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ASIAN ARCHIVES OF ANAESTHESIOLOGY AND RESUSCITATION

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ASIAN ARCHIVES OF ANAESTHESIOLOGY AND RESUSCITATION

Office Address : Room No : 306 - 309, Department of Anaesthesia,3rd Floor, BL Taneja Block, MAMC and LN Hospital, New Delhi (INDIA)

ANAESTHESIA & ALLIED SCIENCES
FOR PARAMEDICS

A Comprehensive textbook of Anaesthesia, Intensive Care, Anatomy, Physiology, Biochemistry, Pharmacology, Pathology and other Special topics

(A Textbook for B.Sc. Operation Theater Students, Trauma Technicians, Nurses, Physiotherapists)

'ANAESTHESIA AND ALLIED SCIENCES FOR PARAMEDICS' is first book of its kind that comprises of six sections. The section one consists of anatomy, physiology and biochemistry for paramedics. The details of all muscles, their actions, nerves and vessels are compiled in tabular form so that it is easily learnt and recapitulated by students. Essential physiology and clinical biochemistry are concised subsections of this section.

The second section provides details of anaesthesia and its various sub-specialities. This section has several chapters and starts from history of anaesthesia till modular operation theatre suit details.

Third section, 'Drugs and Pharmacology' provides details of all the anaesthesia and emergency drugs. Section four covers all the instruments which are used in anaesthetic practice including anaesthesia machine to Automated External Defibrillator. The details of instruments will be very beneficial for the students during examinations and table viva.

Fifth section provides knowledge of unique topics of modern anaesthetic practices that need utmost attention in day - to - day life. The sixth section is a final section highlights all the scoring systems and grading in anaesthesia.

This book will be extremely useful to all paramedics i.e BSc Medical Technology students, operation theatre technicians nurses, physiotherapists and trauma technicians, in all types of examinations, skill development and knowledge augmentation.

The book is a sincere tribute to my father who had this dream for me. I am fortunate enough to have blessings from Almighty, my teachers and parents. All the contributors of this book have provided me a great support and deserve my heartfelt gratitude.

About The Editor

Dr Manpreet Singh is a graduate and post-graduate from Jawaharlal Nehru Medical College, Aligarh. He worked at University College of Medical Sciences and GTB Hospital , Delhi in various capacities as Senior Resident, Sr. Research Associate and Specialist Consultant in Department of Anaesthesia and Critical Care. He has done various fellowships and courses related to Emergency Medicine, Critical Care and Emergency Life Support and is a Fellow of Chest Care Physician (USA), Fellow of Academic College of Emergency Experts in India and Fellow of International Medical Sciences Academy(FIMSA) .

He is an instructor and provider of various courses in India like Advanced Cardiac Life Support (through American Heart Association), Fundamental Critical Care Support , Paediatric and Neonatal Life Support, Trauma Life Support and Advanced Ultrasound trauma Life Support. He is a certificate holder of 'Basic Law and Medicine' (Mumbai) and National Disaster Management (NIDM).He is a course coordinator of various workshops and training courses in Community CPR, Community Trauma and Training & Airway Management courses along with many eminent teachers all over India.

He is a member of more than 10 international and national professional societies, has written more than 52 research papers and presented more than 65 research papers or lectures as invited faculty in various national and international conferences. He is a co-editor of two national journals and reviewer of 8 indexed and non-indexed journals.

Currently, he is Assistant Professor in Department of Anaesthesia and Intensive Care, Govt. Medical College and Hospital-32, Chandigarh .His exceptional skills as a writer ,speaker and editor have been reflected in all major conferences, workshops and publications. His colleagues hold him in high esteem for his academic and professional excellence. Presently he is involved in teaching the students of BSc Medical Technology and Operation Theatre, Trauma Technician course, MBBS and MD (Anaesthesiology).

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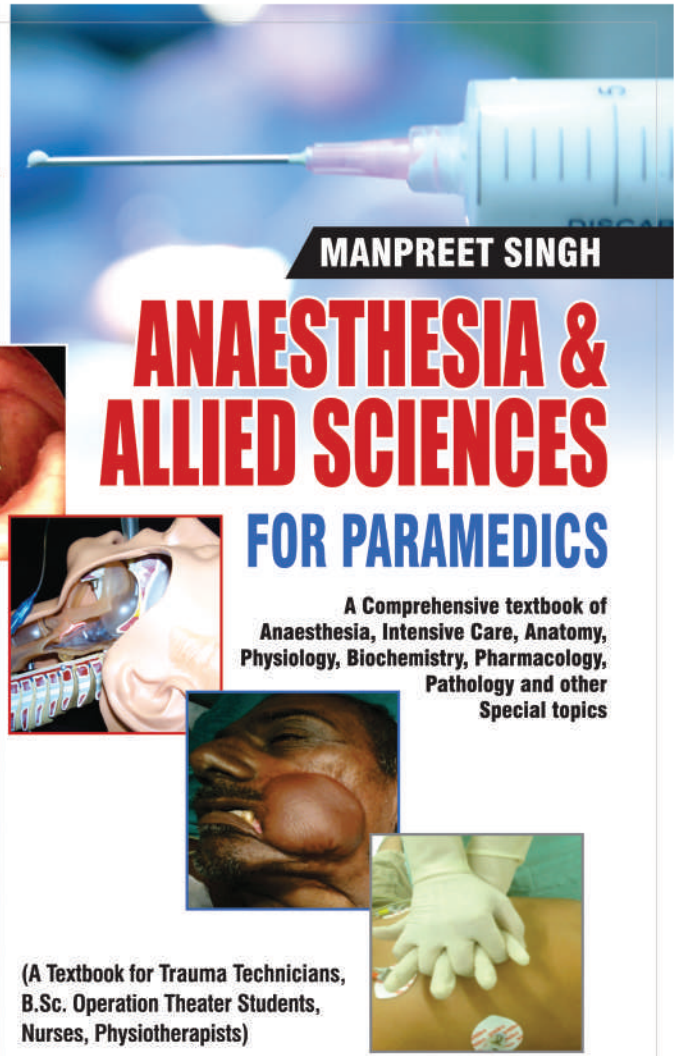
MANPREET SINGH



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DEXMEDETOMIDINE: A NOVEL ANESTHETIC AGENT, ITS PHARMACOLOGY AND USES IN ANESTHESIA AND I.C.U

Ankur Varshney[1], Shahjahan Bano[2], Muazzam Hasan[1], Nadeem Raza[1]

ABSTRACT

The potential uses of dexmedetomidine, a selective alpha 2 adrenergic receptor are many. Although not orally active, bioavailablity when given via other routes like parentral is very good. Dexmedetomidine is a selective α2-adrenoreceptor agonist; it has a α2/α1 selectivity ratio which is eight to 10 times higher than that of clonidine. The ability of dexmedetomidine to reduce the requirements of traditional anaesthetic & analgesic agents is increasingly being used in perioperative period. Also its ability to produce arousable sedation is being utilized frequently in I.C.U's. Dexmedetomidine potentiates the effects of all anesthetics, regardless of method of administration (intravenous, volatile, or even regional block). It provides good sedation and anxiolysis level during surgery as well as it prolongs the duration of analgesia into the post-operative period. These effects have made this drug as most preferred sedative agent in procedural sedation, obstructive sleep apnea patients and sleep studies. Both intravenous and intrathecal dexmedetomidine cause dose dependent prolongation of duration of neuraxial anesthesia. This article reviews the current role of dexmedetomidine in anesthesia and its use in some special scenarios.

Keywords: Anesthesia, sedation, I.C.U

INTRODUCTION

Dexmedetomidine a second generation alpha 2 adrenergic receptor specific, pharmacologically active d- isomer of medetomidine was first synthesized in late 1980's. Alpha 2 agonists are increasingly being used in anaesthesia as they not only decrease sympathetic tone and attenuate the stress response to anaesthesia and surgery but also cause sedation and analgesia. They are also being used as adjuvants during regional anaesthesia.

Clonidine, which was initially introduced as anti hypertensive, is the most commonly used alpha 2 agonists by anaesthesiologist. Dexmedetomidine is the most recent agent found in this group approved by FDA in 1999 for use in humans for analgesia and sedation. Since then it is growing in popularity among anaesthetists and intensivists.

Dexmedetomidine is a selective alpha-2-adrenoreceptor agonist; it has a α2/α1 selectivity ratio which is eight to 10 times higher than that of clonidine [1]. The ability of dexmedetomidine to reduce the requirements of traditional anesthetic & analgesic agents is increasingly being used in perioperative period.

Physical Properties

Dexmedetomidine is a dextro-isomer and

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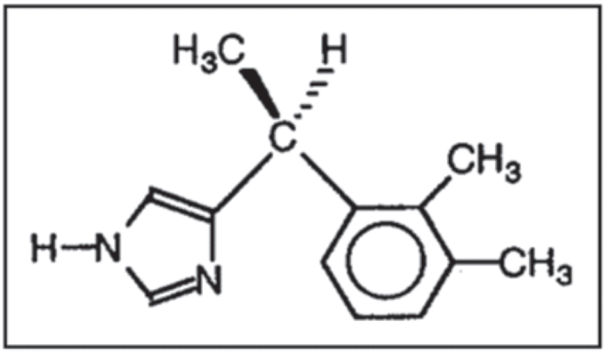
pharmacologically active component of medetomidine, which has been used for many years in vetenary practice for its hypnotic, sedative and analgesic effects. The chemical formula is $C_{13}H_{16}N_2HCl$, which is designated as (*S*)-4-[1-(2, 3-dimethylphenyl) ethyl]-3H-imidazole(Figure 1).



SOURCE: WIKIPEDIA

Figure 1: structure of dexmedetomidine

Pharmacokinetics

Dexmedetomidine has a short half-life[3] (2-3 hours vs. 12-24 hours for clonidine) and is commercially available for intravenous administration. Its context sensitive half life ranges from 4 min after a 10 min infusion to 250 min after 8 hour infusion. Studies have shown that this context sensitive half life is prolonged in elderly patients and with low albumin concentration. This explains the prolonged duration of action in elderly patients[2].

The average protein binding of dexmedetomidine is 94%, with negligible protein binding displacement by fentanyl, ketorolac, theophylline, digoxin, and lidocaine, drugs commonly used during anesthesia and in the ICU [3]. A decreased albumin concentration would cause a higher proportion of unbound dexmedetomidine in blood, which then results in a larger volume of distribution.

Clearance of dexmedetomidine is mainly determined by liver blood flow but less by protein binding. Dexmedetomidine undergoes almost complete hydroxylation through direct glucuronidation and cytochrome P450 (primarily CYP2A6) metabolism in liver. Metabolites are excreted mainly in the urine (about 95%) and a small amount in the faeces (4%). In hepatic failure metabolism of the active drug will be impaired hence dose reduction may be needed.

In cases of renal failure, the metabolites may accumulate and dose reduction is needed only with a creatinine clearance of < 30 ml/min [3].

Pharmacokinetics Extravascular:

Dexmedetomidine absorption in CSF after epidural injection is rapid (Tmax = 5-20 min), although pharmacokinetic modeling suggested a biphasic absorption process. CSF contains only 22% of the injected dose. Intrathecal dexmedetomidine injection produces maximum anlgesia within 20-30 min of injection [4].

Mechanism Of Action

Dexmedetomidine is a highly selective, specific, and potent alpha 2 adrenergic agonist.

— Alpha 2 : alpha 1 ~ 1600 : 1

— Alpha 2 receptors has 3 subtypes-

Subtype A - CNS (locus coerulus) - Responsible for the sedative, analgesic and sympatholytic effects mediated by G-protein inhibition of L-type calcium channels in the post synaptic receptors.

Subtype B - Peripheral vasculature- Responsible for the short-term hypertensive response.

Subtype C - CNS - Responsible for the anxiolytic effect.

Stimulation of the alpha 2-adrenoceptors in the locus coeruleus (important modulator of nociceptive neurotransmission) terminates the propagation of pain signals leading to analgesia. Post synaptic activation of Medullary vasomotor center, alpha 2 receptor results in decrease in sympathetic nervous system outflow from CNS to peripheral tissue. Presynaptic activation of the alpha-2A inhibits the release of norepinephrine

which results in the sedative and hypnotic effects[3]. Substantia gelatinosa of the spinal cord: Stimulation of alpha2-receptors leads to inhibition of nociceptive neurons firing and the release of substance P. Peripheral sympathetic nervous system nerve ending analgesic action is by preventing Nor-ephinephrine release[3].

Pharmacodynamics:
Central nervous system: Cause sedation by decreasing sympathetic nervous system activity and the level of arousal. It results in sedation of the patient who can be aroused readily to full consciousness and are less likely to be disoriented or uncooperative. Unlike other sedative it does not cause clouding of consciousness, depression of ventilation, agitation or delirium. This effect may be mediated by postsynaptic alpha-2a subtype adrenoceptors located in the locus coeruleus, causing a decrease in noradrenergic activity.

Cardiovascular system
Dexmedetomidine does not produce any direct effects on the heart [5.] A biphasic cardiovascular response has been seen after its administration. [3, 6-8]

The bolus of 1 mcg/kg dexmedetomidine initially results in a transient increase of the blood pressure and a reflex fall in heart rate, especially in younger, healthy patients which lasts for about 10-15 min[7]. Stimulation of alpha -2-adrenoceptor in vascular smooth muscle seems to be responsible for the initial rise in the blood pressure, which can be attenuated by a slow infusion. This is followed by a slight decrease in blood pressure due to the inhibition of the central sympathetic outflow. The presynaptic alpha 2-adrenoceptors are also stimulated decreasing the norepinephrine release resulting in fall in blood pressure & heart rate [9]. Alpha-2 adrenoceptor mediated reduction in sympathetic tone and increase in parasympathetic tone results in a reduced heart rate, systemic metabolism, myocardial contractility and systemic vascular resistance. These all result in decrease in the myocardial oxygen requirements.

The beneficial effect on myocardial oxygen balance has been shown to decrease peri-operative myocardial ischemia and infarction in cardiac ,as well as noncardiac surgery [3,10].

Respiratory system
Alpha-2 adrenoceptor agonists do not cause depression of ventilation .It result in a state similar to sleep with mild respiratory depression, but there is no significant effect on hypercapnic or hypoxic ventilatory drive. The respiratory depression caused by dexmedetomidine has been reported to be much less than with other sedatives. [10]

Renal system
Alpha-2 adrenoceptors has a number of effects that promote diuresis and natriuresis. They decrease the secretion of vasopressin and antagonise its effect on renal tubules [3].

Neuroendocrine system
The alpha-2 adenoceptor agonists have a number of neuroendocrine effects, mainly related to their inhibition of sympathetic outflow and the decrease in plasma levels of circulating catecholamines. Stimulation of alpha-2 adrenoceptors located on the cells of the islets of langarhans can temporarily cause direct inhibition of insulin release [3]. Dexmedetomidine can inhibit the secretion of adrenocorticotropic harmones (ACTH) and cortisol during surgery.

Gastrointestinal system
Alpha-2 adrenoceptors regulate vagally mediated increase in gastric and intestinal motility and secretions. Activation of alpha-2 adrenoceptors inhibits water secretion and increases net absorption in the large bowel[3]. Dexmedetomidine is known to reduce salivary secretion and may lead to a dry mouth.

Platelet effects

Selective alpha-2 adrenoceptor agonists as well as adrenaline are known to stimulate platelet aggregation by stimulating alpha-2c receptor on platelets. Dexmedetomidine does not promote platelet aggregation. It also blocks adrenaline induced platelet aggregation. In 1996 Heesen M et al did an invitro study and showed that clinically relevant concentrations of 1 ng/ml clonidine or dexmedetomidine did not alter platelet aggregation or alpha 2-receptor density, even after 24 h exposure. However, 10 ng/ml dexmedetomidine was found to diminish significantly epinephrine-induced platelet aggregation, but did not change alpha 2-receptor density.[12]

Effect on airway dynamics and sleep pattern

Dexmedetomidine have less effect on upper airway tone and airway collapsibility as compared with other sedative agents. It possesses properties that mimic non-REM sleep, without significant respiratory depression or airway obstruction. These properties make dexmedetomidine an attractive agent for MRI sleep study evaluation in children with OSA.[13]

Therapeutic role in anaesthesia

a) Premedication

It can be used as a premedicant for sedation, anxiolysis and antisialogogue action in a dose ranging from 0.5-1.0 mcg/kg. It can also potentiate the anaesthetic action of other agents and reduce anaesthetic requirements during surgery [8]. Scheinin H et al in 1993 showed the intramuscular use of dexmedetomidine as premedication. They suggested that pretreatment with a single intramuscular injection of 2.5 mcg/kg dexmedetomidine is efficacious, but significantly increases the incidence of intraoperative hypotension and bradycardia in ASA physical status 1 or 2 patients [14].

i) Hemodynamic stability and perioperative ischemia

Dexmedetomidine (0.5 mcg/kg over 10-min) was administered prior to induction of general anaesthesia which attenuates the sympathetic response to laryngoscopy and intubation in patients undergoing myocardial revascularization[15].This preanaesthetic dose of dexmedetomidine blunts reflex tachycardia associated with direct laryngoscopy for intubation of the trachea, decreases intraoperative lability of hemodynamic parameters, decreases plasma catecholamine concentrations and dramatically decreases anaesthetic requirements for inhalational and injected drugs.

Emergence from anaesthesia is a period associated with increased sympathetic activity, tachycardia and hypertension. Dexmedetomidine premedication decreases the unwanted hemodynamic effects seen during recovery from anaesthesia. It has been reported in number of studies to improve exercise tolerance in patients with angina pectoris and reduces exercise induced myocardial ischaemia. The hypertensive response to ketamine is also attenuated by dexmedetomidine.

ii) Sedation and anxiolysis

· It has long been known that dexmedetomidine causes sedation. Sedation along with anxiolysis, make alpha-2 adrenoceptor agonist useful premedication drugs. It is generally initiated with a loading dose of 1 mcg/kg over 10 minutes for both procedural sedation and ICU sedation.

· For ICU sedation

Maintenance dosing is initiated at 0.4 mcg/kg/hr and titrated over a dose range of 0.2 to 0.7 mcg/kg/hr.

- **For sedation during surgical and other procedures**

After administration of a 1 mcg /kg loading dose, the maintenance dose is initiated at 0.6 mcg/kg/hr and titrated to achieve the desired clinical effect, with doses ranging from 0.2 to 1 mcg/kg/hr.

iii) Anaesthetic requirements

Dexmedetomidine potentiates the effects of all anesthetics, regardless of method of administration (intravenous, volatile, or even regional block).

Studies have shown that dexmedetomidine use has been associated with approximately 62.5% reduction (0.75 mg/kg) in the induction dose of propofol and 30-50 % less isoflurane requirements for maintenance of anesthesia, while maintaining the adequate depth of anesthesia [16].

b) Analgesia

Alpha-2 adrenoceptor agonists have analgesic properties when given parenterally, epidurally or an intrathecally. It produces intraoperative and postoperative opioid-sparing effect. There is reduced need for additional analgesics in the PACU, thereby reducing the length of stay. It can be used as a sole intravenous analgesic agent preoperative or postoperatively in a dose ranging from 0.5 to 1.0 mcg/kg over 10 min. [8]

1n 1991 Aho MS et al. used dexmedetomidine after laparoscopic tubal ligation and demonstrated the analgesic properties of dexmedetomidine using it as a single agent after minor surgery [17].

c) Use in regional anaesthesia and analgesia:

Dexmedetomidine shortens the onset of action of local anaesthetics with respect to sensory and motor blockade. It has been used intrathecally in various studies in a dose of 3 □g to 15 □g as an adjuvant to local anesthetics [18-26]. It provides good sedation and anxiolysis level during surgery as well as it prolongs the duration of analgesia into the post-operative period. Both intravenous and intrathecal dexmedetomidine cause dose dependent prolongation of duration of neuraxial anesthesia. R. Gupta et al found in their study that dexmedetomidine group had a significantly longer sensory and motor block time than patients in fentanyl group. The mean time of sensory regression to S1 was 476±23 min in group D and 187±12 min in group F (P<0.001). The regression time of motor block to reach modified Bromage 0 was 421±21 min in group D and 149±18 min in group F [21].

While clonidine has been in use as an adjuvant in regional anaesthesia and analgesia, there are only a few studies available on such effects of dexmedetomidine. Epidural/subarachnoid administrations of alpha 2- adrenergic agonists produce analgesia partly by causing spinal acetylcholine and nitric oxide (NO) release since clonidine-induced analgesia is enhanced by subarachnoid neostigmine and inhibited by N-methyl-L arginine (NMLA), a blocker of NO synthesis [27]. Bouaziz H et al [28] in a study administered clonidine and dexmedetomidine in subarachnoid space to ewes, and found that both clonidine and dexmedetomidine produced dose-dependent analgesia with similar potency. Clonidine was potentiated more than dexmedetomidine by neostigmine pretreatment and NMLA did not affect the dexmedetomidine-induced analgesia.

They concluded that it may reflect the lower intrinsic efficacy of clonidine and that analgesia from dexmedetomidine is less dependent on acetylcholine-NO mechanisms than clonidine. In another study, Correa Sales C et al [29] and Fairbanke CA et al [30] concluded that dexmedetomidine produces its analgesic effect by inhibiting the release of C-fibres transmitters and by hyperpolarization of post-synaptic dorsal horn neurons.

d) Use in critical care

Dexmedetomidine has been used in the intensive care for its sedative, anxiolytic, and analgesic properties and does not produce respiratory depression due to its non-opioid mechanism of analgesia. For adult patients, dexmedetomidine is

generally initiated with a loading infusion of 1 Micro g/kg over 10 minutes, followed by a maintenance infusion of 0.2 to 0.7 Micro g /Kg/hr. The bolus dose is not used as it can cause paradoxical increases in blood pressure. Dexmedetomidine produces arousable sedation i.e patient respond quickly and arouse easily when stimulated from sedation and quickly return to their sleep-like state. This property is quite useful in Intensive care units. It is not necessary to stop dexmedetomidine prior to extubation. This sedative and anxiolytic effect of Dexmedetomidine has been continuously utilized in intubated patients in I.C.U prior to extubation, during extubation, and postextubation[10].

e) In Monitored Anesthesia Care:

It can be used in monitored anesthesia care as it causes arousable sedation with ease of orientation, anxiolysis, mild analgesia, lack of respiratory depression and provides hemodynamic stability. It can be used for a broad range of procedures including fiberoptic bronchoscopy, dental procedures, ophthalmological procedures, neurosurgery etc [10]. Its use in monitored anesthesia care has allowed the anesthetist to deliver better anesthesia care with superior patient satisfaction, less opioid requirements, and less respiratory depression as compared with other agents. Candiotti KA et al [31] conducted a study in 2010 comparing dexmedetomidine with midazolam and fentanyl in monitored anesthesia care and found it to be a superior agent as compared to others in MAC care. Dexmedetomidine was well tolerated in a variety of age groups and populations for a broad range of surgical and diagnostic procedures. In addition to its analgesic and sedative properties, its sympatholytic effects attenuate the stress response to surgery. This makes it to have an edge over other agents like midazolam used in MAC care.

f) As a Sole Agent for Total Intravenous Anesthesia (TIVA).

Infusion in increasing doses (up to10 µg/kg/hr) until general anesthesia is attained. This new off-label use of dexmedetomidine is based on its known properties to provide sedation, analgesia while avoiding respiratory depression.

g) Some Special Situations

In sleep studies

Dexmedetomidine have less effect on upper airway tone and airway collapsibility as compared with other sedative agents. It possesses properties that mimic non-REM sleep, without significant respiratory depression or airway obstruction. These properties make dexmedetomidine an attractive agent for MRI sleep study evaluation [32].

Paediatric

There is not much data available in literature for its use in paediatruc patients. Pediatric experiences are in the form of small studies and case reports. However it is being used off label as an adjunctive agent for sedation and analgesia in paediatric patients in the critical care unit and for sedation during non-invasive procedures in radiology.It is being used in lower doses ranging from 0.3 to 0.5 µg/kg loading over 10 min in pediatric patients for sedation and anxiolysis [33].

Awake Fibreoptic Intubation

Awake fibreoptic intubation has now become a technique of choice in anticipated difficult airway. But it requires optimum patient preparation which includes obtundation of airway reflexes, adequate sedation, anxiolysis along with preservation of a patent airway and adequate ventilation. Dexmedetomidine presents as an near ideal agent for conscious sedation, which will ensure spontaneous ventilation with a patent airway, adequate cooperation, smooth intubating conditions and stable hemodynamics without respiratory depression. Studies have shown that dexmedetomidine (1 µg/kg over 10 min) is more effective than fentanyl in producing better

intubation conditions, sedation along with hemodynamic stability and less desaturation during awake fibreoptic [34].

Obstructive Sleep Apnea And Morbid Obesity

Obstructive sleep apnea (OSA) is a syndrome characterized by periodic, partial or complete upper airway obstruction resulting in the disruption of sleep and hypoxemia with potentially serious physiologic consequences. One of the major limitation in anesthetic management of these patients is the use of sedative agents which aggravates the airway obstruction and respiratory depression in these patients. Dexmedetomidine, with beneficial analgesic and sedative properties and limited respiratory depressant side effects, offers an additive advantage over other sedative agents. Thus it may be useful in the preoperative as well as postoperative period for patients with OSA who are susceptible to opioid-induced respiratory depression and undergoing surgical procedures that are associated with significant postoperative pain. Also due to minimal effect on sleep pattern and airway dynamics , it is preffered by many clinicians as sedative in sleep studies for diagnosing OSA.

h) Miscellaneous Uses

It has been reported to preserve renal function before and after anaesthesia for coronary artery surgery.

Dexmedetomidine administered intravenously at the end of surgery attenuated the increase in oxygen consumption and CO2 production due to shivering. The reduction in shivering may be particularly useful in patients with ischaemic heart disease.

Combination Regimens

Although dexmedetomidine can be used as a sole agent for sedation and anxiolysis but most clinicians prefer its combination with other agents like ketamine, propofol, midazolam, fentanyl etc. These combinations not only able to limit the dose of either drugs, but are also associated with lesser side effects.

Various regimens used are-

1. Dexmedetomidine (1 µg/kg) and ketamine (1 mg/kg), to initiate sedation. This can then be followed by a dexmedetomidine infusion (0.5 µg/kg/hr) with supplemental bolus doses of ketamine (0.5 mg/kg) as needed.
2. Dexmedetomidine 1 µg/kg and propofol 1.0 to 2.0 mg/kg bolus to initiate sedation, then an infusion of dexmedetomidine 0.5 □g/kg/h and propofol 2 to 3 mg/kg/h for maintenance.
3. Dexmedetomidine (1 µg/kg) and fentanyl (1-2 µg/kg), to initiate sedation. This can then be followed by a dexmedetomidine infusion (0.5 µg/kg/hr).

Adverse Effects, Contraindications and Interactions

The common adverse effects of dexmedetomidine include hypotension, hypertension, nausea, bradycardia, atrial fibrillation, hypoxia and various atrioventricular blocks [35]. Most of these adverse effects occur during or briefly after bolus dose of the drug. Various studies have shown that omitting or reducing the loading dose can reduce adverse effects[3,10,35].

Anaesthetics/ sedatives/ hypnotics/ opioids have possible pharmacodynamic interactions, when co-administered with dexmedetomidine. It may potentiate their effects so a reduction in dosage of concomitant anaesthetic agent may be required[3,9]. Clinical events of bradycardia or hypotension may be potentiated when dexmedetomidine is used concurrently with propofol or midazolam.

It is contraindicated in patients with a known hypersensitivity to dexmedetomidine. It should be avoided in patients on drugs which cause bradycardia like beta-blockers and digoxin. Caution should be taken in patients with

conduction problems, with reduced ventricular functions (ejection fraction<30 %), hypovolemic or hypotensive patients, patients of diabetes, chronic hypertension and elderly[10].

Dexmedetomidine crosses the placenta and its safety is not established in pregnancy. It should be used during the pregnancy only when the potential benefits exceeds the potential risk to fetus[3].

There is not sufficient human data proving its safety and efficacy in children less than 18 years and in lactating mothers hence caution should be exercised while using it in these patients.

A higher incidence of bradycardia and hypotension was observed in older patients following administration of dexmedetomidine hence dose reduction needed in patients over 65 yrs.

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PREGNANCY INDUCED HYPERTENSION DONE UNDER SUBARACHNOID BLOCK: A CASE REPORT

Manoj Kumar Chaurasia[1], Keshav Govind Rao[2], Aparna Shukla[3]

ABSTRACT

Pregnancy induced hypertension is a hypertensive disorder which occurs in 5% to 7% of all pregnancies. These parturient present to the labour and delivery unit ranging from gestational hypertension to HELLP syndrome. It is essential to understand the various clinical conditions that may mimic preeclampsia and the urgency of caesarean delivery, which may improve perinatal outcome. The administration of general anaesthesia (GA) increases morbidity and mortality in both mother and baby. The provision of regional anaesthesia when possible maintains uteroplacental blood flow, avoids the complications with GA, improves maternal and neonatal outcome. The use of ultrasound may increase the success rate. This review emphasizes on the regional anaesthetic considerations when such parturients present to the labour and delivery unit.

Keywords: Anaesthesia;hypertension; pregnancy; regional

INTRODUCTION

Preeclampsia is a hypertensive disorder of gestation, complicating 5% to 7% of all pregnancies. It is characterized by hypertension (≥140/90 mmHg) and proteinuria that develops after 20 weeks of gestation that usually resolves within 48 h of fetal delivery. It can progress to a severe form in 25% of parturients when it is undiagnosed or untreated.[1] It increases both maternal and fetal morbidity with the occurrence of eclampsia in 0.04-0.05% of the affected parturients[2,3] with an estimated annual mortality rate of 50,000 parturients with preeclampsia world-wide.[4] We present a case of patient with 37 weeks pregnant female, second gravid, Para 1+0 diagnosed as pre-eclamsia booked for emergency caesarean section.

CASE REPORT

A 35-year-old, unbooked, gravida 2 Para 1+0 female (uneducated) was unbooked for caesarean section at 37 weeks for severe pre-eclampsia. Her blood pressure was 200/110 mmHg in supine position with heart rate of 89 beats/minute. Patient went into spontaneous labour on the same day and developed cord prolapse for which she was booked for emergency caesarean section. On examination, she was in distress due to pain with no anasarca, sacral oedema or bilateral pitting pedal oedema. The weight was 70 kg, pulse rate 100 beats per minute, and blood pressure 200/100 mm Hg patient was on 10 mg

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hydralazine. Auscultation of the chest was vesicular breath sounds. She was pale, anicteric. The temporo-mandibular joint was mobile with mallampati grade II. Packed cell volume was 34%. An assessment of ASA IIIE with severe preeclampsia was made. The patient was counselled for surgery and informed consent obtained. Spinal anaesthesia was administered after several attempts with 2 ml of hyperbaric bupivacaine at L4, L5 interspace. The height of block was T5. She was given oxygen by facemask. During surgery, a male baby was extracted birth weight 2.1 kg, Apgar score. Blood pressure at the end of surgery was180/90 mmHg.
Parturients with pregnancy induced hypertension may present to the labour and delivery unit with or without a prior diagnosis of preeclampsia and may pose a significant anaesthetic challenge. The administration of general anaesthesia (GA) in such high risk parturients may cause exaggerated cardiovascular response to intubation leading to cerebral haemorrhage and edema, cardiovascular decompensation causing pulmonary edema; thereby increasing morbidity and mortality in both mother and child.[5,6] Similarly, an exaggerated pressor response to intubation may increase the maternal plasma catecholamine concentration, which in turn impairs the uteroplacental blood flow.[7-9]

The administration of regional anaesthesia (RA) not only avoids the maternal complications with GA like difficult intubation, vasopressor response to intubation, but also improves uteroplacental blood flow and neonatal outcome. This emphasizes on the regional anaesthetic considerations in such parturients presenting to the labour and delivery unit. Electronic search strategies included searching the databases Ovid MEDLINE, Ovid EMBASE (both until Jan 2013) and the Cochrane Library using the key words pregnancy, preeclampsia, eclampsia, regional, anaesthesia and neuraxial.

Spinal anaesthesia

Spinal anaesthesia is a generally preferred anaesthetic technique as it is simple to perform; it provides rapid onset and a dense block. It also provides excellent post-operative analgesia when intrathecal opioids are used.[10,11,12] It has no effect on Apgar scores and umbilical artery pH in preeclampsia as along as the systolic blood pressure is maintained greater than 80% or more of the baseline.[13] The incidence of spinal induced hypotension and the vasopressor requirement were found to be two times lower in preeclamptic parturients when compared with normal parturients undergoing CS delivery.[14,15] The increased production of circulating factors with potent pressor effect and the increased sensitivity to vasopressor drugs in preeclampsia along with the use of hyperbaric bupivacaine (8-12 mg) with opioids could decrease the spinal induced hypotension in preeclamptic parturients.[14] Cardiac output monitoring after spinal anesthesia has shown that neither spinal anesthesia nor the use of phenylephrine to treat hypotension reduce cardiac output during CS delivery, further supporting its safety in preeclamptic parturients.[16] Continuous spinal anesthesia offers the flexibility of titration of local anesthetic agents in small aliquots; thus, graded sympathetic block could be achieved with a lower degree of sympathectomy in these parturients.[17] However, the higher rate of infection, injury to nerve roots, postdural puncture headache and technical difficulty are potential pitfalls and this technique is not frequently used.[18]

Conclusions

Parturients with mild preeclampsia may safely undergo regional anesthetic procedures for labor analgesia and CS delivery. A thorough evaluation to detect underlying coagulopathy or thrombocytopenia is essential prior to considering regional anesthetic procedures in severe preeclamptic parturients. It may be safer to consider non-neuraxial techniques in eclamptic parturients with or without end organ damage.

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INTUBATION IN LATERAL POSITION: A NECESSARY SKILL FOR ANESTHETISTS?

Anju Gupta[1], Nishkarsh Gupta[2]

We as anesthesiologist may be faced with situations in which supine position may be difficult while intubation like large Meningo mylocoele, trauma victims, large tumor in back etc.[2]

We recently were faced with a similar scenario. We had a 60 year old male with large friable soft tissue tumor of the back. (Fig 1) Attempting

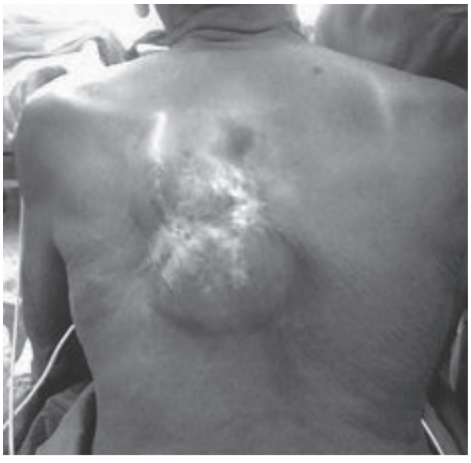

Figure 1: A large soft tissue tumour of back

intubation in supine position may have put excessive pressure on the back resulting in rupture of the soft tissue tumor. So, it was decided to do intubation in left lateral position under anaesthesia. The patient was intubated with size 8 flexometallic tube in first attempt and then made prone for surgery. The surgery lasted for 80 minutes, was uneventful and patient was extubated at the end

Intubation in lateral position may be helpful as a rescue measure when the airway is lost during the surgery in the lateral position. We as anesthesiologists may not be accustomed to lateral position. Thus, one should practice this in elective settings on regular basis before one is able to perform it in emergency scenario.

We believe that lateral positioning is one of important skills for airway management. We anesthesiologist should include it in the curriculum of young postgraduates and residents so that they can safely do intubation in the lateral position.

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THE EFFECT OF PREMEDICATION OF INJECTION DEXMEDETOMIDINE HCL ON HEMODYNAMIC STRESS RESPONSE DUE TO LARYNGOSCOPY AND INTUBATION IN PATIENT UNDERGOING CRANIOTOMY.

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ABSTRACT

Background and objectives:

Induction of anesthesia and tracheal intubation may cause profound alteration of hemodynamic state of patients. This reaction is not prevented by regular pre-medication. Objective of this study is to compare effects of Dexmedetomidine and Fentanyl in attenuation of this pressure response.

METHODS

Fifty ASA I and II status normotensive patients scheduled for elective craniotomy were selected randomly and divided into two groups of 25 each. Group X received 20 ml of normal saline 10 min before induction and group D received Dexmedetomidine 1 mcg/kg infusion 10 min before intubation. Induction of anesthesia was carried out with thiopentone sodium, fentanyl and vecuronium. Hemodynamic variables were defined as follows: before pre-medication, before induction, at induction, 0, 1, 3 ,and 5 minutes after intubation.

RESULTS

In both groups the maximal increase in heart rate and blood pressure occurred after tracheal intubation (0 min) when compared to baseline blood pressure. The increase in heart rate after intubation was 8% in group D and 22% in group X (p=0.00). Similarly significantly increase in systolic blood pressure was observed in group D which was 9% and in group X was 41%.(p=0.00).while increase in diastolic blood pressure was 11% and 27% in group D and group X respectively.(p=0.001).

Conclusion

It is concluded that pre-treatment with Dexmedetomidine is safe and effective for attenuating the sympathetic response to laryngoscopy and intubation.

Keywords: Pressure response, Dexmedetomidine, Fentanyl, craniotomy.

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Introduction

Dexmedetomidine, is an α_2-adrenoreceptor agonist with high selectivity for α_2 compared with $\alpha_1$adrenergic receptors (selectivity ratio 1620:1 compared with 220:1 for clonidine).[1]

It causes reduction in arterial blood pressure and heart rate associated with a decrease in serum noradrenalin concentrations.[2] Laryngoscopy and endotracheal intubation are associated with reflex sympathetic stimulation resulting in tachycardia and hypertension, arrhythmia. It is known as pressure response to laryngoscopy and intubation.[3,4] Hypertension, tachycardia and arrhythmia caused by endotracheal intubation can be deleterious in patients with hypertension [5] myocardial insufficiency [6] and cerebrovascular diseases .[7]

The goals of neuroanesthesia are the appropriate surgical conditions with preservation of autoregulation of the cerebral circulation. Fast recovery from neuroanesthesia allows early neurological examination.[8] When fentanyl is used in repeated doses it causes prolonged respiratory depression after extubation.[9] Dexmedetomidine has recently been used in neuroanesthesia for providing hemodynamic stability.[10]

Methods

After obtaining the ethical committee approval, 50 ASA Grade I&II patients, aged 25-50 years, undergoing craniotomy were enrolled in the study. Patients with cardiovascular disease, epilepsy, hypertension, chronic pulmonary disease, child-bearing potential, taking any antipsychotic medications or having history of allergy to any drug and in whom intubation attempts lasted longer than 25 seconds were excluded from the study. Patients were divided into two groups, group-D and group-X. In pre-operative room, a good intravenous access was secured for drug and continuous administration of ringer lactate and baseline parameter were observed and recorded, which included heart rate systolic and diastolic blood pressure,ECG,SPO_2.Group -D received Dexmedetomidine 1mcg/kg over 10min period 10 minutes before induction. Before induction of anesthesia the state of consciousness and sedative effects were observed. At the time of induction patients of both groups received inj. Glycopyrrolate 3 mcg/kg IV. Group D patients received inj. Fentanyl 2mcg/kg and group X patient received inj. Fentanyl 4 mcg/kg IV 3 minutes before induction. Induction of the anesthesia was carried out with thiopentone sodium in a dose sufficient to abolish the eyelash reflex followed by 0.1mg/kg of vecuronium bromide to provide neuromuscular blockadge. The lungs were ventilated with100%oxygen for the next 3 minutes.Then after sevoflurane concentration 2% was started. All intubations were performed by same anesthesiologist. Hemodynamic variables were recorded again. Times for hemodynamic measurement were defined as follows: Before pre-medication, before induction, at induction,0min,1min,3min,5min after intubation.

Statistical analysis

In our study a power of study is 80% at 5%significance level. Independent t-test was used to compare the study group and control group. The results were expressed as Mean±SD. $p<0.05$ was regarded as statistically significant<0.001 was taken as highly significant. Statistical analysis was conducted with SPSS (version17.0). (SPSS Inc. Chicago, IL<USA).

Results

All patients were included in statistical analysis. Table 1 shows the clinical and demographical data of the patients included in our study. The two groups were comparable in patients characteristics with respect to age, gender. ASA physical status and mean weight. Before induction of anesthesia heart rate, blood pressure and SpO2 values were similar between the groups. About10 minutes after receiving drug all

patients were drowsy but arousable. (Ramsay Sedation score-2). Sedative effects were seen without any significant fall in SpO2.In-group D patients receiving Dexmedetomidine loading infusion, a fall in heart rate and blood pressure was observed, which was not more than5%of the baseline. In both group, the maximal increase in heart rate and blood pressure occurred after tracheal intubation (0min) when compared to baseline blood pressure. The increase in heart rate after intubation was 8% in group D and 22%in group X. (P=0.001). Similarly significantly increase in systolic blood pressure was observed in group D which was 9% and ina group X it was 41% (p=0.00),while increase in diastolic pressure was 11%and 27%in group D and group X respectively (P<0.001) .(figure-1) Mean heart rate, systolic blood pressure and diastolic blood pressure after 1,3 and 5 minutes of intubation again returned to lower values ,but on comparison of rate of decrease and stabilization of hemodynamic parameters, it was highly significant in group D (p<0.001).Thereafter, till the completion of surgery ,no significant difference was noted in this respective parameters.

Discussion

Laryngoscopy and intubation result in increase in all the hemodynamic parameters like heart rate, systolic, diastolic and mean blood pressure. Numerous drugs and their combinations have been tried in the past and studies have highlighted the use of this drugs in varying doses for suppression of stress response but not without the significant incidence of quite a few side effects, especially with higher doses of opioids.[11,12]

Dexmedetomidine is a potent, highly selective α 2 adrenoceptor agonist with sedative, analgesic, anxiolytic, sympatholytic and opioid sparing properties. It provides unique type of sedation, conscious sedation in which patient appear to be sleepy but are easily aroused, cooperative and communicative when stimulated. [13]

We started Dexmedetomidine infusion 10 minutes before induction. It was observed that patients were markedly sedated but all patients were easily arousable on commands. We did not found significant fall in SPO_2. P.E. Tanskan and others observed in their study that Dexmedetomidine

Table-1 Demographic data

	Group D	Group X
Age	33.4(12.4)	38.6(11.9)
Gender	18/7	17/8
Weight	56.4	53.2
ASAI/II	19/6	18/7

Table 2 Ramsay Sedation Score

RAMSAY SEDATION SCORE	
1	Patient is anxious and agitated or restless, or both
2	Patient is cooperative, oriented and tranquil
3	Patient respond to command only
4	Patient exhibits brisk response to light glabellar tap or loud auditory stimulus
5	Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus
6	Patient exhibits no response

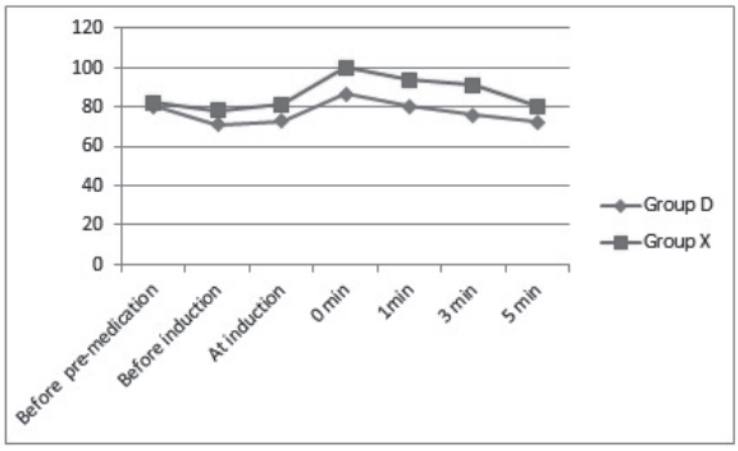

Figure 1 comparison of heart rate between group D and group X

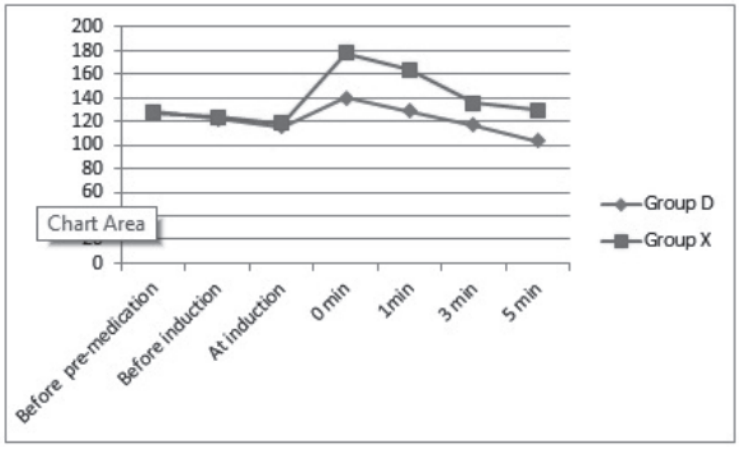

Figure 2 Comparison of systolic blood pressure between group D and group x

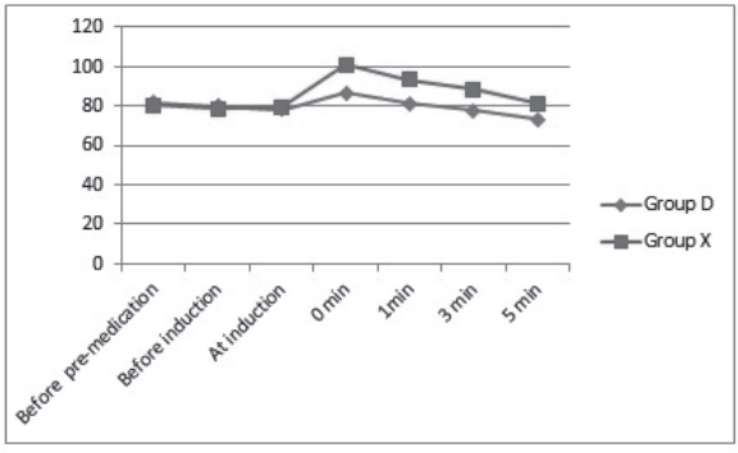

Figure 3 Comparison of diastolic blood pressure between group D and group X

causes markedly sedation without any significant increases in arterial CO_2 partial pressure. So Dexmedetomidine can be used in pts with mass occupying lesions without increase in the risk of increased ICP by hypoventilation.[8]

In the study conducted by OSHMAN and others found no significant difference in SPO_2 before and after Dexmedetomidine infusion. They also observed in their result that there was no significant difference between Dexmedetomidine and Fentanyl groups with regards to pao2 and paco2 values after extubation.[14] With rapid speed of Dexmedetomidine infusion there is higher incidence of side effects such as apnea and irregular ventilation. It is due to increased central sedation rather than direct respiratory depressant effects.[15,16] Dexmedetomidine modulates the response to stress by reducing the neurohumoral response, with decreased serum levels of adrenaline and noradrenalin, as well as the activity of the sympathetic nervous system. However, if it is used for less than 24 hours, it does not seem to reduce significantly the inflammatory response, demonstrated by evaluating adrenocortical activity, cortisol production, and serum levels of interleukin-6 (IL-6).[17]

For intracranial surgeries, the intense surgical stimulation associated with craniotomy frequently causes sympathetic activation and changes in BP, CBF, and ICP.[18] The vascular response can cause an increase in ICP and a reduction in cerebral perfusion pressure. So, prevention and hemodynamic control in response to nociceptive stimuli are extremely important to preserve brain homeostasis in neurosurgical patients.[19] The objective of this study was to review some of the pharmacokinetic and pharmacodynamic characteristics of Dexmedetomidine that are responsible for its applicability in specific situations and types of surgeries, especially neurosurgeries. It does not increase ICP, reduces CBF secondary to cerebral vasoconstriction, maintains hemodynamic stability, and reduces the need for other anesthetics, especially opioid. Besides, Dexmedetomidine can provide sedation without respiratory depression, and allows the fast arousal and neurological evaluation. For all these reasons, Dexmedetomidine is an interesting and promising drug to be used in Anesthesiology, especially in neurosurgeries.[8]

Cerebral vasodilatation induced by isoflurane or sevoflurane is decreased by the prior administration of Dexmedetomidine. Thus, the use of α_2-adrenergic agonists could be useful as adjuvant in inhalational anesthesia for neurosurgeries, in situations where an increase in CBF can be detrimental.[20]

Yildiz M et al.investigated the effect of a single preinduction intravenous dose of Dexmedetomidine (1mcg/kg) on the cardiovascular response from laryngoscopy and endotracheal intubation,need for supplemental anesthetic agent and preoperative hemodynamic stability.Their results showed that preoperative administration of single dose of dexmedetomidine resulted in progressive increase in sedation, blunted the hemodynamic response during laryngoscopy and reduced opioid and anesthetic requirements.[21]

α_2 adrenergic agonists have properties that reduce sympatholytic, sedative, and anesthetic requirements as well as provide hemodynamic stabilization. It is also known that Dexmedetomidine exhibits an analgesic effect without inducing respiratory depression. Tanskanen et al. used Dexmedetomidine on the patients undergoing intracranial tumor surgery. They showed that Dexmedetomidine depresses tachycardia and the hypertensive response developing at intubation and at extubation better than placebo. In the same study, the authors reported that, during the intraoperative period, dexmedetomidine reduced SAP by 20% in comparison to control group levels.[8]

Bekker et al. reported that Dexmedetomidine administered during neuroanesthesia reduces the need for opioid, leads to fewer antihypertensive treatments and provides better hemodynamic stability during incision.[22]

Lawrence et al. Showed that Dexmedetomidine given before the induction as a single dose of 2 µg/kg IV controls the hemodynamic responses to tracheal intubation and extubation as well as HR changes during the intraoperative period.[9] In comparison to control treatment we think that the hemodynamic values remained more stable due to the depressed stress response. In our study, hypertension and tachycardia attacks were more effectively controlled in the Dexmedetomidine group, especially during the periods when the stress response is pronounced.

Taittonen et al. showed that, after premedication with Dexmedetomidine, SAP and DAP decreased by 11% and HR decreased by 18%.[24] In our study, we observed a decrease of 13% in SAP, 9% in DAP, and 9% in HR after the bolus infusion of Dexmedetomidine for 10 minutes. Although these changes represented statistically significant differences, each parameter remained at physiologically acceptable levels.

In our study, we found that hemodynamic values, as well as HR values one minute after the intubation, were lower in group D as compared to group F.

Hypotension and bradycardia are sometimes reported in connection with Dexmedetomidine. Aryan et al. encountered bradycardia in only one of the 39 patients who were infused with Dexmedetomidine and followed up in the intensive care unit; the infusion did not have to be stopped for this patient. Therefore, it was considered that the development of bradycardia did not represent a clinical problem.[25] In our study, we observed only one case of hypotension and one case of bradycardia in group D, and only one case of bradycardia in Group X . Neither hypotension nor bradycardia was observed in any patient during the postoperative period.

Conclusion

It is concluded that pretreatment with Dexmedetomidine at a dose of 1mcg/kg 10 minutes prior to induction of anesthesia is safe and effective for attenuating the sympathetic responses to laryngoscopy and intubation in patient with craniotomy.

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ANAESTHETIC CONSIDERATIONS DURING MANAGEMENT OF A PATIENT WITH MONGOMERY T-TUBE IN SITU

Gaurav Sharma[1], Shivangi Khanna[2], Usha Bafna[3], Pranav Jaitley[4], Divanshu Gupta[5], Manisha Saxena[6]

Introduction

Montgomery tubes are silicon T-tubes used for maintaining patency of the airway as well as a stent after laryngotracheal surgeries. It is a T shaped tube with three limbs, two intratracheal and one extratracheal.[1,2] The long, vertical limb of the tube is inserted into the trachea, while the narrower limb protrudes through the tracheostomy opening.[3,4] The proximal limb is smaller, while the distal limb is longer. The upper limb functions as a stent of the upper airway and the lower limb aids in ventilation. It is available in various sizes 4.5-16mm in diameter. Paediatric tubes are available in sizes from 4.5-8 mm in diameter. The major indications of its use are acute laryngotracheal injuries, to support intra-thoracic tracheal stenosis, to support a reconstructed trachea, tracheal stenosis, segmental resection and anastomosis of trachea and to maintain a cervical trachea that cannot be repaired.[5-7] The limitation of its use to only head and neck surgery specialising institutions and the complexity of usage makes it both intriguing and challenging for the anaesthesiologist.

The problems associated with the Montgomery tube are that the external limb of the tube does not accept a standard 15 mm connector, so the connection of anaesthesia circuit for delivery of anaesthetic gases is a problem. The upper end of the tube remains open, so adequate ventilation becomes a challenge due to leak of gases. Maintenance of depth of anaesthesia also becomes challenging due to continuous dilution of anaesthetic gases with air coming through the upper limb of the tube.

Case report

A 65 kg, 45 yr old male presented with complains of hoarseness of voice and difficulty breathing. He had a Montgomery tube inserted 2 months back after tracheal injury post RTA. He was posted for laryngoscopic assessment of chances of restoration of a physiological airway under general anaesthesia.

On pre-anaesthetic examination, the patient did not have any acute illness. He was ASA grade-I. On airway examination, he was MPG -2 with loose lower central incisors with thyromental distance of 6cm and an interincisor gap of 4.5 cm.

Pre medication was given Inj. Midazolam 1 mg and Inj Glycopyrrolate 0.2 mg prior to the surgery. Modification of bains circuit was done to overcome the aforementioned problems of Montgomery tube. One end of a Y connector(borrowed from the Jackson Rees

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circuit) was connected to the face mask and the other end was connected to one end of the Montgomery tube with the connector of a 7.0mm ET tube which was found to snugly fit the size 10 Montgomery tube in this patient. The third end of the Y piece was connected to the outer tubing of the bains circuit. The fresh gas flow was kept high to compensate for any leaks, the modified version of the circuit and to aid in carbon dioxide elimination. Any leaks from the open upper end of the tube were compensated by fresh gas from the face mask. Due to a compromised upper airway, induction of anaesthesia with the use of inhalation agents was thought appropriate. Inj. Fentanyl 80 mg I.V.was given. Induction was done using 5% Sevoflurane in 100% oxygen with spontaneous breathing. As soon as the patient was anaesthetised, the airway was handed over to the surgeons. The upper end of the tube was blocked with a gauze piece before starting the surgery to prevent any fresh gas leak from the open upper end of the tube. The outer end of the bains circuit was attached directly to the Montgomery tube. Maintenance of anaesthesia was done with 1.5% Sevoflurane in nitrous oxide and oxygen mixture(50:50), with the patient on assisted spontaneous ventilation. The laryngoscopic assessment was performed successfully, but it was decided to retain the T-tube.

After completion of the procedure, the upper end of the T-tube was opened and the patient was mask ventilated again. All anaesthetic gases were withdrawn and 100 % oxygen was administered. The patient was breathing spontaneously. After achievement of adequate tidal volume, the face mask was removed and he was observed for 5 minutes for maintenance of adequate ventilation through the T-tube.

The patient was shifted to the recovery room with a BP-136/96mm Hg, Pulse rate- 100bpm and a saturation of 98% on room air.

Discussion

The Montgomery T-tube was discovered in the mid 1960s by William Montgomery with an aim of tracheal stenting[8] as well as maintenance of airway patency. It has two intratracheal limbs with tapered ends to prevent injury to the tracheal wall. One extraluminal limb projects from the tracheostomy opening, which has a spigot to block the extraluminal limb when ventilation done through the upper end of the tube or during times of spontaneous ventilation.

The causes of tracheal stenosis include an external injury to the throat (RTA, suicidal or homicidal wounds), benign or malignant tumor compressing the trachea, polychondritis, sarcoidosis, papillomatosis,amyloidosis, Wegener's granulomatosis, a scar from the tip of an endotracheal tube or certain bacterial, fungal infections or even tuberculosis. It can also develop as an adverse effect of radiation therapy for treatment of head and neck tumors. The incidence of tracheal stenosis post intubation and tracheostomy ranges from 6-21% and 0.6-21% respectively.[9] The incidence of tracheal injury after penetrating neck injuries ranges from 3-6% and 0.4% after blunt trauma.[10]

Thus, a significant number of patients suffer tracheal injury and stenosis due to injury or disease. A small but significant fraction of these

undergo surgical treatment for the same, which is mostly insertion of a Montgomery T- tube after laryngotracheal repair or tracheal dilatation.

Consequently, it becomes imperative for the anaesthesiologist to be well versed with the functional aspects and practical problems faced while managing a patient with a Montgomery tube in situ. The major concerns of the anaesthesiologist while attending such a patient are as follows. Induction and maintenance of depth of anaesthesia is difficult due to loss of anaesthetic gases from the open upper end of the tube. There is also continuous dilution of gases from the air coming through the open upper end. Also, proper ventilation becomes a challenge due to leakage of oxygen from the upper end. The extraluminal end of the tube does not accept a universal 15 mm connector, so ventilation using a bains circuit is difficult. Also, due to variable sizes and thickness of the Montgomery tubes, it is difficult to predict the size of the 15 mm connector that fits that particular tube.

Many novel methods have been devised over the years to overcome the aforementioned problems. Hypoventilation due to leakage and dilution of anaesthetic gases from the upper end of MT tube was first recognised by William Montgomery.[1]He suggested the use of a Fogarty catheter to aid in ventilation. The catheter was passed from the extraluminal end of the tube upwards into the upper end of the MT tube and the balloon was inflated to block the tube. later a n appropriate sized ET tube was inserted adjacent to the catheter into the trachea for ventilation.

Guha et al[2]inserted a LMA and ventilated the patient through the LMA after occluding the extraluminal limb with the spigot. Uchiyama et al[3] also used the LMA to aid in ventilation, but they occluded the upper end of the LMA and conducted ventilation through the extraluminal limb. Wouter et al[4]inserted a mircolaryngeal tube through the Montgomery tube using awake fibreoptic bronchoscopy for ventilation. Supraglottic jet ventilation can also be used for ventilation through the T tube after occlusion of the extraluminal limb[6]. Sanjay et al[8] blocked the upper end of the T tube with a pharyngeal pack and subsequently ventilated the distal airways through the extraluminal limb. This technique had a disadvantage of leakage around the pack and loss of gases.

Induction and maintenance of anaesthesia remains a challenge while the Montgomery tube is in situ. Although IV induction and TIVA appears to be a more feasible alternative for anaesthetising these patients[11], inhalation induction remains the preferred method regarding patient safety with a compromised upper airway. The transient apnea caused by IV induction agents makes them unsuitable for use here. Also, respiratory paralysis caused by neuromuscular blockers can be hazardous in cases where ventilation and proper blockage of the upper end cannot be ensured. Spontaneous ventilation and inhalation induction provide better airway control.

For induction and ventilation, Guha et al[2]used a modified bains circuit with a Y connector to split the fresh gas flow into two parts, one going into the T-tube and the other into the face mask to compensate for the leak. Empirically, an additional fresh gas flow of 50ml/kg/min to the usual requirements of the bains circuit to compensate for the modified circuit.

As the patient was posted for airway assessment under direct laryngoscopy, insertion of an LMA or occlusion of the upper end with a gauze piece would have obscured the surgeon's view of the airway. Unavailability of a Fogarty catheter and the high resistance ventilation through the small sized ET tube made this technique unsuitable for our use. For adequate pre-medication with Inj. Midazolam and Inj. Fentanyl were used to

attenuate the responses to surgical stimuli and airway manipulation and to decrease the anaesthetic requirements[5]. Volatile anaesthetics were found most appropriate for induction and maintenance of anaesthesia. Thus, spontaneous ventilation with volatile anaesthetics is an acceptable method for anaesthesia in patients posted for short procedures like DL assessment.[7]

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REPLY….. HASAN M, AHMED SM, ALI S, ATHAR M. A UNUSUAL CASE OF HYPERCARBIA DUE TO INTRATRACHEAL RYLE'S TUBE INSERTION.

Rashid M Khan[1], Naresh Kaul[2], Partab Chand[3]

The case report by Hasan et al made interesting reading. The authors very rightly pointed out that each time a nasogastric tube is passed while endotracheal tube is in-situ and abdominal surgery is in progress, one should always confirm its gastric placement by aspiration of gastric contents and asking for its palpation in the stomach by the operating surgeon. In such a situation, we would like to suggest a triad of loss of tidal volume, detection of carbon dioxide from proximal end of the nasogastric tube and presence of humidification in the tube as a pointer to its misplacement in the trachea. When in doubt, always perform diagnostic laryngoscopy to confirm its inadvertent placement in the trachea. The authors used all of these diagnostic modalities except attempting to detect carbon dioxide from the proximal end of the nasogastric tube, albeit in the ICU.

Reference

Hasan M, Ahmed SM, Ali S, Athar M. A unusual case of hypercarbia due to intratracheal Ryle's tube insertion. AAAR 2015; 81: 2471-3.

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Acknowledgements

They should be brief and should include reference to the source of technical help, material support and financial assistance. Individuals named must approve their inclusion in the acknowledgements, before the paper is submitted.

References

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Case Report	2
Brief Report	4
Technical Communication	5
Review Article	10
Medical Intelligence Article	6
Special Article	6
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A Completed checklist must accompany each manuscript submitted to Asian Archives of Anaesthesiology and Resuscitation.

Checklist for submitting the manuscript

General

1. Two complete sets of the manuscripts (including tables) are submitted.

2. A floppy disk or CD is submitted with two files : the complete manuscript and a separate file containing only the title page, abstract, and references.
3. Manuscript is typed double-spaced, with ample, left ,justified, margins.
4. Pages are numbered consecutively, starting with the title page.

Title Page

1. On the first page are typed the title, author name(s) and major degree(s), and affiliation(s).

2. The name, address, telephone and FAX numbers, and E-mail address of the corresponding author are to be given.

3. The manuscript title is no longer than 100 characters (letters and spaces) and does not contain any abbreviations.

4. A short title (no more than 30 characters) is provided at the bottom of the page for use as a running foot.

Summary

* An abstract is provided. For all kind of articles, this abstract is limited to 200-250 words.

References

1. References correspond to the specifications of the Uniform Requirements for Manuscripts Submitted to Biomedical Journals" promulgated by the International Committee of Medical Journal Editors.

2. References are identified in the text by superscript figures, eg., Miller.

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5. Unpublished data, personal communications, submitted manuscripts, statistical programs, papers presented at meetings, and nonpeer review publications are not listed in the bibliography.

6. The bibliography is typed doublespaced.

7. Abbreviations of Journal titles conform to those used in Index Medicus, National Library of Medicine.

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1. Each table is typed on a separate sheet of paper with its title.

2. Tables are numbered with Arabic numerals.

3. Each table contains all necessary information in order that it may stand alone, independent of the text.

4. No table contains data that could be included in the text in several sentences.

5. Vertical lines are not used.

6. Irrelevant and extra tables must not be included

Figures

1. Each figure is cited in the text.

2. Figures have been prepared with the journal column size in mind.

3. Letters and identifying marks are clear and sharp, and the critical areas of radiographs and photomicrographs are identified.

4. Legends and explanatory material appear in the accompanying caption and not no the figure itself.

5. Legends are typed together on one page. Legends for photomicrographs include information regarding stain and magnification.

BOOK REVIEW

ANAESTHESIA AND ALLIED SCIENCES FOR PARAMEDICS,2013,first edition

Editor-Dr Manpreet Singh, MD, FCCP, FIMSA, FACEE,MAMS

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The editor of this book, Dr Manpreet Singh is involved in teaching the students of BSc Medical Technology and Operation Theatre, Trauma Technician courses, MBBS and MD (Anaesthesiology and Intensive care) at Government Medical College, Chandigarh ,India.

'ANAESTHESIA AND ALLIED SCIENCES FOR PARAMEDICS' is first book of its kind and comprises of six sections. All sections are colour coded for easy identification.

Section one consists of anatomy, physiology and clinical biochemistry for paramedics. Details of all muscles, bones and joints along with their actions, nerves and vessels are compiled in a tabular form so that it can be easily learnt and recapitulated by students. Essential physiology and clinical biochemistry are concised subsections of this section.

Second section provides every detail about anaesthesia and its various sub-specialities. This section has 40 chapters i.e from history of anaesthesia till modular operation theatre suit details. Apart from basics of anaesthesia and sub-specialities of anaesthesia, it highlights operation theatre suit, air-conditioning of Operation theatre and ICU, sterilization, pain management, dialysis room management and transportation of patients and anaesthesiologists.

Third section, 'Pharmacology in Anaesthesia' describes intricacies of all anaesthetic drugs and emergency drugs. These drugs are described in tabular forms in easy language. This section will help the students to explain the drugs that are asked in table viva during examination.

Section four covers all the anaesthesia instruments. These includes anaesthesia machine , automated external defibrillator, sutures, vaporizers and all airway management equipments. The details of instruments will be very beneficial for the students during training periods, examination, table viva and day-to-day practice.

Fifth section provides knowledge of 32 unique topics of modern anaesthetic practices that requires utmost attention. It highlights brief knowledge about clinical audit, hospital waste management, ECG, EMG, cardiopulmonary resuscitation 2010 guidelines, intensive care topics and physics in anaesthesia.

The final section 6, highlights all the scoring systems, algorithms and grading in anaesthesia. The students will be elated to read this section as they will feel comfortable to find all gradings at one place.

This book will be extremely useful to all residents of anaesthesiology and paramedics i.e MSc. Operation Theatre, BSc Medical Technology students, operation theatre technicians nurses, physiotherapists and trauma technicians. I assure that the student will not move away from this comprehensive book that will be useful in all types of examinations, skill development and knowledge augmentation.

The book is a sincere tribute to my father who had this dream for me. I am fortunate enough to have blessings from Almighty, my teachers and parents. All the contributors of this book have provided me a great support and deserve my heartfelt gratitude.

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