"EFFECT OF PTERYGOPALATINE FOSSA INJECTION WITH 2% LIGNOCAINE AND 1:80000 ADRENALINE IN MINIMISING BLEEDING DURING ENDOSCOPIC SINUS SURGERY"

By

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In partial fulfilment of the requirements for the degree of

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Under the Guidance of

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"EFFECT OF PTERYGOPALATINE FOSSA INJECTION WITH 2% LIGNOCAINE AND 1:80000 ADRENALINE IN MINIMISING BLEEDING DURING ENDOSCOPIC SINUS SURGERY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. CHANDRAKALA.S,** Associate Professor, Department of Otorhinolaryngology, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M.S. DEGREE IN OTORHINOLARYNGOLOGY**", the examination to be held in May 2017 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

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Dr. REJI MATHEW VARGEHSE

LIST OF ABBREVIATIONS

1.	FESS	Functional Endoscopic Sinus Surgery
2.	PPF	Pterygopalatine Fossa
3.	CRS	Chronic rhinosinusitis
4.	ARS	Acute rhinosinusitis
5.	CRSwNP	Chronic rhinosinusitis with nasal polyps
6.	CRSsNP	CRS without nasal polyps
7.	IPPV	Intermittent positive pressure ventilation
8.	MAP	Mean arterial pressure

ABSTRACT

Background: Functional endoscopic sinus surgery (FESS) is the most physiological and well accepted treatment for chronic sinus disease which is un-responsive to medical treatment[1]. Even with recent advances in technology, controlling the bleeding during endoscopic sinus surgery remains a challenge

Aims and objectives: To study the efficacy of pterygopalatine fossa block with 2% Lignocaine and 1:80000 adrenaline in minimizing bleeding during Endoscopic Sinus Surgery

Materials and methods : This study was done on patients with chronic rhinosinusitis and polyposis from December 2014 to June 2016. Patients were selected based on the inclusion and exclusion criteria and were randomly divided into two groups by a 6 block randomisation method. All patients underwent Functional endoscopic sinus surgery following infiltration of the test drug and placebo in the pterygopalatine fossa .Intra-operatively surgical field assessment was done every 15 minutes to assess the bleeding and was graded using Boezart and Vander Merve scale.

Results: Maximum incidence of disease was seen in the 5th decade. 59.4% were males and 40.6% were females. Ethmoidal polyposis was the most common diagnosis (50%), followed by Chronic rhinosinusitis (25%) and 6.25% had Chronic Rhinosinusitits along with DNS, and fungal sinusitis respectively. Majority of the patient had symptoms less than one year. The mean Blood loss grade on test side was

9.0 ± 3.5 and in control side was 10.9 ± 3.5. and the difference was statistically significant. The Median CT grade and the endoscopic grade of disease in test side and control side were almost similar. There was no significant difference in Median scores between the two groups. Non significant positive correlation was observed between CT scan grade and blood loss grade in both the test and the control side. Significant positive correlation was observed between Endoscopic grade and blood loss grade in the test and control side. No complications were encountered during the procedure.

Conclusion: Infiltration of the pterygopalatine fossa with 2% xylocaine and 1:80000 adrenaline helped in minimizing bleeding during the procedure providing the surgeon with better visualization of all anatomical landmarks and pathology.

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INTRODUCTION

INTRODUCTION

Functional endoscopic sinus surgery (FESS) is the most physiological and well accepted treatment for chronic sinus disease which is unresponsive to medical treatment[1]. Even with recent advances in technology, controlling the bleeding during endoscopic sinus surgery remains a challenge.

The surgical field in endoscopic sinus surgery is very narrow and is surrounded by vital structures such as orbit and brain. Even a small amount of bleed during surgery can soil the tip of the endoscope and obscure the field. Repeated soiling of the tip of the endoscope prolongs the procedure. Operating under such compromised conditions in addition to the narrow and delicate field increases the risk of injury to the adjacent vital structures. Also, if performed under poor visualisation, damage to the mucosa may further lead to post-operative synechiae formation. Hence, an optimal bloodless field is crucial for the surgeon[2].

Various techniques have been used to secure a dry operating field; among them are: Fowler's position, hypotensive agents like beta-adrenergic blockade, and preoperative steroids, topical vasoconstrictors. However, even with all these techniques, excessive bleeding still remains a problem during endoscopic sinus surgery [3, 4].

As the maxillary artery is the major source of supply to the nose, vasoconstriction of this artery can significantly reduce the blood supply to the nose, thus minimizing bleeding during the procedure[2]. This artery can be accessed via greater palatine fossa.

Very few studies have been listed in literature regarding this aspect, the results of which do not confirm the efficacy of infiltration of 2% lignocaine with 1:80000 Adrenaline in the pterygopalatine fossa on minimizing bleeding during endoscopic sinus surgery. Hence we intend to conduct the study to know the effect of pterygopalatine fossa injection with 2% lignocaine and 1:80000 adrenaline in minimizing the nasal bleed during FESS.

OBJECTIVES

OBJECTIVE OF THE STUDY

To study the efficacy of pterygopalatine fossa block with 2 % lignocaine and 1:80000 adrenaline in minimizing bleeding during Endoscopic sinus surgery

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

Throughout the history of medicine, numerous attempts have been made to illuminate and examine the inside of various hollow cavities in our body. The interior of nose and paranasal sinuses, with their narrow passage and fissures, bony walls places heavy demands on the design of instrumentation to be used for this purpose. This sowed the seed for development of nasal endoscopy[5].

In 1915 Killian published a review of the "History of endoscopy from the earliest times to Bozzini "in which he recorded all the attempts to view the upper airways prior to beginning of 19th century. Philip Bozzini in 1806 published an article describing the first "Light conductor, or description of a simple device and its use for illumination of the internal cavities and spaces of living animal body"[5].

Bozzini mentioned in 1806 that he was able to see some areas behind soft palate with the aid of his light conductor. In 1838 Baumes presented to medical society in Lyons a mirror the size of two franc pieces that could be used for the examination of choanae and the larynx[5].

In 1859 in Vienna, Czermak developed a technique similar to laryngoscopy of Turck, which allowed him to view the nasopharynx, the choanae and the posterior aspect of nose with the aid of small mirror. He called this procedure "Rhinoscopy"[5]. The second stage of cystoscopy began with the development of cystoscope by Nitz-Leiter in 1879. A year later Zauful modified the instrument for examining eustachian tube orifice. In 1902, Hirschmann and Valentin followed shortly by Reichert , in 1903 were able to introduce a modified cystoscope directly into the maxillary sinus through an enlarged dental alveolus[5].

During 1951-1956 Hopkins made fundamental improvements in the optics of endoscopy. These included a light source that was separate from the instrument, an excellent resolution with high contrast, a large vision in spite of the small diameter of the endoscopes and perfect fidelity of colour. The Hopkins rod rigid nasal endoscopes made it possible to examine in detail the clefts and recess of the nose. The ability to enter middle meatus of the nose enabled the inspection of anterior ethmoid sinuses and key area of infectious paranasal sinus disease. Today nasal endoscopic examination in combination with tomography allows the identification of small circumscribed changes in paranasal sinuses. These small changes are frequently of considerable pathophysiological significance[5].

Messerklinger was the first to develop and establish a systemic endoscopic diagnostic approach to the lateral wall of nose. His studies beginning in 1950 demonstrated that in most cases the frontal and maxillary sinuses are involved indirectly by primary disease that originates in narrow spaces of the lateral wall of nose and in the anterior ethmoid. This discovery led to the development of endoscopic diagnostic technique that focused on changes on the lateral wall of nose and identified and isolated changes, with the aid of rigid endoscopes and tomography of sinuses. Messerklinger observed that the eradication of primary anterior ethmoid disease by means of a circumscribed, limited endoscopic surgical procedure resulted in recovery of massive mucosal pathology in the adjacent large paranasal sinuses within a few weeks[5].

ANATOMY OF NOSE AND PARANASAL SINUSES

EMBRYOLOGY OF NOSE

Nasal cavity is first recognized in the 4th week as a olfactory or nasal placode. The placode sinks to form the olfactory pit. This then deepens to form the nasal sac. The maxillary process of the 1st arch grows anteriorly and medially to fuse with nasal fold and fronto-nasal process .This closes off the nasal pits to form the primitive nasal cavity[6].

Initially mouth and primitive nasal cavity are separated by bucconasal membrane. This thins as nasal sac extends posteriorly and eventually breaks down to form primitive choana. The floor anterior to the choana is formed from mesenchymal extensions of medial nasal folds to produce premaxilla which gives rise the upper lip, medial crus of lower lateral cartilages[6].

The maxillary process also grows ventrally form dorsal end of mandibular process to join the lateral nasal process around the nasomaxillary groove. The ectoderm in this region canalizes to from nasolacrimal duct[6]. The lateral nasal folds form the nasal bones, upper lateral cartilages, and lateral crus of lower lateral cartilage. Palate begins to form anteriorly with the fusion of the maxillary and fronto nasal process. Nasal septum is formed from the midline ridge developing from the posterior edge of fronto nasal process in roof of oral cavity and extends posterior to the opening of Rakthe's pouch[6].

The palatal process from lateral maxillary mesoderm grows medially towards septum and towards each other. Fusion is complete, except a midline dehiscence which forms site of future incisive canal. It separates the nasal cavity and nasopharynx from oral cavity as they also form soft palate and uvula[6].

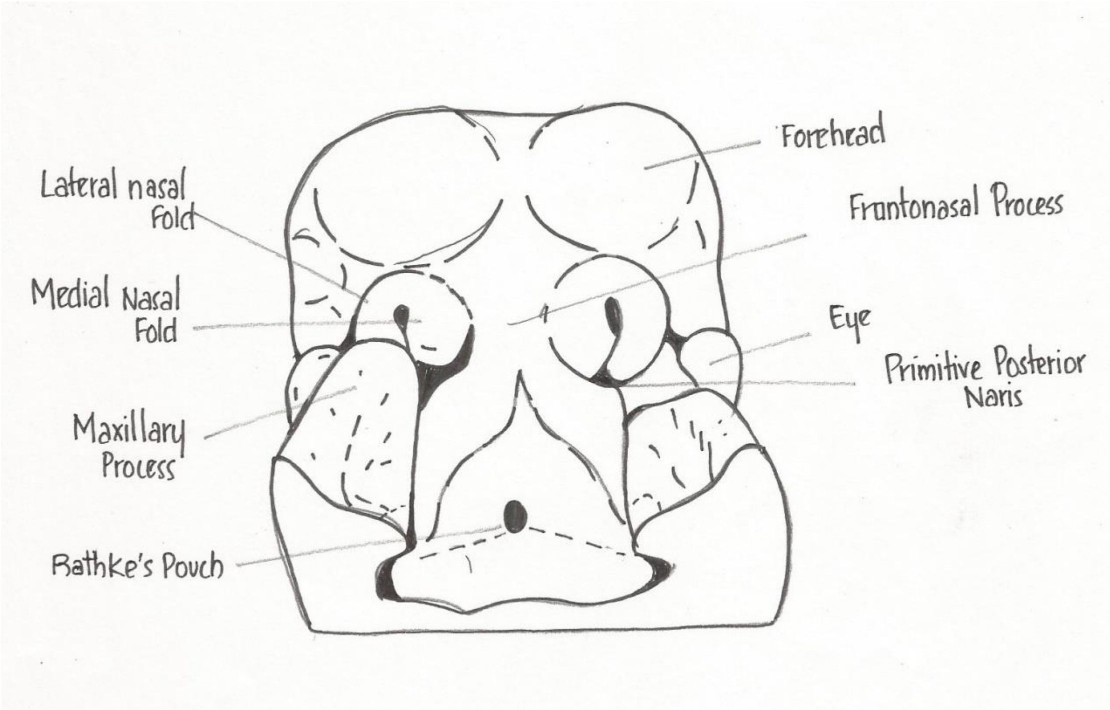

Figure 1: Development of nose

ANATOMY OF NOSE

EXTERNAL NOSE

The external nose is shaped like a triangular pyramid with its root above and base directed downwards. The base is perforated by two nostrils or anterior nares, separated by median septum. Each side of external nose ends in a rounded eminence, the ala nasi , which forms the outer boundary of the nostril. The nasal bones form the bridge, and each is united above with frontal bone and laterally to frontal process of maxilla. Two paired cartilages, the upper and lower lateral cartilage and one unpaired cartilage, the septal, complete the external framework. The chief muscles acting of nose external nose are the compressors and dilators of ala nasi supplied by facial nerve[6].

Blood supply to the external nose is from maxillary and ophthalmic arteries. The anterior facial vein and ophthalmic vein forms the venous supply, lymphatics drain to the submandibular and pre-auricular lymph nodes. The skin of external nose receives its sensory supply form the two upper divisions of the trigeminal nerve; ophthalmic and maxillary.[6]

NASAL CAVITY

Each nasal cavity is divided into three parts i.e., nasal vestibule, olfactory region and respiratory region. Nasal vestibule is the most anterior and it extends from the nostril antero-inferiorly to the nasal valve postero-superiorly. The nasal valve is situated between the caudal end of the upper alar cartilage laterally and the septum medially. The area of demarcation is limen nasi, with skin containing hair follicles, sebaceous and sweat glands. It is a space of importance since it is here that nasal cavity is the narrowest, limited to a triangular shape of only 0.3 cm^2 on each side.

The olfactory region is confined to the upper part of the nasal cavity and the superior turbinate representing an area of 10 cm^2. The rest of the nasal cavity constitutes the respiratory region and its surface may reach 120 sqcm[6].

The lateral wall of each nasal cavity has superior, middle, and inferior turbinates. Each turbinate overhangs a meatus. The space above or medial to superior turbinate is spheno-ethmoidal recess to which sphenoidal sinus open. The posterior ethmoidal cells drain into the superior meatus. The anterior ethmoidal, frontal and maxillary sinuses open into middle meatus. The nasaolacrimal duct opens into inferior meatus[7].

Middle meatus contains several structures of importance. An enlargement is found at anterior end of the middle meatus, which is a part of ethmoidal bone, called as uncinate process. A little further back is another eminence which is called bulla ethmoidalis, which represents a protrusion into the meatus of one of the air cells of the ethmoidal labyrinth[6]. Between these two enlargements is a groove which is known as hiatus semilunaris which leads to a narrowing called infundibulum[7].

Arterial supply is via the lateral branches of sphenopalatine, greater palatine, superior labial, anterior and posterior ethmoidal arteries. venous drainage occurs through the pterygoid plexus. Lymphatics drain into the submandibular nodes anteriorly and to the lateral pharyngeal, retropharyngeal and upper deep cervical nodes posteriorly[7].

Main sensory supply to the nasal cavity is derived from maxillary division of trigeminal nerve through branches arising in pterygopalatine ganglion. The lateral and medial internal nasal branches of the ophthalmic nerve supply anterior part of the nasal cavity while floor and anterior end of middle turbinate supplied by the anterior dental branch of the infra orbital nerve. Sympathetic nerve supply arises from the superior cervical ganglion; it produces vasoconstriction and decreases secretions from nose. Parasympathetic supply arises from the pterygopalatine ganglion via nerve to pterygoid canal. It produces vasodilatation and increased secretion[6].

NASAL SEPTUM:

The nasal septum is formed by the perpendicular plate of ethmoid, the vomer, septal cartilage, nasal crest of maxillary and palatine bones. The main arterial supply of nasal septum arises from the septal branch of sphenopalatine artery. The antero-inferior part of the septum or Little's area is where the septal branches of sphenopalatine, greater palatine, superior labial and anterior ethmoidal artery anastomose. Venous drainage occurs to pterygoid plexus. The anterior septum drains into submandibular nodes which posterior drains into the retropharyngeal and anterior deep cervical nodes. The nerve supply is by nasopalatine nerve posteriorly and anteriorly by the anterior ethmoid branch of nasociliiary nerve and antero-superior alveolar nerve[6].

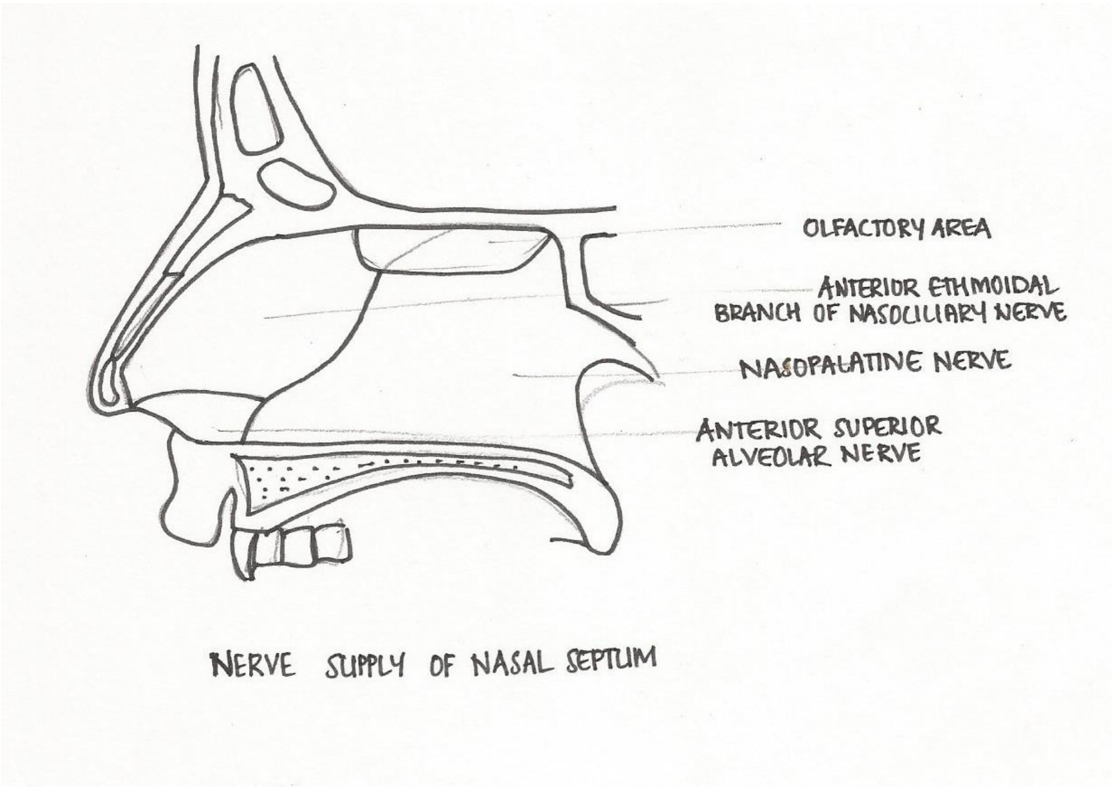

Figure 2 Nerve supply of nasal septum

LATERAL WALL

Lateral wall has a characteristic complex structure as a result of scrolls and projections present on it, which are convoluted to form turbinates. These projections are the superior, middle, and inferior turbinates and occasionally when present the supreme turbinate. The inferior turbinate is a separate bone, whereas middle and superior turbinates are projection from ethmoid bone. Below and lateral to roof of each of respective turbinates, superior middle and inferior meati are found[7].

Superior meatus occupies the posterior third of the lateral wall, the middle meatus occupies the posterior two thirds and the inferior meatus runs the whole length of the lateral wall. Between the middle turbinate and the nasal septum is the space called olfactory cleft. Posterosuperior to the superior choana is the space known as sphenoethmoidal recess to which sphenoid sinus opens. The posterior ethmoidal cells open into the superior meatus. The frontal , the anterior and the middle ethmoidal air cells and the maxillary sinus open into the middle meatus. The nasolacrimal duct opens into the anterosuperior portion of the inferior meatus at the point where the inferior choana contacts lateral wall of the nasal cavity[7].

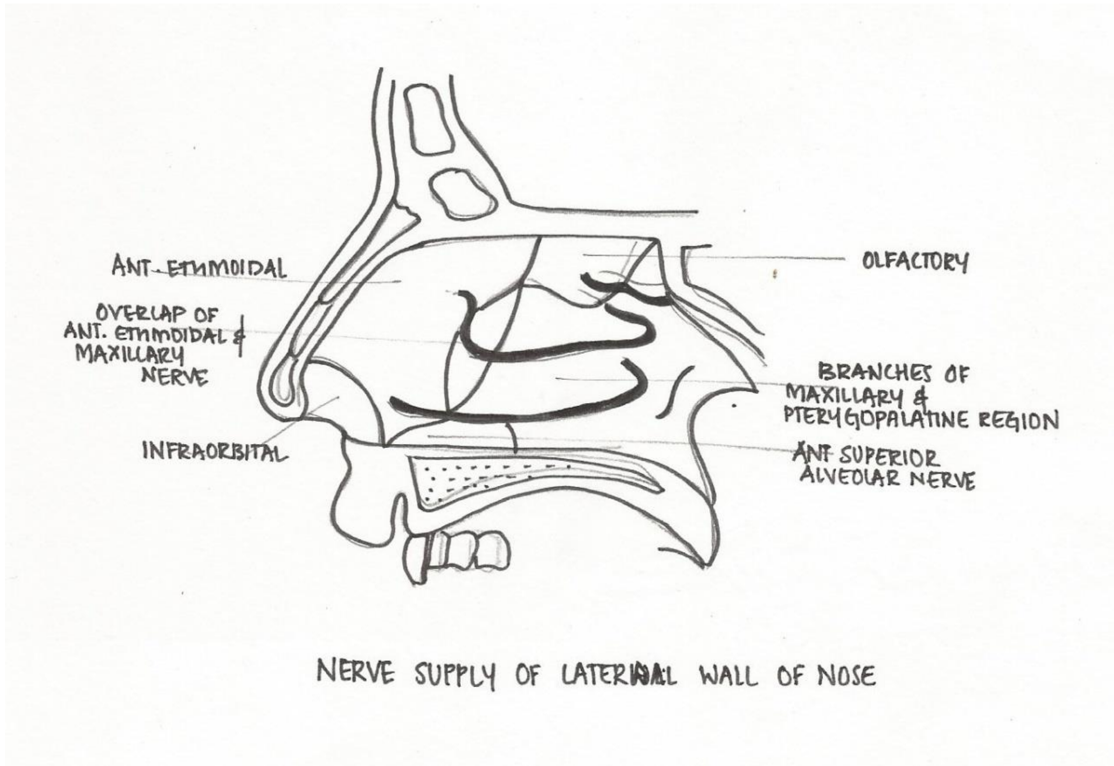

Figure 3: Nerve supply to lateral wall of nose

NASAL MUCOUS MEMBRANE

Nasal mucous membrane consists of fairly dense connective tissue. The mucous membrane is predominantly respiratory epithelium with a small area of olfactory epithelium superiorly adjacent to the cribriform plate. Respiratory epithelium is composed of ciliated and non-ciliated pseudo stratified columnar cells, basal pluripotent stem cells, and goblet cells. Seromucinous glands found in the sub mucosa are more important in mucous production in nasal cavity than goblet cells which are numerous in sinuses[6].

NERVE SUPPLY OF NOSE

Nerve supply to the nose is extremely rich and is via the general sensory, parasympathetic and sympathetic innervations. The main sensory supply comes from maxillary division of the trigeminal nerve from its pterygopalatine branches (nasopalatine, greater palatine, short palatine nerve). The lateral and medial internal nasal branches of the anterior ethmoidal nerve supply the anterior part if the nasal cavity. The floor and the anterior end of the inferior turbinate are supplied by branches of anterior superior alveolar nerve. The secretory nerve fibers are derived from the sympathetic and parasympathetic systems. Sympathetic fibers are derived through superior cervical ganglion via the sympathetic plexus of internal carotid artery and join with the parasympathetic fibers in the pterygoid canal. These supply the nasal vasculature via the greater superficial petrosal nerve to form nerve to pterygoid canal. Parasympathetic fibers are derived from the superior salivary nucleus in the medulla via the nerves intermedius to form the vidian nerve and reach sphenopalatine ganglion, where they relay before reaching the nasal cavity and also supply blood vessels of the nose causing vasodilatation. The special sensory olfactory nerves are distributed in a network in the mucosa in the upper third of nasal septum , roof and corresponding area over the lateral wall of nasal cavity[7]

BLOOD SUPPLY

The nasal cavity derives its blood supply from branches of both internal and external carotid arteries. Anterosuperior quadrant of nose is supplied by anterior and posterior ethmoidal arteries which are branches of sphenopalatine and greater palatine both branches of maxillary artery and by superior labial branch of facial artery. The dividing line between the two carotid system is at the level of the middle turbinate.

Venous drainage is by formation of cavernous plexus beneath mucous membrane and drain through the sphenopalatine and facial veins. Lymphatic drainage from the anterior part of the nose is to the submandibular nodes and to the superior nodes of deep cervical chain. Drainage from the posterior part id to the middle and deep cervical chain.

OSTEOMEATAL COMPLEX

Neumann coined this word to describe the region comprising middle meatus with the anterior air cells. This is the most important area for normal sinus functioning and any pathology in this area will disrupt the physiology and leads to sinus dysfunction. In the middle meatus there are several important structures. Anteriorly the first landmark is a hook shaped bone called the uncinate process. Posterior to the uncinate is a groove known as hiatus semilunaris which leads to the ethmoidal infundibulum. The ethmoidal bulla is a bulge posterior to the hiatus, which is a part of anterior ethmoidal group of cells. The frontal sinus opens into the superior most aspect of the ethmoidal infundibulum called the frontonasal recess, while the anterior ethmoidal cells open into the infundibulum. The ostium of the maxillary sinus opens posteroinferiorly in to the infundibulum.[6]

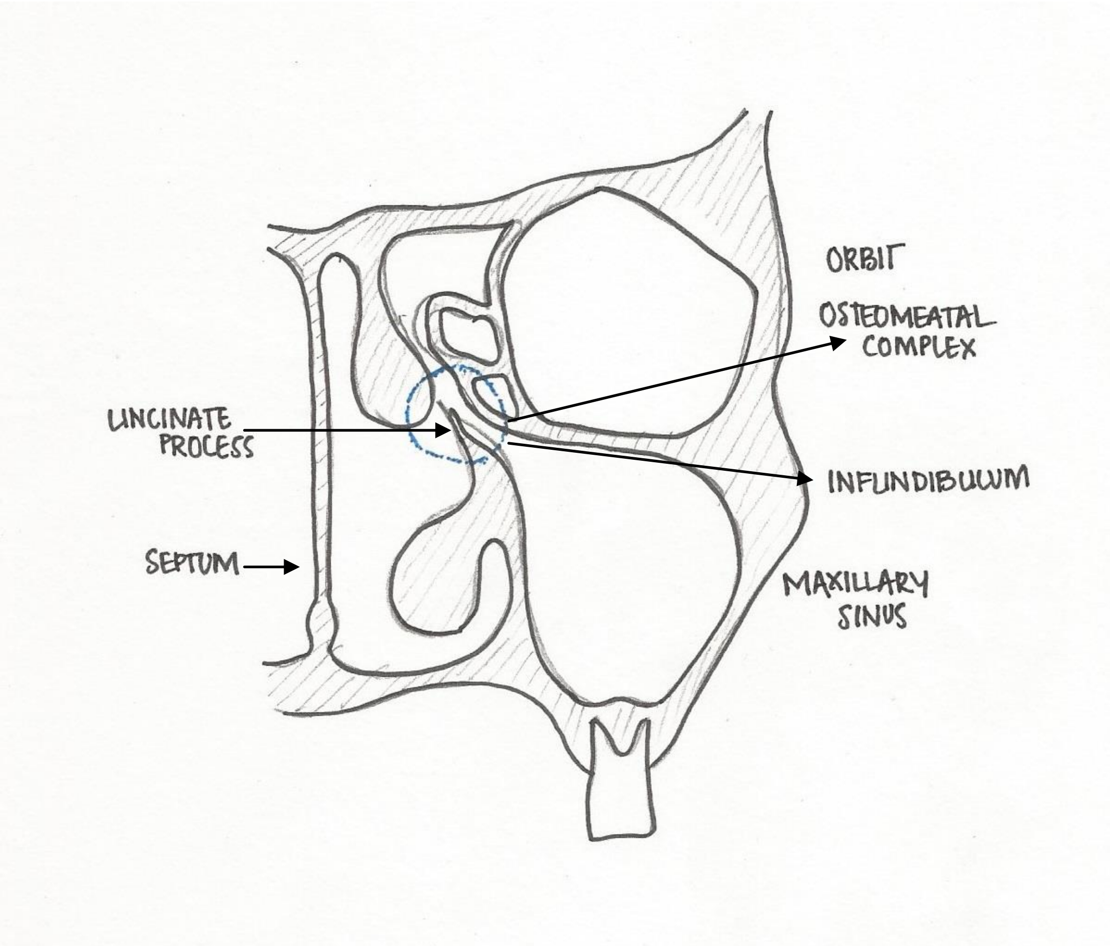

Figure 4: Osteomeatal complex

PHYSIOLOGY OF NOSE

The chief functions of the nose are respiration and olfaction. In addition to being the sensory organ of smell, the nose also plays an important role in cleansing and conditioning of inspired air. It also contributes to heat exchange, humidification, filtration, nasal resistance, nasal fluids and ciliary function, voice modification and the nasal neurovascular reflexes[8].

Filtration: This is accomplished by the nasal mucus blanket which covers the mucosal membrane and is constantly propelled posteriorly by the cilia. Mucus blanket is adhesive and causes the bacteria and dust to adhere to it[8].

Olfactory: The direction of air current ensures that air borne odorous substances reach olfactory area to enhance sense of smell[8].

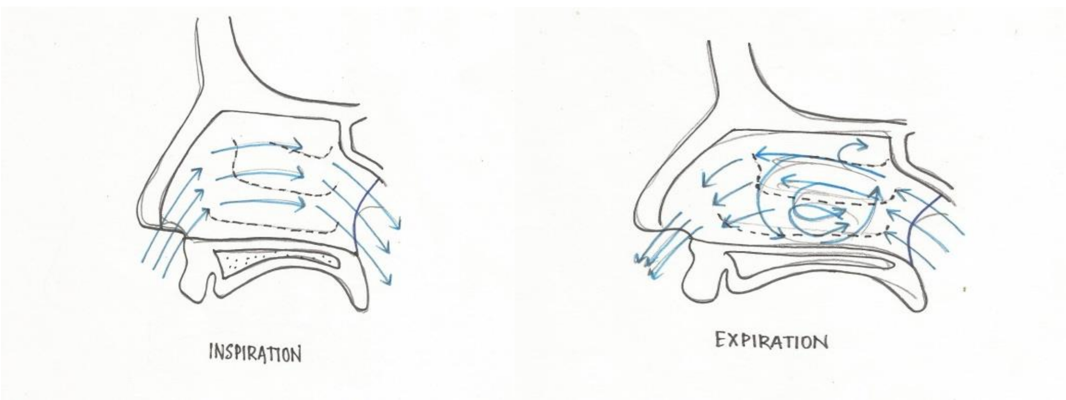

Figure 5: Air currents during inspiration and expiration

EMBRYOLOGY OF PNS

MAXILLARY SINUS

The maxillary sinus is the first sinus to appear at 7-10 weeks as a shallow groove expanding from the primitive ethmoidal infundibulum into the mass of the maxilla. It continues to grow during childhood at an estimated annual rate of 2 mm vertically and 3 mm anteroposteriorly and in particular with development of middle third of face as the dentition erupts. It reaches it final size in the 17-18 year of life .The maxillary sinus in an adult has a volume of around cm^2 and is roughly pyramid shape. The base of the pyramid is formed by the medial wall of the maxillary sinus with apex of pyramid towards the zygomatic recess[6].

ETHMOID SINUS

At 9-10 week of gestation, six major furrows appear on the lateral wall of nose. The furrows are separated by ridges which have an ascending portion called ramus ascendens and a posteroinferior portion called ramus descendens .The inferior turbinate is also called the maxilla-turbinal and is an individual bone. The first ethmoturbinal regress and the descending portion gives rise to the uncinate process, the ascending process forms the agger nasi. The first furrow gives rise to the infundibulum and the frontal recess. Middle turbinate is formed from the second ethmoturbinal, superior from the third. The fourth and fifth ethmoturbinals regress during development. [6,7].

SPHENOID SINUS

The sphenoid sinus is recognizable at around the third intrauterine month as an evagination from the sphenoethmoidal recess and again a small cavity is found at birth. At the third year of life, pneumatisation of the sphenoid bone progresses and at age seven has frequently reach the floor of sella.[6,7]

FRONTAL SINUS

The frontal sinus is the most variable in size and shape and maybe regarded embryologically as an anterior ethmoidal cell. From the most anterior and superior segment of the anterior ethmoid complex, the frontal bone is gradually pneumatised, resulting in frontal sinuses of variable size .At birth the frontal sinuses are small and on x-rays cannot be differentiated from other anterior ethmoidal cells

ANATOMY OF PARANASAL SINUSES

The paranasal sinuses are arranged in pairs and include two groups anterior and posterior. The former includes maxillary sinus, frontal sinus and anterior ethmoidal sinus. The posterior group comprises of posterior ethmoidal and sphenoidal sinus.[7]

MAXILLARY SINUS

It is present since birth, but attains its maximum size around 15 to 17 years of age. The roof is formed by floor of orbit, floor by roots of canine. Posteriorly, it is related to infratemporal and pterygopalatine fossa (PPF), anterolateral walls are superficial and deep to soft tissues of face, medial wall formed by nasal cavity. The maxillary ostium present at the upper part of sinus, drains into the middle meatus. An accessory ostium is present in some people posterior to the main ostium.[6,7]

FRONTAL SINUS

It is rudimentary at birth, being represented by a small upward prolongation from anterior end of middle meatus, the nasofrontal duct. It is bound anteriorly and posteriorly by the outer and inner table of frontal bone, floor by the roof of orbital cavity, medially by the septum between the two frontal sinuses. The ostium of frontal sinus is situated in its floor, drains into middle meatus.

ETHMOIDAL SINUS

It is present at birth and in adult life and they vary in number, size and shape. They are classified into anterior and posterior, depending on whether they communicate with middle or superior meatus. They are bound medially by upper half of nasal cavity, laterally by orbit, anteriorly by frontal process of maxilla and posteriorly by sphenoid bone[7].

SPHENOID SINUS

It is present at birth. The lateral wall is related to internal carotid artery, optic nerve and the cavernous sinus, roof is related to frontal lobe, olfactory tract, optic chiasma and pituitary gland. The floor is related to pterygoid canal, medial wall is between the two sphenoid sinus. The sphenoid ostium is situated high up in the cavity of sinus[6,7].

PHYSIOLOGY OF SINUSES

Air conditioning: They serve as supplementary chambers for conditioning the inspired air by heating and moistening.[6,8]

Vocal response: They act as resonating chambers and add to quality of voice.

Thermal insulators: They protect the structures in orbit and cranial fossa from intra temporal variations.

Balance of head: It reduces the weight of the bones of face, thereby aiding in balance of head[6,8].

MUCOCILLIARY CLEARANCE

Drainage and ventilation and two most important factors in maintenance of normal physiology of paranasal sinuses. It depends upon the amount of mucus produced, composition of mucus, effectiveness of ciliary beat, mucosal resorption, condition of ostia and ethmoidal clefts[8].

The mucus film has two layers: an inner serous layer, called the sol phase, in which cilia beat and an outermost viscous layer, the gel phase, which is transported by the ciliary beat. This functions like a conveyor belt. Normal nasal mucus exists at a pH range of 7.5 to 7.6[8].

In maxillary sinus secretion transport starts from the floor of sinus in a stellate pattern. The mucus from anterior, medial, posterior, lateral wall and roof of sinus converge at the natural ostium. This is finally drained into middle meatus. Frontal sinus has active inward transportation of mucus. Due to whorled pattern of cilia, mucus is circulated again and again. Finally mucus from frontal sinus drains into frontal recess. The anterior ethmoidal cells drain into middle meatus and posterior ethmoidal cells into sphenoethmoidal cells. In the sphenoidal cells, mucus undergoes a spiral transport and drains into sphenoethmoidal cells. All these secretions finally drain into lateral nasal wall and from there to nasopharynx

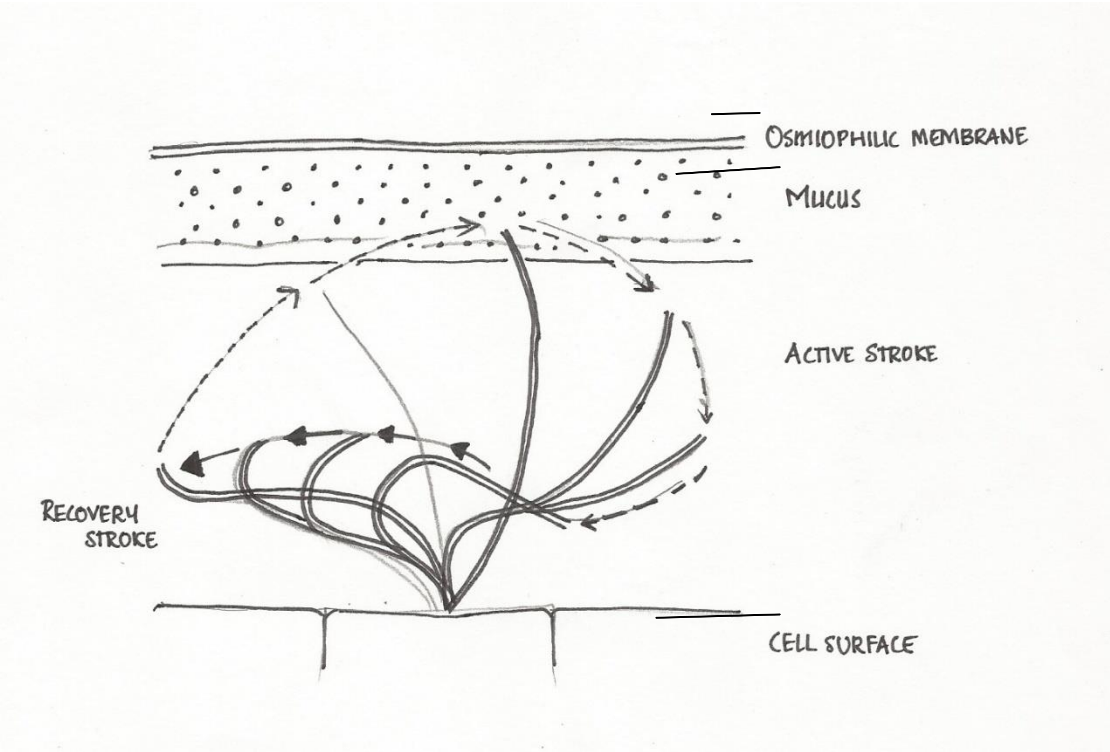

Figure 6: Normal ciliary cycle

PTERYGOPALATINE FOSSA

The PPF is a small pyramidal space below the apex of the orbit on the lateral side of the skull.[10]

The posterior boundary is the root of the pterygoid process and adjoining anterior surface of the greater wing of the sphenoid, the anterior boundary is the superomedial part of the infra-temporal surface of the maxilla. The perpendicular plate of the palatine bone, with its orbital and sphenoidal processes forms the medial boundary, and the pterygomaxillary fissure is the lateral boundary. The fossa communicates with the nasal cavity via the sphenopalatine foramen, with the orbit via the medial end of the inferior orbital fissure, and with the infratemporal fossa via the pterygomaxillary fissure, which lies between the back of the maxilla and the pterygoid process of the sphenoid and transmits the maxillary artery. It also communicates with the oral cavity via the greater palatine canal, which opens in the posterolateral aspect of the hard palate. There are two openings in the posterior wall of the PPF, the foramen rotundum, which transmits the maxillary nerve, and the pterygoid canal, which transmits the nerve of the pterygoid canal (Vidian nerve). When the anterior aspect of the pterygoid plate is examined in a disarticulated sphenoid, the foramen rotundum is seen to lie above and lateral to the pterygoid canal.[10]

The main contents of the PPF are the third part of the maxillary artery, the maxillary nerve and many of its branches, and the pterygopalatine ganglion.

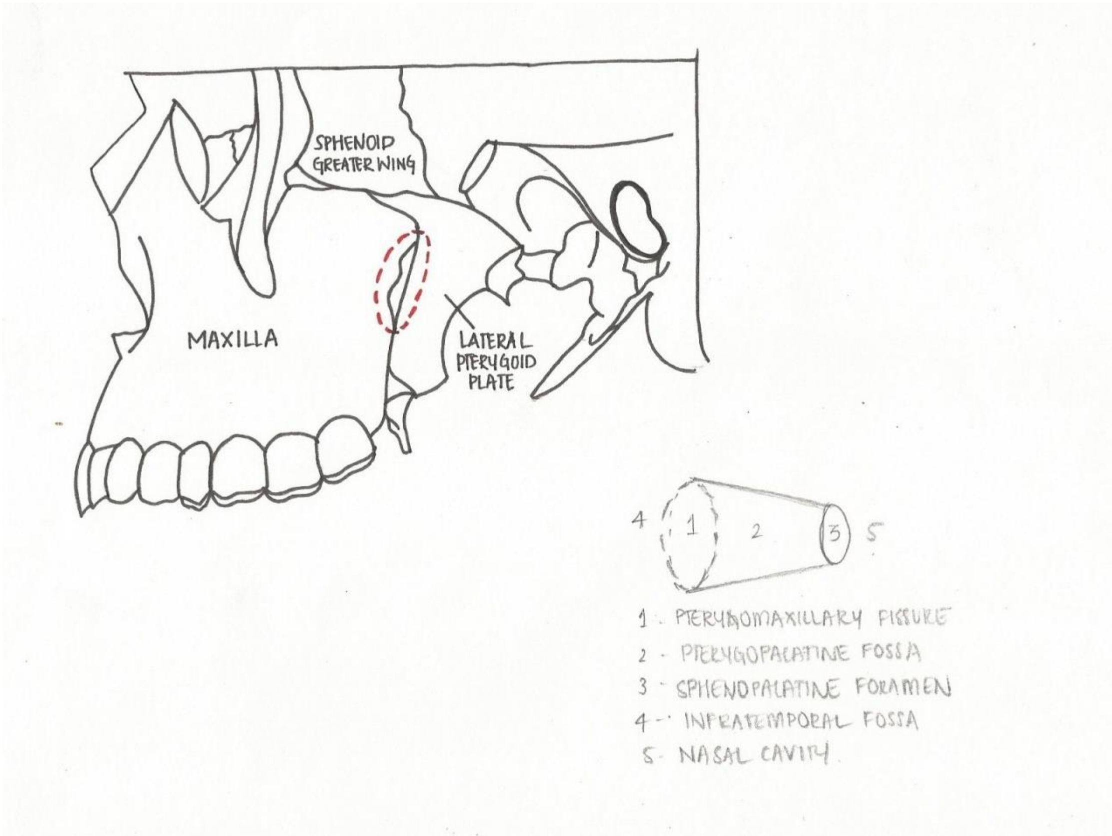

Figure 7: Relations of pterygopalatine fossa

Maxillary artery

The maxillary artery passes through the pterygomaxillary fissure from the infratemporal fossa into the PPF, where it terminates as the third part of the maxillary artery. It gives off numerous branches including the posterior superior alveolar, infraorbital, sphenopalatine and greater palatine arteries.[10]

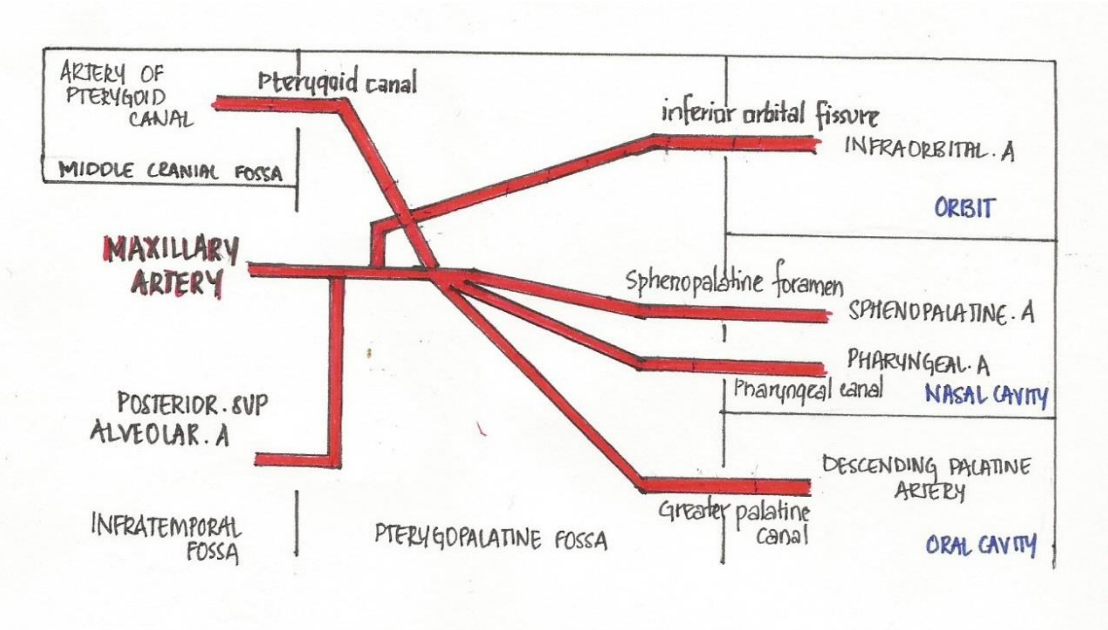

Figure 8: Maxillary artery in pterygopalatine fossa

Posterior superior alveolar artery

The posterior superior alveolar artery arises from the maxillary artery within the PPF and runs through the pterygomaxillary fissure onto the maxillary tuberosity. It gives off branches which penetrate the bone here to supply the maxillary molar and premolar teeth and the maxillary air sinus, and other branches that supply the buccal mucosa. Occasionally the posterior superior alveolar artery arises from the infraorbital artery.[10]

Infraorbital artery

The infraorbital artery enters the orbit through the inferior orbital fissure. It runs on the floor of the orbit in the infraorbital groove and infraorbital canal and emerges onto the face at the infraorbital foramen to supply the lower eyelid , part of the cheek, the side of the external nose, and the upper lip. While within the infraorbital canal it gives off the anterior superior alveolar artery which runs downwards to supply the anterior teeth and the anterior part of the maxillary sinus. A middle superior alveolar artery is also often described. When present, it branches from the infraorbital artery within the infraorbital canal and runs inferiorly along the lateral wall of the maxillary sinus toward the region of the canine and lateral incisor teeth and anastomoses with the anterior and posterior superior alveolar arteries.[10]

Artery of the pterygoid canal

The artery of the pterygoid canal (Vidian artery) arises as a branch of either the distal part of the maxillary artery (70%) or the petrous segment of the internal carotid artery (30%). It passes through the pterygoid canal and anastomoses with the pharyngeal, ethmoidal and sphenopalatine arteries in the PPF and with the ascending pharyngeal, accessory meningeal, ascending palatine and descending palatine (occasionally) arteries in the oropharynx and around the pharyngotympanic tube. Through these complex anastomoses, the artery of the pterygoid canal contributes to the supply of part of the pharyngotympanic tube, the tympanic cavity, and the upper part of the pharynx. It may also anastomose with the artery of the foramen rotundum, and so communicate with branches of the cavernous portion of the internal carotid artery.[10]

Pharyngeal artery

The pharyngeal branch of the maxillary artery passes through the palatovaginal canal, accompanying the nerve of the same name, and is distributed to the mucosa of the nasal roof, nasopharynx, sphenoidal air sinus and pharyngotympanic tube.

Greater (descending) palatine artery

The greater palatine artery leaves the PPF through the greater (anterior) palatine canal, within which it gives off two or three lesser palatine arteries. The greater palatine artery supplies the inferior meatus of the nose, then passes onto the roof of the hard palate at the greater (anterior) palatine foramen and runs forwards to supply the hard palate and the palatal gingivae of the maxillary teeth. It gives off a branch that runs up into the incisive canal to anastomose with the sphenopalatine artery, and so contribute to the arterial supply of the nasal septum. The lesser palatine arteries emerge onto the palate through the lesser (posterior) palatine foramen, or foramina, and supply the soft palate.[10]

Sphenopalatine artery

The sphenopalatine branch of the maxillary artery passes through the sphenopalatine foramen and enters the nasal cavity posterior to the superior meatus. From here its posterior lateral nasal branches ramify over the conchae and meatuses, anastomosing with the ethmoidal arteries and nasal branches of the greater palatine artery to supply the frontal, maxillary, ethmoidal and sphenoidal air sinuses. The sphenopalatine artery next crosses anteriorly on the inferior surface of the sphenoid and ends on the nasal septum in a series of posterior septal branches which anastomose with the ethmoidal arteries. A branch descends on the vomer to the incisive canal to anastomose with the greater palatine artery and the septal branch of the superior labial artery.[10]

Maxillary nerve

The maxillary division of the trigeminal nerve is wholly sensory. It leaves the skull via the foramen rotundum, which leads directly into the posterior wall of the PPF. Crossing the upper part of the PPF, the nerve then gives off two large ganglionic branches which contain fibres destined for the nose, palate and pharynx, and they pass through the pterygopalatine ganglion without synapsing. It then inclines sharply laterally on the posterior surface of the orbital process of the palatine bone and on the upper part of the posterior surface of the maxilla in the inferior orbital fissure (which is continuous posteriorly with the PPF) , it lies outside the orbital periosteum, and gives off its zygomatic, and then posterior superior alveolar branches. About halfway between the orbital apex and the orbital rim the maxillary nerve turns medially to enter the infraorbital canal as the infraorbital nerve.

The maxillary nerve gives off many of its branches in the PPF. They can be subdivided into those that come directly from the nerve, and those that are associated with the pterygopalatine parasympathetic ganglion. Named branches from the main trunk are meningeal, ganglionic, zygomatic, posterior, middle and anterior superior alveolar and infraorbital nerves. Named branches from the pterygopalatine ganglion are orbital, nasopalatine, posterior superior nasal, greater (anterior) palatine, lesser (posterior) palatine and pharyngeal.[10]

Meningeal nerve

The meningeal branch of the maxillary nerve arises within the middle cranial fossa and runs with the middle meningeal vessels. It contributes to the innervation of the dura mater.

Ganglionic branches

There are usually two ganglionic branches that connect the maxillary nerve to the pterygopalatine ganglion.

Zygomatic nerve

The zygomatic branch of the maxillary nerve leaves the PPF through the inferior orbital fissure together with the maxillary nerve.

Posterior superior alveolar nerve

The posterior superior alveolar nerve leaves the maxillary nerve in the PPF. Its subsequent course and distribution is described in detail in Chapter 30.

Infraorbital nerve

The infraorbital nerve can be regarded as the terminal branch of the maxillary nerve. It leaves the PPF to enter the orbit at the inferior orbital fissure

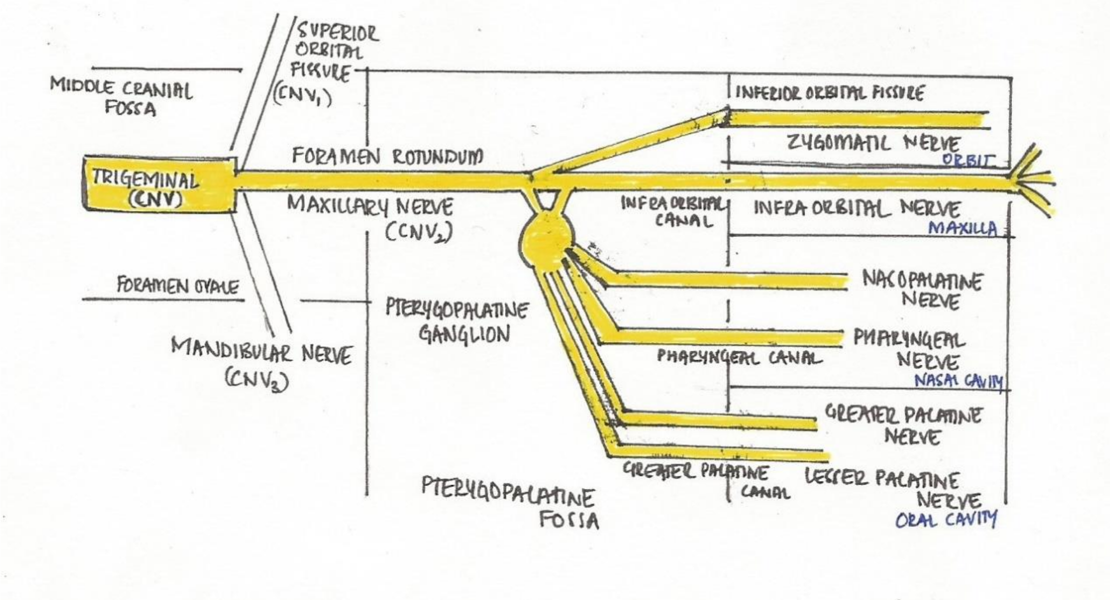

Figure 9: Maxillary nerve in pterygopalatine fossa

Pterygopalatine ganglion

The pterygopalatine ganglion is the largest of the peripheral parasympathetic ganglia. It is placed deeply in the PPF, near the sphenopalatine foramen, and anterior to the pterygoid canal and foramen rotundum (Fig. 31.16B). It is flattened, reddish-grey in colour, and lies just below the maxillary nerve as it crosses the PPF. The majority of the 'branches' of the ganglion are connected with it morphologically, but not functionally, because they are primarily sensory branches of the maxillary nerve. Thus they pass through the ganglion without synapsing, and enter the maxillary nerve through its ganglionic branches, but they convey some parasympathetic fibres to the palatine, pharyngeal and nasal mucous glands.[10]

Preganglionic parasympathetic fibres destined for the pterygopalatine ganglion run initially in the greater petrosal branch of the facial nerve, and then in the nerve of the pterygoid canal (Vidian nerve), after the greater petrosal unites with the deep petrosal nerve. The nerve of the pterygoid canal enters the ganglion posteriorly. Postganglionic parasympathetic fibres leave the ganglion and join the maxillary nerve via a ganglionic branch, then travel via the zygomatic and zygomaticotemporal branches of the maxillary nerve to the lacrimal gland. Preganglionic secretomotor fibres of uncertain origin also travel in the nerve of the pterygoid canal. They synapse in the pterygopalatine ganglion, and postganglionic fibres are distributed to palatine, pharyngeal and nasal mucous glands via palatine and nasal branches of the maxillary nerve.[10]

Postganglionic sympathetic fibres pass through the ganglion without synapsing and supply blood vessels and orbitalis. They arise in the superior cervical ganglion and travel via the internal carotid plexus and deep petrosal nerve to enter the pterygopalatine ganglion within the nerve of the pterygoid canal.[10]

General sensory fibres destined for distribution via orbital, nasopalatine, superior alveolar, palatine and pharyngeal branches of the maxillary division of the trigeminal nerve run through the ganglion without synapsing.

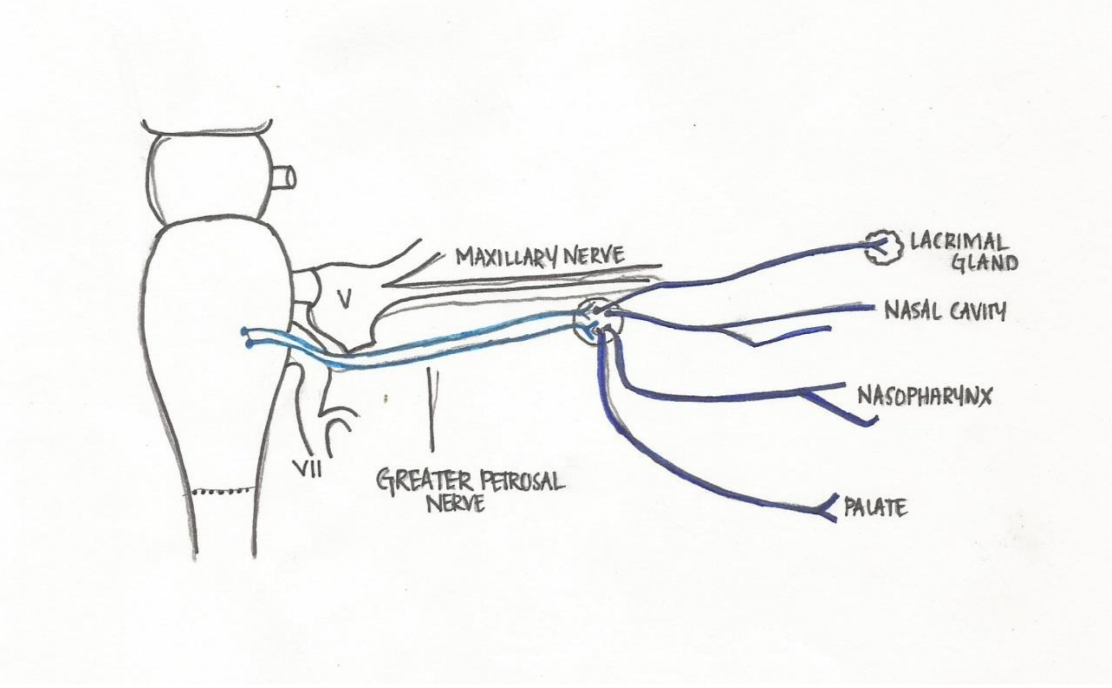

Figure 10: Pterygopalatine ganglion

Orbital branches

Fine orbital branches enter the orbit through the inferior orbital fissure and supply orbital periosteum. Some fibres also pass through the posterior ethmoidal foramen to supply the sphenoidal and ethmoidal sinuses. The orbital branches probably join branches of the internal carotid nerve to form a ‘retro-orbital’ plexus from which orbital structures such as the lacrimal gland and orbitalis receive an autonomic innervation.

Nasopalatine nerve

The nasopalatine nerve leaves the PPF through the sphenopalatine foramen and enters the nasal cavity. It passes across the cavity to the back of the nasal septum, runs downwards and forwards on the septum in a groove in the vomer, and then turns down through the incisive fossa in the anterior part of the hard palate to enter the roof of the mouth. When an anterior and a posterior incisive foramen exist in this fossa, the left nasopalatine nerve passes through the anterior foramen, and the right nerve passes through the posterior foramen. The nasopalatine nerve supplies the lower part of the nasal septum and the anterior part of the hard palate, where it communicates with the greater palatine nerve.[10]

Posterior superior nasal nerves (lateral and medial)

The posterior superior alveolar nerves enter the back of the nasal cavity through the sphenopalatine foramen. Lateral posterior superior nasal nerves (about 6) innervate the mucosa lining the posterior part of the superior and middle nasal conchae and the posterior ethmoidal sinuses. Two or three medial posterior superior nasal nerves cross the nasal roof below the opening of the sphenoidal sinus to supply the mucosa of the posterior part of the roof and of the nasal septum.[10]

Palatine nerves (greater and lesser)

The greater and lesser palatine nerves pass downwards from the pterygopalatine ganglion through the greater palatine canal. The greater palatine nerve descends through the greater palatine canal, emerges on the hard palate from the greater palatine foramen and runs forwards in a groove on the inferior surface of the bony palate almost to the incisor teeth. It supplies the gingivae, mucosa and glands of the hard palate and also communicates with the terminal filaments of the nasopalatine nerve. In the greater palatine canal, it gives off posterior inferior nasal branches that emerge through the perpendicular plate of the palatine bone and ramify over the inferior nasal concha and walls of the middle and inferior meatuses. As it leaves the greater palatine canal, it gives off branches which are distributed to both surfaces of the adjacent part of the soft palate.

The (middle and posterior) palatine nerves are much smaller than the greater palatine nerve. They descend through the greater palatine canal from which they diverge low down to emerge through the lesser palatine foramina in the tubercle or pyramidal process of the palatine bone. They innervate the uvula, tonsil and soft palate.[10]

Fibres conveying taste impulses from the palate probably pass via the palatine nerves to the pterygopalatine ganglion. They pass through the ganglion without synapsing, and leave via the greater petrosal nerve. Their cell bodies are located in the facial ganglion and their central processes pass via the sensory root of the facial nerve (nervus intermedius) to the gustatory nucleus in the nucleus of the tractus solitarus.

Pharyngeal nerve

The pharyngeal branch of the maxillary nerve leaves the pterygopalatine ganglion posteriorly. It passes through the palatovaginal canal with the pharyngeal branch of the maxillary artery and supplies the mucosa of the nasopharynx behind the pharyngotympanic tube.

CHRONIC RHINOSINUSITIS

The term "sinusitis" refers to a group of disorders characterized by inflammation of the mucosa of the paranasal sinuses. Because the inflammation nearly always also involves the nose, it is now accepted that "rhinosinusitis" is the preferred term to describe this inflammation of the nose and paranasal sinuses[11].

To highlight the role of inflammation better, newer definitions have been applied to rhinosinusitis,:that is, a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses. Chronic rhinosinusitis (CRS) is rhinosinusitis of at least 12 consecutive weeks duration[12].

Therefore, CRS is a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses of at least 12 consecutive weeks duration[12,13].

A widely accepted set of classifications or definitions was developed by the Rhinosinusitis Task Force of the American Academy of Otolaryngology-Head and Neck Surgery and reported by Lanza and Kennedy[12,14,15].

Table 1.classification of Rhinosinusitis

CLASSIFICATION	DURATION
Acute (ARS)	7 Days to <4 weeks
Sub acute	4-12 weeks
Recurrent acute	> 4 episodes of ARS per year
Chronic(CRS)	>12 Weeks
Acute exacerbation of chronic	Sudden worsening of CRS with return to baseline after

The taskforce in order to accommodate the different needs gave definitions that can be applied in appropriate studies. In this way the taskforce tried to improve the comparability of studies and hence enhanced the evidence based diagnosis and treatment of patients with rhinosinusitis and nasal polyps.

CRS is best considered as a group of heterogeneous disorders due to a multitude of causes that result in mild to severe symptomatic inflammation of the sinonasal mucosa. The management of this complex disease is therefore a challenge. The most simplified classification divides CRS into those patients who have nasal polyps (CRSwNP) and those without (CRSsNP) .[11]

Table 2. CRS differentiation by inflammatory mediators.[11]

CRS with nasal polyps (CRSwNP)	• Tissue oedema, low tumour growth factor -β and lot T-reg activity • High tissue eosinophillia and IgE increased IL-5 and IL-13 (Th2 polarisation)
CRS without nasal polyps (CRSsNP)	• Fibrosis, less eosinophillic infiltration • Increased interferon -ɣ, tumour growth factor-β and T--regulatory activity (Th1 polarisation)

Although all cases of rhinosinusitis involve inflammation of the mucosal linings, in practical setting, the main focus is on those patients in whom this inflammation leads to symptoms. Because of this important relationship to symptoms, the Rhinosinusitis Task Force's definitions include a group of symptoms to be applied to these conditions to allow for clinical diagnosis .[12]

Rhinosinusitis symptoms/signs (requires two major factors, or one major and two minor).

Table 3. Symptoms of Chronic rhinosinusitis.[11]

MAJOR SYMPTOMS	MINOR SYMPTOMS
Facial pain /Pressure	Headache
Facial congestion/Fullness	Fever (Non acute)
Nasal Obstruction /Blockage	Halitosis
Nasal discharge/Purulence/Discoloured Posterior drainage	Fatigue
Hyposmia/Anosmia	Dental pain
Purulence on nasal examination	Cough
Fever (Acute RS only)	Ear pain/Pressure/Fullness

Epidemiology

CRS is the fifth most common diagnosis for an antibiotic prescription worldwide.[11,16]. Despite its prevalence, there is a paucity of accurate epidemiologic data for CRS, especially for CRSsNP. Patient surveys in the United States have found a 15%–16% prevalence of CRS. A study conducted in Canada, Korea, Scotland, Europe, and Sao Paulo shows prevalence of CRS ranges from 1%–11%. [11,17,18]

Men and women are both affected by CRSwNP. In general, nasal polyps occur in all races and become more common with age, with the average age of onset being 42 years.

Etiology

Numerous hypotheses have been proposed with a great deal of overlap, supporting a multifactorial basis. One classification method separates potential contributing entities into host and environmental factors. The heterogeneous nature of CRS is important to understand when planning treatment for this diverse group of patients whose disease may have arisen from very different underlying etiologies.[11]

Table 4. Factors associated with CRS.[11]

SYSTEMIC HOST FACTORS	**LOCAL HOST FACTORS**	**ENVIORMENTAL FACTORS**
1) Allergy	1) Anatomic	1) Microorganism (bacteria, fungi, virus)
2) Immunodeficiency	2) Neoplasm	2) Noxious chemicals
3) Mucociliary dysfunction	3) Acquired mucociliary dysfunction	3) Medications
4) Cystic fibrosis	4) Previous trauma or surgery	
5) Granulomatous diseases		
6) GERD		
7) Aspirin intolerance		

CRSwNP in the Caucasian population is associated more closely with high tissue eosinophilia and increased T helper (Th)-2 cytokine expression (interleukin [IL]-5 and IL-13) as well as nasal obstruction and smell loss, whereas CRSsNP may have more Th-1 polarization and less eosinophilic infiltration.[11,19]

Defects in the coordinated mechanical barrier and/or the innate immune response of the sinonasal epithelium has also been proposed as a mechanism for CRS. This susceptibility may be based on host genetic factors, predisposing some individuals to mechanical barrier failure in the presence of environmental stress. CRS is a common problem in patients with Kartagener's syndrome, primary ciliary dyskinesia, and cystic fibrosis. Inability of the sinonasal cilia to transport viscous mucus causes ciliary malfunction leading to CRS. Epithelial damage or host barrier dysfunction will result in colonization of the sinonasal mucosa with Staphylococcus aureus. Subsequent secretion of super-antigenic toxins may lead to a skewed Th-2 host inflammatory response with generation of local polyclonal immunoglobulin E (IgE), promotion of eosinophil survival and mast cell degranulation with alteration of eicosanoid metabolism. The sum of these local tissue effects may lead to polyp formation.[20]

The role of microbes as causative agents in CRS is not clear, but microbial infection and biofilms may contribute to the propagation of CRS. S. aureus is the most common bacterial pathogen identified in CRS patients. Coagulase-negative Staphylococcus and anaerobic and Gram-negative bacteria are also commonly cultured from CRS patients.[11]

Diagnosis

Symptoms

At the first notification of the problem, the diagnosis of rhinosinusitis is presumed on symptoms alone. The symptoms are mainly the same in acute rhinosinusitis (ARS), CRSsNP and CRSwNP, but the pattern and intensity may vary. Litvack et al in their study reported a significantly increased risk of hyposmia (odds ratio = 2.4 and anosmia (Odds ration =13.2) in nasal polyposis patients compared to CRSsNP. (14) After inquiring the symptoms, anterior rhinoscopy remains the first step in clinical examination, although it is of limited value [12]

Examinations

1) Nasal endoscopy

Nasal endoscopy involves passing a frequently rigid, or sometimes flexible, endoscope through the nostril to examine the nasal cavity, middle and superior meati, nasopharynx and mucociliary drainage pathways. Nasal endoscopy has a major contribution in the diagnosis of CRS and affords significantly better illumination and visualization of the nasal cavity compared to anterior rhinoscopy.[12]

2) Imaging

The plain sinus x-ray has limited usefulness for the diagnosis of rhinosinusitis and for evaluation of the response to therapy. CT scanning is the modality of choice for the paranasal sinuses due to optimal display of differences between air, bone and soft tissue. As mentioned before, CT scanning is not the primary step in the diagnosis of rhinosinusitis, but has the aim to affirm the symptoms and findings of endoscopic examination after failure of medical therapy. Because of many insignificant abnormalities found in the normal population during scans.[15], the diagnosis of CRS based on imaging, in absence of symptoms, is inappropriate.[12]

3) Nasal cytology, biopsy and bacteriology

Generally, cytology has not proven a useful tool in diagnosis of rhinosinusitis. However, lavage with 0.9% saline, microsuction, nasal brushes, nasal tampons, disposable scrapers, etc. are techniques which are largely used for clinical research.[12]

CRS is diagnosed based on clinical symptoms and objective evaluation. Symptoms must be present for at least 12 consecutive weeks. Several studies have shown using symptoms alone to diagnose CRS can be nonspecific. Therefore, nasal endoscopy or imaging must also be used to confirm the presence of sinonasal disease to increase the specificity of diagnosis.[12]

Endoscopic findings suggestive of CRS include mucopurulent discharge, nasal polyps or polypoid change, and/or mucosal edema obstructing the middle meatus.

Computed tomography (CT) is considered the gold standard for imaging in CRS. Although CT scans cannot distinguish between inflammation and infection, they do seem to correlate fairly well with the extent of disease. Findings consistent with CRS include isolated or diffuse mucosal thickening, bone changes, or air-fluid levels.[15]

Treatment

Aim of the treatment is to Improve osteomeatal complex drainage.

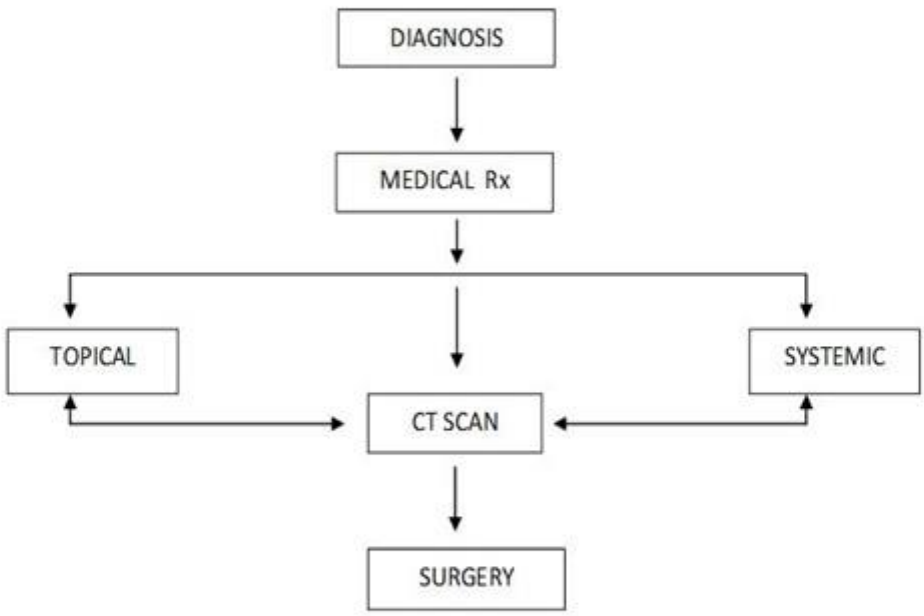

Figure 11: Treatment Algorithm

Medical treatment. [11]

Medical treatment involved in CRS include:

- Allergen and/or irritant avoidance
- Douching
- Corticosteroids
- Decongestants
- Antibiotics
- Antifungals
- Antileukotrienes
- Aspirin
- Immunotherapy
- Other therapies

Corticosteroids

Systemic corticosteroids (oral, intramuscular) can reduce the size of nasal polyps to an extent that is comparable with surgery. Topical corticosteroids reduce the recurrence of nasal polyps and should be used routinely in the long term, preferably employing a molecule with low systemic absorption in drop form in the head upside down position In non-polypoid chronic rhinosinusitis, topical corticosteroid shows modest efficacy in reducing symptoms during acute exacerbations when combined with antibiotics.

Antibiotics

Antibiotics are needed for acute severe bacterial sinusitis; their place in the chronic form is controversial.

Two routes are available: topical and oral.

Oral short course is used in the treatment of acute exacerbations of CRS.

Antihistamines /Antileukotrienes

There is little evidence of the efficacy of antihistamines in chronic rhinosinusitis, presumably because the majority have little or no effect on nasal blockage

Leukotriene antagonists, such as Montelukast, Zafirlukast, and Zileuton, have been evaluated in numerous studies involving patients with CRSwNP

Surgical treatment

Functional endoscopic sinus surgery (FESS)

FESS has now become well established for the treatment of chronic rhinosinusitis refractory to medical treatment

Steps:

1) Uncinectomy

2) Middle meatal antrostomy

3) Anterior Ethmoidectomy

4) Posterior ethmoidectomy

5) Clearance of frontal recess and frontal sinusotomy

6) Sphenoidotomy

Table 5. Complications of FESS[21]

SITE	COMPLICATION
Orbit	Nasolacrimal duct damage Extra-occular muscle injury Intra-orbital haemorrhage/ Emphysema Optic nerve damage
Intracranial	Haemorrhage Cerebrospinal fluid leak--> meningitis
Nasal	Haemorrhage

Control of bleeding

The presence of significant bleeding in the surgical field is a critical factor in the potential success or failure of FESS.[22,23,24,25] When significant bleeding is present, recognition of anatomical landmarks becomes difficult. [23,24,25]

Bleeding obscures surgical planes and makes the identification of the drainage pathways of the sinuses difficult. Cell walls become difficult to distinguish from the lamina papyracea or skull base and the risk of causing complications increases.[24,25] If the patient has significant inflammation of the sinuses, from chronic infection or the presence of pus/fungal debris, increased vascularity will often contribute to more bleeding.[23,26] If the surgeon attempts to manipulate an instrument in the surgical field after the discernible anatomy is covered in blood, the risk of a complication increases. In addition, greater surgical trauma may occur, cells may be left behind and there is an increased likelihood of postoperative scarring and failure of the surgical procedure. It is therefore critical to optimize the surgical field and, in so doing, make the surgical dissection as easy as possible.[23,24,25]

Bleeding is more common close to large vessels. Stamberger has included 3 areas which are responsible for extensive bleeding during sinus surgery.

1. Anterior ethmoidal artery located in an osseous channel close to ethmoid roof

2. Branch of sphenopalatine artery close to the posterior end of middle turbinate. This is more prone for injury in patients with well pneumatized middle turbinate (concha bullosa)

3. Damage to sphenopalatine artery while attempting to widen the sphenoidal ostium

Surgical bleeding during FESS has been classified into: Arterial, Venous and Capillary Out of these three types of bleeding it is the capillary bleed that causes the most trouble during FESS [27]

Different methods have been tried to control bleeding during sinus surgery, which are.[28]

1) Controlled Hypotension.

Controlled hypotension technique includes various modalities associated with different potency and adverse effects. Its safety is dependent on a thorough knowledge about the mechanism of action for each modality, adequate monitoring of the patient, and choosing the appropriate modality with consideration to history of drug allergies and co-morbidities.

2) Patient's Positioning

Reverse Trendelenburg or anti-Trendelenburg position is a common surgical position in which the head is up and feet are down. Head elevation reduces mean arterial pressure in the elevated part by about 2 mm Hg for each 2.5 cm above the cardiac level.[29] A reverse Trendelenburg position is used in numerous surgical procedures and presents multiple benefits. It reduces venous return from the lower extremities, therefore, reducing total blood loss, blood loss per minute ($P < 0.001$) and improving haemostasis of the surgical field when compared with the supine position.[30] Sudden shift in blood pressure is a serious complication of the reverse Trendelenburg position; the patient must be tilted in and out slowly to avoid this complication.[28]

3) Using a Laryngeal Mask Airway

The laryngeal mask airway (LMA) is a supraglottic device that is associated with less respiratory and cardiovascular reflex responses due to reduced stimulation of the larynx as compared to endotracheal intubation. Moreover, LMA facilitates controlled hypotension. One study suggested that LMA is more effective than endotracheal intubation in regard to rapid onset to achieve a target systolic arterial blood pressure ($P< 0.05$), less blood loss), and use of lower doses of remifentanil ($P < 0.05$). Visibility of the operative field improved in the first 15 minutes ($P < 0.05$) .[31]

4) Technique of Ventilation

Ventilation with normocapnia or mild hypocapnia has been advocated to minimize bleeding and optimize the surgical field during endoscopic sinus surgery

Mode of ventilation is important to control hypotension. Traditional intermittent positive pressure ventilation (IPPV) has a troublesome hemodynamic effect due to high intra-thoracic pressures and reduced venous return to the heart.[32] This result provides decreased blood circulation from the upper part of the body; therefore, it is a high risk for intra-operative bleeding. Conversely, high-frequency jet ventilation is small volume ventilation that provides adequate gas exchange at a lower pressure than intermittent positive pressure ventilation. A recent study compared efficacy between intermittent positive pressure ventilation and high frequency jet ventilation demonstrating that the total mean blood loss in the high-frequency jet ventilation group (170 ml) was significantly lower than the intermittent positive pressure ventilation group (318.8 ml; P = 0.017). The quality of the surgical field in high-frequency jet ventilation was significantly better than the intermittent positive pressure ventilation group (P = 0.012) .[33]

5) *Medications for Controlled Hypotension.*[28]

(a) Inhalation Anaesthetics. Controlled hypotension with inhalation agents (e.g., Isoflurane, Sevoflurane, and Desflurane) decreases arterial blood pressure through peripheral vasodilatation due to blockage of α-Adrenoceptors. However, higher concentrations can increase cerebral blood flow, increase the intracranial pressure, and deteriorate cerebral auto regulation. Therefore, a combination of inhalation anaesthetics with other drugs has been advocated to help reduce the concentration and adverse effects of each agent.[34]

(b) Intravenous Anaesthesia. Intravenous anaesthesia has been introduced for analgesia, hypnosis, sedation, and general anaesthesia (induction phase or maintenance phases). Agents currently used for general anaesthesia include propofol and opioids. Propofol has a depressant effect on the central nervous system via direct activation of the gamma-aminobutyric acid (GABA- [A]) receptors, inhibition of the n-methyl d-aspartate (NMDA) receptor, and modulation of the calcium influx through slow calcium ion channels. Propofol has rapid onset of action and recovery time with a dose-related effect. However, dose-dependent hypotension is its most common complication, especially high-dose infusions that are associated with propofol infusion syndrome. This condition is a potentially fatal complication with severe metabolic acidosis and circulatory collapse.[22]

Traditional opioids have been used as analgesic drugs and they bring some hypotensive effect. However, this effect is difficult to use for controlled hypotension due to their long half-life. Remifentanil is a new potent ultra short-acting μ opioid agonist with a short half-life; therefore, its action has a rapid onset and offset. It offers a reduction in sympathetic nervous system tone and dose-dependent effects, decreasing heart rate and blood pressure. In general anaesthesia, opioids are often used as an adjunct of intravenous-based technique (opioids combined with antihypertensive drugs or propofol) or inhalation-based technique (opioids combined with an inhalation agent). There is controversy regarding whether intravenous- or inhalation-based technique is more efficient for controlled hypotension.[28]

(c) Antihypertensive Drugs. Antihypertensive drugs have numerous classifications and mechanisms of action to control blood pressure.[35] Controversy exists regarding which is the ideal mean arterial pressure (MAP) in controlled hypotension to reduce bleeding and the correlation between blood loss and MAP. MAP at a very low level did not correlate with decreased intra-operative blood loss. However, severe hypotension may further reduced blood supply to vital organs.[28]

Surgical Considerations

There are many methods to deal with intra-operative bleeding during endoscopic sinus surgery. Their choice often depends on whether the bleeding is from venous or arterial origin, the size of the vessel, and its location.[36]

1. Topical Vasoconstrictors.

The aim of topical vasoconstrictors is to decongest the nasal cavity, thus widening the nasal corridor and minimizing bleeding. Commonly used topical vasoconstrictors include cocaine, epinephrine, phenylephrine, and oxymetazoline. All topical vasoconstrictors have potential adverse effects; therefore, the property of each agent should be considered.[28]

2. Local Anesthetic with Vasoconstrictor Injection.

Infiltration of a solution of local anaesthetic with vasoconstrictor has been introduced to minimize intraoperative bleeding. Haemostatic efficacy of local anaesthetic with vasoconstrictor was demonstrated in a study that showed decreased bleeding when lidocaine/epinephrine was injected as compared to injection of placebo ($P < 0.05$).[37] However in another study the intraoperative estimated blood loss was not significantly different between the anaesthetic/epinephrine and control groups ($P > 0.05$) .In another study conducted by Wormald et all they found that direst injection of, epinephrine into pterygopalatine fossa (containing the main nasal arterial supply) results in significantly better haemostasis ($P= 0.01$).[2] Arterial pressure and heart rate were affected immediately after injection of lidocaine/epinephrine but were not elevated over the normal range.[37]

3. Haemostatic Biomaterials.

Low-flow bleeding (capillary, venous, and small arteries) can be inhibited by the topical application of absorbable biomaterials. Recent development of numerous biomaterials has provided new methods for effective intraoperative and postoperative haemostasis, while avoiding complications, such as adhesions, excessive granulation tissue, and crusting.[28]

4. Topical Antifibrinolytics

Topical antifibrinolytics (i.e., epsilon-aminocaproic acid, tranexamic acid) mechanism of action is competitive binding with the lysine site on plasminogen. This prevents fibrinolysis and stabilizes the blood clot potentially decreasing further bleeding. However, the epsilon-aminocaproic acid was demonstrated to be ineffective in reducing intraoperative bleeding. Conversely, a low dose (100 mg) of tranexamic acid provided haemostasis and improved quality of the surgical field after application.[38]

5. Gelatin-Thrombin Matrix

Topical matrix sealant consists of human thrombin and gelatin matrix granules of bovine or porcine gelatin. It provides tamponade of injured vessels and rapid clot formation on the tissue surface. Topical gelatin-thrombin matrixes have been modified to allow their use during endoscopic endonasal skull base surgery. It stops bleeding on an average of 2 minutes (range 1–5 minutes) after its application. A recent study showed that bovine gelatin could effectively stop bleeding from the venous sinus. but has adverse effects such as extensive loss of cilia on the epithelium, significant increase of adhesion ($P < 0.05$) and granulation tissue formation ($P < 0.05$).[39.40.41]

6. Micro porous Polysaccharide Hemispheres

Micro porous polysaccharide hemispheres are a novel haemostatic biomaterial agent produced from purified potato starch that acts to dehydrate blood and concentrate blood components, including platelets, red blood cells, and clotting factors. Adverse effects, including synechiae formation, oedema and infection were noted

Other biomaterial agents such as oxidized methyl cellulose, fibrin glue , microfibrillar collagen , and gelatin sponges can be used to control intra-operative bleeding. However, there is a lack of scientific evidence comparing the efficacy of these agents.[28]

7. Hot Water Irrigation

Hot water irrigation was originally introduced as a treatment of epistaxis. The haemostatic mechanism of hot water irrigation is unclear but may include

1) Oedema and narrowing of the intranasal lumen that contributes to the compression of the leaking vessel

2) Decreasing the flow and the intraluminal blood pressure due to mucosal vasodilatation; and

3) Cleaning of blood coagulates from the nose.[42]

Hot water irrigation for epistaxis is simple and effective, less painful, and less traumatic to the nose than nasal packing therefore, this technique was adopted to reduce intraoperative bleeding. Hot water irrigation with 40^{o} –42^{o} saline reduces diffuse oozing from sinonasal mucosa as well as intracranial bleeding from minor vessels. Another benefit of warm water irrigation is that it allows the cleaning of the endoscopic lens.[28]

MATERIALS & METHODS

MATERIALS AND METHODS

Methods

A total of 32 patients presenting to the department of Otorhinolaryngology and Head and Neck surgery of R L JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR who were diagnosed as chronic Rhinosinusitis or polyposis from December 2014 to June 2016 was taken up for the study.

Written informed consent was obtained from all patients for study who were willing for the surgery.

Patients were randomised into test and control side based on 6 block randomization method. A series of random numbers were generated using computer software and based on the series, using 6 blocks patients were randomised on which side the test and control drug was to be given

Functional Endoscopic Sinus surgery was done on both sides . Test side were given 2% lignocaine with 1:80000 adrenaline and the control side Normal saline was used as placebo.

Surgeon was blinded as to which side was the test drug and placebo was given

PPF infiltration was done using Mercuri technique after identifying the greater palatine foramen .Standard 26 gauge $1^{1/2}$ inch needle which was bend 45 degree at 25 mm from the tip was used to infiltrate the PPF under sterile precautions

Surgical field assessment was done every 15 minutes to assess the bleeding and was graded using Boezart and Van der Merve scale. Other parameters like mean arterial pressure, Heart rate, blood pressure , Residual CO_2 were recorded and maintained constant

Data was compiled in Microsoft excel data sheet and was analyzed using SPSS software. Qualitative data was represented in the form of frequencies and proportions. Chi-square was used as the test of significance for qualitative data. Quantitative data was represented in the form of Mean and Standard deviation. Student's t test was the test of significance for quantitative data. P value < 0.05 will be considered as statistically significant

Sample size: 32 patients were included into the study

Randomization : Block randomization

Type of study design : Prospective Single blind comparative study

Table 6.Boezaart and Van Der Merve Endoscopic Grading of Nasal Bleeding

Grade	Endoscopic Grading of Nasal Bleeding-
0	No bleeding (cadaveric conditions)
1	Slight bleeding—no suctioning required
2	Slight bleeding—occasional suctioning required
3	Slight bleeding—frequent suctioning required; bleeding threatens surgical field a few seconds after.
4	Moderate bleeding—frequent suctioning required and bleeding threatens surgical field directly after
5	Severe bleeding—constant suctioning required; bleeding appears faster than it can be removed by suction is removed suction is removed suction; surgical filed severely threatened and surgery usually is not possible

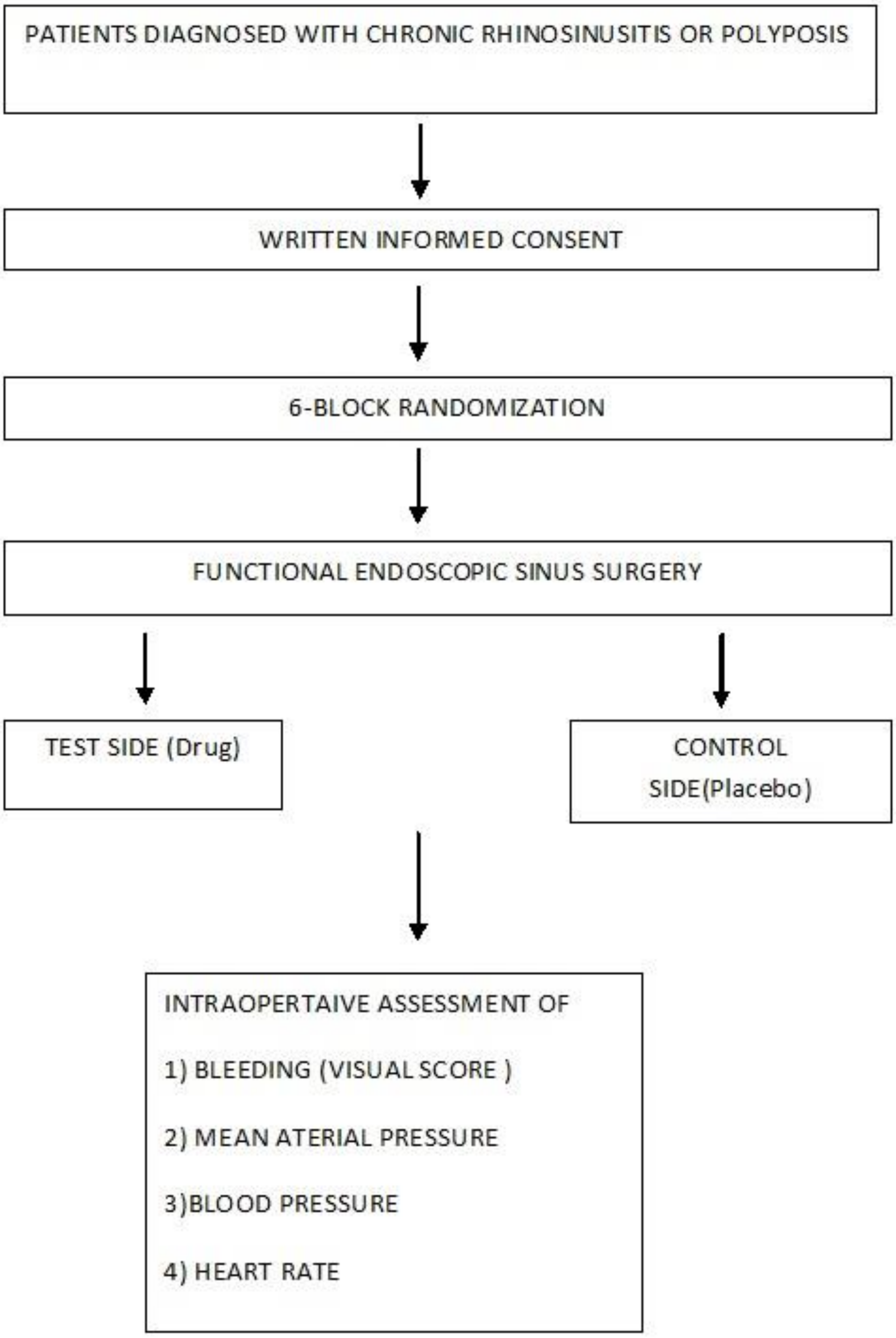

Figure 12 : Study protocol

Inclusion criteria:

1. All patients above the age 18 years with chronic rhinosinusitis or polyposis who are undergoing endoscopic sinus surgery

Exclusion criteria:

1. Asymmetrical disease
2. Poorly controlled hypertension
3. Bleeding disorders
4. Use of Anticoagulants
5. Acute infections

Method of collection of data:

Cases selected for the study was subjected to a detailed clinical history and complete ENT examination. All patients underwent diagnostic nasal endoscopy preoperatively to confirm the diagnosis and was graded based on Lund and Mackay system. Disease were also staged based on CT imaging using the Lund and Mackay system

Table 7.The Lund and Mackay Staging System: CT Appearance score

SINUS SYSTEM	LEFT	RIGHT
Maxillary (0/1/2)		
Anterior Ethmoids (0/1/2)		
Posterior Ethmoids (0/1/2)		
Sphenoid (0/1/2)		
Frontal (0/1/2)		
Osteomeatal complex (0 or 2)		
Total points :		

0, No abnormalities; **1,** Partial opacification**; 2,** Total opacification.
0, Not occluded**; 2,** Occluded.

Table 8. The Lund and Mackay staging system: endoscopic appearance score

Characteristic	Baseline
Polyp **--** > Left / Right (0 ,1,2,3)	
Oedema --> Left / Right (0 ,1,2,3)	
Discharge --> Left / Right (0 ,1,2,3)	

Polyps: 0, Absence of polyps; 1, Polyps in middle meatus only; 2,Polyps beyond the middle meatus, but not completely obstructing the nose; 3, Polyps completely obstructing the nose.
Oedema: 0, Absent; 1, Mild; 2, Severe.
Discharge: 0, No discharge; 1, Clear, thin discharge; 2, Thick purulent discharge.

Cases were investigated in the following manner :

1) Haemoglobin , Total leucocyte count , Differential leucocyte count , Bleeding time, Clotting time ,Blood grouping and Rh typing

2) Urine for sugar , Albumin and Microscopy

3) Chest x-ray

4) ECG

5) X-ray of paranasal sinuses - water's view to note the condition of the paranasal sinuses

A correlation was established between clinical features and radiological findings. After complete pre-operative assessment, patients were subjected to surgical intervention.

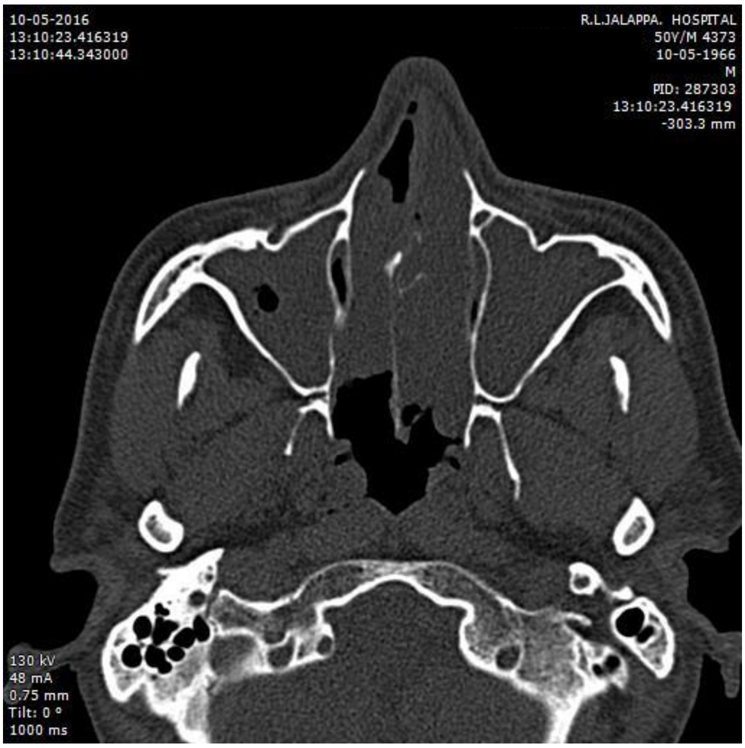

Figure 13: CT-Axial section showing bilateral maxillary sinus polyp

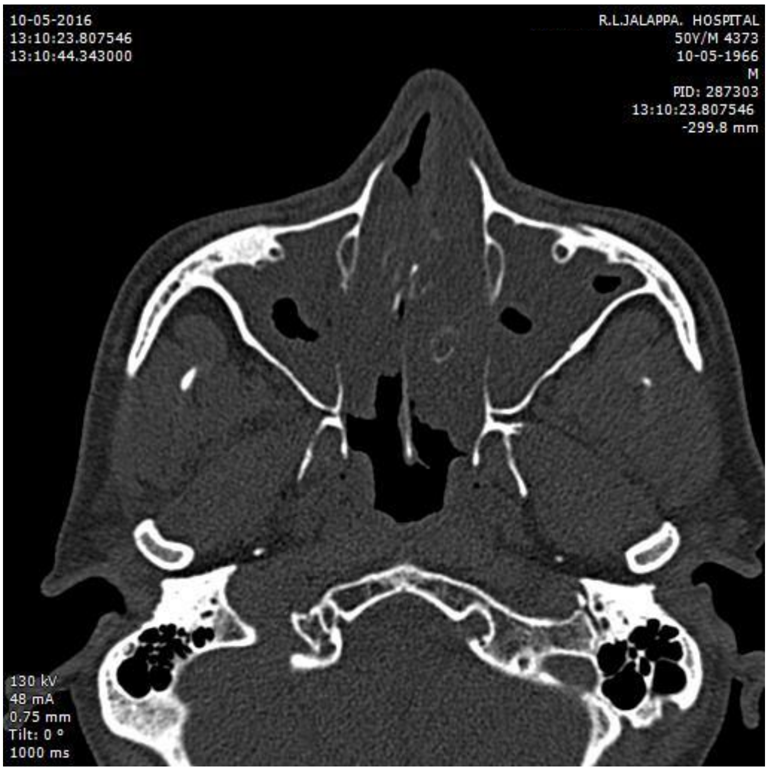

Figure 14: CT-Axial section showing bilateral osteomeatal complex occlusion

PROCEDURE

INJECTION TECHNIQUE

The greater palatine foramen is located over the hard palate just anterior to the posterior edge of the hard palate opposite the second molar tooth, halfway between the tooth and the midline of the hard palate.

The greater palatine foramen was identified by palpating the palate with a finger, after depressing the tongue using a tongue depressor. An endoscope was used for visualizing. The posterior free edge of the hard palate was first palpated and then moved anteriorly over this ridge onto the hard palate. The foramen can be felt as a depression directly anterior to the free edge about midway between the second molar tooth and the midline of the palate

This region on the hard palate is identified on the monitor as the finger is withdrawn from the mouth.

The needle used for infiltration was bent at 25 mm from the tip at an angle of 45 degrees in order to perform an effective infiltration of the pterygopalatine fossa. This enables the tip of the needle to just penetrate the pterygopalatine fossa without putting any of its contents at risk (The opening of the foramen into the canal is funnel shaped and the canal is angled at 45 degrees to the hard palate)

With the needle bent at 25 mm and at a 45 degree angle, the needle was inserted into the palate. If the needle struck the bone, then a small amount of lignocaine was infiltrated and the needle was withdrawn assuming that the needle had just missed the foramen and that a slight adjustment was needed to locate the foramen.

If repeated attempts to identify the foramen failed, then the landmarks for the foramen were reassessed and it was relocated.

After aspirating to ensure that the needle was not in a blood vessel, the pterygopalatine fossa was infiltrated with the test drug (2% Xylocaine and 1:80000 Adrenaline) and the placebo (Normal saline).

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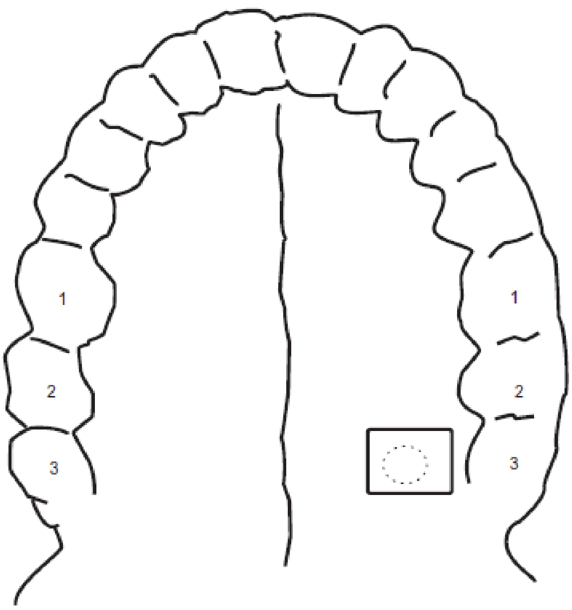

Figure 15 : Schematic drawing demonstrating the position of the left greater palatine foramen in line with the third upper molar tooth on the left hand side

Figure 16 : Needle used to perform the procedure

Statistical analysis:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi-square was used as test of significance. Continuous data was represented as mean and standard deviation. Independent t test or Mann Whitney U test was used as test of significance to identify the mean difference between two groups. p value <0.05 was considered as statistically significant.

Table 9. Correlation Coefficient and Interpretation

Correlation coefficient (r)	**Interpretation**
0 - 0.3	**Positive Weak correlation**
0.3-0.6	**Positive Moderate correlation**
0.6-1.0	**Positive Strong correlation**
0 to (-0.3)	**Negative Weak correlation**
(-0.3) to (-0.6)	**Negative Moderate Correlation**
(-0.6) to – (1)	**Negative Strong Correlation**

RESULTS

Table 10**:** Side of the Test and Control nose

		Group			
		Test side		Control Side	
		Count	%	Count	%
Side of Drug	Left	12	37.5%	20	62.5%
	Right	20	62.5%	12	37.5%

In our study after 6 block randomization 12 patients received the test drug on the left side and the remaining 20 patients received the drug on the right side

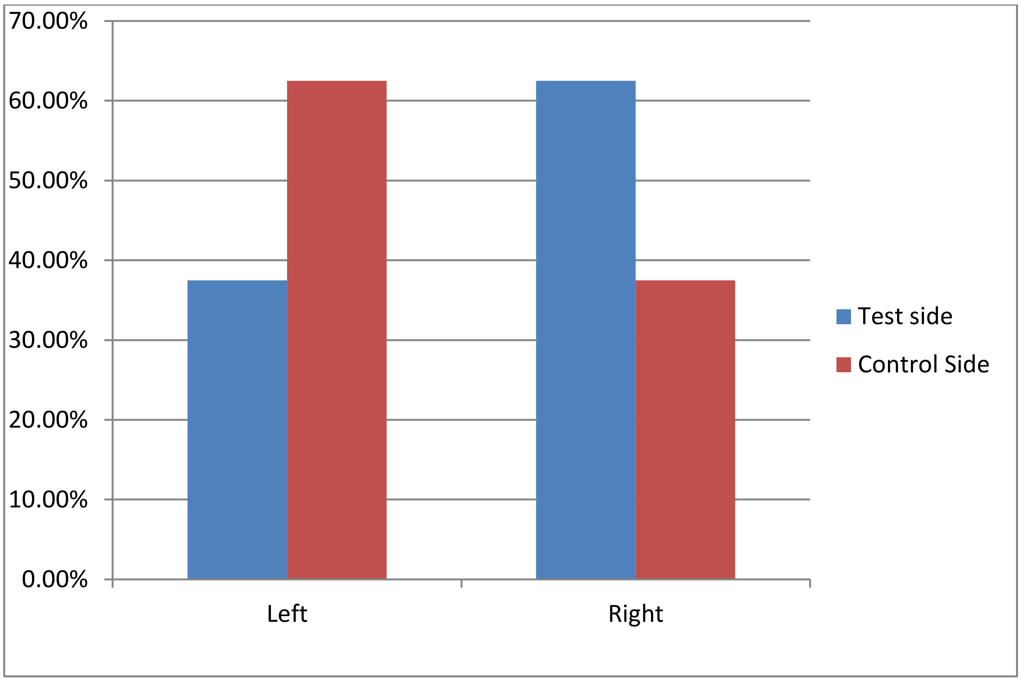

Figure 17: Bar diagram showing Side used as test and control

Table 11: Age distribution of the subjects in the study

		Frequency	Percent
Age	< 20 years	4	12.5
	21 to 30 years	6	18.8
	31 to 40 years	7	21.9
	41 to 50 years	8	25.0
	> 50 years	7	21.9
	Total	32	100.0

The mean age of subjects in the study was 39.1 ± 15.2 years.

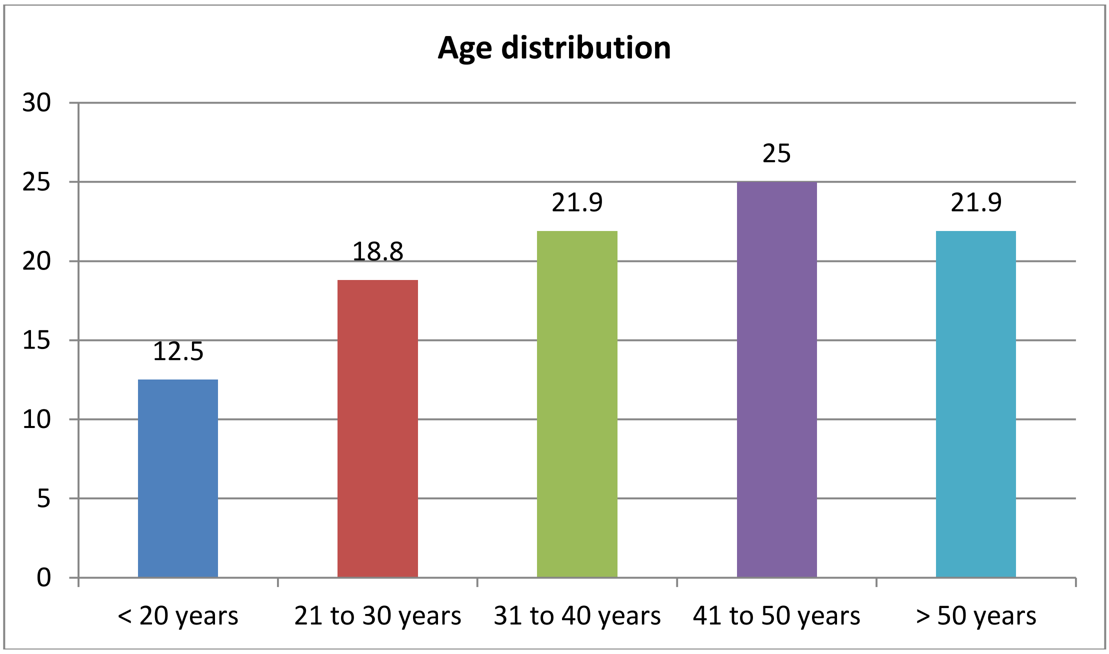

Figure 18: Bar diagram showing Age distribution of the subjects in the study

Table 12: Gender distribution of subjects in the study

		Frequency	Percent
Gender	Female	13	40.6
	Male	19	59.4
	Total	32	100.0

In our study 59.4% were males and 40.6% were females.

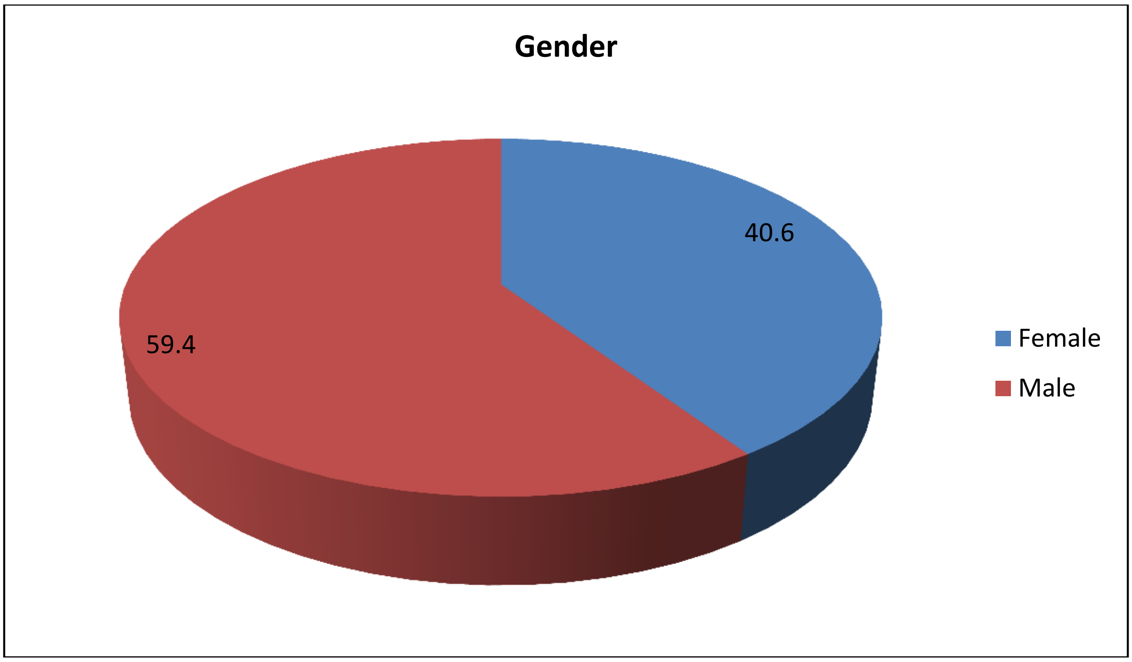

Figure 19: Pie diagram showing gender distribution of subjects

Table 13: Duration of lesion in subjects

		Frequency	Valid Percent
Duration	< 6 months	12	37.5
	6 months to 1 year	12	37.5
	> 1 year	8	25.0
	Total	32	100.0

In our study the Mean duration of illness was 1.47 years. Majority of subjects 75% had lesion<1 year.

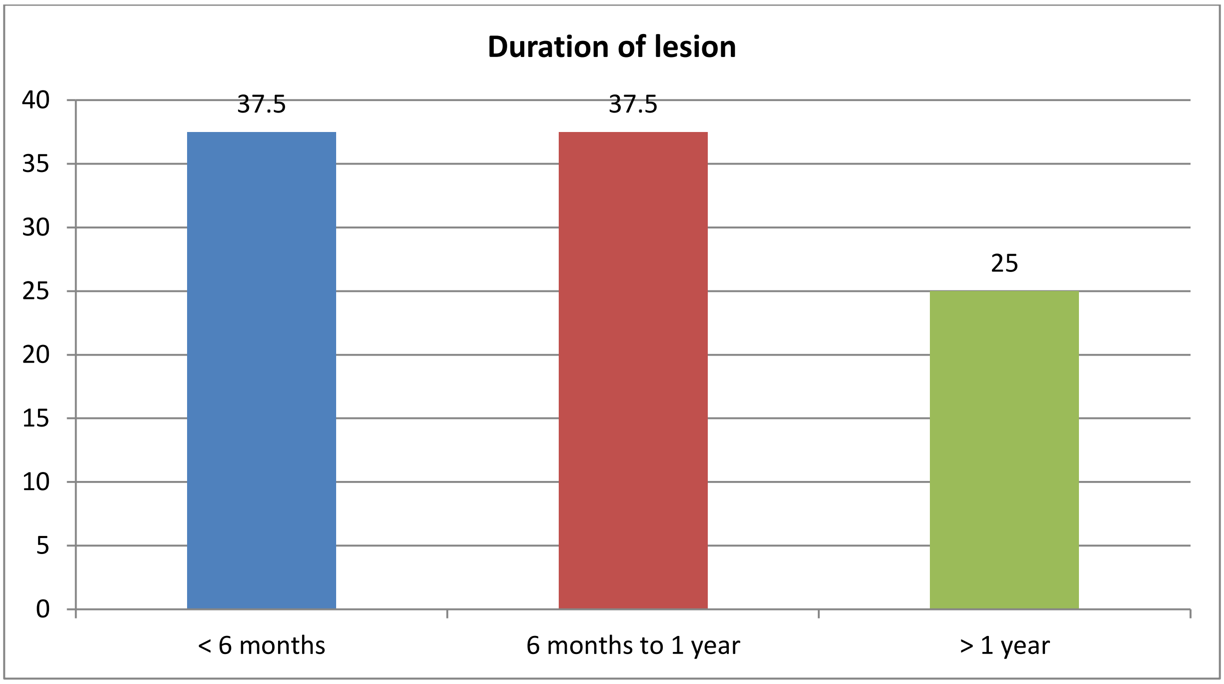

Figure 20: Bar diagram showing Duration of lesion in subjects

Table 14 : Diagnosis in study subjects

		Count	%
Diagnosis	Ethmoidal polyposis	18	56.25
	Chronic Rhinosinusitits	10	31.25
	Chronic Rhinosinusitits with DNS	2	6.25
	Fungal sinusitis	2	6.25
	Total	32	100.0

In our study 50% of subjects were diagnosed to have Ethmoidal polyposis, 25% with Chronic Rhinosinusitits and 6.25% with Chronic Rhinosinusitits with DNS, fungal sinusitis and respectively.

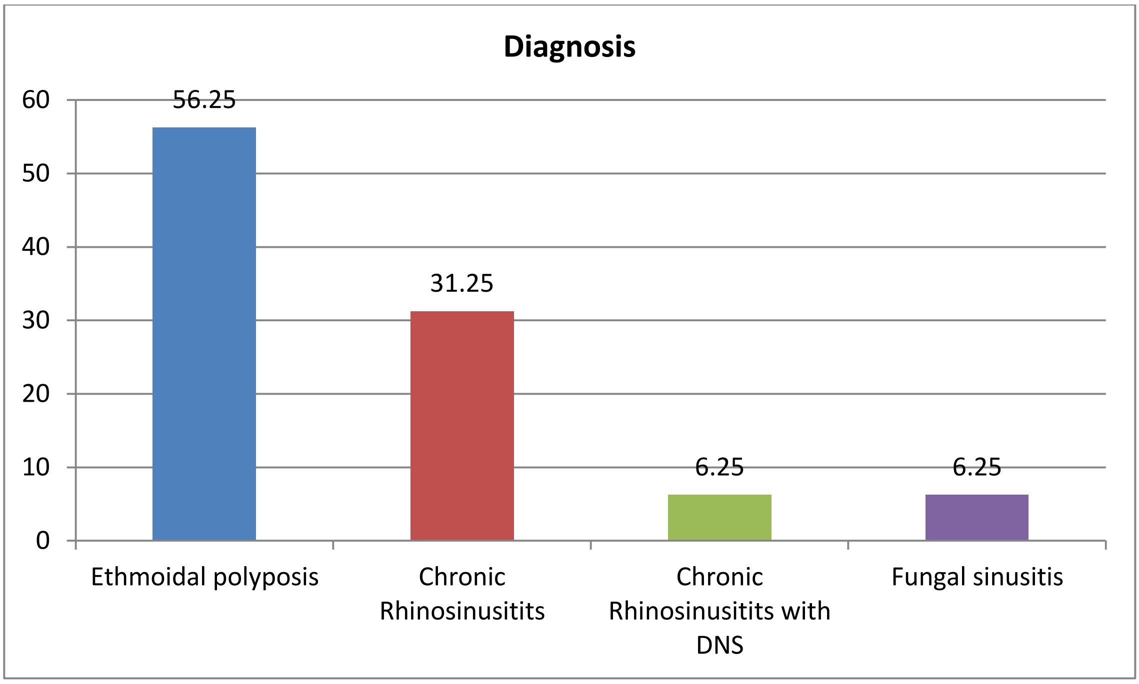

Figure 21: Bar diagram showing Diagnosis in study subjects

Table 15: Vital signs in subjects

	Mean	SD
Heart Rate	76.0	8.6
SBP	123.2	10.7
DBP	82.4	8.1
MAP	89.3	6.4

Mean Heart rate was 76 ± 8.6 bpm, SBP was 123.2 ± 10.7 mm Hg, DBP was 82.4 ± 8.1 and MAP was 89.3 ± 6.4.

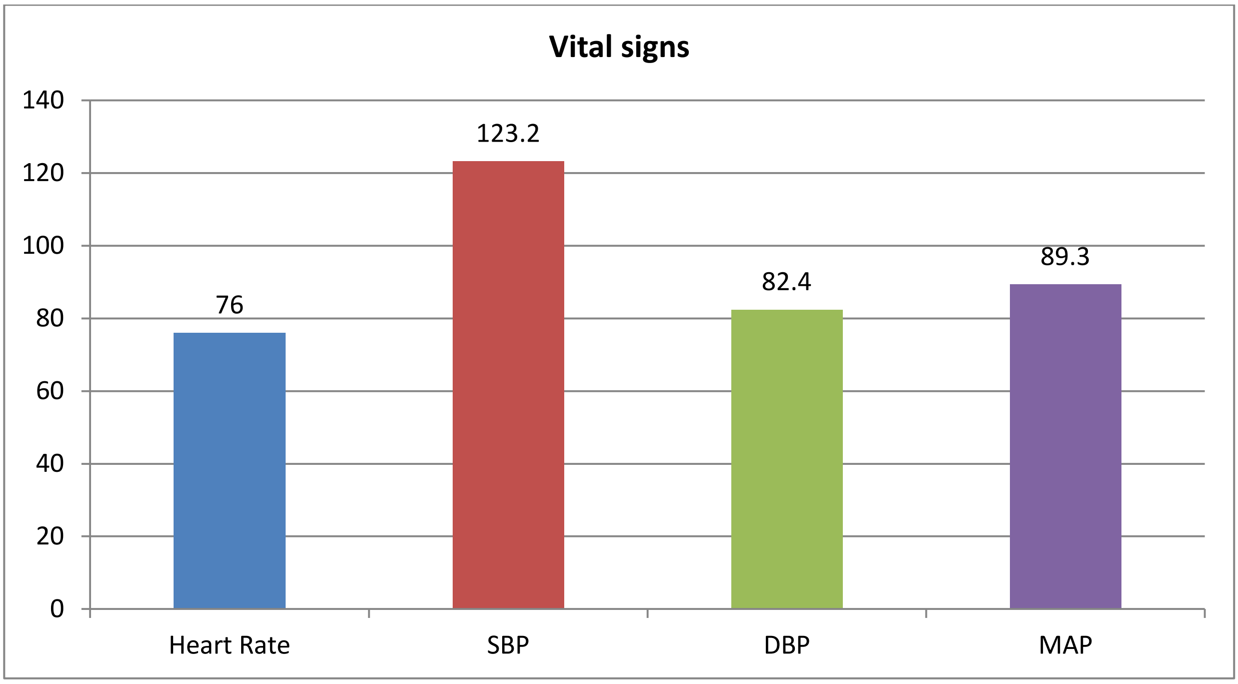

Figure 22: Bar diagram showing Vital signs in subjects

Table 16: Blood loss in subjects between two groups

	Group				P value
	Test side		Control Side		
	Mean	SD	Mean	SD	
Blood Loss Grade	9.0	3.5	10.9	3.5	0.034*

Mean Blood loss grade on test side was 9.0 ± 3.5 and in control side was 10.9 ± 3.5. This difference in mean blood loss was statistically significant.

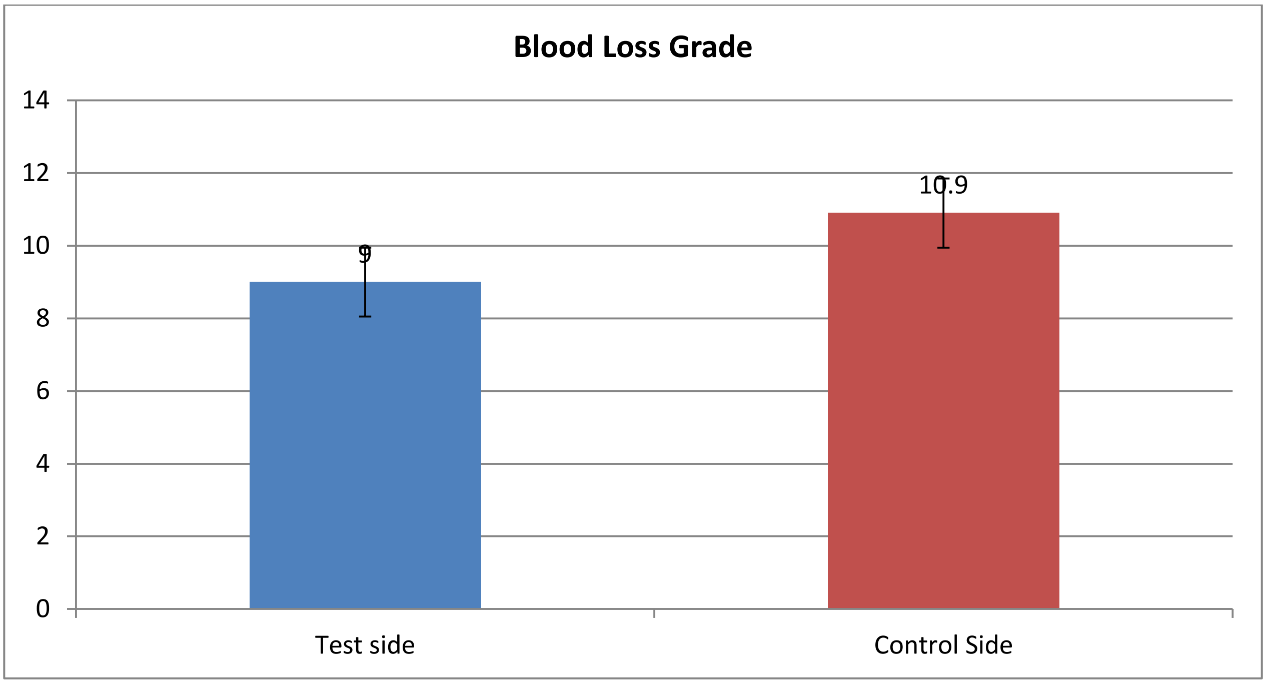

Figure 23: Bar diagram showing Blood loss in subjects between two groups

Table 17: Comparison of CT scan grade and Endoscopic grade between two groups

	Group						P value
	Test side			Control Side			
	Mean	SD	Median	Mean	SD	Median	
CT Scan	4.5	1.3	4	4.7	1.5	5	0.710
Endoscopic Grading	1.9	1.0	2	1.8	1.0	2	0.487

In our study the Median CT grade in test side was 4 and in control side was 5. Median endoscopic grade in Test side was 2 and in control side was 2. There was no significant difference in Median scores between two groups.

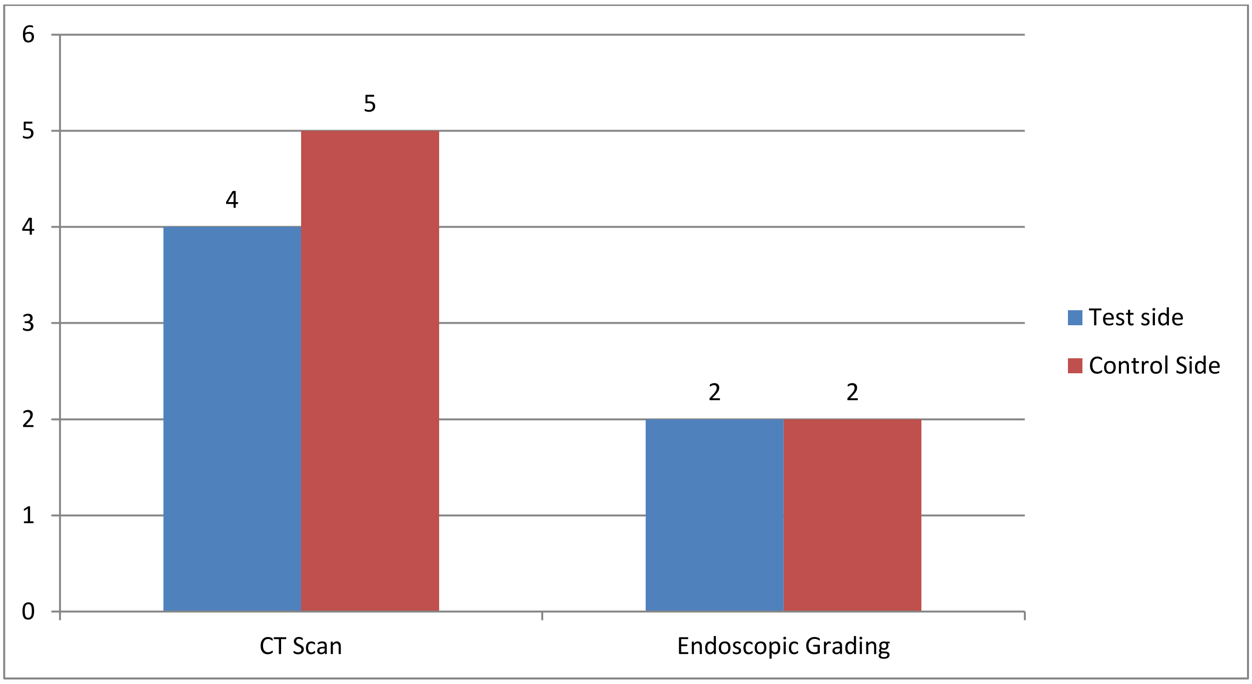

Figure 24: Bar diagram showing Comparison of CT scan grade and Endoscopic grade between two groups

Table 18: Spearman's Correlation between CT scan, Endoscopic grade with blood loss grade on test side

			Blood loss grade
Spearman's rho	CT Scan grade	Correlation Coefficient	0.260
		P value	0.150
		N	32
	Endoscopic Grading	Correlation Coefficient	0.464**
		P value	0.007
		N	32

In our study non significant positive correlation was observed between CT scan grade and blood loss grade. I.e. with increase in CT grade there was increase in blood loss grade.

Were as significant positive correlation was observed between Endoscopic grade and blood loss grade. I.e. with increase in Endoscopic grade there was increase in blood loss grade and vice versa.

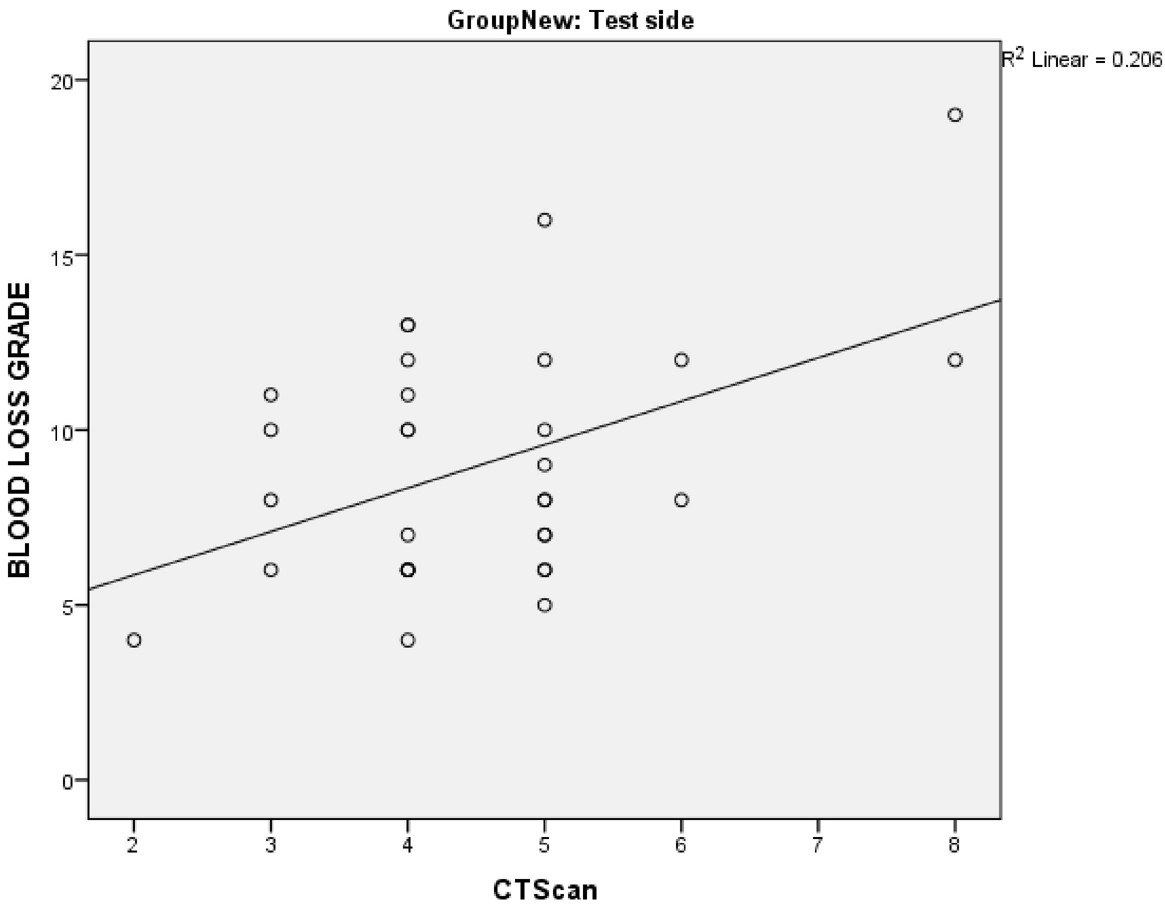

Figure 25: Scatter plot showing correlation between CT scan grade and Blood loss grade in test side

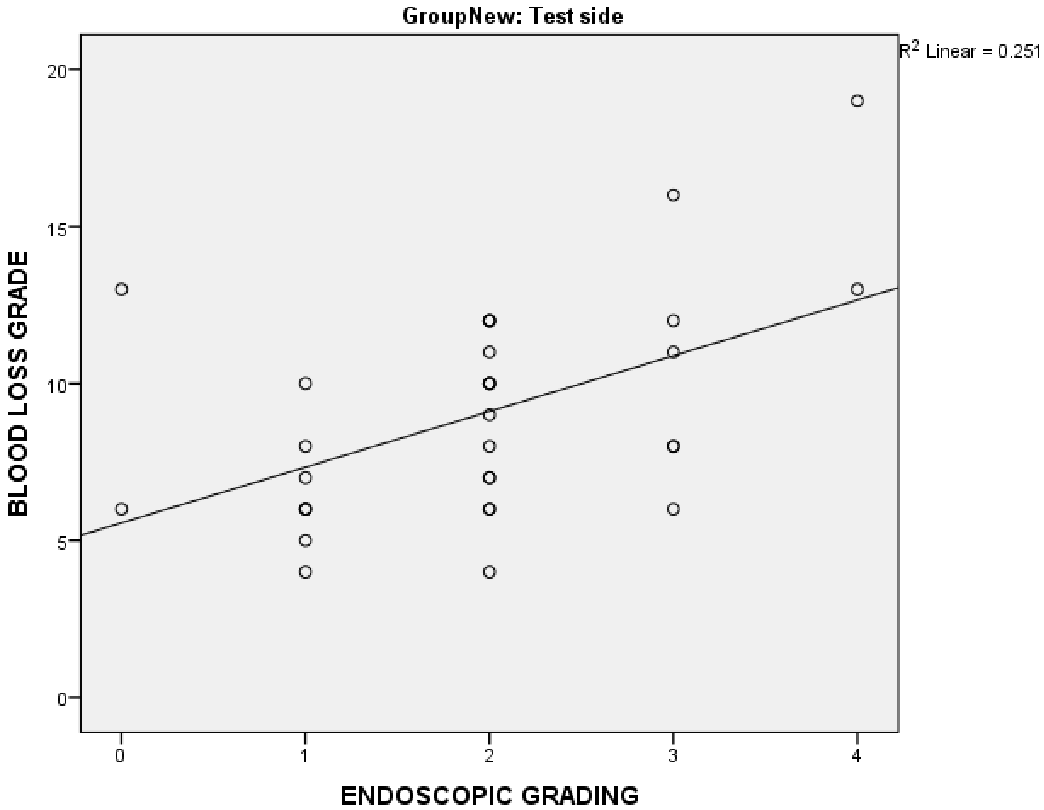

Figure 26: Scatter plot showing correlation between Endoscopic grade and Blood loss grade in test side

Table 19: Spearman’s Correlation between CT scan, Endoscopic grade with blood loss grade on control side

			Blood Loss Grade
Spearman's rho	CT Scan grade	Correlation Coefficient	0.172
		P value	0.347
		N	32
	Endoscopic Grading	Correlation Coefficient	0.410*
		P value	0.02*
		N	32

In the study non significant positive correlation was observed between CT scan grade and blood loss grade in control side, i.e., with increase in CT grade there was increase in blood loss grade.

Were as **significant** positive correlation was observed between Endoscopic grade and blood loss grade in control side, i.e., with increase in Endoscopic grade there was increase in blood loss grade and vice versa.

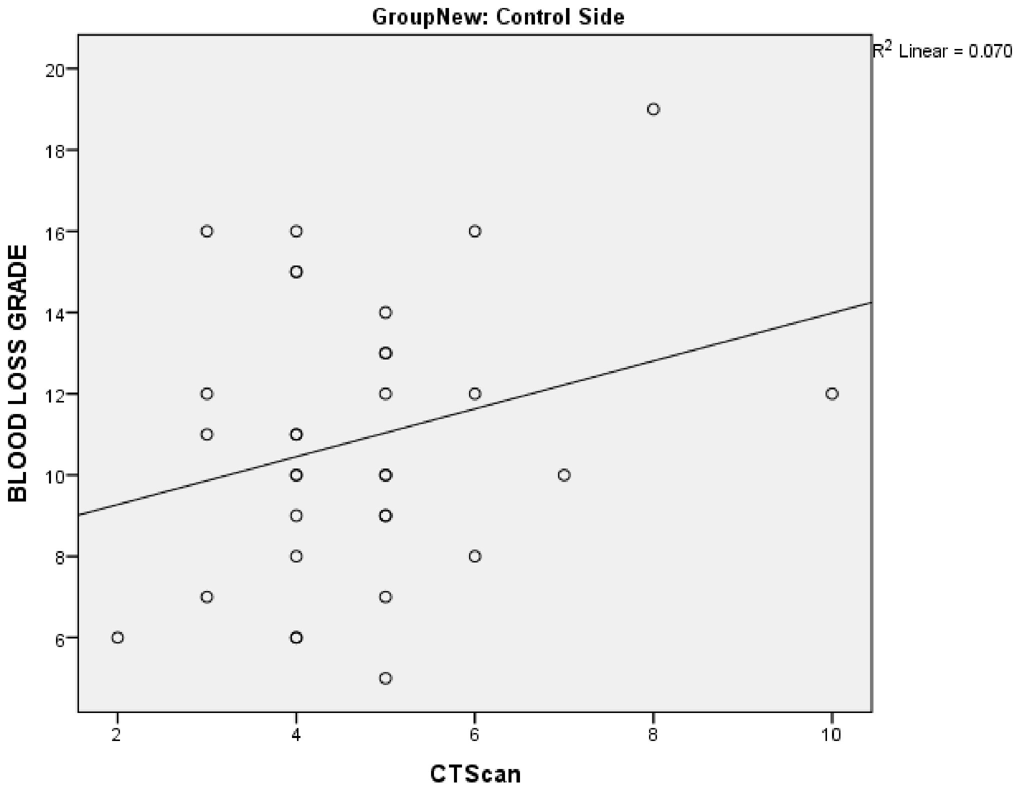

Figure 27: Scatter plot showing correlation between CT scan grade and Blood loss grade in control side

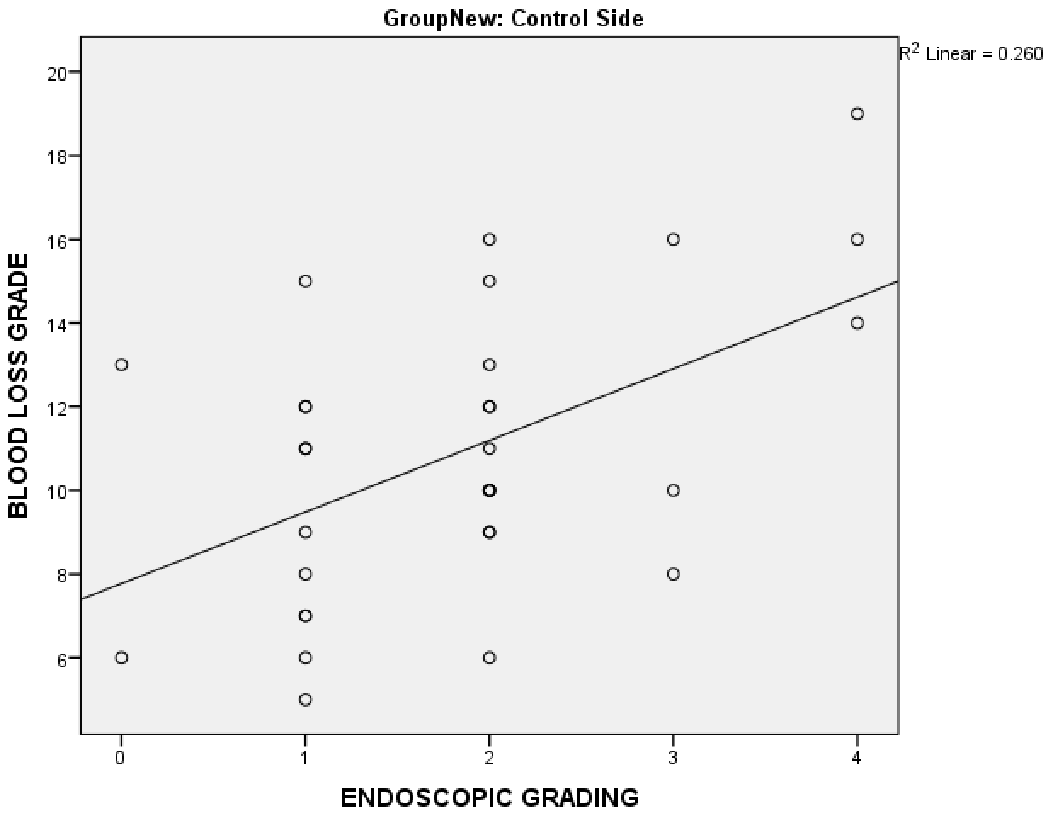

Figure 28: Scatter plot showing correlation between Endoscopic grade and Blood loss grade in control side

Table 20: Correlation between blood loss grade and vital parameters in subjects

		Heart Rate	SBP	DBP	MAP
Blood Loss Grade	Pearson Correlation	0.549**	0.399*	0.353*	0.530**
	P value	0.001	0.024	0.047	0.002
	N	32	32	32	32

In our study Significant positive correlation was observed between blood loss grade and heart rate, SBP, DBP and MAP respectively. That is, with increase in Blood loss there was increase in Heart rate, SBP, DBP and MAP respectively and Vice versa.

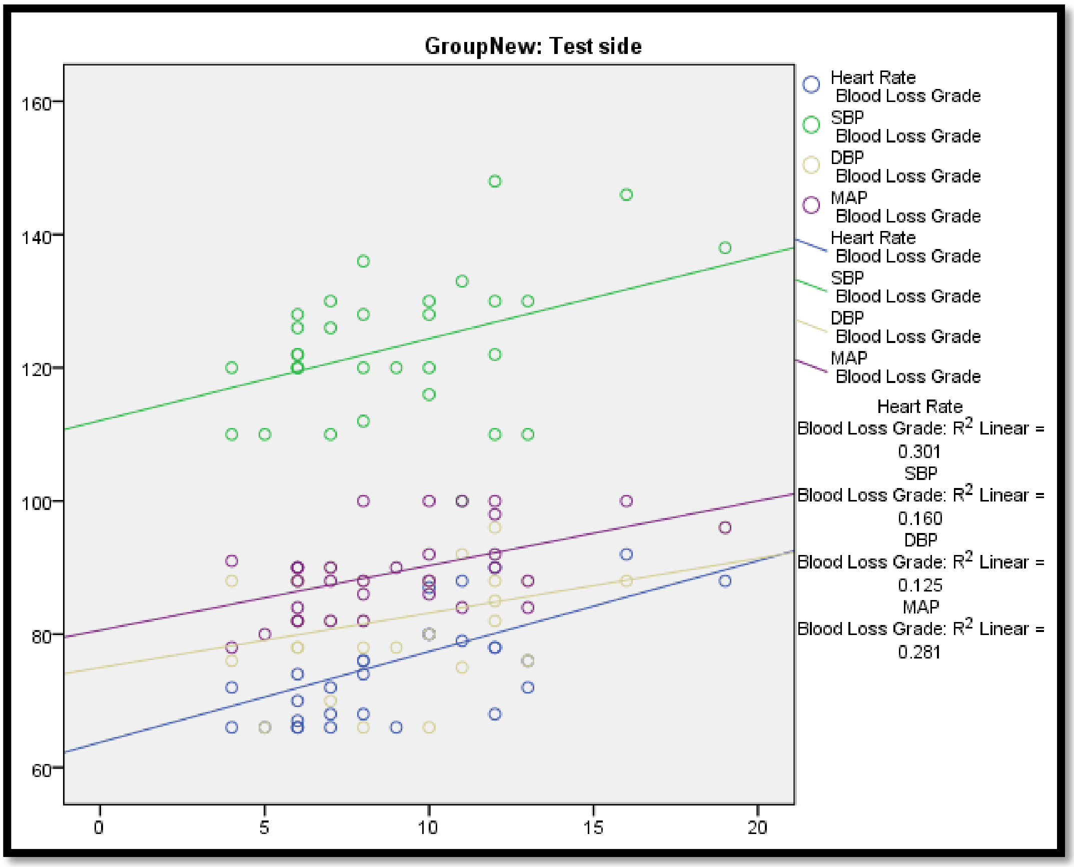

Figure 29: Over lay Scatter plot showing correlation between blood loss grade and vital parameters in test side

DISCUSSION

DISCUSSION

Functional endoscopic sinus surgery is one of the most common operative ENT procedures for chronic rhinosinusitis with nasal polyposis which is refractory to medical treatment . Visualization of the nasal anatomy is vital to the procedure, allowing complete dissection, and lessening complications. Bleeding from the nasal mucosa during ESS interferes with the surgical field, prolongs operative time, and increases the incidence of incomplete surgery. Hence, most surgeons agree that haemostasis is important.[43]

In our study most of the patients were adults in the age group of 20-50 years. The mean age of the subjects our study was 39.1 ± 15.2 years .Similarly in a study conducted by Wormald et al the median age was 50 years (range , 20-78 years).[2]In another study conducted it was found that majority of the patients who presented were also in the age group of 16 to 45 years.[44]

In our study males were more in number with 19 patients (59.4%) being males and 13 patients (40.62%) being females . In all other studies that were compared males were predominant in number. This is probably due to the number of males coming forward seeking surgical treatment in response to symptoms. In our study the mean duration of illness was 1.47 years. Majority of subjects, i.e., 75% had symptoms for < 1 year.

In our study 50% of subjects were diagnosed to have Ethmoidal polyposis, 25% had Chronic Rhinosinusitits and 6.25% had Chronic Rhinosinusitits with DNS, Bilateral Ethmoidal polyposis, fungal sinusitis and respectively. In another study conducted the most common condition was chronic rhinosinusitits (50%) and the remaining 50 % were diagnosed as polyposis and fungal sinusitis.[2]

In our study the Mean Heart rate was 76 ± 8.6 bpm, SBP was 123.2 ± 10.7 mmHg, DBP was 82.4 ± 8.1 and MAP was 89.3 ± 6.4. In our study however significant positive correlation was observed between blood loss grade and heart rate, SBP, DBP and MAP respectively, that is with increase in Heart rate, SBP, DBP and MAP there was proportional increase in blood loss . However this is contradictory to another study done by Wormald et al. where they have not found any statistically significant correlation between MAP and surgical grade (P value = 0.724).[2]

It was found that, in spite of higher vital parameters (MAP,BP, HR) we could achieve a reasonably bloodless surgical field after infiltration of the PPF with the test drug

In a similar study done by Sieśkiewicz A et al, they concluded that significant correlation was present between mean arterial pressure and conditions in the operating field (p value =0.003).[45] They attained good operative field with a mean arterial pressure in the range of 65-78 mm Hg . In this study no correlation was found between heart rate and bleeding in the operating field as the authors had maintained heart rate in the range of 60 / min 2.[45]

In our study the mean Blood loss grade on test side was 9.0 ± 3.5 and in control side was 10.9 ± 3.5. This difference in mean blood loss was statistically significant . P =0.034.

Wormald et al, conducted a study in 2005 which showed statistically significant benefit in favour of the injected site when comparing endoscopic surgical field. The overall mean blood loss grade of the injected site was 2.59 when compared to 2.99 for the non injected site.[2]

In another study conducted by Valdes et al they found no statistical difference between injected and non injected site in terms of surgical grade and blood loss . Furthermore there was no correlation between surgical field improvement ,MAP , heart rate either.[46]

In a study conducted by Salah A. Ismail they concluded that bilateral sphenopalatine ganglion block via the greater palatine fossa provided a satisfactory operative field with less blood loss.[47]

In our study, the Median CT grade in test side was 4 and in control side was 5 (P =0.710). Median endoscopic grade in test side was 2 and in control side was 2 (P = 0.487) There was no significant difference in Median scores between the test and control sides

In our study, non significant positive correlation was observed between CT scan grade and blood loss grade in both the test and the control side. I.e. with increase in CT grade there was increase in blood loss grade. P Value was 0.150 and the P Value was 0.347 respectively

Significant positive correlation was observed between Endoscopic grade and blood loss grade in the test and control side, i.e. , with increase in Endoscopic grade there was increase in blood loss grade and vice versa. The P value was 0.007 and P value was 0.02 respectively

CONCLUSION

CONCLUSION

Achieving a blood less field during functional endoscopic sinus surgery remains a challenge to date

In our study we found, that infiltration of the pterygopalatine fossa with 2% Xylocaine and 1:80000 adrenaline helped in minimising bleeding during the procedure providing the surgeon with better visualization of all anatomical landmarks and pathology . We found that the mean blood loss between test side and the control side was statistically significant and was found to be in agreement with the breakthrough study conducted by Wormald et al.

Furthermore it was found that the grade of bleeding was dependent on various factors like mean arterial pressure, blood pressure and heart rate. There was a proportional increase in bleeding with increase in the above mentioned parameters. Hence to achieve a relatively blood less field during the procedure it is imperative that the above vital parameters be maintained

We also found out that in extensive diseases such as fugal sinusitis, bleeding could not be minimized even with infiltration of the pterygopalatine fossa

Conducting a study on a larger sample size will help us to better understand the significance of infiltration of pterygopalatine fossa with 2% Xylocaine and 1:80000 Adrenaline in minimizing bleeding during functional endoscopic sinus surgery.

SUMMARY

SUMMARY

This study was done on patients with chronic rhinosinusitis and polyposis attending to the ENT outpatient department at R L Jalappa Hospital, Kolar from December 2014 to June 2016.

Patients were selected based on the inclusion and exclusion criteria and were randomly divided into two groups by a 6 block randomisation method. All patients underwent Functional endoscopic sinus surgery following infiltration of the test drug and placebo in the pterygopalatine fossa .Intra-operatively surgical field assessment was done every 15 minutes to assess the bleeding and was graded using Boezart and Vander Merve scale.

- Maximum incidence was seen in the 5th decade.
- 59.4% were males and 40.6% were females.
- Ethmoidal polyposis was the most common diagnosis (50%), followed by Chronic rhinosinusitis (25%) and 6.25% had Chronic Rhinosinusitits along with DNS, and fungal sinusitis respectively.
- Majority of the patient had symptoms less than one year
- Mean Blood loss grade on test side was 9.0 ± 3.5 and in control side was 10.9 ± 3.5. and the difference was statistically significant.

- The Median CT grade and the endoscopic grade of disease in test side and control side were almost similar. There was no significant difference in Median scores between the two groups.

- Non significant positive correlation was observed between CT scan grade and blood loss grade in both the test and the control side.

- Significant positive correlation was observed between Endoscopic grade and blood loss grade in the test and control side

- No complications were encountered during the procedure.

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ANNEXURES

ANNEXURE

PROFORMA

Effect of pterygopalatine fossa with 2% lignocaine and 1:80000 adrenaline in minimizing bleeding during endoscopic sinus surgery
SRI DEVARAJA URS MEDICAL COLLEGE

PERSONAL DETAIL:
Name: Age: sex: | M | F |
Hospital Number: Date: __/__/__
Address:

Telephone number:

PRESENTING COMPLAINTS:

COMPLAINTS	YES/NO	SINCE
Nasal obstruction		
Nasal discharge		
Headache		
Hyposmia/ Anosmia		
Sneezing		
Postnasal drip		
Facial pain		
Others(specify) • •		

PAST HISTORY:
H/O Hypertension, Allergies, Bleeding disorder, Asthma, Drug Allergy,

H/O any previous | Yes | No | surgeries

Treatment History : (if any)

FAMILY HISTORY: | Contributory | Non Contributory |

GENERAL PHYSICAL EXAMINATION:
Built: Poor/Medium/Well Built Nutritional status: Poor/Satisfactory
Temperature: Pulse: BP: RR:
Pallor :Y/N Icterus:Y/N Cyanosis:Y/N
Clubbing: Y/N Lymphadenopathy:Y/N Oedema:Y/N

ENT Examination :

Examination of Nose:
External framework:
Vestibule:
Anterior Rhinoscopy:

Nasal cavity:
Septum:
Mucosa:
Turbinates:
Floor of nose:

Sinuses:
Oral cavity and Oropharynx:
Ear:

DNE findings:

	Right side	Endoscopic grading	Left side	Endoscopic grading
1st pass				
2nd pass				
3rd pass				

CT scan findings (Lund Mackay grading)

Sinus	Left	Right
Maxillary(0/1/2)		
Anterior ethmoids(0/1/2)		
Posterior ethmoids(0/1/2)		
Sphenoid(0/1/2)		
Frontal(0/1/2)		
Osteomeatal complex(0/2)*		
TOTAL		

0 No Abnormality, **1** partial opacification, **3** total opacification
*0 Not occluded, 2 occluded

SYSTEMIC EXAMINATION:

Cardio Vascular System: Respiratory System:
Per Abdomen: Central Nervous System:

OPERATIVE FINDINGS:

INTRAOPERATIVE FINDINGS:

GRADING OF BLEEDING: Boezaart and Van Der Merv scale)

TIME	RIGHT	LEFT
0 Mins		
15Mins		
30Mins		
45Mins		
1hour		
1:15Mins		
1:30Mins		
1:45Mins		
2Hours		
2:15Mins		
2:30Mins		

0 No bleeding (cadaveric condition)
1 slight bleeding-no suctioning required
2 Slight bleeding-occasional suctioning required,
3Slight bleeding-frequent suctioning required, bleeding threatens surgical field a few seconds after
4Moderate bleeding- frequent suctioning and bleeding threatens surgical field directly after suctioning.
5 Severe bleeding – constant suctioning required bleeding faster than it can be removed by suctioning, surgery not possible

OTHER PARAMETERS

TIME	HR	PULSE	BP	MAP	END TIDAL CO2
0Mins					
15Mins					
30Mins					
45Mins					
1Hour					
1:15Mins					
1:30Mins					
1:45Mins					
2hours					
2:15Mins					
2:30Min					

INFORMED CONSENT

TITLE OF THE PROJECT:

EFFECT OF PTERYGOPALATINE FOSSA INJECTION WITH 2% LIGNOCAINE AND 1:80000 ADRENALINE IN MINIMISING BLEEDING DURING ENDOSCOPIC SINUS SURGERY– A PROSPECTIVE STUDY

I understand that I remain free to withdraw from the study at any time and this will not change my future care.

I have read the consent form or has been read to me and I understand the purpose of this study, the procedures that will be used, the risks and benefits associated with my involvement in the study and the confidential nature of the information that will be collected and disclosed during the study.

I have had the opportunity to ask questions regarding various aspects of this study and my questions have been answered to my satisfaction.

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of my personal information as outlined in this consent form.

Subject's name and signature/ Thumb impression Date:

Name and signature of witness Date:

Name and signature of the principle investigator Date:

SL NO	HOSPITAL NO:	AGE	SEX	CLINICAL PRESENTATIONS	CLINICAL FINDINGS	DIAGNOSIS	CT SCAN GRADING		ENDOSCOPIC GRADING		HEART RATE	BLOOD PRESSURE	MAP	SIDE OF DRUG	BLOOD LOSS GRADE	
							RIGHT	LEFT	RIGHT	LEFT					TEST	CONTROL
1	259874	17	MALE	Nasal obstruction	Gross DNS to left , Polypoidal mass	Bilateral Ethmoidal polyposis	4	4	4	4	76/min	110/76 mm hg	84	RIGHT	13	16
				nasal discharge	seen in bilateral nasal cavity											
				headache SINCE 6 MONTHS	arising from middle meatus ,											
				sneezing	insenstitive to touch											
				heaviness of head	mucopus seen bilaterally											
2	126251	45	Female	nasal obstruction												
				nasal discharge SINCE 2 YEARS	Polyps seen in both nasal cavity in	ethomoidal polyposis	4	4	2	2	66/min	120/82mm hg	90	RIGHT	6	10
				headache	middle meatus											
					clear thin discharge present											
					bilteral maxillary sinus tenderness (+)											
3	287303	50	Male	nasal obstruction	Multiple grape like mass seen arising											
				nasal discharge SINCE 1 YEAR	from middle meatus partially	bilateral ethmoidal polyposis	5	4	2	2	80/min	120/80 mm hg	86	LEFT	10	10
				headache	obstructing nasal cavity											
					bilateral maxiallry ,ethmoidal &frontal sinus											
					tenderness (+)											
4	148565	29	female	nasal obstruction	DNS to left with bony spur	Chronic Rhinosinusitits	6	5	2	1	66/min	110/70mm hg	82	RIGHT	7	7
				nasal discharge SINCE 1 YEAR	paradoxical middle turbinate											
				headache	left concha bullosa											
				sneezing	bilateral maxillary and frontal tenderness (+)											
5	55317	29	male	nasal obstruction	multiple polyps seen in bilateral nasal cavity	ethmoidal polyposis	7	6	3	3	74/min	136/88mm hg	100	RIGHT	8	16
				nasal discharge SINCE 6 MONTHS	arisisng from the middle meatus, pale											
				headache	insensitive to touch											
				postnasal drip SINCE 2 WEEKS	watery discharge (+)											
6	16895	45	female	nasal obstruction	right ITH (+)	chronic rhinosinusitits	4	4	3	1	79/min	100/75 mm hg	84	RIGHT	11	15
				headache SINCE 2 WEEKS	Mucosa congested											
				sneezing	mucopus (+) in right nasal cavity											
				postnasal drip	biltaral maxiallry and frontal tenderness (+)											
7	57617	34	Female	nasal obstruction SINCE 3 MONTHS	bilateral grape like mass arising from middle	ethmoidal polyposis	2	2	2	2	72/min	120/88 mm hg	91	LEFT	4	6
				breathing difficulty	meatus occluding nasal cavities											
8	64731	23	female	nasal discharge (+)	bilateral roomy nasal cavities	Fungal sinusitis	7	5	2	2	80/min	130/88mm hg	100	LEFT	10	10
				anosmia	foul smelling discharge (+)											
				facial pains	tenderness + in b/l maxiallry and ethmoidal,											
					frontal sinuses											
9	249929	45	male	nasal obstruction												
				nasal discharge SINCE 2 MONTHS	DNS to right with spur to Left	chronic rhinosinusitis	3	3	1	1	87/min	128/66mm hg	88	RIGHT	10	12
				breathing difficulty	mucosa congested											
					left ITH (+)											
					Bilateral maxiallry tenderness (+)											
10	4497	58	male	nasal obstructin	DNS to left with bony spur	ethmoidal polyposis	5	4	2	2	78/min`	130/82 mm hg	92	RIGHT	12	15
				nasal discharge SINCE 8 MONTHS	Polyps seen in bilateral nasal cavity arising											
				sneezing	from middle meatus partially occluding											
					nasal cavity											
11	1019859	50	male	nasal obstruction	multiple polyp seen srising form bilateral	ethomoidal polyposis	2	4	1	1	67/min	120/82mm hg	88	RIGHT	6	10
				postnasal drip SINCE 5 MONTHS	nasal cavity . Pale and insensitive to touch											
				hawking sensation	maxillary tenderness (+)											
12	93435	16	female	nasal obstruction	DNS ro right	chronic rhinosinusitis	5	3	2	2	88/min	133/92mmhg	100	RIGHT	11	16
				nasal discharge SINCE 4 MONTHS	mucosa congestes											

				sneezing	mucopus (+)											
13	76008	30	male	nasal obstruction	DNS to Right	chronic rhinosinusitis	4	3	2	2	76/min	128/66mm hg	86	RIGHT	8	11
				headache SINCE 20 DAYS	left ITH (+)											
				postnasal drip	Thick purulent discharge (+)											
					ppw - postnasal drip (+)											
					bilateral maxiallry sinus tenderness (+)											
14	109352	75	male	nasal obstruction	Caudal dislocation to left	ethmoidal polyposis	8	8	4	4	88/min	138/96mmhg	96	LEFT	19	19
				nasal discharge SINCE 2 YEARS	DNS to right											
				sneezing	multipe polyps seen in bilateral nasal cavity											
				postnasal drip	completely obstructing the cavity											
					discharge (+)											
15	103309	22	male	nasal obstruction	DNS to left , left ITH (+), concha bullosa L	ethomoidal polyposis	3	3	1	1	74/min	126/78 mm h g	82	LET	6	7
				nasal discharge SINCE 2 YEARS	Poylps arising from maxiallry sinus											
				hyposmia	bilateral maxiallry and frontal tenderness (+)											
16	99224	21	male	nasal discharge	DNS to left with spur	chronic rhinosinusitits	4	5	0	0	66/min	128/88mm hg	90	RIGHT	6	13
				nasal obstruction SINCE 3 YEARS	mucosa congested											
				headache	bilateral maxiallry sinus tenderness (+)											
17	190441	32	male	nasal obstruction	DNS to left with spur	pansinusitis	4	4	0	0	72/min	130/88mm hg	88	RIGHT	13	6
				nasal discharge SINCE 2 YEARS	paradoxical turbinate											
				headache	right accessory ostia (+)											
					bilateral maxiallry and frontal sinus											
					and ethmoidal tenderness (+)											
18	1020441	60	male	nasal obstruction	DNS to right	chronic sinusitits	5	5	1	1	66/min	110/66mm hg	80	LEFT	5	5
				nasal discharge SINCE 4 MONTHS	mucosa congested											
				headache	bilateral maxillary and frontal tenderness (+)											
				Cough												
				breathing difficulty												
19	17996	58	male	nasal obstruction	fouls smelling discharge (+)	Fungal Sinusitis	5	5	3	4	92/min	146/88mm hg	100	RIGHT	16	14
				nasal discharge	crusting (+)											
				headache	polyp (+) arising from middle meatus in right											
				anosmia	nasal cavity											
20	116516	42	female	nasal obstruction	DNS to left	Ethmoidal Polyposis	4	5	3	3	70/min	122/78mm hg	84	RIGHT	6	10
				nasal discharge SINCE 1 YEAR	multiple grape like masses seen from bilateral											
				headache	nasal cavity arising from middle meatus and											
				sneezing	beyond but not obstructing nasal cavity											
					discharge (+)											
21	306378	52	male	nasal obstruction	DNS to right	ethmoidal polyposis	10	8	2	2	90/min	148/88mmhg	98	LEFT	12	12
				sneezing SINCE 6 MONTHS	polypoidal tissue seen arising from the											
				recurrent URTI	middle meatus biltaeraly pale and insensitive											
					to touch											
					mucosa congested											
22	298098	36	female	nasal obstruction	DNS to left	ethmoidal polyposis	5	4	2	1	88/min	122/88mm hg	90	RIGHT	6	11
				breathing difficulty	polyps seen in biltaeral middle meatus											
					pale and insensitive to touch											
23	132986	12	female	nasal obstruction	DNS to right	ethmoidal polyposis	4	4	1	1	66/min	110/76mm hg	78	RIGHT	4	6
				snoring since 6 months	grape like polyps seen in bilateral middle											
				headache	meatus											
24	157275	35	female	nasal discharge	multiple polypoidal mass seen in bilateral	ethmoidal polyposis	6	5	3	3	76/min	120/82mm hg	88	LEFT	8	8
				recurrent URTI SINCE 6 MONTHS	nasal cavity partially obstructing cavity											
				Headache	discharge (+)											

				heaviness of head	cogested mucosa (+). PNS tenderness (+)										
25	361951	39	female	nasal obstruction	DNS to right with spur	Pan sinusitis	6	6	2	2	68/min	110/85 mm hg	90	RIGHT	12	12
				nasal discharge SINCE 6 MONTHS	left middle turbinate hypertrophy (+)										
				sneezing	mucopus (+)										
				fever SINCE 3 DAYS	maxillary frontal and ethmoidal sinus										
					tenderness(+)										
26	190441	32	male	nasal discharge	Right side : multiple polyps see arising from	ethmoidal polyposis	5	5	2	2	72/min	130/90mm hg	88	LEFT	7	13
				recurrent URTI SINCE 2 YEARS	middle meatus and partially obstructing										
				Headache	nasal cavity										
				sneezing	left side: polyps seen arising from the middle										
				postnasal drip SINCE 3 MONTHS	meatus										
					watery discharge (+) biltaerally										
					frontal sinus tenderness (+)										
27	323801	46	male	nasal discharge	DNS to left	ethmoidal polyposis	4	4	1	1	68/min	126/82mm hg	90	LEFT	7	11
				nasal obstruction SINCE 6 MONTHS	right ITH (+)										
					Multiple polyps seen in bilateral nasal cavity										
					bilateral maxiallry sinus tenderness (+)										
28	103309	40	male	headache	DNS to right , caudal dislocation to left	chronic rhinosinusitits	4	4	1	1	88/min	120/88mmhg	82	LEFT	6	8
				nasal obstruction since 2 years	mucopus (+)										
				breathing difficulty	biltaeral maxiallry sinus tenderness (+)										
29	67751	19	male	nasal obstruction since 3 years	DNS to right	chronic rhinosinusitis with gross DNS	5	5	1	1	68/min	112/78mm hg	82	RIGHT	8	12
				breathing difficulty	ITH (+) Left side	to Right									
				headache since 6 months	bilateral maxially and frontal sinus										
				Heaviness of head	tenderness (+)										
30	307754	55	Female	nasal obstruction	Right side: polyps arisisng from middle meatus	ethmoidal polyposis	6	5	3	1	78/min	122/96mm hg	100	RIGHT	12	9
				headache SINCE 3 MONTHS	partially obstructing nasal cavity										
				facial pain	left side : polyps arising from middle meatus										
					bilateral maxiallry and ethmoidal tenderness										
31	152661	45	female	nasal discharge	Bilateral nasal polyps arising from middle	ethmoidal polyposis	5	4	2	2	88/min	116/88mm hg	92	RIGHT	10	9
				nasal obstruction SINCE 6 MONTHS	meatus										
				recurrent URTI	discharge (+)										
32	181254	58	male	nasal obstruction	DNS to left										
				nasal discharge	mucosa congested	chronic rhinosinusitis with DNS to left	5	5	2	2	66/min	120/78 mm hg	90	LEFT	9	9
				breathing difficulty	mucopus seen biltaeral nasal cavity										
					blateral maxiallry ethomidal and frontal										
					sinus tenderness (+)										