Upper Airway Obstruction During Sedation

Essay

Submitted for the partial fulfillment of master degree in anesthesiology

By

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بسم الله االرحمن الرحيم

وأَنزَلَ اللَّهُ عَلَيْكَ الكِتَابَ والْحِكْمَةَ وعَلَّمَكَ مَا لَمْ تَكُن تَعْلَمُ وكَانَ فَضْلُ اللَّهِ عَلَيْكَ عَظِيماً

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List of Abbreviations

ABC	Airway, breathing, circulation.
ASA	American Society of Anesthesia.
BMI	Body mass index.
BVM	Bag valve mask.
Bi – PAP	Bilevel continuous positive airway pressure.
COPD	Chronic obstructive pulmonary disease.
CPAP	Continuous positive airway pressure.
CT	Computed tomography.
ECG	Electrocardiography.
EEG	Electroencephalography.
EET	Endotracheal tube.
FIO_2	Fraction of inspired oxygen.
FRC	Functional residual capacity.
GABA	Gamma amino butyric acid.

Hb – O_2	Oxy – hemoglobin.
LMA	Laryngeal mask airway.
MAC	Monitored Anesthesia care.
MRI	Magnetic resonance imaging.
nCPAP	Nasal Continuous airway pressure.
NPO	Nothing per osium.
NREM	Non-rapid eye movement sleep.
OSAS	Obstructive sleep apnea syndrome.
$PaCO_2$	Partial pressure of carbon dioxide.
P_{crit}	Critical closing pressure.
$P_{downstream}$	Downstream pressure.
Peep	Positive end expiratory pressure.
P_n	Nasal pressure.
PO_2	Partial pressure of oxygen.
P_{tr}	Tracheal pressure.
$P_{upstream}$	Upstream pressure.
$R_{downstream}$	Downstream resistance.

REM	Rapid eye movement sleep.
Rupstream	Upstream resistance.
SPO2	Oxygen saturation.
TTJV	Transtracheal Jet Ventilation.
UAO	Upper airway obstruction.
UAR	Upper airway resistance.

Introduction

Invasive diagnostic and minor surgical procedures outside the traditional operating room setting have increased in the last decade. As a consequence of this change and the increased awareness of the importance of providing analgesia and anxiolysis, the need for sedation for procedures in physician offices, dental offices ,subspeciality procedure suites, imaging facilities, emergency departments, and ambulatory surgery centers has also markedly increased ***(Law et al., 2003)*** .

Upper airway obstruction is common during sedation. Obstruction is caused by loss of muscle tone present in the awake state. Patients with tendency to upper airway obstruction during sleep are vulnerable during sedation and anesthesia. Loss of wakefulness is compounded by depression of the ability to arouse, so they can not respond adequately to asphyxia ***(Hillman et al., 2003)*** .

Identifying the patient at risk is vital. Previous anesthetic history and investigations of the upper airway are helpful, and a history of upper airway compromise during sleep (snoring, obstructive sleep apnea) should be sought. Beyond these, risk identification is essentially a search for factors that narrow the airway. These include obesity, maxillary hypoplasia, mandibular

retrusion, bulbar muscle weakness and specific obstructive lesions such as nasal obstruction or adenotonsillar hypertrophy. Such abnormalities not only increase vulnerability to upper airway obstruction during sleep or anesthesia, but also make intubation difficult ***(Hillman et al., 2003) .***

Appropriate drug selection for the intended procedure as well as the presence of an individual with the skills needed to rescue a patient from an adverse response is essential. Appropriate physiological monitoring and continuous observation by personnel not directly involved with the procedure allow for accurate and rapid diagnosis of complications and initiation of appropriate rescue interventions ***(Hoffman et al., 2002).***

Only recently quantitative methods were identified as being reliable in the evaluation of sedation induced upper airway obstruction. Methods used to evaluate sleep apnea & related breathing disorders are now being employed for study the effect of sedation and general anesthesia ***(Litman , 2005)*** .

Identification of risk and caution are keys to management, and the airway should be secured before sedation where doubt exists. if tracheal intubation is needed, spontaneous breathing until intubation is important principles . Every anesthesiologist should have in mind a plan for failed intubation or ,worse failed ventilation ***(Hillman et al., 2003)*** .

Clinical trials are needed to evaluate oral sedative drugs and combinations , as well as to develop discharge criteria with objective quantifiable measures of home readiness . Courses devoted to airway management should be developed for dentists who provide conscious sedation . Services state regulation of enteral administration of sedatives to achieve conscious sedation is needed to ensure safety ***(Dionne et al., 2006) .***

Aim of the work

The aim of the current work is to highlight how sedation can cause upper airway obstruction, which should be clearly differentiated from hypoventilation. It is reassuring that significant unpleasant events can be prevented by careful preoperative assessment, along with attentive intraoperative monitoring and postoperative care. And also-the aim of this review is to focus on upper airway collapse during sedation in relation to different drugs used in sedation in different clinical conditions with a close reference to patient related factors augmenting the problem.

Anatomy of the upper airway

Upper airway is a compartment that has two opening: the nose which leads to the nasopharynx and the mouth which leads to the oropharynx . These passages are separated anteriorly by the palate, but they join posteriorly in the pharynx ***(Morgan et al., 2006)***

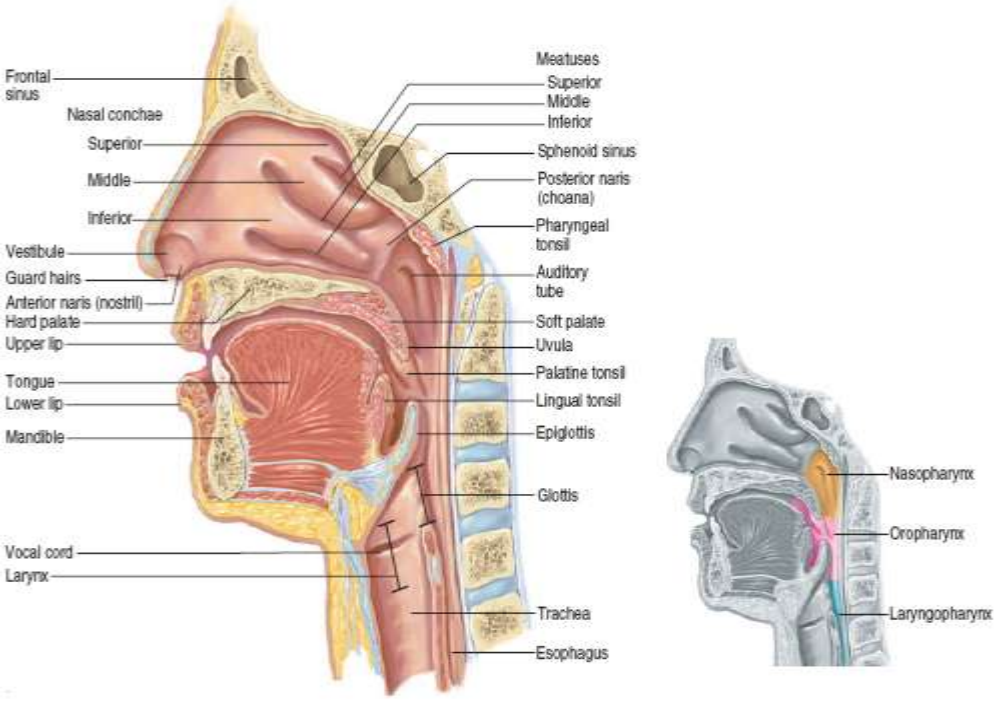

Fig (1): Anatomy of the Upper Respiratory Tract.

(Kavangh, 2008).

The nose:

The nose is divided anatomically into external nose and nasal cavity.

The external nose is formed by an upper framework of bone (made up of the nasal bones, the nasal part of frontal bones and the frontal processes of the maxilla),a series of cartilages in the lower part, and a small zone of fibro-fatty tissue that form the lateral margin of the nostril (the ala). The cartilage of the nasal septum comprises the central support of this framework.

The nasal cavity is subdivided by the nasal septum into two separate compartments that open to the exterior by the nares and into the nasopharynx by the posterior nasal apertures or choanae. Each side of the nose formed by a roof, a floor, medial wall and lateral wall. The roof first slopes upwards and backwards to form the bridges of the nose (the nasal and the frontal bones) then has a horizontal part (the cribriform plate of the ethmoid), and finally a downward sloping segment (the body of sphenoid). The floor is concave from side to side and formed by the palatine process of the maxilla and the horizontal plate of the palatine bone. The medial wall is the nasal septum which formed by septal cartilages, the perpendiclar plate of the ethmoid and the vomer. The lateral wall has a bony framework made up principally of the nasal aspect of the ethmoidal labyrinth above, the nasal surface of the maxilla