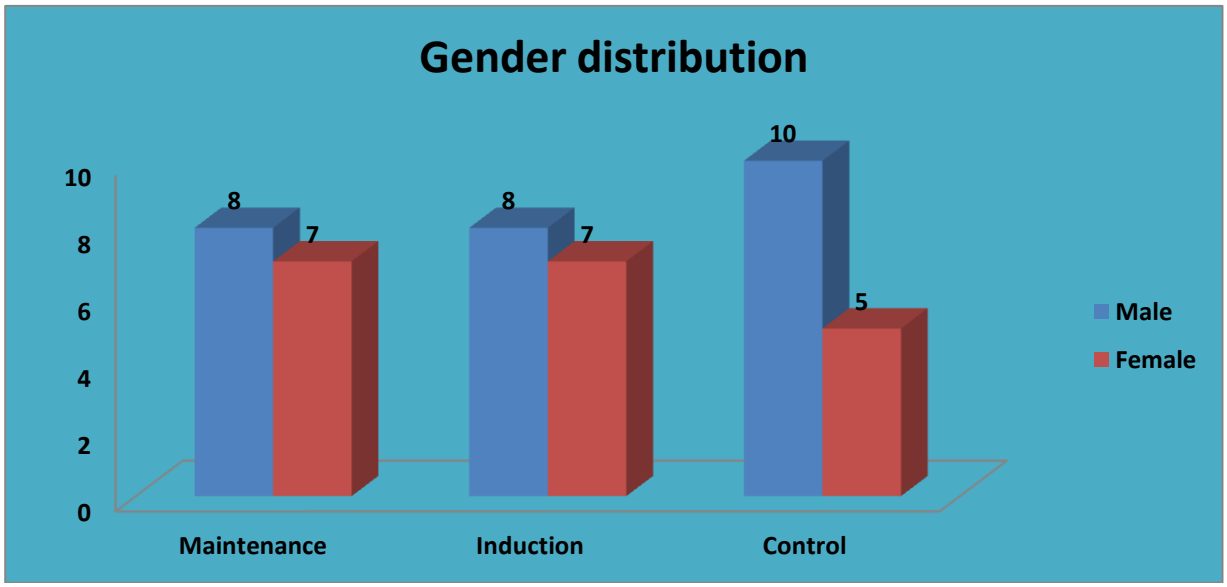
	N	Mean	Std. Deviation
DEX - 0.4	15	38.80	13.991
DEX - 0.2	15	36.53	9.538
DEX - C	15	38.80	14.872



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 3: Gender Distribution of the patients

	MALE	FEMALE
DEX - 0.4	8(53.3%)	7(46.7%)
DEX - 0.2	8(53.3%)	7(46.7%)
DEX - C	10(66.7%)	5(33.3%)
Total	45	100.0



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 4: Fromme et al bleeding score

Duration	Group	N	Mean	Std. Deviation	P value
5 min	DEX - C	15	.87	.640	.104
	DEX - 0.2	15	.80	.676	
15 min	DEX - C	15	1.33	.488	.039
	DEX - 0.2	15	1.27	.594	
30 min	DEX - C	15	1.07	.458	.081
	DEX - 0.2	15	1.20	.561	
45 min	DEX - C	15	1.27	.458	.028*
	DEX - 0.2	15	1.67	.488	
60 min	DEX - C	15	1.07	.594	.039
	DEX - 0.2	15	1.40	.507	
	DEX - C	15	1.07	.594	.030

75 min	DEX - 0.2	15	1.27	.458	
90 min	DEX - C	15	1.07	.704	.087
	DEX - 0.2	15	1.47	.516	
120 min	DEX - C	15	.93	.704	.105*
	DEX - 0.2	15	1.47	.516	
150 min	DEX - C	15	.80	.414	.091
	DEX - 0.2	15	1.00	.655	

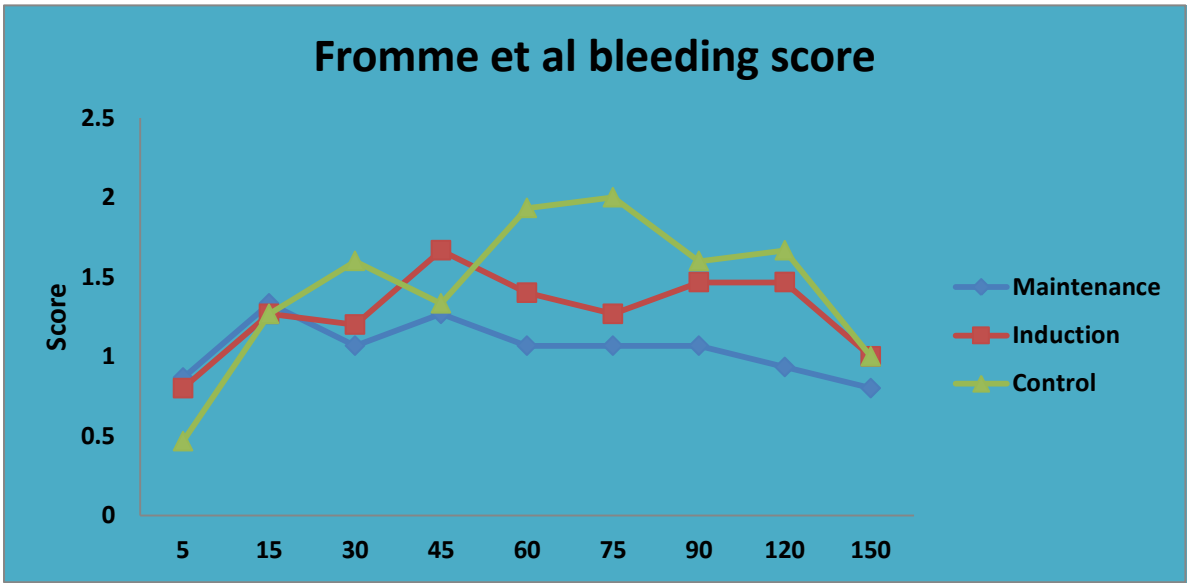
*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration	Group	N	Mean	Std. Deviation	P value
5 min	DEX - 0.2	15	.80	.676	.096
	DEX - 0.4	15	.47	.640	
15 min	DEX - 0.2	15	1.27	.594	.042
	DEX - 0.4	15	1.27	.704	
30 min	DEX - 0.2	15	1.20	.561	.050
	DEX - 0.4	15	1.60	.507	
45 min	DEX - 0.2	15	1.67	.488	.012
	DEX - 0.4	15	1.33	.617	
60 min	DEX - 0.2	15	1.40	.507	.003*
	DEX - 0.4	15	1.93	.594	
75 min	DEX - 0.2	15	1.27	.458	.001*
	DEX - 0.4	15	2.00	.655	
90 min	DEX - 0.2	15	1.47	.516	.051
	DEX - 0.4	15	1.60	.507	
120 min	DEX - 0.2	15	1.47	.516	.044
	DEX - 0.4	15	1.67	.617	
150 min	DEX - 0.2	15	1.00	.655	.098
	DEX - 0.4	15	1.00	.535	

*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration	Group	N	Mean	Std. Deviation	P value
5 min	DEX - C	15	.87	.640	.038
	DEX - 0.4	15	.47	.640	
15 min	DEX - C	15	1.33	.488	.005
	DEX - 0.4	15	1.27	.704	
30 min	DEX - C	15	1.07	.458	.005*
	DEX - 0.4	15	1.60	.507	
45 min	DEX - C	15	1.27	.458	.039
	DEX - 0.4	15	1.33	.617	
60 min	DEX - C	15	1.07	.594	.001*
	DEX - 0.4	15	1.93	.594	
75 min	DEX - C	15	1.07	.594	.001*
	DEX - 0.4	15	2.00	.655	
90 min	DEX - C	15	1.07	.704	.024*
	DEX - 0.4	15	1.60	.507	
120 min	DEX - C	15	.93	.704	.005*
	DEX - 0.4	15	1.67	.617	
150 min	DEX - C	15	.80	.414	.062
	DEX - 0.4	15	1.00	.535	

*-STATISTICALLY SIGNIFICANT (P<0.05)



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 5: Mean systolic blood pressure

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	122.73	5.934	.613
	DEX - 0.2	15	121.53	6.865	
Start of surgery	DEX - C	15	124.40	8.034	.002*
	DEX - 0.2	15	114.40	7.799	
5 min	DEX - C	15	118.40	6.555	.000*
	DEX - 0.2	15	109.13	5.866	
10 min	DEX - C	15	118.73	6.881	.000*
	DEX - 0.2	15	108.67	4.761	
15 min	DEX - C	15	115.20	7.794	.030*
	DEX - 0.2	15	110.20	3.364	
20 min	DEX - C	15	111.40	8.175	.843
	DEX - 0.2	15	112.13	11.643	
30 min	DEX - C	15	110.27	11.793	.537
	DEX - 0.2	15	112.87	10.954	
60 min	DEX - C	15	104.73	12.612	.015*
	DEX - 0.2	15	115.73	10.573	
90 min	DEX - C	15	105.27	11.640	.006*
	DEX - 0.2	15	117.73	11.061	
120 min	DEX - C	15	106.53	11.218	.001*
	DEX - 0.2	15	119.07	6.552	
150 min	DEX - C	15	111.13	9.187	.000*
	DEX - 0.2	15	122.13	5.592	
180 min	DEX - C	15	119.20	6.349	.554
	DEX - 0.2	15	120.53	5.817	
210 min	DEX - C	15	120.07	7.401	.356
	DEX - 0.2	15	122.67	7.771	

*-STATISTICALLY SIGNIFICANT (P<0.05)

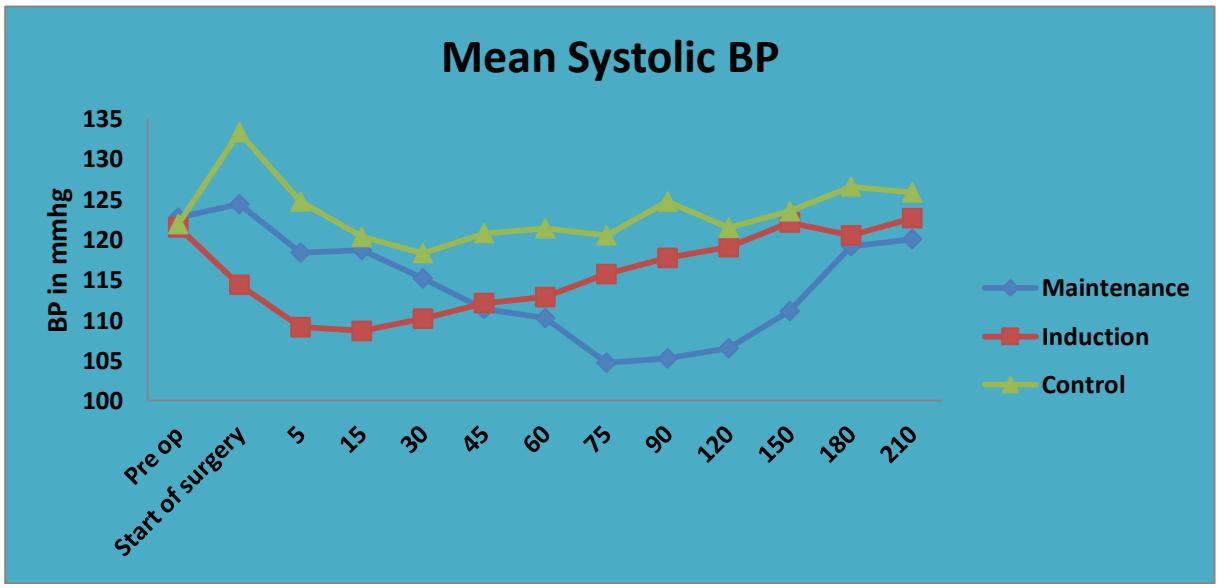
Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - 0.2	15	121.53	6.865	.899
	DEX - 0.4	15	121.93	9.975	
Start of surgery	DEX - 0.2	15	114.40	7.799	.000*
	DEX - 0.4	15	133.33	7.499	
5 min	DEX - 0.2	15	109.13	5.866	.000*
	DEX - 0.4	15	124.73	7.995	
10 min	DEX - 0.2	15	108.67	4.761	.000*
	DEX - 0.4	15	120.33	6.195	
15 min	DEX - 0.2	15	110.20	3.364	.000*
	DEX - 0.4	15	118.33	6.821	
20 min	DEX - 0.2	15	112.13	11.643	.021*
	DEX - 0.4	15	120.80	7.193	
30 min	DEX - 0.2	15	112.87	10.954	.026*

	DEX - 0.4	15	121.40	8.862	
60 min	DEX - 0.2	15	115.73	10.573	.172
	DEX - 0.4	15	120.53	8.017	
90 min	DEX - 0.2	15	117.73	11.061	.127
	DEX - 0.4	15	124.73	13.210	
120 min	DEX - 0.2	15	119.07	6.552	.132
	DEX - 0.4	15	121.47	6.749	
150 min	DEX - 0.2	15	122.13	5.592	.196
	DEX - 0.4	15	123.53	12.552	
180 min	DEX - 0.2	15	120.53	5.817	.014*
	DEX - 0.4	15	126.60	6.843	
210 min	DEX - 0.2	15	122.67	7.771	.171
	DEX - 0.4	15	125.87	4.172	

*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	122.73	5.934	.791
	DEX - 0.4	15	121.93	9.975	
Start of surgery	DEX - C	15	124.40	8.034	.004*
	DEX - 0.4	15	133.33	7.499	
5 min	DEX - C	15	118.40	6.555	.025
	DEX - 0.4	15	124.73	7.995	
10 min	DEX - C	15	118.73	6.881	.009
	DEX - 0.4	15	120.33	6.195	
15 min	DEX - C	15	115.20	7.794	.051
	DEX - 0.4	15	118.33	6.821	
20 min	DEX - C	15	111.40	8.175	.002*
	DEX - 0.4	15	120.80	7.193	
30 min	DEX - C	15	110.27	11.793	.007*
	DEX - 0.4	15	121.40	8.862	
60 min	DEX - C	15	104.73	12.612	.000*
	DEX - 0.4	15	120.53	8.017	
90 min	DEX - C	15	105.27	11.640	.000*
	DEX - 0.4	15	124.73	13.210	
120 min	DEX - C	15	106.53	11.218	.000*
	DEX - 0.4	15	121.47	6.749	
150 min	DEX - C	15	111.13	9.187	.005*
	DEX - 0.4	15	123.53	12.552	
180 min	DEX - C	15	119.20	6.349	.005*
	DEX - 0.4	15	126.60	6.843	
210 min	DEX - C	15	120.07	7.401	.013*
	DEX - 0.4	15	125.87	4.172	

*-STATISTICALLY SIGNIFICANT (P<0.05)



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 6: Mean diastolic blood pressure

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	75.53	5.963	.776
	DEX - 0.2	15	76.27	7.869	
Start of surgery	DEX - C	15	72.13	5.527	.075
	DEX - 0.2	15	69.40	7.744	
5 min	DEX - C	15	69.40	7.529	.085
	DEX - 0.2	15	65.40	4.290	
10 min	DEX - C	15	63.60	4.763	.022
	DEX - 0.2	15	64.93	1.870	
15 min	DEX - C	15	66.40	4.290	.047
	DEX - 0.2	15	68.33	8.715	
20 min	DEX - C	15	66.40	7.337	.011
	DEX - 0.2	15	67.73	6.861	
30 min	DEX - C	15	64.80	7.636	.083
	DEX - 0.2	15	65.47	5.263	
60 min	DEX - C	15	64.60	6.479	.108
	DEX - 0.2	15	70.27	11.517	
90 min	DEX - C	15	64.33	6.543	.089
	DEX - 0.2	15	68.60	6.706	
120 min	DEX - C	15	64.80	6.971	.163
	DEX - 0.2	15	68.07	5.405	
150 min	DEX - C	15	66.87	7.210	.066
	DEX - 0.2	15	71.33	5.473	
180 min	DEX - C	15	66.40	4.657	.236
	DEX - 0.2	15	68.33	4.065	
210 min	DEX - C	15	70.00	5.412	.254
	DEX - 0.2	15	72.33	5.563	

*-STATISTICALLY SIGNIFICANT (P<0.05)

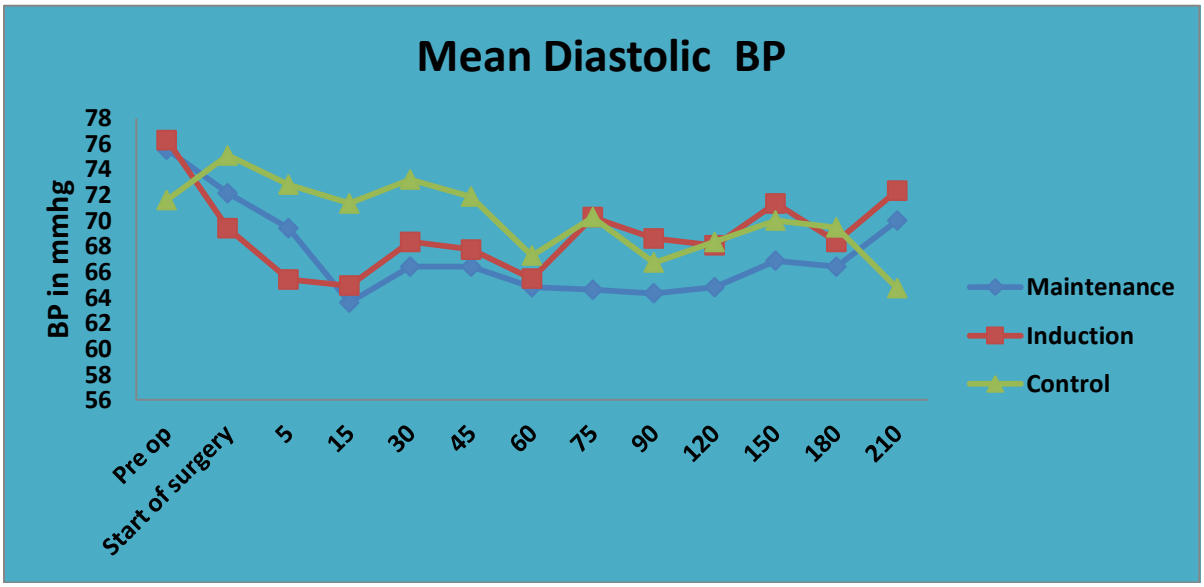
Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - 0.2	15	76.27	7.869	.106
	DEX - 0.4	15	71.60	7.434	
Start of surgery	DEX - 0.2	15	69.40	7.744	.117
	DEX - 0.4	15	75.07	11.132	
5 min	DEX - 0.2	15	65.40	4.290	.008*
	DEX - 0.4	15	72.80	9.151	
10 min	DEX - 0.2	15	64.93	1.870	.034*
	DEX - 0.4	15	71.33	10.952	
15 min	DEX - 0.2	15	68.33	8.715	.083
	DEX - 0.4	15	73.20	10.712	
20 min	DEX - 0.2	15	67.73	6.861	.193
	DEX - 0.4	15	71.87	9.833	
30 min	DEX - 0.2	15	65.47	5.263	.039
	DEX - 0.4	15	67.27	7.156	
60 min	DEX - 0.2	15	70.27	11.517	.090
	DEX - 0.4	15	70.27	7.166	
90 min	DEX - 0.2	15	68.60	6.706	.08
	DEX - 0.4	15	66.73	6.464	
120 min	DEX - 0.2	15	68.07	5.405	.026
	DEX - 0.4	15	68.33	9.634	
150 min	DEX - 0.2	15	71.33	5.473	.148
	DEX - 0.4	15	70.00	9.761	
180 min	DEX - 0.2	15	68.33	4.065	.100
	DEX - 0.4	15	69.47	7.210	
210 min	DEX - 0.2	15	72.33	5.563	.030*
	DEX - 0.4	15	64.73	3.973	

*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	75.53	5.963	.121
	DEX - 0.4	15	71.60	7.434	
Start of surgery	DEX - C	15	72.13	5.527	.038
	DEX - 0.4	15	75.07	11.132	
5 min	DEX - C	15	69.40	7.529	.076
	DEX - 0.4	15	72.80	9.151	
10 min	DEX - C	15	63.60	4.763	.018*
	DEX - 0.4	15	71.33	10.952	

15 min	DEX - C	15	66.40	4.290	.030*
	DEX - 0.4	15	73.20	10.712	
20 min	DEX - C	15	66.40	7.337	.095
	DEX - 0.4	15	71.87	9.833	
30 min	DEX - C	15	64.80	7.636	.059
	DEX - 0.4	15	67.27	7.156	
60 min	DEX - C	15	64.60	6.479	.031*
	DEX - 0.4	15	70.27	7.166	
90 min	DEX - C	15	64.33	6.543	.021
	DEX - 0.4	15	66.73	6.464	
120 min	DEX - C	15	64.80	6.971	.060
	DEX - 0.4	15	68.33	9.634	
150 min	DEX - C	15	66.87	7.210	.066
	DEX - 0.4	15	70.00	9.761	
180 min	DEX - C	15	66.40	4.657	.177
	DEX - 0.4	15	69.47	7.210	
210 min	DEX - C	15	70.00	5.412	.005*
	DEX - 0.4	15	64.73	3.973	

*-STATISTICALLY SIGNIFICANT (P<0.05)



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 7: Mean arterial pressure

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	90.87	4.824	.948
	DEX - 0.2	15	91.00	6.188	
Start of surgery	DEX - C	15	89.27	4.773	.033*
	DEX - 0.2	15	84.13	7.472	
5 min	DEX - C	15	85.33	6.366	.007*
	DEX - 0.2	15	79.60	4.137	
10 min	DEX - C	15	82.20	4.395	.020*
	DEX - 0.2	15	79.13	1.922	
15 min	DEX - C	15	82.40	4.808	.068
	DEX - 0.2	15	82.07	6.041	
20 min	DEX - C	15	81.13	5.718	.057
	DEX - 0.2	15	82.27	7.932	
30 min	DEX - C	15	79.73	7.667	.019
	DEX - 0.2	15	81.00	6.047	
60 min	DEX - C	15	77.47	7.918	.024*
	DEX - 0.2	15	85.13	9.635	
90 min	DEX - C	15	77.67	7.509	.042*
	DEX - 0.2	15	83.73	8.093	
120 min	DEX - C	15	78.33	7.678	.006*
	DEX - 0.2	15	84.80	3.427	
150 min	DEX - C	15	81.13	7.200	.005*
	DEX - 0.2	15	87.73	4.301	
180 min	DEX - C	15	83.80	4.379	.276

	DEX - 0.2	15	85.40	3.460	
210 min	DEX - C	15	86.73	4.652	.176
	DEX - 0.2	15	89.07	4.559	

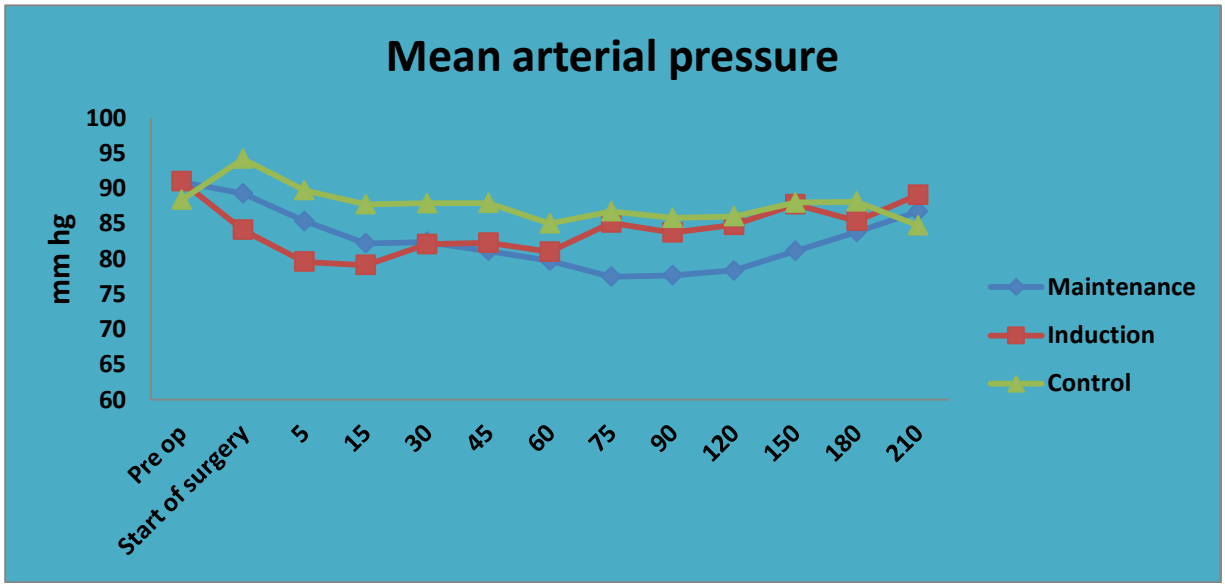
*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - 0.2	15	91.00	6.188	.265
	DEX - 0.4	15	88.38	6.439	
Start of surgery	DEX - 0.2	15	84.13	7.472	.002*
	DEX - 0.4	15	94.20	8.334	
5 min	DEX - 0.2	15	79.60	4.137	.000*
	DEX - 0.4	15	89.73	7.995	
10 min	DEX - 0.2	15	79.13	1.922	.000*
	DEX - 0.4	15	87.73	6.808	
15 min	DEX - 0.2	15	82.07	6.041	.022*
	DEX - 0.4	15	87.87	7.070	
20 min	DEX - 0.2	15	82.27	7.932	.066
	DEX - 0.4	15	87.93	8.268	
30 min	DEX - 0.2	15	81.00	6.047	.075
	DEX - 0.4	15	85.02	5.860	
60 min	DEX - 0.2	15	85.13	9.635	.065
	DEX - 0.4	15	86.73	4.543	
90 min	DEX - 0.2	15	83.73	8.093	.032
	DEX - 0.4	15	85.80	5.955	
120 min	DEX - 0.2	15	84.80	3.427	.085
	DEX - 0.4	15	86.04	8.031	
150 min	DEX - 0.2	15	87.73	4.301	.030
	DEX - 0.4	15	87.96	8.692	
180 min	DEX - 0.2	15	85.40	3.460	.097
	DEX - 0.4	15	88.13	5.097	
210 min	DEX - 0.2	15	89.07	4.559	.006*
	DEX - 0.4	15	84.73	3.218	

*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	90.87	4.824	.241
	DEX - 0.4	15	88.38	6.439	
Start of surgery	DEX - C	15	89.27	4.773	.057
	DEX - 0.4	15	94.20	8.334	
5 min	DEX - C	15	85.33	6.366	.107
	DEX - 0.4	15	89.73	7.995	
10 min	DEX - C	15	82.20	4.395	.013*
	DEX - 0.4	15	87.73	6.808	
15 min	DEX - C	15	82.40	4.808	.020*
	DEX - 0.4	15	87.87	7.070	
20 min	DEX - C	15	81.13	5.718	.014*
	DEX - 0.4	15	87.93	8.268	
30 min	DEX - C	15	79.73	7.667	.043*
	DEX - 0.4	15	85.02	5.860	
60 min	DEX - C	15	77.47	7.918	.001*
	DEX - 0.4	15	86.73	4.543	
90 min	DEX - C	15	77.67	7.509	.003*
	DEX - 0.4	15	85.80	5.955	
120 min	DEX - C	15	78.33	7.678	.012*
	DEX - 0.4	15	86.04	8.031	
150 min	DEX - C	15	81.13	7.200	.027*
	DEX - 0.4	15	87.96	8.692	
180 min	DEX - C	15	83.80	4.379	.019*
	DEX - 0.4	15	88.13	5.097	
210 min	DEX - C	15	86.73	4.652	.182
	DEX - 0.4	15	84.73	3.218	

*-STATISTICALLY SIGNIFICANT (P<0.05)



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 8: Mean Oxygen saturation in bloods

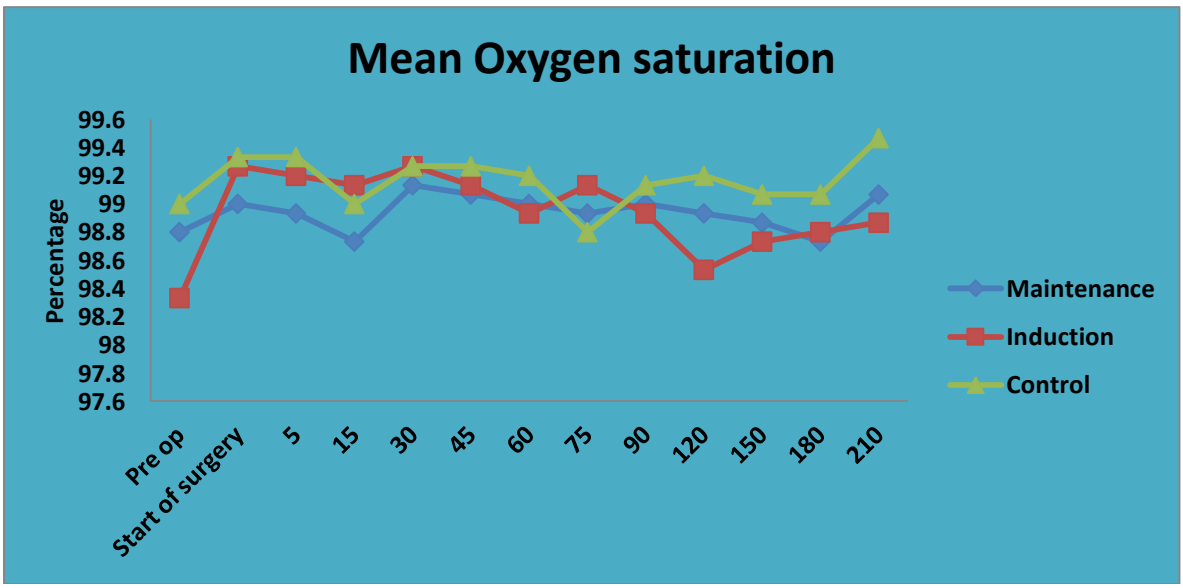
Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	98.800	1.0142	.225
	DEX - 0.2	15	98.333	1.0465	
Start of surgery	DEX - C	15	99.000	.7559	.026
	DEX - 0.2	15	99.267	.7037	
5 min	DEX - C	15	98.933	.9612	.010
	DEX - 0.2	15	99.200	.7746	
10 min	DEX - C	15	98.733	.7988	.141
	DEX - 0.2	15	99.133	.6399	
15 min	DEX - C	15	99.133	1.0601	.088
	DEX - 0.2	15	99.267	.7037	
20 min	DEX - C	15	99.067	.8837	.041
	DEX - 0.2	15	99.133	.9155	
30 min	DEX - C	15	99.000	1.0000	.063
	DEX - 0.2	15	98.933	1.0998	
60 min	DEX - C	15	98.933	.7988	.055
	DEX - 0.2	15	99.133	.6399	
90 min	DEX - C	15	99.000	.9258	.026
	DEX - 0.2	15	98.933	.7037	
120 min	DEX - C	15	98.933	1.0328	.053
	DEX - 0.2	15	98.533	.8338	
150 min	DEX - C	15	98.867	.8338	.068
	DEX - 0.2	15	98.733	.7988	
180 min	DEX - C	15	98.733	.8837	.083
	DEX - 0.2	15	98.800	.8619	
210 min	DEX - C	15	99.067	.7037	.548
	DEX - 0.2	15	98.867	1.0601	

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - 0.2	15	98.333	1.0465	.255
	DEX - 0.4	15	99.000	.7559	
Start of surgery	DEX - 0.2	15	99.267	.7037	.085
	DEX - 0.4	15	99.333	.6172	
5 min	DEX - 0.2	15	99.200	.7746	.006
	DEX - 0.4	15	99.333	.6172	
10 min	DEX - 0.2	15	99.133	.6399	.006
	DEX - 0.4	15	99.000	.7559	
15 min	DEX - 0.2	15	99.267	.7037	.100
	DEX - 0.4	15	99.267	.4577	
20 min	DEX - 0.2	15	99.133	.9155	.074
	DEX - 0.4	15	99.267	.7988	
30 min	DEX - 0.2	15	98.933	1.0998	.030
	DEX - 0.4	15	99.200	.6761	
60 min	DEX - 0.2	15	99.133	.6399	.066

	DEX - 0.4	15	98.800	.9411	
90 min	DEX - 0.2	15	98.933	.7037	.022
	DEX - 0.4	15	99.133	.6399	
120 min	DEX - 0.2	15	98.533	.8338	.040*
	DEX - 0.4	15	99.200	.8619	
150 min	DEX - 0.2	15	98.733	.7988	.263
	DEX - 0.4	15	99.067	.7988	
180 min	DEX - 0.2	15	98.800	.8619	.361
	DEX - 0.4	15	99.067	.7037	
210 min	DEX - 0.2	15	98.867	1.0601	.059
	DEX - 0.4	15	99.467	.5164	

*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	98.800	1.0142	.545
	DEX - 0.4	15	99.000	.7559	
Start of surgery	DEX - C	15	99.000	.7559	.097
	DEX - 0.4	15	99.333	.6172	
5 min	DEX - C	15	98.933	.9612	.086
	DEX - 0.4	15	99.333	.6172	
10 min	DEX - C	15	98.733	.7988	.356
	DEX - 0.4	15	99.000	.7559	
15 min	DEX - C	15	99.133	1.0601	.058
	DEX - 0.4	15	99.267	.4577	
20 min	DEX - C	15	99.067	.8837	.021
	DEX - 0.4	15	99.267	.7988	
30 min	DEX - C	15	99.000	1.0000	.065
	DEX - 0.4	15	99.200	.6761	
60 min	DEX - C	15	98.933	.7988	.079
	DEX - 0.4	15	98.800	.9411	
90 min	DEX - C	15	99.000	.9258	.050
	DEX - 0.4	15	99.133	.6399	
120 min	DEX - C	15	98.933	1.0328	.049
	DEX - 0.4	15	99.200	.8619	
150 min	DEX - C	15	98.867	.8338	.508
	DEX - 0.4	15	99.067	.7988	
180 min	DEX - C	15	98.733	.8837	.263
	DEX - 0.4	15	99.067	.7037	
210 min	DEX - C	15	99.067	.7037	.087
	DEX - 0.4	15	99.467	.5164	



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 9: Mean pulse rate

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	88.400	10.4663	.000*
	DEX - 0.2	15	72.000	7.4066	
Start of surgery	DEX - C	15	92.200	12.8630	.000*
	DEX - 0.2	15	75.867	8.8871	
5 min	DEX - C	15	86.200	10.6315	.000*
	DEX - 0.2	15	71.000	5.0709	
10 min	DEX - C	15	77.000	8.4684	.002*
	DEX - 0.2	15	67.733	5.9698	
15 min	DEX - C	15	74.400	7.9534	.038*
	DEX - 0.2	15	68.467	6.9062	
20 min	DEX - C	15	70.800	7.4852	.256
	DEX - 0.2	15	67.667	7.3062	
30 min	DEX - C	15	68.000	5.8797	.303
	DEX - 0.2	15	71.667	12.1870	
60 min	DEX - C	15	68.133	6.9165	.146
	DEX - 0.2	15	72.600	9.2567	
90 min	DEX - C	15	65.800	8.0640	.094
	DEX - 0.2	15	70.533	6.8543	
120 min	DEX - C	15	67.467	5.7801	.110*
	DEX - 0.2	15	71.333	6.9864	
150 min	DEX - C	15	69.600	6.3449	.056
	DEX - 0.2	15	74.400	6.8222	
180 min	DEX - C	15	73.067	5.7504	.084
	DEX - 0.2	15	77.867	8.6426	
210 min	DEX - C	15	76.867	9.2957	.464
	DEX - 0.2	15	79.267	8.4046	

*-STATISTICALLY SIGNIFICANT (P<0.05)

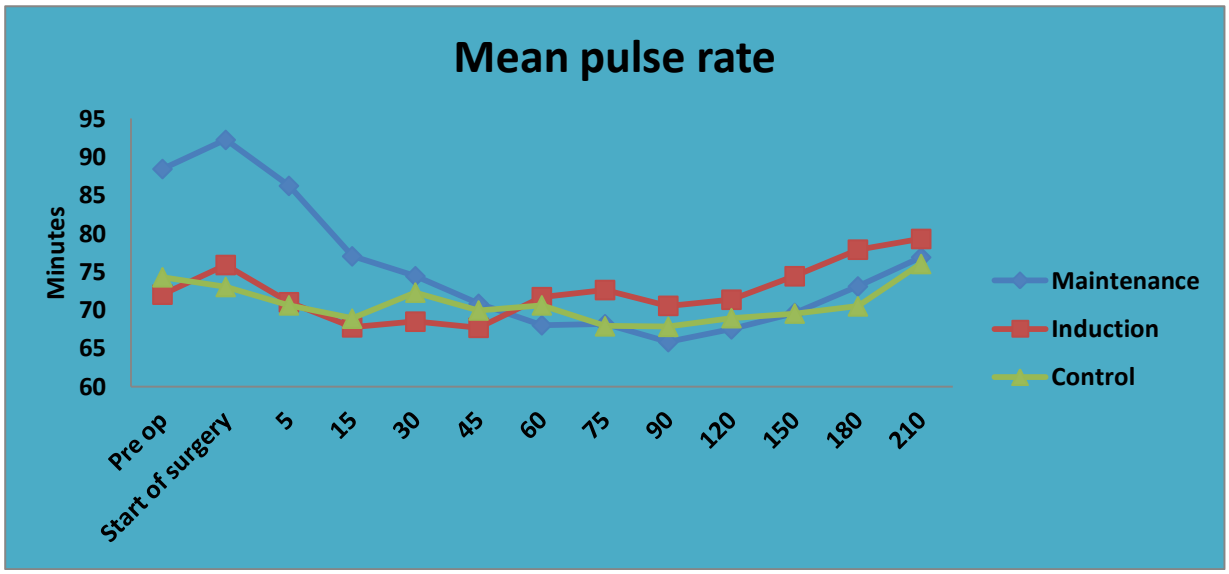
Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - 0.2	15	72.000	7.4066	.426
	DEX - 0.4	15	74.267	7.9504	
Start of surgery	DEX - 0.2	15	75.867	8.8871	.352
	DEX - 0.4	15	73.000	7.6625	
5 min	DEX - 0.2	15	71.000	5.0709	.055
	DEX - 0.4	15	70.600	6.7061	
10 min	DEX - 0.2	15	67.733	5.9698	.061
	DEX - 0.4	15	68.867	5.7553	
15 min	DEX - 0.2	15	68.467	6.9062	.055
	DEX - 0.4	15	72.267	7.3335	
20 min	DEX - 0.2	15	67.667	7.3062	.010
	DEX - 0.4	15	69.933	7.5448	
30 min	DEX - 0.2	15	71.667	12.1870	.088
	DEX - 0.4	15	70.600	9.1324	
60 min	DEX - 0.2	15	72.600	9.2567	.064
	DEX - 0.4	15	67.867	8.8952	
90 min	DEX - 0.2	15	70.533	6.8543	.205
	DEX - 0.4	15	67.800	4.4110	
120 min	DEX - 0.2	15	71.333	6.9864	.009
	DEX - 0.4	15	68.933	8.6145	
150 min	DEX - 0.2	15	74.400	6.8222	.105
	DEX - 0.4	15	69.533	8.9671	
180 min	DEX - 0.2	15	77.867	8.6426	.020*
	DEX - 0.4	15	70.467	7.8182	
210 min	DEX - 0.2	15	79.267	8.4046	.291
	DEX - 0.4	15	76.000	8.2115	

*-STATISTICALLY SIGNIFICANT (P<0.05)

Duration		N	Mean	Std. Deviation	P value
Pre op	DEX - C	15	88.400	10.4663	.000*
	DEX - 0.4	15	74.267	7.9504	
Start of surgery	DEX - C	15	92.200	12.8630	.000*
	DEX - 0.4	15	73.000	7.6625	
5 min	DEX - C	15	86.200	10.6315	.000*
	DEX - 0.4	15	70.600	6.7061	
10 min	DEX - C	15	77.000	8.4684	.005*

	DEX - 0.4	15	68.867	5.7553	
15 min	DEX - C	15	74.400	7.9534	.451
	DEX - 0.4	15	72.267	7.3335	
20 min	DEX - C	15	70.800	7.4852	.054
	DEX - 0.4	15	69.933	7.5448	
30 min	DEX - C	15	68.000	5.8797	.362
	DEX - 0.4	15	70.600	9.1324	
60 min	DEX - C	15	68.133	6.9165	.028
	DEX - 0.4	15	67.867	8.8952	
90 min	DEX - C	15	65.800	8.0640	.007
	DEX - 0.4	15	67.800	4.4110	
120 min	DEX - C	15	67.467	5.7801	.588
	DEX - 0.4	15	68.933	8.6145	
150 min	DEX - C	15	69.600	6.3449	.081
	DEX - 0.4	15	69.533	8.9671	
180 min	DEX - C	15	73.067	5.7504	.308
	DEX - 0.4	15	70.467	7.8182	
210 min	DEX - C	15	76.867	9.2957	.789
	DEX - 0.4	15	76.000	8.2115	

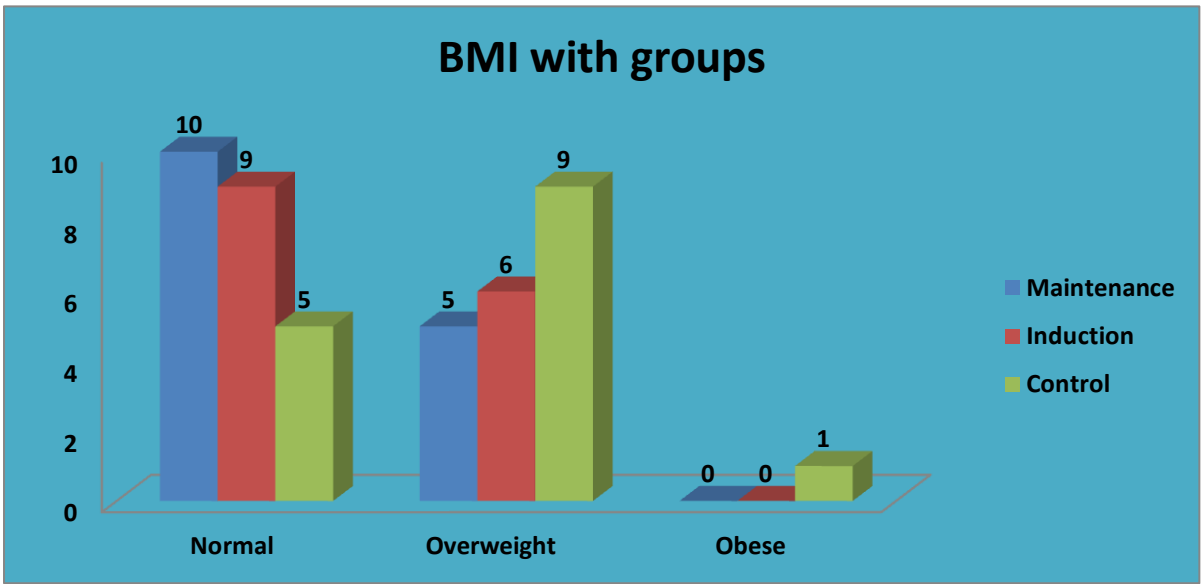
*-STATISTICALLY SIGNIFICANT (P<0.05)



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

Table 10:

	BMI			
	NORMAL	OVERWEIGHT	OBESE	P VALUE
DEX - 0.4	10(66.7%)	5(33.3%)	0(0.0%)	
DEX - 0.2	9(60.0%)	6(40.0%)	0(0.0%)	.248
DEX - C	5(33.3%)	9(60.0%)	1(6.7%)	



*Maintenance – DEX-0.4, Induction – Dex-0.2, Control – Dex-C.

V. Discussion

Several studies were done with various pharmacologic agents to produce controlled hypotension in FESS and to study various aspects includes sedation score, hemodynamic variables, surgeons comfort, rescue analgesic requirements. In this study we compared two different dexmedetomidine doses for bringing controlled hypotension in endoscopic sinus surgeries. After obtaining our institutional ethical committee clearance, the study was started and informed consent was obtained from all participants.

Those who didn't give consent were omitted from the study. Out of total 45 participants, 15 were separated for each group by random allocation through computer generated random assignation table. Group Dex-0.2 patients received only bolus dexmedetomidine 1 µg/kg iv over 10-20 mins and continous iv infusion of 0.2 µg/kg/h dexmedetomidine. Group Dex-0.4 received bolus dose of 1 µg/kg iv and continous iv infusion of 0.4 µg/kg/h dexmedetomidine and Group Dex-C recieved saline infusion as maintainance and bolus dexmedetomidine.

Both the groups are demographically comparable with respect to the age, ASA physical status, duration of surgery, duration of anesthesia and recovery characteristics including extubation time .

Fromme et al. bleeding score

In our study fromme et al bleeding score was used to assess the surgical field induced by dexmedetomidine. In all three groups bleeding score was recorded from the beginning to the end of surgery using a separate proforma. At all assessment time the difference between the three groups were statistically significant with (P< 0.05). Bleeding score observed was lowest in Dex-0.4 group than other groups. Between the DEX-C and DEX-0.2 group, the bleeding was significantly reduced in the DEX-0.2 group at 45 min (p < 0.028), 60 min (p <.039) and 75 min (p <.030) apart from that there is no significance difference for remaining assessment time points between these groups. Between the DEX-0.2 and DEX-0.4 group, bleeding was significantly reduced in the DEX-0.4 group with p values 60 min (p< .003*), 75 min (p<.001*). With DEX-C and DEX-0.4 group , p values are < 0.05 at 15 min and 20 min, at 60 min and 75 min (p < 0.01) , 90 min (p = .024*) and the results are supported with the study done by DK bharathwaj and Kamath SS[11] and Tarek Shams, and Ragaa El-Masry [12].

MEAN ARTERIAL BLOOD PRESSURE

There was remarkable difference between the three groups which was statistically significant with (P< 0.05) at all assessment time points for the target mean arterial pressure. In Group Dex-0.4, target MAP achieved throughout the surgery, p values were highly significant with p < 0.013 at 10 min, p <0.001at 60 min, p < 0.019 at 180 min with the DEX-C group. For Group Dex-0.2 group, target MAP was attained most of the times but the effect was not consistent, Injection Nitroglycerin was infused in few circumstances to achieve target MAP with values of p <0.06 at 20 min, p<0.08 at 120 min with the DEX-C group. This correlates with the study conducted by Neamat I. Abel Rahman, Eman A. Fouad et al [2] and Gurbet A, Basagan-Mogol E et al [9].

MEAN SYSTOLIC AND DIASTOLIC BLOOD PRESSURE

Significant fall in systolic blood pressure and diastolic blood pressure was noted in Group Dex-0.4 during most of time intervals during the study than other two groups. Incidences of hypotension noted and doses of ephedrine required were significantly greater with Group Dex-0.4 than remaining groups. This correlates with the study conducted by DK bharathwaj and Kamath et al [11] and Esmail Moshiri, Hesameddin Modir et al [14].

MEAN PULSE RATE

The baseline heart rate recorded was not significantly differed in three groups. Heart rate significantly reduced in Group Dex-0.4 from T5* till T90* ($p< 0.05$) with group DEX-C. Thereafter from T90* till T120 no significant difference noted in either groups. Heart rate was significantly reduced in Group Dex-0.4 when compared to other groups. In the control group there was higher heart rate reduction in Dex-0.4 group at most of times except at T120, T180 and T210 minutes of observation. These results are comparable with the study conducted by Vineela , Ganapathi et al [8].

*T- Time of assessment in minutes.

SEDATION LEVEL

Ramsay sedation score was significantly higher in Group Dex-0.4 and Group Dex-0.2 in post assessment unit during the post-operative period when compared to DEX-C group. This results were comparable to previous studies conducted by Neamat I. Abel Rahman, Eman et al [2] and Richa F, Yazigi et al [5].

RESPIRATORY PARAMETERS

No statistically significant difference was noted in all three groups with regard to the oxygen saturation or respiratory rate at all time intervals.

ADVERSE EFFECTS

Hypotension and bradycardia was more in dexmedetomidine Group Dex-0.4 group than other two groups. Hypotension was more pronounced in the group DEX-0.4 managed with crystalloids and injection Ephedrine 30mg in titrated doses. In DEX-0.4 group hypotensive episodes noted in more than half of the study population during middle of the surgery. Dexmedetomidine infusion was stopped in instances in addition to rescue measures. Bradycardia was more pronounced in DEX-0.4 patients which was managed with atropine 0.6mg iv.

Nausea and vomiting was observed for two participants during the extubation period in DEX-0.4 group even with the routine anti emetic prophylaxis to all participants treated empirically. Post-operative dry mouth was noted in few patients which was not alarming.

VI. Summary

Fromme et al bleeding score was significantly reduced in DEX-0.4 group (score 0 to 2) during most of the intraoperative time period and in DEX-0.2 (score 0 to 2) when compared to DEX-C (score 1 to 3). Target MAP was well maintained in DEX-0.4 (62 ± 33) and DEX-0.2 groups (66± 11) when compared to DEX-C group (72±15). The mean pulse rate in DEX-0.4 is (59±13) and in DEX-0.2 group the value is (64±12), (76 ± 14) with DEX-C group. The incidence of hypotension and bradycardia was very high in DEX-0.4 which requires aggressive intervention. Only 2 out of 15 participants in DEX 0.2 group had intra-op hypotension which was not much alarming and managed with reduction of the infusion dose and bolus crystalloids. DEX-0.2 group provided an optimal surgical field with less adverse events when compared to other two groups.

VII. Conclusion

Dexmedetomidine provides better surgical operative condition during FESS, bolus dose of dexmedetomidine 1µg/kg iv followed by continous iv infusion of 0.2µg/kg/hr provides an optimal surgical field with less side effects. Bolus dose of dexmedetomidine 1µg/kg iv followed by continous iv infusion of 0.4µg/kg/hr provides better operative conditions at the expense of higher incidence of adverse hemodynamic events.

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