Dissertation On

AN OBSERVATIONAL STUDY COMPARING THE EFFECT OF SPHENOPALATINE ARTERY BLOCK ON BLEEDING IN ENDOSCOPIC SINUS SURGERY

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Under the guidance of

DR. F ANTHONY IRUDHAYARAJAN, M.S., D.L.O
Professor & HOD,
Department of ENT & Head and Neck Surgery,
Govt. Stanley Medical College,
Chennai.

GOVERNMENT STANLEY MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

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CERTIFICATE

This is to certify that this dissertation titled **AN OBSERVATIONAL STUDY COMPARING THE EFFECT OF SPHENOPALATINE ARTERY BLOCK ON BLEEDING IN ENDOSCOPIC SINUS SURGERY** is the original and bonafide work done by **Dr. NIGIL SREEDHARAN** under the guidance of Prof **Dr F ANTHONY IRUDHAYARAJAN**, M.S., DLO Professor & HOD, Department of ENT & Head and Neck Surgery at the Government Stanley Medical College & Hospital, Chennai – 600 001, during the tenure of his course in M.S. ENT from July-2014 to April- 2017 held under the regulation of the Tamilnadu Dr. M.G.R Medical University, Guindy, Chennai – 600 032.

Prof Dr F Anthony Irudhayarajan, M.S., DLO
Professor & HOD,
Department of ENT & Head and Neck Surgery
Government Stanley Medical College & Hospital,
Chennai – 600 001.

Place : Chennai
Date : .10.2016

Dr. Isaac Christian Moses M.D, FICP, FACP
Dean,
Govt.Stanley Medical College,
Chennai – 600 001.

Place: Chennai
Date : .10.2016

CERTIFICATE BY THE GUIDE

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PROF. DR F ANTHONY IRUDHAYARAJAN M.S, DLO
Professor and HOD
Department of ENT & Head and Neck Surgery
Government Stanley Medical College
Chennai- 600 001.

Place : Chennai
Date : .10.2016

DECLARATION BY THE CANDIDATE

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Place: Chennai
Date: .10.2016

Signature by the candidate
(Dr Nigil Sreedharan)

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ABSTRACT

This is a cross sectional observational study done in a tertiary care centre. 55 patients coming to the ENT department of Government Stanley Medical College from 2015 – 2016 were included in this study. Of these there were 28 males and 27 females. All the patients met the inclusion criteria decided upon at the beginning the study. Ethical committee clearance for the study was obtained and written informed consent for the study was taken from each patient. All patients had bilateral nasal sinus disease and endoscopic sinus surgery was performed on both sides. The procedure done on both sides were the same in each case. 20 minutes prior to surgery one side was chosen randomly and sphenopalatine artery block was administered via the greater palatine canal approach. A mixture of lignocaine (2%) and adrenaline (1:80000) was used for infiltration. The surgery was done in an alternating fashion where the surgeon would operate for 15 minutes on one side and then move onto the other side. The field was graded for bleeding at 30 minute intervals. Wormald Grading System was used. The results were tabulated and the Wilcoxon Signed Rank Test was done at each time interval to see if there was a statistically significant difference in the grades of bleeding on both sides at each time interval. It was found that for each time interval up to 120 minutes there was a significant decrease in the bleeding on the blocked side. However after 120 minutes the bleeding on both sides appeared to be same. In conclusion sphenopalatine artery block given prior to surgery will be effective in reducing bleeding in FESS for the first 2 hours after which the effect of the block wears away.

AIMS AND OBJECTIVES

The aim of this study is to observe if there is any significant difference in bleeding during functional endoscopic sinus surgery (FESS), when giving sphenopalatine artery block and when not giving. Sphenopalatine artery is the main feeding vessel to the lateral wall of nasal cavity and most of the septum. Blocking this artery is believed to reduce the amount of bleeding, and there by enhancing the visibility during surgery. The block involves injecting a local anesthetic – lignocaine, along with a vaso constrictive agent – adrenaline into the pterygopalatine fossa.

INCLUSION CRITERIA

- All patients between ages 20 yrs and 45 yrs.
- Bilateral chronic sinusitis or bilateral sinus disease requiring the same procedure in both nasal cavities.

EXCLUSION CRITERIA

- Patients with unilateral disease.
- Patients with systemic hypertension
- Patients taking anti coagulant drugs or medication affecting their coagulation profile
- Patients having bleeding or clotting disorders.
- Patients below 20 yrs of age or above 45 yrs
- Patient known to have allergic reactions to lignocaine
- Patients having undergone nasal surgery previously

- Patients whose intra op BP rises above 130 mmHg systolic and 80 mmHg diastolic at any point during surgery.
- Patients operated by more than 1 surgeon.
- Patients under going septal correction and if septal correction is started prior to FESS on both sides
- Patients assessed under PSA II and greater

INTRODUCTION

Visualisation of the nose and paranasal sinuses has evolved over the past 2 centuries. Starting with Philip Bozzini's first "Light conductor" to the modern day high powered endoscopes, a plethora of devices were invented for seeing 'behind the corners' of the upper aero digestive tract. However, the works of Messerklinger, Wigand, Stammberger et al, have proved without doubt that the rigid Hopkins endoscope was superior to any other device in dealing with pathologies related to the nose and paranasal sinuses.

Messerklinger is credited with being the first to develop and establish a systemic endoscopic diagnostic approach to the lateral wall of the nose. His pioneering work began in the 1950s and was based on the extensive anatomical studies of the lateral wall of the nose by Zuckerkandl, Onodi and Grunwald. He proved that in most cases the larger sinuses that is the frontal and maxillary were actually involved secondary to a primary pathology lying elsewhere. The location of the primary lesion was usually the ethmoidal clefts. He developed a diagnostic approach wherein one could identify diseased mucosa in these important primary areas. Surgical clearance of disease was then carried out by a circumscribed limited endoscopic surgery directed toward the ethmoid sinuses. He noticed that eradication of disease in these areas resulted in complete resolution of even massive mucosal pathologies in the larger sinuses within a few weeks post operatively even though no procedure was directly done on them. Thus started the era of conservative nasal surgeries where mucosal preservation was the

dictum. The term functional endoscopic sinus surgery (FESS) was coined by David Kennedy.

FESS is now considered the treatment of choice for chronic sinusitis not responding to maximal medical management or causing complications thereby requiring urgent surgical intervention. Various different techniques have evolved over the years to address the removal of each structure during FESS. However, the ultimate aim is to widen natural ostia to the sinuses, correct any anatomic variation predisposing to chronic sinusitis, preserve as much normal mucosa as possible at the same time removing any extensive polypoidal tissue, improve ventilation of sinuses so that diseased mucosa can recover and ultimately relieve the patient from his or her ailments.

One of the persistent problems that prevent us from achieving these goals during sinus surgery is intraoperative bleeding. There are a number of ways in which bleeding during FESS may be reduced. Firstly and most importantly hypotensive anaesthesia is mandatory throughout the procedure. Correct positioning of the patient during surgery. Adequate premedication and control of hypertension. Use of decongestant and vasoconstrictor soaked packs pre and intra operatively also helps. Frequent irrigation with saline helps remove debris as well as control mucosal bleeds. Infiltration of the nasal cavity mucosa with adrenaline based solution have been controversial. Some authors are of the opinion that infiltration provides no added benefit and is potentially harmful, others continue to use it to good measure.

Sphenopalatine artery block is another widely used method to control bleeding prior to surgery. Since major bleeding is encountered via the branches of the sphenopalatine artery, injecting a vasoconstrictor, usually adrenaline, into the sphenopalatine fossa where the artery originates will reduce bleeding. Various approaches to achieving this block has been described. The most popular one is the endoscopic approach where the posterior end of the middle turbinate is identified, this is where the opening of the sphenopalatine foramen is found and infiltration is given. This method however cannot be used in extensive mucosal disease where the posterior end of the turbinate is inaccessible to us prior to surgery. An alternative method is via the trans oral route through the greater palatine foramen in the hard palate.

Regardless of the route taken to achieve the block, the efficacy of the block is still doubted by many. Some use this block routinely for all endoscopic procedures, some reserve it for cases with extensive disease or in case where extra blood loss is anticipated, some do not use it altogether. Very few studies have been conducted in literature regarding the actual efficacy of sphenopalatine block in controlling bleeding in FESS.

REVIEW OF LITERATURE

RELEVANT ANATOMY

EMBRYOLOGY OF THE LATERAL NASAL WALL

Paranasal sinuses develop from lateral nasal wall ridges called the Ethmoturbinals. In the 8th week of intrauterine life, 5-6 ridges are formed which undergo regression and fusion after which 3-4 persist. The 1stethmoturbinal regresses, with its ascending part forming the Agger nasi and the descending part forming the Uncinate. The second and third ethmoturbinals give rise to Middle and Superior turbinates, whereas the fourth and fifth fuse to form the supreme turbinate.

The Maxilloturbinal ridge arises inferior to these structures and gives rise to the Inferior turbinate. Primary furrows between the ethmoturbinals form nasal meati and recesses. The first furrow lies between the first and second ethmoturbinals, the descending part of which forms the Ethmoidal infundibulum, Middle meatus and Hiatus semilunaris. Its ascending part forms the Frontal recess. The second and third primary furrows form the superior and supreme meati respectively. Secondary lateral nasal wall Evagination gives rise to the Bulla ethmoidalis, and the Secondary lateral wall Invagination gives rise to the Supra and Retrobullar recesses.

Primordial maxillary sinus develops as a shallow groove from the inferior aspect of the Ethmoidal Infundibulum into the mass of the maxilla.[2,3] The evolution of maxillary sinus depicted in the following

figure shows why the maxillary ostium is found at the floor of the ethmoidal infundibulum and why the drainage and ventilation of the maxillary sinus pass through it. Maxillary ostium lies on the medial wall of the ethmoidal infundibulum at the transition of its middle to posterior third.

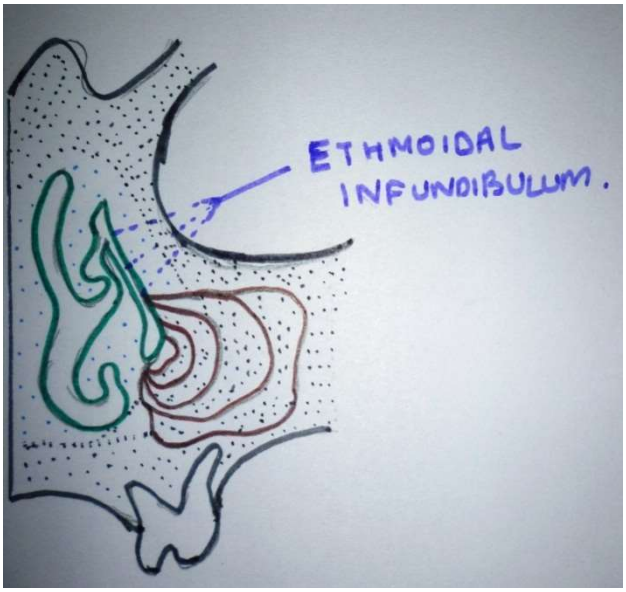


Fig 1 - Development of maxillary sinus from ethmoidal infundibulum

Maxillary sinus is the first sinus to appear (7 – 10 weeks).[3] It is around 7 x 4 x 4 mm^3 at birth. It grows at a rate of 2 mm vertically and 3 mm anteroposteriorly.[3] Its growth slows down around the age of 7 with a second growth phase thereafter.[3] At 12 years , pneumatisation reaches just under the lateral orbital wall at the junction of the zygomatic process. Inferiorly it reaches up to the level of the nasal floor , and , after 2^{nd} dentition , till below the nasal floor. [3] Its final size ~15 ml is attained at the age of 17 years.[3] Relative enlargement of the sinus occurs in old age due to resorption of the alveolus.

ANATOMY OF LATERAL WALL

OSTEOLOGY

The Maxilla

Forms the framework or base on which the lateral nasal wall is built. The medial surface of the maxilla forms the lateral wall of nose. The large opening into the maxillary sinus is what is most obvious in the lateral nasal wall. In the live patient the maxillary sinus opening is not easily seen and rather small. The reason for this is that the processes of different bones narrow and partially close off the large opening in the maxillary bone.

These processes are:

• Anteriorly, the lacrimal bone with its descending process.

• Anterior and inferiorly - uncinate process of the ethmoid bone

• Inferiorly the inferior turbinate and its maxillary process

• Posteriorly the palatine bone with its perpendicular plate.

Only a double layer formed by mucosa of the maxillary sinus and nasal cavity covers certain areas of the maxillary sinus opening. The posterior and anterior fontanelles are examples of such areas. In certain cases, the double layers of the mucosa may become dehiscent producing accessory ostia. Hidden deep

behind the intermediate portion of the uncinate process is the natural maxillary sinus ostium.

The frontonasal process of the maxilla is formed by the drawing up of the maxilla anterior to the maxillary hiatus. This extends superiorly and the upper border of this process articulates with the frontal bone. The anterior border articulates with the nasal bone and hence the name. Two crests are seen on the medial surface of this process. The ethmoidal crest is the upper one and the most anterior part of the middle turbinate gets attached to this. Anterior to the attachment of the middle turbinate, the agger nasi cells also overlies this crest. The formation of the agger nasi cells is contributed by the pneumatization of the ethmoidal crest and the adjacent lacrimal bone. The conchal crest is the lower one and gives attachment to the inferior turbinate. Part of the inferior meatus is formed by the smooth area below the conchal crest. A groove, found immediately behind the frontonasal process that is closed by the lacrimal process of the inferior turbinate and by the lacrimal bone contributes to the formation of the nasolacrimal duct. The thick bone is the frontonasal process and the thin bone is the lacrimal bone. Hence the lateral wall of the nasolacrimal duct is formed by thick bone whilst its medial wall is formed by fairly thin bone. The medial bone can hence be easily damaged. Maxillary tuberosity is a roughened area found posterior to the hiatus, at the junction of the posterior and medial wall of the maxilla. The greater palatine canal that transmits the greater palatine vessels and nerves is formed in this area. An oblique groove over the tuberosity is closed off

by the perpendicular plate of the palatine bone leading to the formation of the greater palatine canal. The roof of the maxillary sinus is formed by the orbital surface of the maxilla. The infraorbital canal is found in the roof. This may sometimes be dehiscent exposing its contents, namely, the infraorbital nerve and vessels. Being composed of a fairly thin bone, the posterolateral wall of the maxillary sinus separates the infratemporal fossa laterally and the pterygopalatine fossa medially. It is rather smooth and featureless.

The Frontal Bone

By looking at its basal view, the contribution made by the frontal bone to the lateral nasal wall is best explained. In the living person the cribriform plate of the ethmoid fills the hiatus at the centre of the frontal bone. A variable number of air cells are found on either side of this hiatus. These are called the posterior and anterior ethmoid air cells. The ethmoid fovea or fovea ethmoidalis which is part of the skull base forms the roof of these air cells. The fovea ethmoidalis, thus lies at a higher level than the cribriform plate. The lamina papyracea of the ethmoid bone gives attachment to the lateral border of these air cells. The posterior and anterior ethmoidal foramens transmitting their associated arteries is found at the junction of these suture lines between the lamina and the frontal bone. The orbit is found lateral to the lamina papyracea. In the midline and anteriorly, the nasal spine is formed by the elongation of the frontal bone. The nasal bones articulates with this spine, which helps in formation of the anterior most portion of the lateral nasal wall.

The Ethmoid Bone

A single delicate bone consisting of numerous air cells forming the ethmoidal sinuses. It consists of a vertical plate in the midline called the perpendicular plate of ethmoid and a horizontal plate formed by the cribriform plate. Posterior part of the septum is formed by the perpendicular plate. The cribriform plate separates the nose from the anterior cranial fossa. Its fits into the notch in the frontal bone and specifically separates the olfactory bulb and the gyrus rectus from the nasal cavity. Olfactory nerves as well as the anterior and posterior ethmoidal arteries perforate the cribriform plate creating many foramina. The crista galli forms a midline projection on the upper surface of the cribriform plate. It is occasionally pneumatized. The cribriform plate shows an oblique or vertical lateral lamella and a horizontal medial lamella. The frontal bone articulates with this lateral lamella. The skull base in this region is hence formed laterally by the frontal bone, which is thick and medially by the lateral lamella of the cribriform plate, which is in contrast a very thin bone. The lateral lamella of the cribriform plate is 0.2 mm. The frontal bone forming the ethmoid fovea is 0.5 mm in thickness. The thinnest area in the base skull is only 0.05 mm in thickness and is the region where the anterior ethmoidal artery pierces the dura medially. The depth of the olfactory fossa and length of the lateral lamella are classified by Keros into 3 types:

- Type I which is 1-3 mm
- Type II which is 4-7 mm

- Type III which is 8-17 mm

Two masses of air cells are found lateral to the perpendicular plate on either side. These are the ethmoidal air sinuses. They are bounded laterally by the lamina papyracea that is paper thin and medially by the superior and middle turbinate. The lamina papyracea separates the orbit from the ethmoids. A supreme turbinate maybe occasionally found above the superior turbinate. The superior and middle turbinate are part of the ethmoid bone. The inferior turbinate is a separate bone. The area under and lateral to the middle turbinate but medial to the lateral nasal wall is called the middle meatus and that under the superior turbinate is called the superior meatus.

Similar to a dried leaf which curves in different planes the middle turbinate forms a thin sheet of bone that takes it anterior most attachment in the sagittal plane to the frontonasal process of the maxilla and superiorly to the cribriform plate. In the coronal plane it is attached laterally to the lamina papyracea the attachment being referred to as the basal or ground lamella. The attachment in the horizontal plane, posteriorly is along the lamina papyracea and the perpendicular plate of the palatine bone up to choana near its posterior roof. The uncinate process is a gently curved bony process. It lies almost free within the middle meatus covering the maxillary sinus opening partially. It articulates posteriorly with the perpendicular plate of palatine bone and the inferior turbinate and anteriorly with the lacrimal bone.

The ethmoid cells are divided into two groups:

1. Anterior ethmoid cells

These open in the middle meatus and lie anterior to the ground lamella of the middle turbinate.

2. Posterior ethmoid cells

These open into the superior meatus or sphenoethmoidal recess and lie behind the ground lamella.

Depending on the relative extent of pneumatisation of the posterior and anterior ethmoidal air cells, the ground lamella may be displaced posteriorly or anteriorly. The ethmoidal bulla is a fairly constant and large anterior ethmoid air cells. Superiorly and posteriorly the ethmoidal cells are incomplete. Posteriorly they are completed by the sphenoid bone and superiorly by the frontal bone. Variable patterns of pneumatization are developed by the ethmoid cellsas they tend to migrate into the surrounding bones. Paths of pneumatization include:

- Into the frontal bone to form the frontal sinus, anterosuperiorly
- Above the ethmoidal bulla, behind the frontal sinus and over the orbit to form the supraorbital cell, posterior and superiorly
- As the Haller cell, into the roof of the maxillary sinus, inferolaterally
- As the Onodi cell, posteriorly, above the sphenoid sinus.
- As the agger nasi cells, anteriorly, into the frontonasal process of the maxilla and the lacrimal bone.

- Into the frontal recess forming different types of frontal cells, superiorly
- Isolated cells formed within the ethmoid infundibulum are called infundibular cells

The Sphenoid Bone

It separates the anterior and middle cranial fossa from the nasal cavity by closing off the back of the nasal cavity. It is in relation with very important structures such as the carotid artery and the optic nerve. The sphenoid bone looks like a bat with outstretched wings, when seen from the front. The body of the sphenoid forms the central portion. It is pneumatized by the two sphenoid sinuses. A strong triangular process called the rostrum articulates with the vomer anteriorly, in the midline.

On either side are a pair of lesser and greater wings, extending laterally from the central body. Lying between the lesser and greater wings is the retort-shaped superior orbital fissure. Two roots attach the lesser wing to the body. Between these lies the optic canal. Along with its meninges and the ophthalmic artery, this canal transmits the optic nerve. The pterygoid process is a stout process extending downwards on either side at the junction of the body and greater wing of the sphenoid. It divides into the lateral and medial pterygoid plates. The lateral wall of the posterior choana is formed by the medial pterygoid plate. The lateral pterygoid plate has no direct relation to the nasal cavity proper. The posterior wall of the pterygopalatine fossa forms the anterior surface of the pterygoid

process. Presenting as foramina on the anterior surface, two canals traverse the pterygoid process:

1. The inferomedial foramen

It is the funnel-shaped opening of the vidian canal transmitting the vidian nerve.

2. The superolateral foramen

Which transmits the maxillary nerve and is called the foramen rotundum.

The foramen rotundum lies just a few millimetres below the superior orbital fissure. The sphenoid sinuses are very often asymmetrical, variably pneumatized and show right- or left-sided “sphenoidal dominance”. The sphenoid sinus can be classified into the following types depending on the pneumatization of the sphenoid bone:

- A small pit-like depression called Conchal
- Extending up to the anterior wall of the pituitary fossa called Presellar.
- Extending up to the clivus called Sellar. In the roof of the sinus the pituitary forms a distinct bulge.
- Mixed.

In the sellar type of pneumatisation, posterosuperiorly in the midline of the roof lies the pituitary bulge. The optic canal can be visualized, laterally and superiorly, which at times is dehiscent. The internal carotid artery bulge produces a prominence in the lateral wall, posteriorly and inferiorly. Septae may divide the sphenoid sinus. These septae are usually found attached to important structures on the lateral wall of the sinus like the internal carotid artery or optic nerve. The

carotico-optic recess exists between the internal carotid artery and optic nerve. This recess especially deep and the optic nerve may be dehiscent when the anterior clinoid process is pneumatized.

The Inferior Turbinate

A separate scroll-like bone. It runs a fairly straight course from posterior to anterior, unlike the middle and superior turbinates. Overhanging the inferior meatus its inferior margin lies free. The superior margin of the turbinate is attached to the palatine bone posteriorly and the maxilla anteriorly. In the inferior meatus the nasolacrimal duct opens at a peak found approximately 1 cm behind its anterior end on its superior margin.

The inferior turbinate has 3 processes:

1. The Lacrimal Process

Extends anteriorly, from its superior margin, this process articulates with the descending process of the lacrimal bone. It hence assists in forming the canal for the nasolacrimal duct.

2. The ethmoid process

Arising from near the superior margin a little behind the lacrimal process. It articulates with the uncinate process of the ethmoid bone.

3. The maxillary process

Arising from the superior border it curves laterally to attach to the maxilla. It forms part of the lateral wall of the inferior meatus and closes off part of the maxillary hiatus.

Lacrimal Bone

It is the most fragile and smallest of the cranial bones. It separates the nasal cavity from the lacrimal fossa. Anteriorly it articulates with the frontonasal process of the maxilla, superiorly with the frontal bone, posteriorly with the uncinate process and inferiorly it gets drawn into a process called the descending process of the lacrimal bone.

Palatine Bone

Forming the floor of the nasal cavity and the posterior part of the lateral nasal wall the palatine bone is a fragile L-shaped bone. It consists of two plates. The horizontal plate which forms the posterior part of the nasal floor. A perpendicular plate, which forms the posterior part of the lateral nasal wall.

Being covered by nasal mucosa the perpendicular plate has a smooth medial surface. Two crests running across this surface is divide it into three sections:

- The Conchal Crest is the lower crest.
- The Ethmoidal Crest is the upper crest.

The conchal crest is attached to the inferior turbinate. The ethmoidal crest is attached to the middle turbinate. Hence the area between the two crests forms the posterior part of the middle meatus and the area below the conchal crest forms part of the inferior meatus. Part of the superior meatus lateral wall is formed by a narrow groove above the ethmoidal crest. Just above the posterior most

attachment of the middle turbinate, the sphenopalatine foramen opens up into the nasal cavity. By detaching the middle turbinate from the ethmoidal crest the fossa can, however, be approached through the middle meatus.

The medial wall of the pterygopalatine fossa is formed by the smooth upper lateral surface of the perpendicular plate. The lower lateral surface articulates with the maxillary tuberosity and is rough. Between these two bones lies the canal for the greater palatine nerves and vessels. Articulation with the surrounding bones is as follows:

Perpendicular plate

- The maxillary hiatus is closed by the articulation of the maxillary process of the inferior turbinate with the prolongation from the anterior border of the perpendicular plate called the maxillary process of the palatine bone.
- Posteriorly forms the lateral wall of the posterior choana by articulating with the medial pterygoid plates.
- The horizontal plate attaches to it inferiorly.
- Superiorly it has two process. The orbital process attaches to the maxilla while the sphenoidal process to the sphenoid.

Horizontal plate

- Anteriorly forms the nasal floor by articulating with the horizontal process of the maxilla.

- Posterior free border forms the posterior end of the hard palate.

Process of the Palatine Bone

The palatine bone has 3 processes:

1. The orbital process

Is anterior and arises from the upper part of the perpendicular process. So called because a small portion of the orbital floor which near the posterior end of the inferior orbital fissure is formed by it.

2. The posterior process

Also called the sphenoidal process. It articulates with the body of the sphenoid. A deep notch called the sphenopalatine notch lies between these two processes. Completed superiorly by the body of the sphenoid bone forming the sphenopalatine foramen.

3. The pyramidal process,

Extends posterolaterally from the junction of the horizontal and perpendicular plates. Articulating with notch between the two pterygoid plates it does not take part in the formation of the nasal cavity.

LATERAL NASAL WALL

The complex ethmoidal labyrinth can be reduced into a series of lamellae which corresponds to the following - 1st- uncinate, 2nd – ethmoidal bulla, 3rd – basal or ground lamella and 4th – lamella of superior turbinate.

Agger Nasi

Anterior to the attachment of the middle turbinate to the lateral nasal wall, it is seen as a prominence. This region may be pneumatised by an anterior ethmoidal cell giving rise to the Aggernasi cell.

Ethmoidal Bulla

It is the most constant and largest of all anterior ethmoidal cells, located in the middle meatus , posterior to the uncinate anterior to the basal lamella. It is based on the lamina papyracea (LP). In 8% of subjects , it is unpneumatised and appears as a bony projection from the LP , known as the Torus Lateralis.

Hiatus Semilunaris

Hiatus Semilunaris Inferior of Grunwald is a 2 dimensional sagittaly oriented crescent shaped gap between the posterior free margin of the uncinate process and the anterior free margin of the bulla ethmoidalis. It communicates with the ethmoidal infundibulum.

Hiatus Semilunaris Superior

It is cleft between the posterior wall of bulla and the basal lamella where the middle meatus communicates with the Lateral Sinus (Retrobullar and Suprabullar recess)

Ethmoidal Infundibulum

It is a 3 dimensional funnel shaped passage through which secretions from Anterior ethmoid, Maxillary and frontal sinus are transported and channeled into the middle meatus.

Frontal Recess

It is the most anterosuperior part of the anterior ethmoidal sinus that forms a connection with the frontal sinus. It is bounded laterally by Lamina Papyracea, medially by middle turbinate, anteriorly by the posterosuperior wall of Agger nasi and posteriorly by the anterior wall of the ethmoidal bulla.

Osteomeatal Unit

It is a functional designation (Naumann) referring to all the middle meatal structures ,viz. – the uncinate , ethmoid infundibulum , anterior ethmoidal cells , ostia of anterior ethmoidal , maxillary and frontal sinuses.

Uncinate process

It is a sickle shaped sagittal oriented structure paralleling the ethmoidal bulla and with a free posterior border. It is attached anterosuperiorly to the ethmoidal crest of maxilla. Directly inferior to this it is attached to the posterior aspect of the lacrimal bone. Posteroinferiorly its attached to the ethmoidal process of inferior turbinate ,and posterosuperiorly to the lamina perpendicularis of palatine bone. Superiorly it is attached to lamina papyracea / skull base / vertical attachment of the middle turbinate.

Fontanelles[1,2,3,8,35]

Anterior and posterior to its attachment to the inferior turbinate, uncinate process has no bony attachments. In a disarticulated skull there is a large opening, the maxillary hiatus, on the medial wall of the maxillary bone which in life is filled by the maxillary process of inferior turbinate, uncinate process, bulla ethmoidalis, perpendicular plate of palatine bone and lacrimal bone. Nevertheless a portion of this maxillary hiatus is devoid of bony structures and in life is covered by dense connective tissue which is a continuation of the periosteum and the mucous membranes of the middle meatus and the maxillary sinus. *Zuckerkandl* called these structures the *Anterior and Posterior Fontanelles* (AF &PF) in relation to the uncinate process. This part of the lateral nasal wall is also known as its *Membranous Area.* PF is larger and more distinct than AF and AO are

frequently encountered in the PF. They are considered to be formed as a consequence of infection. Their formation is often likened to that of perforations on the tympanic membrane[3]. Their natural incidence is difficult to confirm yet there are views that it could be congenital. Their incidence have been found to be 4-5 % in the general adult population and 25 % in those with Chronic rhinisinusitis[3].

Anatomy of the Pterygopalatine Fossa

A small pyramidal space below the apex of the orbit on the lateral side of the skull forms the pterygopalatine fossa. Anteriorly it is bounded by superomedial part of the infratemporal surface of the maxilla. Posteriorly by the root of the pterygoid process and the adjoining anterior surface of the greater wing of sphenoid.

The perpendicular process of the palatine bone at its upper end has two processes separated by a groove. The anterior most process is the orbital process which attaches to the maxilla, ethmoid and sphenoid bones. Being slightly deviated medially the posteriormost process is called the sphenoidal process. It articulates with the medial pterygoid plate and the undersurface of the sphenoid concha. Between these two process there is a space called the sphenopalatine notch. With the sphenoid taking attachment this groove is converted to the sphenopalatine foramen. This transmits the sphenopalatine vessels and posterior superior nasal nerves.

The medial boundary of the pterygopalatine fossa is formed by the orbital and sphenoidal processes of the palatine bone with the sphenopalatine foramen. The lateral boundary has a fissured opening into the infra temporal fossa called the pterygopalatine fissure.

Apart from the above mentioned communications the fossa also communicates with the orbit via the medial end of the inferior orbital fissure. It connects to the oral cavity through an opening in the posterior lateral aspect of the hard palate, forming the greater palatine canal. The posterior part has two openings, one containing the maxillary nerve in the foramen rotundum and the other the vidian nerve in the pterygoid canal. The foramen rotundum lies superior and lateral to the vidian canal.

Contents of the Pterygopalatine fossa

(1) Maxillary Artery and its branches

The artery enters through the pterygopalatine fissure, gives off numerous branches and terminates as the third part. The branches are:

a) Posterior Superior Alveolar Artery – this artery supplies the maxillary molar and pre molar as well as the mucosa of the maxillary sinus and buccal cavity. It arises within the pterygopalatine fossa and exits through the pterygomaxillary fissure to pierce the bone over the maxillary tuberosity.

b) Infraorbital Artery - Found in the infraorbital groove and canal in the floor of the orbit it supplies the lower eyelid, part of the cheek, upper lip and side of the external nose. It enters the orbit through the inferior orbital fissure. The artery emerges on the face through the infraorbital foramen.

c) Artery of the Pterygoid Canal (Vidian Artery): passes through the Vidian canal and anastomoses with pharyngeal, ethmoidal and sphenopalatine arteries in the pterygopalatine fossa. It also anastomoses with ascending pharyngeal, accessory meningeal, ascending palatine and descending palatine arteries in the oropharynx and around the eustachian tube.

d) Pharyngeal Artery: supplies the mucosa of the nasal roof, nasopharynx, Eustachian tube and sphenoid sinus.

e) Greater Palatine Artery: Supplies the inferior meatus of the nose, hard palate, and palatal gingivae of the maxillary teeth. It leaves the pterygopalatine fossa through the greater palatine canal.

f) Sphenopalatine Artery: it is branch of the maxillary artery. It leaves the sphenopalatine fossa through the sphenopalatine foramen and enters the nose posterior to the superior meatus. It supplies the lateral wall of the nose where its branches anastomose with ethmoidal arteries.

2) Maxillary Nerve

This is purely a sensory nerve, leaving the skull through the foramen rotundum, and entering the sphenopalatine fossa through its posterior wall. It has the following named branches:

a) Meningeal Nerve – arises within the middle cranial fossa and runs with the middle meningeal vessels
b) Ganglionic Branches – usually two branches that connect to the pterygopalatine ganglion
c) Zygomatic Branch
d) Posterior Superior Alveolar Nerve
e) Infraorbital Nerve

3) Pterygopalatine Ganglion

The largest parasympathetic ganglia placed deeply in the pterygopalatine fossa. It is found close to the sphenopalatine foramen and anterior to the pterygoid canal and foramen rotundum. It lies below the maxillary nerve as it crosses the pterygopalatine fossa. It flattened and reddish-grey in colour. The majority of the branches are connected morphologically to the ganglion but plays no functional role. They pass through the ganglion without synapsing. The ganglion has the following nerves:

a) Orbital branches
b) Nasopalatine Nerve

c) Medial Posterior Superior Nasal Nerve

d) Lateral Posterior Superior Nasal Nerve

e) Greater and Lesser Palatine Nerves

Blood Supply of Nose

Nasal cavity is supplied by branches of both internal and external carotid arteries. Posteroinferior part of the nasal cavity is supplied by the sphenopalatine and greater palatine branches of the internal maxillary artery which is a branch of external carotid artery. Anteroinferior part is supplied by external labial branches of the facial artery which is a branch of external carotid artery. Superior part of nasal cavity is supplied is by anterior and posterior ethmoidal arteries which are the branches of ophthalmic artery which is a branch of internal carotid artery.

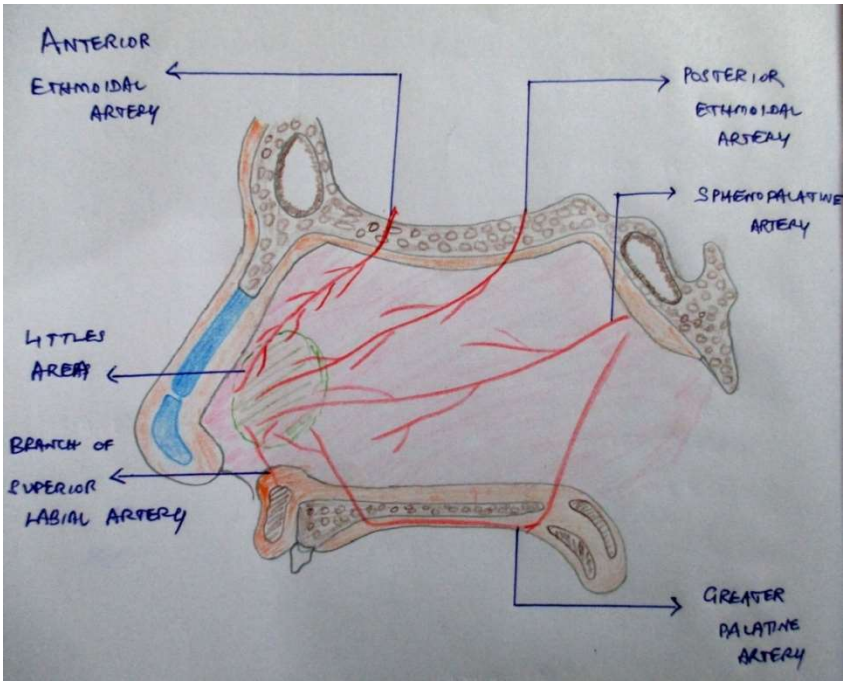


Fig 2 – shows the blood supply of nasal septum

Submucosal Vascular Plexus

The nasal cavity mucosa has rich submucosal vascular plexus which consists of arterioles, capillaries, venules and venous sinusoids. This structure is comparable to the erectile tissues of external genitals. They are under autonomous nervous control. Parasympathetic stimulation will cause vasodilatation and congestion of submucosal plexus.

Nerve Supply of Nasal Cavity

Olfactory nerves carry sensation of smell from olfactory region of nose. This consists of upper one third of nasal septum and its corresponding areas of lateral nasal wall. Nerves of common sensation are anterior ethmoidal nerve, sphenopalatine and branches of infra orbital nerve. Parasympathetic and sympathetic nerves pass through the sphenopalatine ganglion.

Anatomy of the greater palatine canal

The greater palatine canal is a passage that transmits the descending palatine vessels and lesser and greater palatine nerves from the pterygopalatine fossa to the oral cavity. Starting at the inferior part of the pterygopalatine fossa, it travels through the palatine and sphenoid bones to reach the palate, ending at the greater palatine foramen. Accessory canals branch off from this canal and these are known as the lesser palatine canals. Formed by a vertical groove on the posterior part of the maxillary surface of the palatine bone; it is converted into a canal by articulation with the maxilla. It transmits the descending palatine vessels, the lesser palatine nerve, and the greater palatine nerve.

RELEVANT PHYSIOLOGY

MUCOCILIARY CLEARANCE[2]

Paranasal sinuses are lined by pseudostratified ciliated columnar epithelium with goblet cells. The maxillary sinus has the highest density of goblet cells compared to other paranasal sinuses (9700/mm^2).[3] Seromucinous glands are relatively infrequent , concentrated around the ostium. The thickness of the sinus mucosa is 0.2 – 0.8 mm.

Nasal cilia are relatively short (5 μ) and are found at the rate of 200/cell. They are formed of 9 paired outer microtubules surrounding a single inner pair of microtubules. They have a beat frequency of 12 Hz [2] (7-16Hz) [3] at body temperature , which propels materials at the rate of 3 – 25 mm / minute by their metachronous movement. Maxillary sinus produces remarkable quantities of Nitric oxide. The production is increased in cases of allergic rhinitis and decreased in chronic rhinosinusitis and Kartagener's syndrome. In chronic sinusitis ultrastructural changes in the mucosa with secondary ciliary dysfunction is seen , with 23% mucosal samples showing absent ciliary activity. Isotope methods show that there is a lesser drainage of tracer substances in sinuses with

a. Retention of fluid

b. Thick mucosa

Quantitative and qualitative changes in the secretion, including the delicate periciliary fluid layer is of greater importance for the impairment of mucociliary transport during sinus inflammation than the structural abnormality of the cilia or their retarded beat rate. Ciliary impairment in the presence of purulent secretions is due to-

a. High proteolytic enzyme activity

b. Low pH

c. Anaerobic mucosal metabolism

d. Impairment of mucosal oxygen supply (rare)

SECRETION AND TRANSPORTATION[1]

Principles

Drainage and ventilation are two important factors for the normal physiology of the paranasal sinuses and their mucus membranes. Drainage depends on secretion and transport mechanisms which in turn depend on -

- Amount of mucus
- Composition of mucus
- Effectiveness of ciliary beat
- Mucosal resorption
- Condition of ostia and ethmoidal clefts
- Free flow of inspired air
- Mucosal pulsations and movements of fontanelles (in case of inflammation)

The nasal mucosa is lined by a mucous blanket which is produced by the mucoserous nasal glands and intraepithelial goblet cells. Mucus is a complex non Newtonian fluid whose quality and quantity are important and requires an intact blood supply and nervous system. It is composed of -

1. Water & ions

2. Glycoproteins (sialomucins , fucomucins , sulphomucins)

3. Enzymes (lysozymes, lactoferrin)

4. Circulatory proteins (Complement , α 2 macroglobulin , CRP)

5. Ig- IgA , IgE , IgG , IgM , IgD

6. Cells – surface epithelium , basophils , eosinophils , leucocytes.

The mucus film has two layers – the *sol* phase , which is the *inner serous* layer (water and ions) and the *gel* phase , which is the *outer* viscous layer (glycoproteins). Cilia beat in the inner sol phase and the outer gel phase also moves along over the sol phase like a "carpet". Dust particles adhere to the gel phase for being transported out of the paranasal sinuses. A pH of range of 7.5 – 7.6 is required for maintaining an equilibrium between the sol phase and gel phase. The production and composition of this mucus is dependent on –

- Humidity
- Pollution
- Airborne irritants

The mucosal glands are controlled mainly by the parasympathetic fibers. The nerve fibers from the Superior Salivatory Nucleus via the Greater Petrosal Nerve reach the Pterygopalatine ganglion, from where the postganglionic fibers supply the mucosal glands.

The sympathetic fibers arise from the Lateral Horn of the spinal cord. The postsynaptic fibres via the Carotid plexus form the Deep Petrosal Nerve which joins the Greater Petrosal Nerve to form the Vidian Nerve which ultimately supplies the nasal and sinus mucosa.

Substance P secreted from type C fibers via local reflexes also have great effect on mucosal glands. They are found to produce Hypersecretion , Vasodilation and extravasation of plasma.

On an average the maxillary sinus mucus layer is renewed every 20 – 30 minutes. The mucosal secretions form a homogenous layer all along the walls of the sinuses except at the ostia where the viscous layer is found to be thicker as secretions from the whole sinus converge there.

When mucosal surfaces come close to each other leaving a recess in between, the gap is filled by the mucosal blanket by a the *Bridging phenomenon*, whereby the cohesive forces in the gel phase bridge the gap, while the sol phase fills the recess in between. Similarly the flow over a small mucosal defect also goes unhindered due to cohesive nature of the mucus carpet.

But when the mucus is too viscous, this defect can prove to be an obstacle with the secretion being retained at the site of the defect. Similarly at the region of crests a thick secretion will be retained for a while and finally drain away under the influence of gravity. A highly

viscous mucus can block the primary maxillary ostium ,later fall down into the sinus only to be transported again towards the primary ostium. If the sinus ostium is oval or oblong , theciliary beat works on the mucus from two or three sides and the mucus passes through the corners of the ostium.

In cases where an Accessory ostium of up to 4mm is present , a normal secretion bypasses it, and a viscous secretion moves over it without leaving the sinus through it. But in larger accessory ostia the part of the mucus passing through its middle gets transported out, whereas that moving around the corners get bypassed. This same phenomenon can be visualized in inferior meatal antrstomies. Accessory ostia also are sites of what is called *'recirculation'* i.e. Mucus from the ethmoidal infundibulum moves into the maxillary sinus via the accessory ostium and then out through the natural ostium only to return back through the accessory ostium.

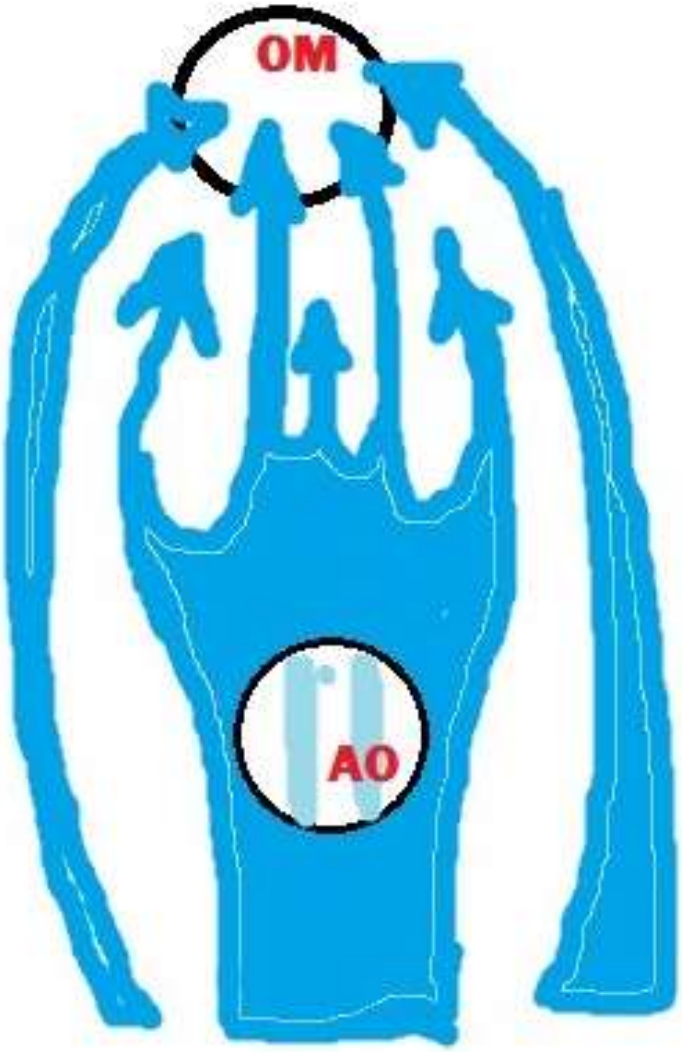


Fig 3 - Mucous Blanket moving over and across the Accessory Ostium towards the Natural Ostium

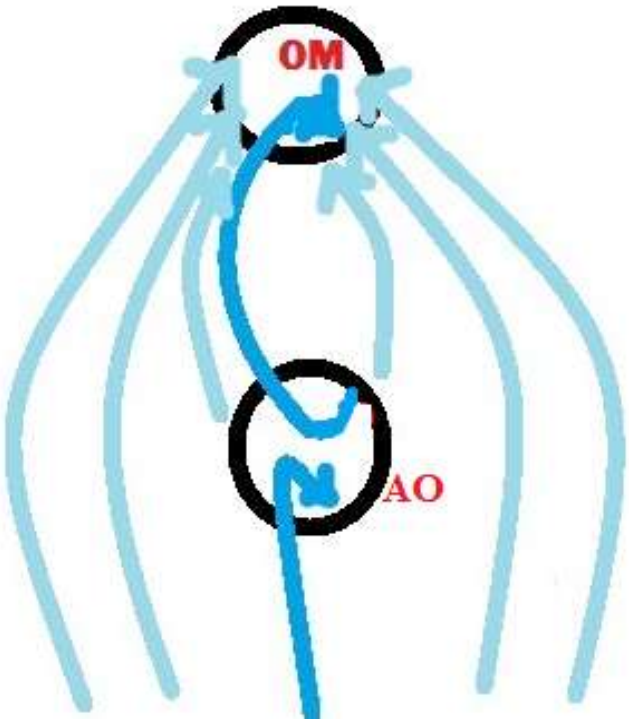


Fig 4 - Blue arrow shows circular transport of mucous

SECRETION TRANSORT PATHOLOGY

Hypersecretion causes the mucus to flow into the deepest part of a sinus due to gravity. In a normally composed mucus , the gel layer persists on the surface. Active cilia in the non drowned part of the mucosa may pull away the mucus carpet on the surface because of cohesion , provided the cilia beat normally and the direction of transport in the corresponding areas are not opposing each other.

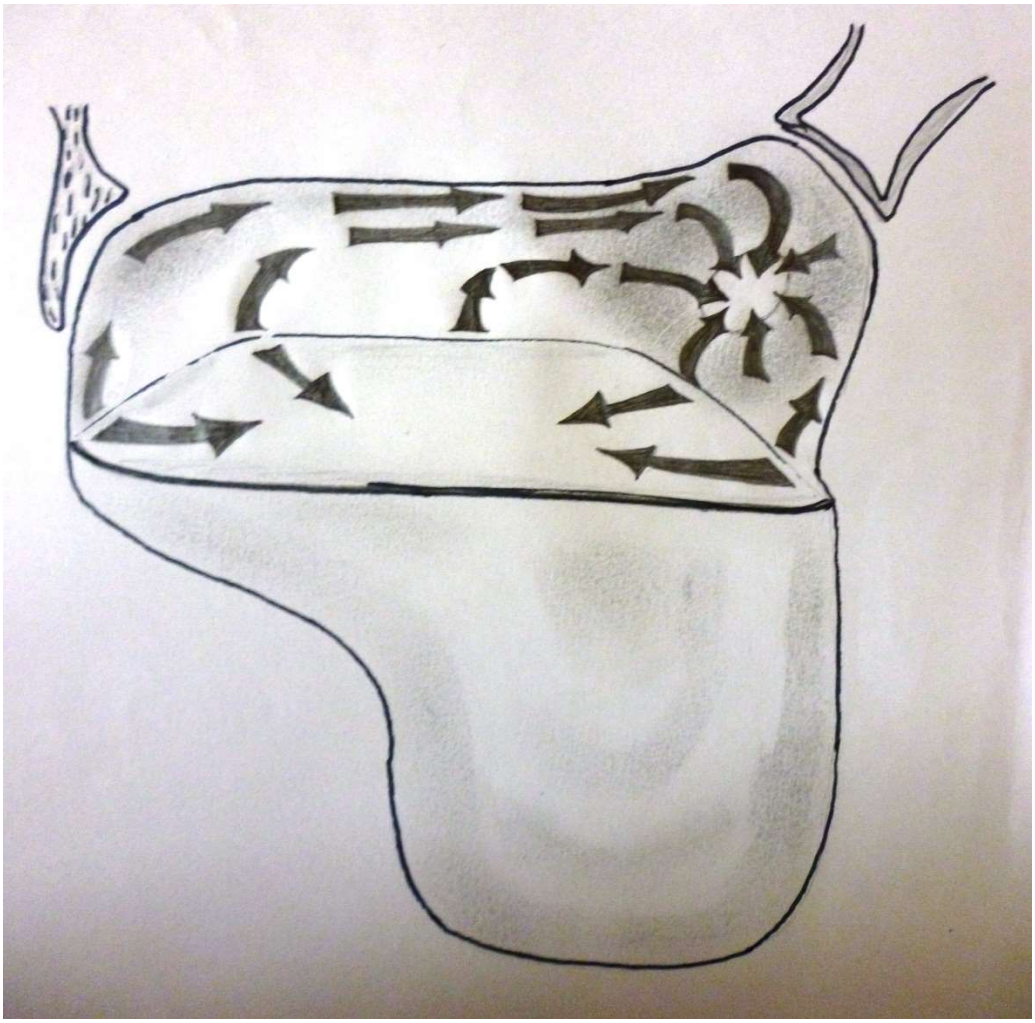


Fig 5 - A half filled maxillary sinus. The cohesional forces of the gel phase are holding back the transportation of the mucus. The still intact cilia are not powerful enough to 'tear off' the gel layer from the surface

In case of decreased secretions or reduced humidity at the surface , sol phase becomes rather thin. The mucus becomes very viscous and the gel phase comes in direct contact with the cilia , thereby impeding their action resulting in a worm like movement of the mucus layer.

During inflammation of the sinuses , the mucosa gets inflamed rapidly and even pulsate. Also increased movements in the region of the membranous portions of the fontanelles may occur. Such mucosal movements may also assist the transport of secretions out of the maxillary sinus.

There is another phenomenon known as "*secretion expressways"* which is found in both cases of abnormal secretions as well as apparently normal sinuses. This means that the transport of secretion is not uniform throughout a sinus. From time to time the mucosa of one region transports secretion faster than the neighboring areas. In some time the slower areas catch up speed and the faster region slows down.

The normal ciliary beat frequency ranges between 8 – 20 beats per second. Optimal mucociliary clearance system requires –

- Normal ventilation
- Humidification
- Normal Metabolism
- Osmotic pressure
- Optimal pH – 7-8
- Optimal temperature – around 33^0 C
- protection from noxious stimuli

TRANSPORT OF SECRETION IN THE MAXILLARY SINUS

Hilding Sr. and later Messerklinger made the discovery that secretions from sinuses follow a genetically predetermined route to reach their respective ostia. In the maxillary sinus the secretions are transported in a *stellate* pattern from the floor. The secretions pass along the anterior , medial posterior , lateral walls and the roof of the sinus and finally converge at its natural ostium. From there it moves to the floor of the posterior third of the ethmoidal infundibulum where the natural ostium usually opens. In the ethmoidal infundibulum the secretions from the frontal and anterior ethmoidal sinuses usually join. The ethmoidal infundibulum via the Hiatus

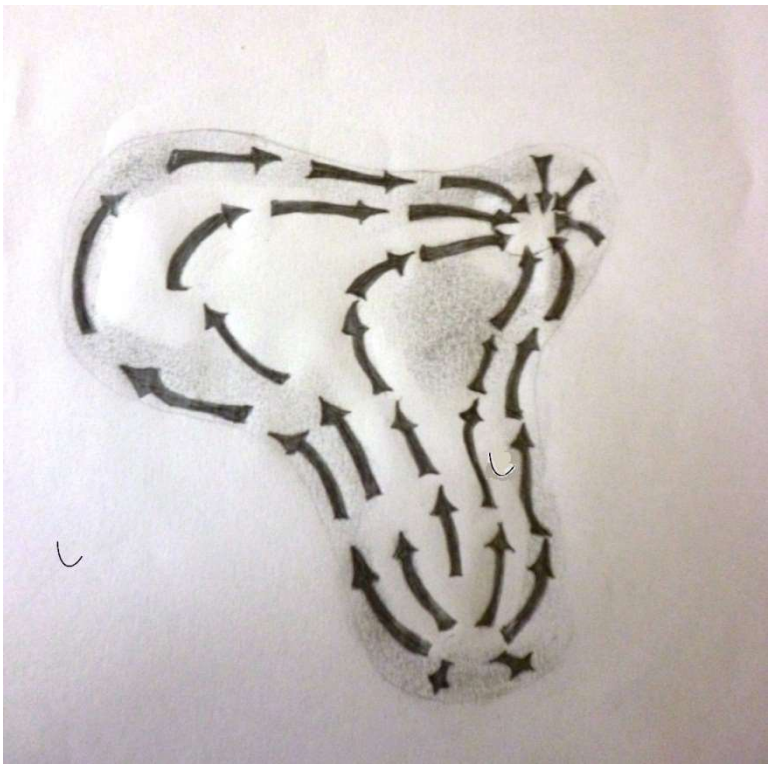


Fig 6 - Mucous Transportation Pathways inside Maxillary Sinus

semilunaris drains into the middle meatus. From here the secretion passes over the posterior free border of the 41ntermoda process and along the medial face of the inferior turbinate and then into the nasopharynx posteriorly where they pass anterior and inferior to the Eustachian tube orifice. Up to the border of the ciliated and squamous epithelium active transport continues after which secretions move downwards by gravity aided by the swallowing mechanism.

Secretions from the maxillary sinus always tend to pass through the natural ostium even in the presence of a single or multiple accessory ostia or a surgically created inferior meatal antrostomy.

MECHANISM OF MAXILLARY SINUSITIS

During the transport of secretions through the ethmoidal infundibulum, which is a prechamber through which the maxillary sinus communicates with the middle meatus, if its opposing mucosal surfaces come into intense contact and firmly pressed against each other pathologically , the cilia in the region get immobilized and mucus transport is hampered. Obstruction in such a key area acts like a 'bottle neck' and affects the ventilation and drainage of the whole maxillary sinus resulting in retained secretions. When this area of obstruction expands or an infection ensues the retained secretions acts as a culture medium for bacteria or virus , thus perpetuating the vicious cycle.

Also poor ventilation decreases the pH of the sinus , further slowing down its ciliary movement with the resultant formation of viscous mucous. Such viscous mucous may remain in the sinus for a considerable length of time due to the blockade in the prechamber. This again is a favorable condition for microbial growth, toxins of which may cause further impairment of the mucosal function. This is an instance where an otherwise normal sinus ends up diseased due to a pathology that lies in the outflow tract.

Inhaled pathogens deposited at the entrance of the middle meatus adhere to the mucus. Due to the confluence of the mucus pathways of the ethmoidal infundibulum and the frontal recess , the microbes get transported into either of the sinuses. In the presence of a suboptimal self healing capacity of the sinus mucosa or inadequate antibiotic therapy an Acute or a Chronic recurring sinusitis results.

Maxillary sinuscopic studies have revealed that viscous mucus sometimes enters the sinus through accessory ostiapresent in either of the fontanelles. This mucus is transported along the natural pathways towards the natural ostium where it exits the sinus. This mucus may again re enter the sinus via the accessory ostium and this may continue endlessly. As long as the natural ostium is patent this is not of much significance. But if it is blocked or in the presence of a nasal infection this plays an important role in the transport of pathogens into the maxillary sinus from

the nose. Due to ostial blockade infected secretions are unable to leave the sinus resulting in maxillary sinusitis.

Thus the cause of infections of the large sinuses like maxillary sinus is mostly rhinogenic. It usually spreads from the nose through the ethmoid clefts and prechambers into the maxillary and frontal sinuses. An exception to this is dentogenic sinusitis (<2%). Other such exceptions are foreign bodies in the maxillary sinus (aberrant root filling material) , blood in the sinus following trauma, cholesterol or mucus retention cysts (depending on their location).

ADRENERGIC DRUGS (Sympathomimetics)

Drugs with actions similar to that of adrenaline or of sympathetic stimulation are called Adrenergic drugs.

Direct sympathomimetics

These act directly as agonists on β or/and α adrenoceptors examples are noradrenaline, isoprenaline, xylometazoline, phenylephrine, adrenaline, methoxamine, salbutamol etc.

Indirect sympathomimetics

These first act on adrenergic neurone to release noradrenaline, which then acts on the adrenoceptors examples are amphetamine, tyramine.

Mixed action sympathomimetics

These act directly and indirectly examples are dopamine, ephedrine, mephentermine etc.

ACTIONS

Depending on the predominant receptor type present in a given tissue, the peripheral actions of adrenaline have been clearly differentiated into those that are mediated by β or α receptors. Actions of a particular sympathomimetic amine are depend on its relative activity at different types of adrenergic receptors.

The overall actions are —

1. Heart

By increasing the slope of slow diastolic depolarization of cells in the SA node, adrenaline increases the heart rate. The latent pacemakers in A-V node and Purkinje fibres are activated, hence arrhythmias can occurs. With high doses the BP raises markedly. Reflex depression of the SA node occurs due to the raised BP and this unmasks the latent pacemakers. Anaesthetics like halothane and chloroform sensitizes the heart to the arrhythmic action of adrenaline. In patients with complete heart block the idioventricular rate is increased. The force of cardiac contraction is increased and the development of tension as well as relaxation are accelerated. Systole is hence shortened more than the diastole. This markedly enhances the oxygen consumption of the heart and the cardiac output. There is increased conduction velocity through bundle of His, A-V node,

ventricular and atrial fibres. Hence administration of these drugs may be able to overcome a partial A-V block. There is reduced Refractory period (RP) of all types of cardiac cells. Cardiac actions are all predominantly β1 receptor mediated. BP rises markedly – stimulation of vagus – reflex bradycardia occurs. When NA is injected i.v this is the usual response seen.

2. Blood vessels

Both vasodilatation (β2) and vasoconstriction (α) may occur depending on the vascular bed, the drug that is used and its dose. In mucous membrane, cutaneous and renal beds constriction predominates. Through both α2 and α1 receptors vasoconstriction can occur. However, only circulating Cas are able to activate α2 (extrajunctional) receptors due to its location. The α1 (junctional) receptors on the other hand primarily mediate responses to NA that is neuronally released. In liver, skeletal muscles and coronaries dilatation predominates. The direct effect on cerebral vessels is not prominent— change in BP causes changes in blood flow through these beds. The most marked action is on arterioles however at higher doses larger arteries and veins are affected.

3. BP

The amine, its dose and the rate of administration determines the effect on BP.

• Rise in systolic, diastolic and mean BP is caused by NA. It does not however cause vasodilatation as there is no β2 action, hence the peripheral resistance increases consistently due to α action alone.

• Isoprenaline causes marked fall in diastolic BP but rise in systolic BP (β2—vasodilatation, β1—cardiac stimulation). The mean BP however generally gets reduced.

• Adrenaline given by s.c. injection or slow i.v. infusion causes a fall in diastolic BP but rise in systolic BP. Because vascular β2 receptors are more sensitive than α receptors the peripheral resistance decreases. Mean BP however generally rises. Pulse pressure is also increased.

• Marked increase in both systolic as well as diastolic BP is seen after rapid i.v. injection of adrenaline (at high concentration vasoconstriction occurs even in skeletal muscles and α response predominates). Within a few minutes the BP returns to normal and this is followed by a secondary fall in the mean BP. The mechanism is as follows:

- rapid uptake and dissipation of drug
- concentration of adrenaline is reduced
- Lower concentrations are unable to act on α receptors but still continue to act on β2 receptors.
- Only fall in BP is seen when an α blocker has been given this is called *vasomotor reversal of Dale.*

4. Respiration

Isoprenaline and adrenaline are very potent bronchodilators. But NA is not. When the bronchi are constricted this action is more marked. Decongestion

of bronchial mucosa occurs when given as aerosol by action on alpha receptors. Direct stimulation of the respiratory centre (RC) can occur. However, this action is rarely manifest at doses that are clinically used. Transient apnoea can occur due to reflex inhibition of the respiratory centre. This occurs when rapid i.v. injection in animals is carried out. By shifting blood from systemic to pulmonary circuit, toxic doses of adrenaline can cause pulmonary edema.

5. Eye

When the radial muscles of iris contract, mydriasis occurs. After topical application, however, this action is minimal, because penetration of the cornea by adrenaline is poor. The intraocular tension usually tends to fall. This effect is most marked in wide angle glaucoma.

6. GIT

Relaxation occurs through activation of both β and α receptors, in isolated preparations of gut. However in vivo, both in animals and in man, sphincters are constricted and peristalsis is reduced. These effects however are of no clinical significance.

7. Bladder

Overall actions of adrenaline on the bladder tend to restrict micturition

8. Uterus

Contraction or relaxation of the uterine muscles can occur due to adrenaline. Gestational status, effect of hormones and type of species decides the overall effect.

9. Splenic capsule

In animals it contracts the splenic capsule and hence pours more RBCs into circulation. However this action is not seen in man.

10. **Skeletal muscle**

Facilitates neuromuscular transmission. On motor nerve endings acetyl cholinesterase release is augmented. This is in contrast to action on autonomic nerve endings. Effect on muscle fibres differs according to the type of fibre and is exerted through β2 receptors. Due to incomplete fusion of individual responses less tension is developed in the slowly contracting red fibres and the active state is abbreviated. This is responsible for the tremors produced by β2 agonists along with enhanced firing of muscle spindles. Increasing the tension developed and prolonging the active state is the main action on rapidly contracting white fibres.

11. CNS

Adrenaline can cause apprehension, restlessness, and tremor. However, in clinically used doses there is poor penetration in brain and it does not produce any marked CNS effects. Fall in BP and bradycardia may occur due to activation of α2 receptors and consequent decrease in sympathetic outflow in the brainstem.

12. Metabolic

Glycogenolysis leads to hyperglycaemia and hyperlactacidaemia. Rise in plasma free fatty acid results from lipolysis. Adrenaline also causes calorigenesis and transient hyperkalaemia which followed by hypokalaemia due to direct action on muscle, liver and adipose tissue. Other metabolic effects result from augmentation of glucagon secretion reduction of insulin.

Biochemical mediation of adrenergic responses

β *actions*

Mediated through cAMP. Through a regulatory protein Gs adrenaline activates membrane bound enzyme adenylyl cyclase. At the inner face ATP is broken down to cAMP. A number of intracellular cAMP-dependent protein kinases are in turn phosphorylated and this initiates a series of reactions:

(i) Liver and Muscle

Glycogen synthetase is inhibited and glycogen phosphorylase is activated, in liver and muscle. The result is hyperlactacidemia and hyperglycaemia. In liver the response is augmented by neoglucogenesis. Hyperkalaemia initially results from potassium being released from liver. This is then followed by uptake in muscle and later in liver itself and so a more prolonged hypokalaemia ensues.

(ii) Adipose tissue

Increased plasma free fatty acids results from activation of triglyceride lipase. Action on brown adipose tissue result in increased heat production and O2 consumption. This action is mediated by the predominant β3 receptors.

(iii) Heart

Phosphorylation of proteins like troponin and phospholamban occur. The phosphorylation of troponin results in increased interaction with calcium at the myofilaments. This in turn increases the force of contraction. The

phosphorylation of phospholamban, however, causes sequestration of calcium in sarcoplasmic reticulum, which causes more rapid relaxation. In addition, the activated protein Gs promotes influx of calcium by interacting directly with the calcium channels in the membrane. This reinforces the positive inotropic action that is exerted through cAMP.

(iv) Gut and Bronchial muscle

Though the intermediate steps are not clear relaxation is induced along the hyperpolarization

(v) Pancreatic islets

Action on α cells increases glucagon secretion by activation of β2 receptors. Action on β cells increases insulin secretion. Both are mediated by raising intracellular cAMP. Augmentation of insulin secretion is, however, weak.

Adverse effects and contraindications

- After s.c./i.m. injection of adrenaline palpitation, restlessness, tremor, anxiety, pallor that are transient, occurs.
- Inadvertant i.v. injection or large doses of adrenaline can cause marked rise in BP leading to ventricular tachycardia/fibrillation, angina, cerebral haemorrhage and myocardial infarction.
- In hyperthyroid, hypertensive, and angina patients, adrenaline is contraindicated.

- During anaesthesia with halothane, adrenaline should not be given. Further, patients receiving β blockers should also be avoided from being administered adrenaline.

Local Anesthetics (LAs)

These are drugs which upon topical application or local injection in a restricted area of the body will cause reversible loss of sensory perception, especially of pain. Without causing any structural damage they block generation and conduction of nerve impulse, where they come in contact with, at all parts of the neuron. When applied to a mixed nerve, not only sensory but also motor impulses are interrupted, resulting in loss of autonomic control as well as muscular paralysis.

CHEMISTRY

Being weak bases with amphiphilic property, all clinically useful Las have a lipophilic aromatic residue on one side and a hydrophilic secondary or tertiary amine on the other, joined by an alkyl chain via an amide or ester linkage. Ester-linked Las Cocaine, tetracaine, benzocaine, procaine, chloroprocaine. Amide-linked Las Lidocaine, dibucaine, bupivacaine, ropivacaine, prilocaine.

Features of Amide Local Anesthetics

• Known to produce more longer lasting and intense anesthesia

• In plasma found to bind to α1 acid glycoprotein

• Plasma esterase do not cause hydrolysis

• No cross sensitivity with ester Las and rarely known to cause hypersensitivity reactions

The ester-linked Las are rarely used for infiltration or nerve block because of their less intense analgesia, shorter duration of action and higher risk of hypersensitivity. They are mainly still used as topical preparations on mucous membranes.

MECHANISM OF ACTION

During upstroke of action potential (AP), by decreasing the entry of Na+ ions, the Las block nerve conduction. As the concentration of the LA is increased, maximum depolarization and the rate of rise of AP decreases causing slowing of conduction. Conduction block ensues when finally, local depolarization fails to reach the threshold potential.

A receptor is situated within the voltage sensitive Na+ channel. It raises the threshold of channel opening. The Las interact with these receptors. In response to an impulse or stimulus, Na+ permeability fails to increase. When the Na+ channels over a critical length of the fibre that is 2–3 nodes of Ranvier in case of myelinated fibres, are blocked the impulse conduction is interrupted. The LA molecule is partly ionized at physiological pH. The equilibrium between the ionized cationic form (BH+) and the unionized base form (B) depends on the pKa of the LA. The cationic form of LA is the predominant active species which is

able to approach its receptor only when the channel is open at the inner face and it binds more avidly to the inactive state of the channel, prolonging the inactive state. The refractory period of the fibre is increased as the channel takes longer to recover. Blockade develops rapidly when the nerve is stimulated and a resting nerve is rather resistant to blockade.

The Na+ channel has an inactivation gate ('h' gate) at the intracellular mouth and an activation gate ('m' gate) near its extracellular mouth. In the resting state the activation gate is closed. When threshold depolarization of the membrane is reached it opens up the activation gate allowing the Na+ ions to flow in along the concentration gradient. The inactivation gate closes within a few milliseconds and ion flow ceases. In a time-dependent manner the channel recovers to the resting state. Within the channel, the local anesthetic (LA) receptor is located in its intracellular half. The LA traverses the membrane in its unionized lipophilic from (B). It then reionizes in the axoplasm. Then it approaches the LA receptor through the intracellular mouth of the channel. It is the cationic form (BH+) of the LA which primarily binds to the receptor. When compared to the resting state, the receptor is more accessible to or has higher affinity for the LA in the activated state. Binding of the LA to its receptor stabilizes the channel in the inactive state. This reduces the probability of channel opening. The neuronal Na+ channel is actually a 300 KD glycoprotein composed of a quite large (α) and two rather small (β1, β2) subunits. The α – subunit encloses the sodium ion selective pore within its 4 homologous domains (I to IV).

Each domain has exactly 6 membrane spanning helical segments (S1 to S6) that are connected alternately by intracellular and extracellular loops. The wall of the pore is now formed by all four S5-S6 segments. The short non-helical loops connecting S5-S6 on the extracellular surface serve as the activation gate after folding into the pore. Voltage sensors that are located in the S4 segments move vertically on depolarization. They then open the activation gate by allosteric conformational change. A few milliseconds later, the short intracellular loop which are connecting domains III and IV fold into the inner mouth of the pore thereby inactivating the channel. The LA receptor is mostly located in the S6 segment of domain IV. Channel activation transforms the LA receptor to a higher affinity conformation. It may also expose it on the wall of the pore. This may then persist during the subsequent inactivation phase. Degree of blockade is frequency dependent. Greater blockade is achieved at higher frequency of stimulation. Moreover, exposure to higher concentration of Ca2+ can reduce inactivation of Na+ channels and hence lessens the degree of block. Blockade of conduction by LA is not quite due to hyperpolarization. In fact the resting membrane potential is not changed because K+ channels are blocked only at much higher concentrations of LA. The onset time of blockade is related mainly to the pKa of the LA. Those that have lower pKa (7.6–7.8), for example mepivacaine and lidocaine, are actually fast acting, because 30–40 percent of the LA is in the un dissociated base form when at pH 7.4 and this is the form which penetrates the axon. Tetracaine, procaine, bupivacaine have higher pKa (8.1–8.9). Only 15 percent or even less is unionized at a pH of 7.4. These are hence slow

acting. Chloroprocaine is an exception. Chloroprocaine has rapid onset despite high pKa (9.1).

LOCAL ACTIONS

The clinically used Las have no or have minimal local irritant action. They block sensory nerve endings, neuromuscular junction, nerve trunks, receptors and ganglionic synapse non-selectively. That is to say that they are structures which function through increased Na+ permeability. They also are able to reduce release of acetylcholine from motor nerve endings. When injected around a mixed nerve they cause anesthesia of skin along with paralysis of the voluntary muscle that is supplied by that nerve.

Motor and sensory fibres are equally sensitive. The sensitivity is determined by fibre type as well as by diameter of the fibres. When the diameter is remaining the same, the non – myelinated nerves are blocked after the myelinated nerves. In general, larger fibres are less sensitive than smaller fibres. Different fibres have different critical lengths of axons to which the LA needs to be exposed to for blockade. Smaller fibres usually tend to have shorter critical lengths. This is because in them voltage changes propagate passively for shorter distances. More slender axons have shorter internodal distances. Las can hence easily enter the axon at the nodes of Ranvier. At these nodes the density of Na+ channel is much higher. Moreover, smaller sensory fibres generate high frequency longer lasting action potentials than the motor fibres. The frequency

dependence of blockade hence makes them more vulnerable to Las. Somatic fibres are generally less susceptible than autonomic fibres. Pain then temperature sense then touch and finally deep pressure sense. This is the order of blockade among the somatic afferents. Pain is the first modality to be affected, since pain is generally carried by fibers having smaller diameter, than those carrying motor impulses or other sensations. When applied to the tongue the order of loss of taste sensations are first bitter then sweet then sour and finally salty. Mostly fibres that are more susceptible to local anesthetics are the first to be blocked and also the last to recover. Furthermore the location of the fibre within a nerve trunk determines often the depth of local anesthesia, the duration and latency. Fibres in the outer layers are blocked earlier than the inner or core fibres. This is because the nerve sheaths restrict diffusion of the LA into the nerve trunk. The axons that supply the more proximal areas are located more peripherally in the nerve and those that supply the distal areas are located in the core. Hence the more proximal areas that are supplied by a nerve are affected earlier. The differential arrangement in a mixed nerve, of various types of sensory and motor fibres, may partly account for the differential blockade.

The local anesthetic fails to afford adequate pain control in inflamed tissues.

The possible reasons are:

a. Lower pH in inflamed tissues — greater fraction of the LA is in the ionized form.

b. Increased blood supply to the inflamed tissue — hence the drug is removed more efficiently from the site.

c. Effectiveness of adrenaline that is injected along with the local anesthetic is significantly reduced at the site of inflammation.

d. The products of inflammation may reduce the effectiveness of the local anesthetic. .

Adding a vasoconstrictor like adrenaline to the local anesthetic has the following actions:

• By decreasing their rate of removal from the local site into the general circulation, it prolongs duration of action of local anesthetics.

• Intensity of the nerve block is enhanced.

• Systemic toxicity of local anesthetics is reduced as the rate of absorption is reduced and the plasma concentration is kept low due to metabolism.

• The infiltration is made more painful.

• A bloodless field is provided for surgery.

• The chances of subsequent necrosis and local tissue edema is increased. It also delays wound healing by enhancing oxygen consumption in the affected area and reducing oxygen supply.

• The drug can promote arrhythmia and raise BP in certain susceptible individuals.

SYSTEMIC ACTIONS

Local anesthetics that are applied locally or injected is finally absorbed and can produce systemic effects. This depends on what concentration is attained in the tissues and plasma.

C.N.S.

All local anesthetics are capable of producing a sequence of stimulation which is then followed by depression. Cocaine is a powerful CNS stimulant. It causes a sequence of euphoria followed by excitement then mental confusion leading to restlessness, tremor and twitching of muscles. Then convulsions, unconsciousness, respiratory depression and even death can occur, in a dose-dependent manner.

Procaine and other synthetic local anesthetics are less potent in this regard. They produce little apparent CNS effects at safe clinical doses. Accidental i.v. injection or higher dose produces CNS stimulation which is then followed by depression. On the contrary, Lidocaine, can initially cause lethargy and drowsiness. At higher doses it may produce excitation followed by depression like other Las.

All Las basically cause neuronal inhibition. The stimulation seen initially is apparently due to inhibition of the inhibitory neurons. At higher doses, all neurons are inhibited and EEG waves are flattened.

C.V.S.

Heart

Local anesthetics cause cardiac depression. At conventional doses no very significant effects are observed. Inadvertent i.v. injection or at very high doses, they decrease excitability, automaticity, conductivity, contractility, and increase the effective refractory period (ERP). They have an antiarrhythmic action similar to that of quinidine. Procaine is not used as antiarrhythmic because of its propensity to cause CNS side effects and due to the short duration of action. The amide derivative of procaine, however, called procainamide, is a classical antiarrhythmic. Electrophysiological properties of heart may be markedly altered at high plasma concentrations. The Las can themselves induce cardiac arrhythmias and the QTc interval may be prolonged. Bupivacaine has produced fibrillation or ventricular tachycardia and is relatively more cardiotoxic. Lidocaine has very little effect on conductivity and contractility. It is used as an antiarrhythmic as it abbreviates effective refractory period.

Blood vessels

Local anesthetics tend reduce BP, which is primarily due to sympathetic blockade. At high concentrations, however, they cause direct relaxation of

arteriolar smooth muscle. Such concentrations are obtained locally at the site of injection. Bupivacaine causes more vasodilation than lidocaine. Prilocaine causes the least vasodilation. Toxic doses of any local anesthetic will produce cardiovascular collapse.

PHARMACOKINETICS

Soluble surface anaesthetics like lidocaine and tetracaine are quite rapidly absorbed from mucous membranes and from abraded areas. However absorption from intact skin is very poor. Procaine is a local anesthetic that does not significantly penetrate mucous membranes. The rate of absorption of the local anesthetic will depend on the blood flow to the area of injection or application. The absorbed local anesthetic being lipophilic is widely distributed. It then rapidly enters highly perfused heart, brain, kidney and liver. Following this it enters the muscle and other viscera.

Amide local anesthetics are bound to plasma protein α1 acid glycoprotein. Procaine, however, is negligibly bound to plasma proteins. Local anesthetics bind rapidly to tissues but only temporarily, especially nerves. Local anesthetics that are ester-linked example procaine are quite rapidly hydrolyzed by plasma pseudo cholinesterase. The remaining are hydrolyzed by esterase in the liver. Local anesthetics that are amide-linked are degraded by hydrolysis and dealkylation only in the liver microsomes. The metabolism of lidocaine is hepatic blood-flow dependent. The maximal safe dose of local anesthetic in the elderly and in

patients with hepatic disease is much lower than normal individuals. These groups of people have decreased liver function. After oral ingestion both lidocaine and procaine have very high first pass metabolism in the liver. For the same reason they cannot be used orally for antiarrhythmic purposes.

ADVERSE EFFECTS

The intrinsic anaesthetic potency of the LA determines the systemic toxicity on rapid i.v. injection. Toxicity after regional injection or topical application is, however, influenced by the relative rates of absorption and metabolism. Those that are slowly metabolized but relatively rapidly absorbed tend to be more toxic.

(1) CNS effects are dizziness, light-headedness, auditory and visual disturbances, disorientation, mental confusion, twitchings, shivering, involuntary movements, convulsions and finally respiratory arrest. This can be prevented as well as treated by diazepam.

(2) Cardiovascular toxicity of local anesthetics is manifested as hypotension, bradycardia, cardiac arrhythmias leading to cardiovascular collapse.

(3) Local tissue toxicity of local anesthetics is low. However, local injection of the may be painful. Wound healing may be sometimes delayed. The addition of vasoconstrictors to the local anesthetics may enhance the local tissue damage. Rarely it may lead to necrosis. Vasoconstrictors should never be added for ring block of fingers, hands, penis, feet, toes, and in the pinna.

(4) Hypersensitivity reactions like angioedema, rashes, contact sensitization, dermatitis, asthma and very rarely anaphylaxis may occur. Such reaction are more common with local anesthetics that are ester-linked. However they are rare with lidocaine or its congeners.

Precautions and interactions

1. Aspirate lightly before injecting the LA, to avoid intravascular injection

2. Take care not to exceed the maximum safe dose especially in children. Inject the LA very slowly.

3. Use of concurrent propranolol may result in the reduction of metabolism of lidocaine and other amide Las by reducing blood flow to the liver.

4. Local anesthetic containing vasoconstrictor agents like adrenaline should be avoided for patients with cardiac arrhythmia, uncontrolled hypertension, ischaemic heart disease, thyrotoxicosis, and also those receiving tricyclic antidepressants or β blockers

Lidocaine (Lignocaine)

Introduced back in 1948, lidocaine is currently the most widely used local anesthetic. It is very versatile and good both for injection as well as surface application. It is available in a variety of forms. When injected around a nerve, it blocks conduction within 3 minutes. The anesthesia is more intense and longer lasting than procaine. Vasodilatation usually occurs in the injected area. It is used

for nerve block, surface application, epidural, infiltration, spinal and intravenous regional block anesthesia. It has no cross sensitivity with ester linked local anesthetics. As compared to other Las, early central effects of lidocaine are mental clouding, drowsiness, tinnitus and altered taste. Like other local anesthetics overdose causes convulsions, muscle twitching, fall in BP, cardiac arrhythmias, coma and respiratory arrest.

MATERIALS AND METHODS

55 Patients (Male and female in the age group of 20-45 years) with chronic bilateral sinusitis who attended the ENT Out Patient Department and were planned for endoscopic sinus surgery on both sides, during the period March 2015 to August 2016 , who satisfied the inclusion criteria were enrolled for the study after getting an informed written consent.

Study design: Cross Sectional Observational Study

Study place: Department of ENT, Government Stanley Medical College and Hospital.

Study and Follow-up period: March 2015 to August 2016.

Sample size: 55 patients

Equipment Used:

1. 2ml syringe
2. Sterile Gloves
3. Sterile Gauze
4. Sterile Cotton
5. Jobson Horne Ear Probe
6. Tongue Depressor

7. Lignocaine with Adrenaline (2% lignocaine with 1:80000 adrenaline) Solution
8. Headlight

Table for recording the readings

Name: Age: Sex:

S. No.	Time (minutes)	Right Side Grade	Left Side Grade
2	30		
4	60		
6	90		
8	120		
10	150		
12	180		

The Wormald Grading System for Bleeding During Endoscopic Sinus Surgery

Grade	Surgical Field
0	No bleeding
1	1–2 points of ooze
2	3–4 points of ooze
3	5–6 points of ooze
4	7–8 points of ooze

5	9–10 points of ooze (sphenoid fills after 60 seconds)
6	>10 points of ooze, obscuring surface (sphenoid fills between 40 – 60 seconds)
7	Mild bleeding/oozing from entire surgical surface with slow accumulation of blood in postnasal space (sphenoid fills in 40 seconds)
8	Moderate bleeding from entire surgical surface with moderate accumulation of blood in postnasal space (sphenoid fills in 30 seconds)
9	Moderately severe bleeding with rapid accumulation of blood in postnasal space (sphenoid fills in 20 seconds)
10	Severe bleeding with nasal cavity filling rapidly (sphenoid fills in <10 seconds)

METHOD OF STUDY

Patients who came with bilateral chronic sinusitis or bilateral sinonasal polyposis and who required similar surgical intervention on both sides and who also fit the inclusion criteria were included in this study.

Being an observational study the sample size is not defined prior to the study. All patients fitting the inclusion criteria during the study period are included in the study. This yielded a total of 55 patients. Of these 28 were males and 27 females.

Written informed consent regarding the procedure, the potential benefits and side effects were taken from all patients. For all patients undergoing an endoscopic sinus surgery a computed tomography of the paranasal sinuses were taken. This provided information about the extent of the disease on each side and helped in planning what kind of procedure was to be done on each side. If exactly the same procedure was planned on each side then the patient was included in the study.

Prior to surgery each patient was routinely tested for hypersensitivity to the infiltration solution i.e. lignocaine with adrenaline (2% lignocaine and 1:80000 adrenaline). This was done by injecting 0.2ml of the solution intradermally on the right forearm of the patient and then testing for induration, erythema or other signs of local hypersensitivity reaction after 30 minutes and then again after 120 minutes. Those patients showing sensitivity to the mixture were excluded from the study.

THE SPHENOPALATINE ARTERY BLOCK

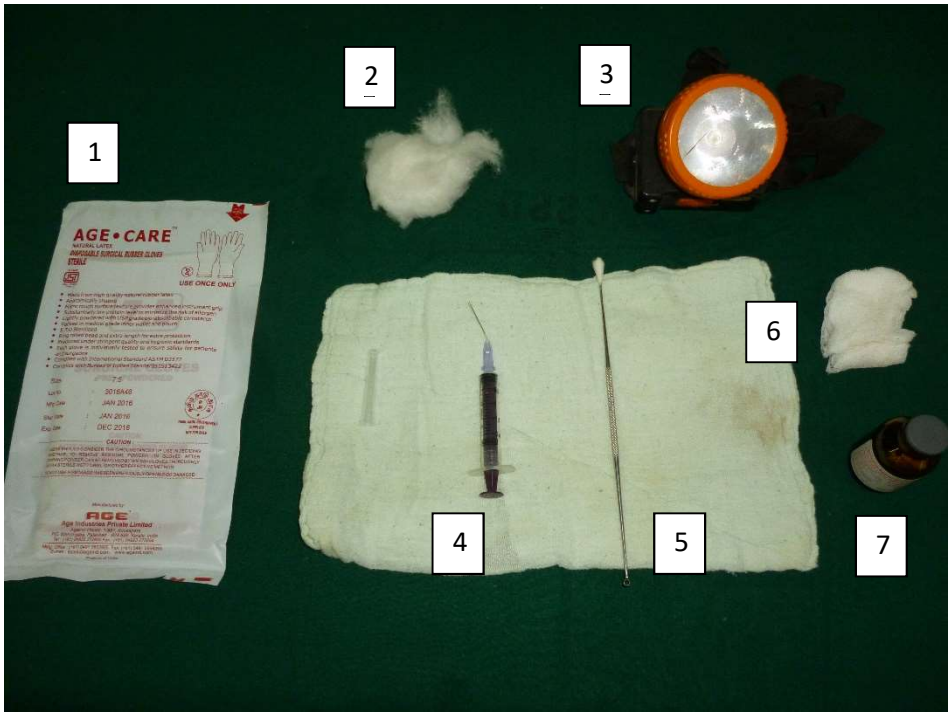


Fig 7 – showing the equipment needed for the sphenopalatine artery block.

1 – Gloves, 2 – Cotton, 3 – Headlight, 4 – 2ml syringe with needle bent to 60 degrees, 5 – Jobson Horne Ear Probe with cotton, 6 – Gauze, 7 – Lignocaine and Adrenaline Solution for infiltration

This was given via the intra oral route for all patients. The procedure was first explained to the patient thoroughly. The patient was then kept in supine position and was asked to keep the mouth open continuously until the block is given. The tongue depressor maybe used in certain patients whose palates are difficult to visualize. A sterile gauze is the used to wipe the saliva off the hard palate. A wick

of cotton is threaded around the Jobson Horne ear probe and using this the greater palatine foramen is probed for.

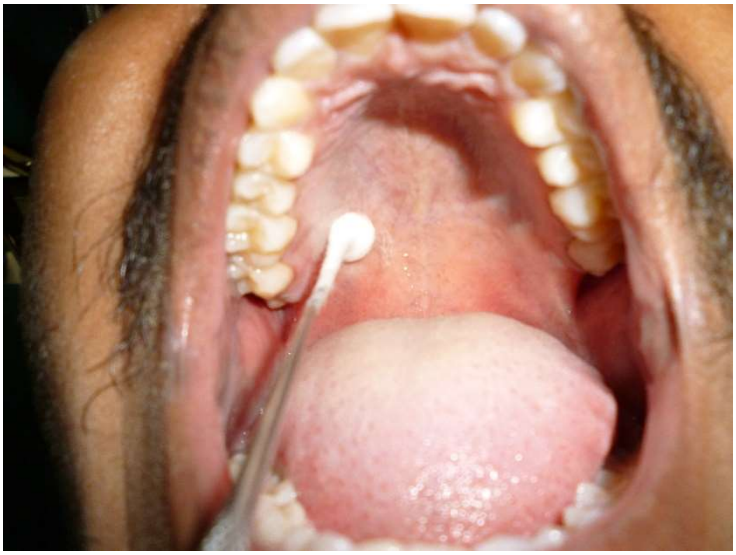


Fig 8 – The right greater palatine foramen is being probed for using the Jobson Horne probe

The greater palatine foramen has a constant location posteromedial to the third maxillary molar and anteromedial to the maxillary tuberosity and pterygoid hamulus. When probing in this region, a distinct depression can be noted. 2ml of the infiltration is loaded into a sterile 2ml syringe with a 25 gauge needle. The needle is bent to about 60 degrees about 25 mm from the tip. This helps in easily manoeuvring the needle into the canal. Keeping the tip of the probe within the palpated depression, the tip of the needle is inserted and 0.1 ml of infiltration is given submucosally so that the area is anesthetized while probing for the canal. With negative pressure the canal is probed for and when it is found the needle easily slips in. Aspiration of any air or blood indicates wrong positioning. If so the needle is withdrawn and repositioned. The needle is inserted for about 25mm

into the canal. The 2ml of block is then very slowly injected into the pterygopalatine fossa. Bleeding during the procedure is usually very minimal. For 3 patients the greater palatine foramen could not be correctly identified and so the procedure was abandoned and these cases were excluded from the study. 8 patients complained of persistent numbness over the teeth on the side of the block. This however returned to normal in all these patients within the first 24 hours post-surgery. A number of complications have been described in literature when doing this block. They include injury to the contents of the pterygopalatine fossa namely the pterygopalatine ganglion and the also the infra orbital neve. Hematoma may develop at the site of injection. The injection maybe inadvertently be injected into the artery directly resulting in systemic effects. Hypersensitivity reactions may occur. Fortunately apart from the transient numbness none of our cases had any such complications. The block maybe given after intubation under general anaesthesia or prior to intubation in the pre op room. Whether given under general or local anaesthesia the aim was to give the block 20 minutes prior to the onset of surgery. The pulse rate was constantly monitored throughout the procedure. Post procedure blood pressure was measured for all patients after the block.

The side of the block was decided randomly. The surgeon or the person grading the field during the surgery was unaware of the side being blocked.

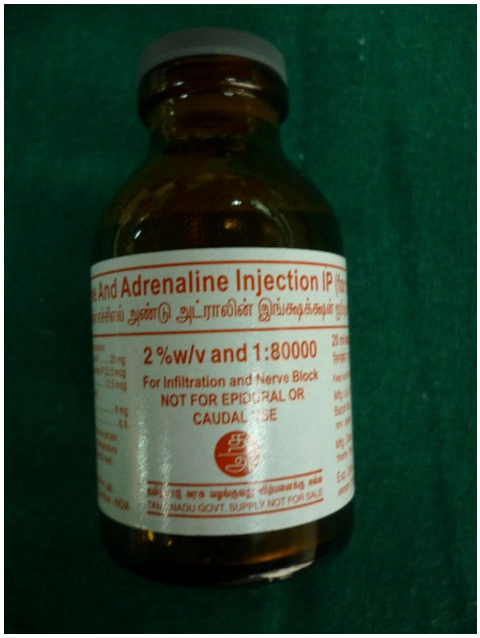


Fig 9 – Lignocaine and Adrenaline (2% and 1:80000), used for infiltration

GRADING THE FIELD

The endoscopic sinus surgery for patients included in this study was done in a “ping-pong” fashion. Patient was operated for 15 minutes first on a side that is chosen by the surgeon. He/she was not aware of which side was blocked. After completing 15 minutes on one side the then surgeon moved on to operate on the opposite side for 15 minutes and after which a saline wash was given on both sides and the field graded for the 30 minute mark. This was done alternatively until the surgeon completes the surgery on both sides. Readings were obtained for every 30 minutes of surgery. The maximum time for which a patient was operated was 3 hours (180 minutes) and the minimum time was one and half hours (90 minutes).

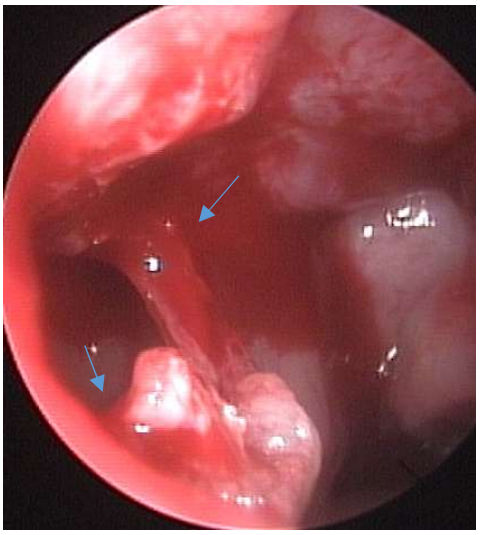


Fig 10 – Intraoperative picture showing 2 points of ooze i.e. Grade 1 bleeding

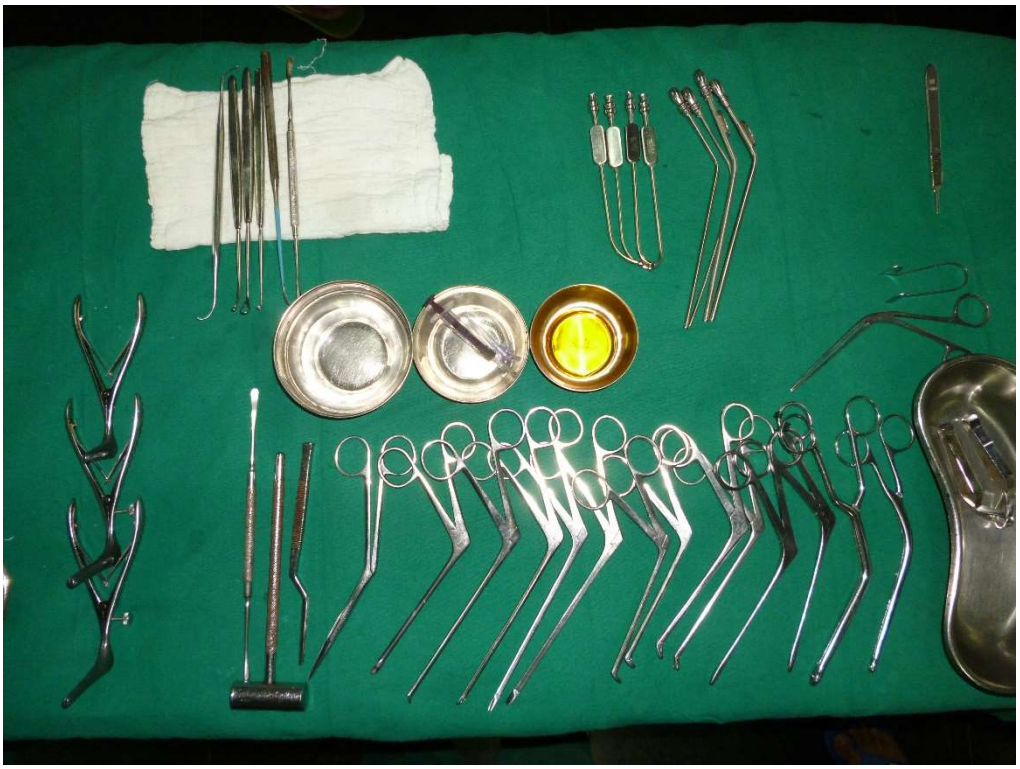


Fig 11 – Table set for endoscopic sinus surgery

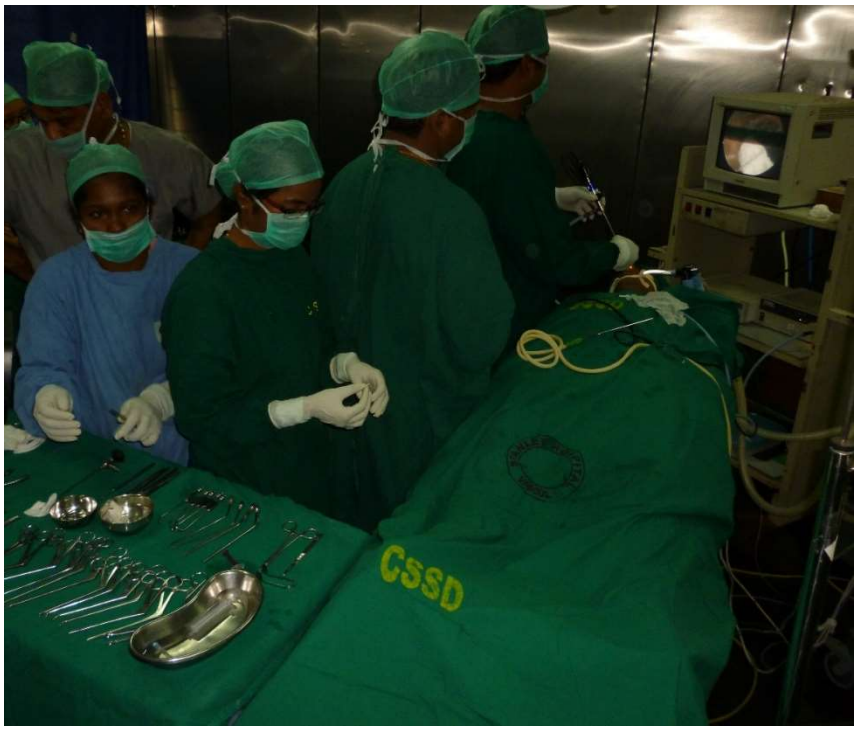


Fig 12 – Showing the intraoperative setup during Endoscopic sinus surgery

The intra operative parameters are assumed to be kept at a constant for every 30 minute intervals. If the intra operative blood pressure were to rise above 130 mmHg systolic and 80 mmHg diastolic at any point during surgery, then that case was excluded from the study. The use of gauze packs impregnated with vasoconstrictors or decongestants were completely avoided for all cases included in this study, both pre and intra operatively. No local infiltration was also given other than the initial sphenopalatine block on one side.

Tabulation of Results

S.no	Age	Sex	Blocked Side						Unblocked Side					
			30 min	60 min	90 min	120 min	150 min	180 min	30 min	60 min	90 min	120 min	150 min	180 min
1	28	M	1	3	3	4			2	2	4	4		
2	35	F	1	2	2	5			1	2	2	5		
3	30	M	1	2	4	6			1	2	4	5		
4	31	M	1	2	3				2	3	4			
5	26	F	2	3	4	6	7		2	3	4	6	8	
6	29	M	2	3	4				1	3	4			
7	30	M	2	2	4	5			2	4	4	6		
8	32	F	2	3	3	6			2	3	4	5		
9	38	F	3	3	3	5	8		3	3	4	5	8	
10	42	F	2	2	3				1	2	4			
11	27	M	2	2	4				2	3	4			
12	41	M	1	3	4				2	3	4			
13	40	F	2	3	4	5	8		2	4	5	5	8	
14	29	M	2	4	4	6			2	3	4	6		
15	32	F	1	3	5				2	4	4			
16	36	M	2	3	4	6			2	4	5	7		
17	30	M	2	3	4	5	8		2	3	4	5	8	
18	28	M	1	2	4	7			2	2	5	6		
19	42	M	1	2	4	5			2	3	4	5		
20	40	M	2	2	4	6	7	8	1	3	4	7	7	8
21	27	F	2	4	4	6			3	4	4	6		
22	33	M	2	3	5				2	3	4			
23	40	F	3	4	4				2	4	5			
24	43	F	2	3	4	6	7		2	3	4	6	7	
25	38	M	2	3	4				2	3	4			
26	30	F	1	3	4	7			1	4	4	7		
27	27	F	1	3	4	5	7		2	4	4	5	7	

28	36	F	2	3	4				2	3	4			
29	39	M	2	2	5	7			2	3	4	7		
30	44	F	1	3	4	6			2	2	4	6		
31	27	F	1	2	4				2	3	5			
32	32	F	1	3	4	5	7		2	3	5	6	7	
33	40	M	1	3	4	6			1	3	4	6		
34	30	F	2	3	5	5			2	3	5	6		
35	36	F	1	2	4				2	3	4			
36	29	F	2	3	5	6			2	3	5	6		
37	35	M	2	4	4	5	7	8	2	4	5	6	7	8
38	36	M	2	3	4	5	8		2	3	4	6	8	
39	44	M	2	3	5	6			2	4	5	6		
40	28	F	1	2	4				1	3	4			
41	34	M	2	4	4				2	4	5			
42	32	F	2	3	4				2	3	4			
43	36	F	1	3	4	5			2	4	5	6		
44	42	M	2	3	4				2	3	4			
45	43	M	1	3	5	6	7		2	3	5	7	7	
46	27	M	2	3	4	5			2	3	4	6		
47	29	F	2	4	5				2	4	5			
48	30	F	2	4	5				2	4	5			
49	31	F	1	3	4	5	7		1	3	4	6	8	
50	33	M	2	2	4	5			2	3	5	6		
51	43	M	1	3	4	6			2	4	5	7		
52	26	F	2	4	5				2	4	5			
53	31	M	2	2	4	5			2	3	4	6		
54	29	M	2	4	4	6			2	4	5	7		
55	41	F	1	2	4	5			1	3	4	6		

STATISTICAL ANALYSIS OF DATA

The Normality tests Kolmogorov-Smirnov and Shapiro-Wilks tests results reveal that the variables do not follow Normal distribution. Therefore to analyse the data Non-Parametric methods are applied. To compare between Blocked and Non-blocked sides at each time point Wilcoxon Signed Rank test is applied. SPSS version 22.0 is used to analyse the data. Significance level is fixed as 5% ($\alpha = 0.05$).

Descriptive Statistics

Time point	Statistics	Blocked Side	Non -Blocked Side
30 mins	N	55	55
	Mean	1.65	1.85
	Std. Dev	.552	.448
	1st Quartile	1.00	2.00
	Median	2.00	2.00
	3rd Quartile	2.00	2.00

Wilcoxon Signed Ranks Test to compare grading between blocked and Non-blocked side at 30 mins

Time point	Z-Value	P-Value
30 mins	2.524	0.012

Descriptive Statistics

Time point	Statistics	Blocked Side	Non -Blocked Side
60 mins	N	55	55
	Mean	2.87	3.20
	Std. Dev	.668	.621
	1st Quartile	2.00	3.00
	Median	3.00	3.00
	3rd Quartile	3.00	4.00

Wilcoxon Signed Ranks Test to compare grading between blocked and Non-blocked side at 60 mins

Time point	Z-Value	P-Value
60 mins	3.530	<0.001

Descriptive Statistcs

Time point	Statistics	Blocked Side	Non -Blocked Side
90 mins	N	55	55
	Mean	4.05	4.31
	Std. Dev	.591	.573
	1st Quartile	4.00	4.00
	Median	4.00	4.00
	3rd Quartile	4.00	5.00

Wilcoxon Signed Ranks Test to compare grading between blocked and Non-blocked side at 90 mins

Time point	Z-Value	P-Value
90 mins	3.130	0.002

Descriptive Statistics

Time point	Statistics	Blocked Side	Non -Blocked Side
120 mins	N	36	36
	Mean	5.56	5.92
	Std. Dev	.695	.732
	1st Quartile	5.00	5.50
	Median	5.50	6.00
	3rd Quartile	6.00	6.00

Wilcoxon Signed Ranks Test to compare grading between blocked and Non-blocked side at 120 mins

Time point	Z-Value	P-Value
120 mins	2.982	0.003

Descriptive Statistics

Time point	Statistics	Blocked Side	Non -Blocked Side
150 mins	N	12	12
	Mean	7.33	7.50
	Std. Dev	.492	.522
	1st Quartile	7.00	7.00
	Median	7.00	7.50
	3rd Quartile	8.00	8.00

Wilcoxon Signed Ranks Test to compare grading between blocked and Non-blocked side at 150 mins

Time point	Z-Value	P-Value
150 mins	1.414	0.157

Descriptive Statistics

Time point	Statistics	Blocked Side	Non -Blocked Side
180 mins	N	2	2
	Mean	8.00	8.00
	Std. Dev	.000	.000
	1st Quartile	8.00	8.00
	Median	8.00	8.00
	3rd Quartile	8.00	8.00

Wilcoxon Signed Ranks Test to compare grading between blocked and Non-blocked side at 180 mins

Time point	Z-Value	P-Value
180 mins	0.000	1.000

Descriptive Statistics

Variable	Statistics	Blocked Side	Non -Blocked Side
Average Grading	N	55	55
	Mean	3.49	3.76
	Std. Dev	.679	.695
	1st Quartile	3.00	3.00
	Median	3.50	3.71
	3rd Quartile	4.00	4.40

Wilcoxon Signed Ranks Test to compare average grading between blocked and Non-blocked side

Time point	Z-Value	P-Value
Average Grading	4.686	<0.001

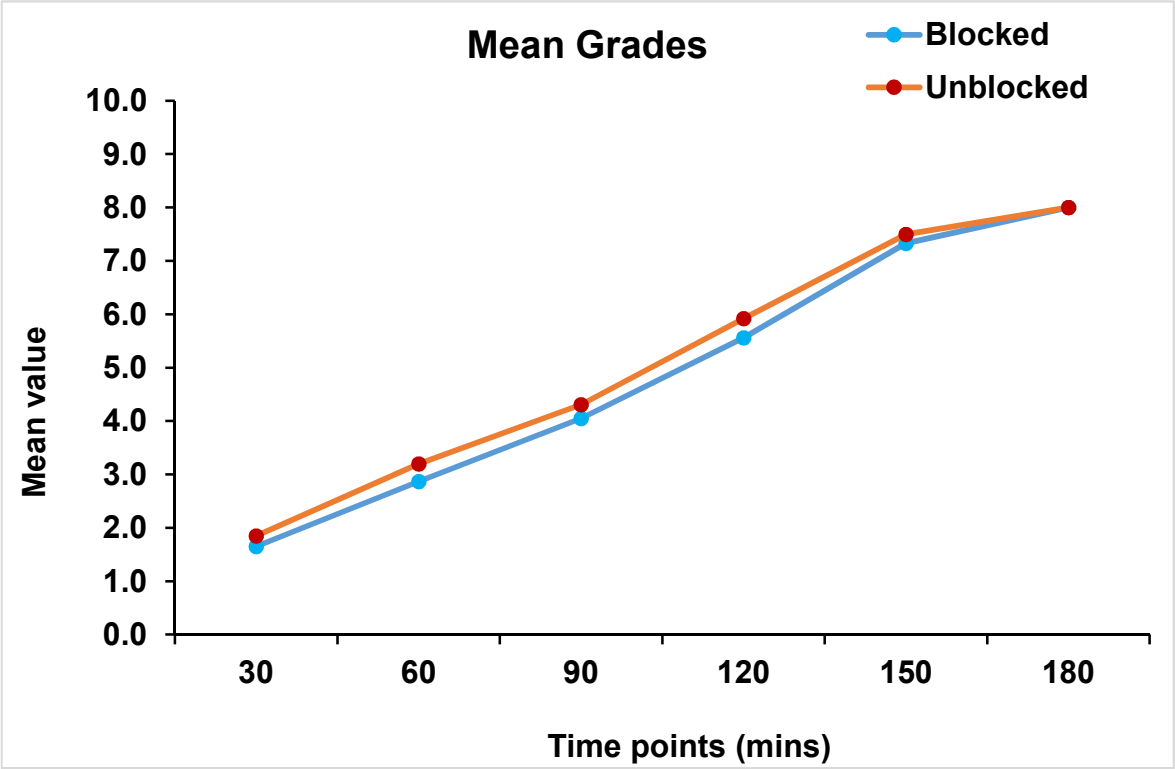
RESULTS AND DISCUSSION

The Wilcoxon Signed Rank Test was used at each time interval to know if there was any significant difference in the bleeding on the blocked and unblocked side. The level of significance was fixed at P </= 0.05.

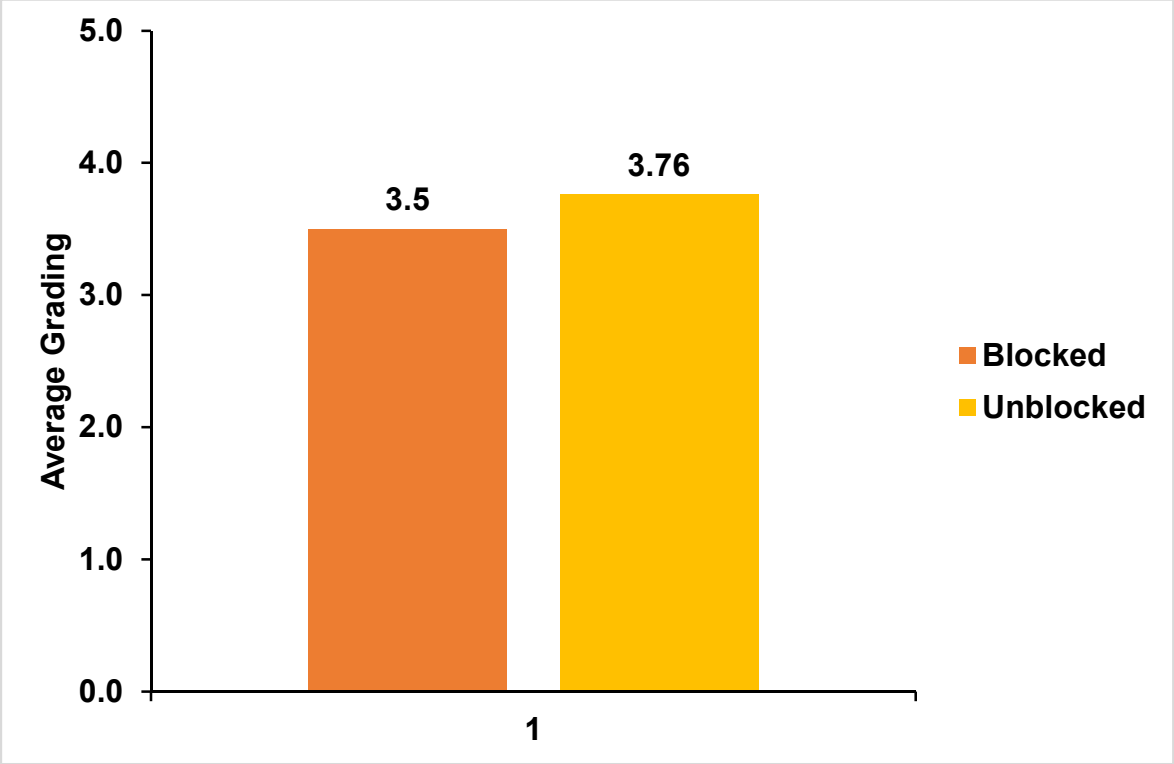
For the time interval starting from 30 minutes to 120 minutes the P value was less than 0.05. This indicates that for each of the readings i.e. 30 min, 60 min, 90 min, and 120 min the grades of bleeding on the unblocked side were significantly higher than those on the blocked side.

However after 120 minutes this difference ceases to exist. At time points 150 minutes and 180 minutes there seems to be no significant difference in the bleeding on both sides.

The line graph below shows the mean grades of bleeding on both sides (Y – axis) plotted against each time interval (X – axis). It can be seen graphically that there is a small but definite gap between the blocked and unblocked sides which disappears after 120 minutes.



The Wilcoxon signed Rank test was also done to see if there was a significant difference in the average grades of bleeding on each side. This also has a P value less than 0.05 showing that overall the bleeding on the blocked side is lesser than the unblocked side. The bar chart below shows the difference between the average grading overall on both sides



The above results and observations indicate that when sphenopalatine artery block is given via the greater palatine route using a combination of lignocaine and adrenaline there is a significant difference in the bleeding between the sides. The effect of the block however gradually wears away over a period of 2 hours. After this period the bleeding between the sides were almost similar. This could be because with time the vasoconstrictor and aesthetic is gradually absorbed into systemic circulation and hence does not produce vasoconstriction of the sphenopalatine vessels.

The sphenopalatine artery is the major blood vessel supplying the lateral wall of the nose as well as the septum. The injection of a vasoconstrictor and lignocaine around this vessel's main trunk results in transient vasoconstriction

and so reduced blood flow. This probably accounts for the difference in bleeding seen.

A probable source of error in this study and also its limitation is that we are comparing the results on the same individual. Hence block given to one side gradually may diffuse to the opposite side as well and result in vasoconstriction on that side too, through the extensive vascular anastomosis in the nasal mucosa. This may alter the results by showing a falsely low bleeding on the unblocked side.

Another probable source of error is that after the surgeon finishes 15 minutes on one side he moves onto the opposite side for the next 15 minutes. During this time the first side is left undisturbed. This side may show an abnormally low grade of bleeding due to haemostatic mechanisms kicking in. Hence when taking the reading at the end of 30 minutes the side finished second will most likely show greater bleeding. This source of error is for a certain degree accounted for by giving a saline wash on both sides before grading the field.

A review of literature also suggests results consistent with this study. A study by PJ Wormald et al showed a small but significant reduction in bleeding during FESS using this block. Salah et al also concluded that sphenopalatine artery block improves surgical field and improves post op analgesia. Others like Hassan et al have reported similar findings.

CONCLUSION

In conclusion, even while keeping in mind the above limitations of this study, there seems to be a statistically significant difference in bleeding in FESS when using the sphenopalatine artery block and can be safely and routinely used for improving surgical field during FESS.

ANNEXURES

A. BIBLIOGRAPHY

1. Functional endoscopic sinus surgery, The Messerklinger Technique, H.Stammberger, History of Nasal Endoscopy pg 1- 13.
2. Diseases of the sinuses- diagnosis and management, David W. Kennedy, James Zinreich ;Anatomy of Paranasal Sinuses Pg 1-12.
3. Scott Brown's Otorhinilaryngology Head and Neck Surgery 7th edition, vol. 2, part 13- Anatomy of Nose and PNS. Pg-1315-1343
4. Computational modeling of flow and gas exchange in models of the human maxillary sinus. C. M. Hood, R. C. Schroter, D. J. Doorly, E. J. S. M. Blenke and N. S. Tolley*J ApplPhysiol*107: 1195–1203, 2009. First published july16,2009;doi:10.1152 /japplphysiol .91615.2008
5. Accessory maxillary ostia: Topography and clinical application, Kumar et al, Deartment of Anatomy, Lady Hardinge Medical College, New Delhi, India; J Anat. Soc. India 50(1) 3-5 (2001)
6. Development of maxillary accessory ostium following sinusitis in rabbits.; SelhattinGenc, MugeOzcan, Ali Titiz, Adnan Unal ; Kecioren Education and Research hospital ENT clinic & Ankara NumuneEduction and Research Hospital, Ankara, Turkey, Rhinology , 46,121-124, 2008.
7. Diseases of the sinuses- diagnosis and management, David W. Kennedy, James Zinreich; chapter 21: Paediatric Sinus Surgery ;David S. Parsons,GaryNishioka; pg 275
8. Otolaryngology Clinics of North America ,Sinusitis,April 2004, vol.37;No.2; pg 221-242 –Aanatomic Variations and Pearls- Functional Endoscopic Surgery.
9. Otolaryngology Clinics of North America ,Sinusitis,April 2004, vol.37 ; pg 243-252- Adult Chronic Rhinosinusitis : Diagnosis; Anand K. Deviah

10. Diagnostic nasal endoscopyBy Dr. T. Balasubramanian M.S. D.L.O. drtbalu's otolaryngology online

11. Scott Brown's Otorhinilaryngology Head and Neck Surgery 7th edition, vol. 2, part 13-Nasal Endoscopy pg1344-1354

12. Scott Brown's Otorhinilaryngology Head and Neck Surgery 7th edition, vol. 2, part 13-chapter 113; Rhinosinusitis –Michael S. Benninger ; pages 1439-1448.

13. Head & Neck Surgery--otolaryngology, Volume 1 ,section ii – Rhinology and allergy ;pg412; Byron J. Bailey, Jonas T. Johnson, Shawn D. Newlands.

14. Otolaryngology Clinics of North America ,Sinusitis,April 2004, vol.37; pg-253-266 –Microbiology and antimicrobial management of sinusitis; Itzhak Brook, MD MSc

15. Otolaryngology Clinics of North America ,Sinusitis,38(2005) , pg 1143-1153; Classification, management of rhinosinusitis and its complications.

16. Otolaryngology Clinics of North America ,Sinusitis,38(2005) pg 1193-1201- Bacterial biofilm:do they play a role in chronic sinusitis; James N. Palmer MD

17. The Two Hole Symdrome- Mladina et al- American journal of Rhinology and Allergy 23:6 pg602-4; PubMed NCBI

18. 'Two Hole Syndrome is present in more than half of post nasal discharge patients?'R.Mladina et al ,ActaOtolaryngologia 05/2010;130(11):1274-7; PubMed.

19. Anatomy of Accessory Maxillary Ostium with Clinical Application-Manju Deepak Singhal; International journal of Medical Sciences and public Health 2014;3(3):327-329

20. 'How Frequent are Accessory Ostia?'Jog.M,McGarry GW; Journal of Laryngology otology 2003April; 117(4):270-2.

21. ‘Endoscopic Cadaver Study- Accessory Maxillary Ostium’ AlperSindel et al, 2014;41(2):262-267 Dicle med j -0921.2014.02.0413: http://www.readperiodicals.com/201406/3386392721.html

22. ‘Study of anatomical variation of lateral nasal wall by endoscope’ – Kolvekar, Khusru, Pradeep:Unique journal of Medical and Dental Sciences 2014,02(2) 117-121.

23. ’Ostium MaxillareAccessorium- A Morphological Study’ ;Manjulapatil ;National Journal of Clinical Anatomy vol 1(4) pg 171-175

24. Lang J. *Clinical Anatomy of the Nose, Nasal Cavity and Paranasal Sinuses.* Stuttgart: Georg ThiemeVerlag, 1989.

25. Sinus Surgery : Endoscopic and microscopic approaches. edited by Howard L. Levine, M. Pais Clemente ; pg 31

26. ‘Iatrogenic maxillary sinus recirculation and beyond’;Gutman M[1], Houser S.;Ear Nose Throat J. 2003 Jan;82(1):61-3:abstract.

27. Scott Brown’s Otorhinilaryngology Head and Neck Surgery 7th edition, vol. 2, part 13-chapter 117; Surgical Management of Rhinosinusitis; pg-1478 – 1499.

28. Diseases of the sinuses- diagnosis and management, David W. Kennedy, James Zinreich ; Functional Endoscopic Sinus Surgery –Anaesthesia, Technique, postoperative management; pg:218.

29. Endoscopic sinus surgery- Anatomy, 3D Reconstruction and Surgical Technique chapter 5 ; Peter John Wormald ;Thieme publication.

30. Minimally Invasive Endonasal Sinus Surgery-principles, Techniques, Results, Complications, Revision Surgery - Werner G. Hosemann, Rainer K. Weber, Rainer E. Keerl, Valerie J.Lund - Maxillary Sinus procedures -Endonasal Maxillary Sinus Fenestration

31. Peterson's principles of Oral and Maxillofacial Surgery. 2nd edition, vol 1;MichaelMiloro, peter Larsen

32. Sinus Surgery : Endoscopic and microscopic approaches. edited by Howard L. Levine, M. Pais Clemente; chapter 10.

33. Anatomical Principle Of Endoscopic Sinus Surgery; RenukaBradoo. Pg111.

34. Micro Endoscopic Surgery Of The Paranasal Sinuses And The Skull Base: Aldo C. Stamm, Wolfgang Draf; Surgical Approaches To Sinusitis:Overview Of Techniques, Pg187

35. Endoscopic Surgery Of The Paranasal Sinuses And Anterior Skull Base ;Malte Erik Wigand; Pg 43

36 Complications In Endoscopic Sinus Surgery Diagnosis, Prevention And Management, S.K Kaluskar ;Pg 29 – 30

37. Manual of Endoscopic Sinus Surgery: and its Extended Applications ; Daniel Simmen , Nick S. Jones , pg114

38. Minimally Invasive Maxillofacial Surgery, Maria J. Troulis, Leonard B. Kaban pg53 – 55

39. Closure of accessory maxillary ostium during endoscopic sinus surgery for CRS - M.A. Penttilä; ENT, Centro HospitalarLisboaOcidental, Lisbon, Portugal - http://www.europeanrhinologicsociety.org /Congress/Abstracts/ERS-0496.pdf

40. Effects of Maxillary Sinus Accessory Ostia on Mucosal Ciliary Area- Marcus D. Shaffer, MD; Hassan H.Ramadan,MD; Diane Schwegler-Berry,MS; Jonathan Franks, MS;2012 Combined Sections Meeting; The American Laryngological Rhinological and OtologicalSociety.Inc; http://www.triomeetingposters.org/poster/effects-of-maxillary-sinus-accessory-ostia-on-mucosal-ciliary-area-the/

41. Nasal ventilation system and method of using same-inventor- Patrick C. Melder Patent WO2010025299A2 - Nasal ventilation system and method of using same - Google Patents.

42. Head & Neck Surgery--otolaryngology, Volume 1,edited by Byron J. Bailey, Jonas T. Johnson, Shawn D. Newlands, Section ii Rhinology and Allergy , chapter 34- Complications of sinus surgery , pg 480

42. Diseases of the sinuses- diagnosis and management, David W. Kennedy, James Zinreich; chapter 16A –Functional Endoscopic Sinus Surgery:

Concepts, Surgical indications, and instrumentation. 197-210 : David W.Kennedy.

43. “A nasal endoscopic study of anatomical variations of the human nasal ostiomeatalcomplex"Dr. Bharathi D, Dept. of Anatomy © Rajiv Gandhi University of Health Sciences, Karnataka

44. “Essentials of Medical Pharmacology”, 6th Edition, K D Tripathi.

B. PROFORMA

NAME: **AGE**/SEX **I.P.No**:

D.O.A:

D.O.S:

DOD

CHIEF COMPLAINTS

1.Facial pain/pressure

2.Nasal obstruction

3.Nasal discharge/discolored postnasal drip

4.Hyposmia/anosmia

5.Headache

6.Fever (all nonacute)

7.Halitosis

8.Dental pain

9.Fatigue

10.Cough

11.Ear pain/pressure/fullness

H/O OF PRESENT ILLNESS

PAST HISTORY

H/o DM/ epilepsy/ PTB/ drug allergy/ bleeding diathesis /SHT/Cardiac disorder

FAMILY H/O:

PERSONAL H/O: Diet

Bladder and bowel habits

Addictions

GENERAL EXAMINATION:

Anemia	Jaundice	Cyanosis	Clubbing	Pedal edema	GLNE

CVS: RS: P/A: CNS:

VITALS:

LOCAL EXAMINATION:

NOSE:

EXTERNAL CONTOUR

DORSUM

VESTIBULE

SEPTUM

ANTERIOR RHINOSCOPY:

	Septum	Inf. Turbinate	Inf. meatus	Mid turbinate	Mid meatus
RIGHT					
LEFT					

PNS TENDERNESS :

	RIGHT	LEFT
COTTON WOOL TEST		
COLD SPATULA TEST		
COTTLE'S TEST		

<u>E/O THROAT</u>:

ORAL Cavity

OROPHARYNX

IDL EXAMINATION

POST NASAL EXAMINATION

Examination of Ear:

	PRE AURICULAR AREA	PINNA	POST AURICULAR AREA	EAC	TM
RIGHT					
LEFT					

DIAGNOSTIC NASAL ENDOSCOPY

ANAESTHESIA:

TECHNIQUE:

ACCESSORY OSTIUM

AO +/-	Single/double/multiple	Side – left/ right	Location- ANF/PNF	Shape	Recirculation

C. ETHICAL COMMITTEE APPROVAL LETTER

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : An observational study comparing the effect of sphenopalatine artery block on bleeding in Endoscopic sinus surgery.

Principal Investigator : Dr Nigil Sreedharan

Designation : PG M S (E N T)

Department : Department of E N T
Stanley Medical College
Chennai -01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 29.09.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

D. PATIENT INFORMATION SHEET

தகவல் படிவம்

தங்களுக்கு செய்த பரிசோதனைகள் மூலம் தங்களுக்கு._____

_____.

நோய் உள்ளது தெரியவந்துள்ளது. இந்த நோயை குணப்படுத்த பலவகை அறுவைசிகிச்சை முறைகள் உள்ளன. அதில் உங்களுக்கு

.

_____.

முறை பயன்படுத்தப்படவுள்ளது. இந்த அறுவை சிகிச்சையில் அண்ணம் வழியாக ஊசி போட்டு மருந்து(அட்ரெனலின் + லிகநோகின்) கொடுத்து மூக்கிற்கு ரத்தம் வழங்கும் முக்கிய ரத்த குழாயான ஸ்பிநோ பலடின் ரத்த குழாயை தற்காலிகமாக சுருங்க செய்யப்படும் முறை மேற்கொள்ளபடுகிறது. மேலும் இந்த முறையின் மூலம் ஏற்படும் விளைவுளை ஒப்பிட ஆய்வு மேற்கொள்ளப்பட உள்ளது. இது குறித்த உங்களின் நோய் மற்றும் அதன் விவரங்களை ஆய்வில் பயன்படுத்த விரும்புகிறோம். தாங்கள் விரும்பினால் மருத்துவ ஆய்விலிருந்து எந்த சட்டசிக்கலுக்கும் இடமில்லாமல் எப்பொழுது வேண்டுமானாலும் ஆய்விலிருந்து விலகிக் கொள்ளலாம். இந்த ஆய்வின்மூலம் கிடைக்கும் தகவல்களும் பரிசோதனை முடிவுகளும் தங்களின் ஒப்புதலின்மூலம் மட்டும் ஆய்வில் பயன்படுத்தப்படும்.

ஆய்வாளரின் கையொப்பம்:

ஆய்வாளரின் பெயர்:

இடம்:

நாள்:

E. INFORMED CONSENT FORM

சுய ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் : காது, மூக்கு, தொண்டை பிரிவு

ஸ்டான்லி அரசு பொது மருத்துவமனை மற்றும் மருத்துவ கல்லூரி

பங்கு பெறுபவரின் பெயர்:

பங்கு பெறுபவரின் எண் :

மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டுள்ளது. எனது நோய்_____மற்றும் அதன் நிலை தெரியபடுத்தப்பட்டது. எனது நோய் பற்றிய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. இந்த நோயை குணப்படுத்தும் அறுவை சிகிச்சை முறைகள்_____. விளக்கப்பட்டுள்ளது. இந்த அறுவை சிகிச்சையில் அண்ணம் வழியாக ஊசி போட்டு மருந்து(அட்ரெனலின் + லிகநோகின்) கொடுத்து மூக்கிற்கு ரத்தம் வழங்கும் முக்கிய ரத்த குழாயான ஸ்பினோ பலடின் ரத்த குழாயை தற்காலிகமாக சுருங்க செய்யப்படும் முறை பற்றி விளக்கப்பட்டது. இதனால் ஏற்படும் விளைவுகள் எனக்கு விரிவாக விளக்கப்பட்டன. இம்முறையை எனது அறுவை சிகிச்சையின் போது மேற்கொள்ள சுயநினைவுடன் சம்மதிக்கிறேன்.

இந்த அறுவை சிகிச்சையின் விளைவுகளை ஆய்வில் பயன்படுத்தவும் தன்னிச்சையாக சம்மதிக்கிறேன். எக்காரணத்தினாலும் எந்தக் கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் இவ்வாய்வில் இருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த சோதனை மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்க தேவைப்பட்டால் என்னையும் எனக்கு நடக்கும் அறுவை சிகிச்சையையும் புகைப்படம் எடுக்கவும் நான் முழு மனதுடன் சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்: நாள்:

கட்டை விரல் ஒப்பம் இடம்:

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்:

ஆய்வாளரின் கையொப்பம்: நாள்:

ஆய்வாளரின் பெயர்: இடம்:

F. LIST OF TABLES

G. LIST OF GRAPHS

H. LIST OF FIGURES

I. MASTER CHART

S.no	Age	Sex	Blocked Side						Unblocked Side					
			30 min	60 min	90 min	120 min	150 min	180 min	30 min	60 min	90 min	120 min	150 min	180 min
1	28	M	1	3	3	4			2	2	4	4		
2	35	F	1	2	2	5			1	2	2	5		
3	30	M	1	2	4	6			1	2	4	5		
4	31	M	1	2	3				2	3	4			
5	26	F	2	3	4	6	7		2	3	4	6	8	
6	29	M	2	3	4				1	3	4			
7	30	M	2	2	4	5			2	4	4	6		
8	32	F	2	3	3	6			2	3	4	5		
9	38	F	3	3	3	5	8		3	3	4	5	8	
10	42	F	2	2	3				1	2	4			
11	27	M	2	2	4				2	3	4			
12	41	M	1	3	4				2	3	4			
13	40	F	2	3	4	5	8		2	4	5	5	8	
14	29	M	2	4	4	6			2	3	4	6		
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18	28	M	1	2	4	7			2	2	5	6		
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21	27	F	2	4	4	6			3	4	4	6		
22	33	M	2	3	5				2	3	4			

23	40	F	3	4	4				2	4	5			
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25	38	M	2	3	4				2	3	4			
26	30	F	1	3	4	7			1	4	4	7		
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33	40	M	1	3	4	6			1	3	4	6		
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41	34	M	2	4	4				2	4	5			
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43	36	F	1	3	4	5			2	4	5	6		
44	42	M	2	3	4				2	3	4			
45	43	M	1	3	5	6	7		2	3	5	7	7	
46	27	M	2	3	4	5			2	3	4	6		
47	29	F	2	4	5				2	4	5			
48	30	F	2	4	5				2	4	5			
49	31	F	1	3	4	5	7		1	3	4	6	8	

50	33	M	2	2	4	5			2	3	5	6		
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53	31	M	2	2	4	5			2	3	4	6		
54	29	M	2	4	4	6			2	4	5	7		
55	41	F	1	2	4	5			1	3	4	6		

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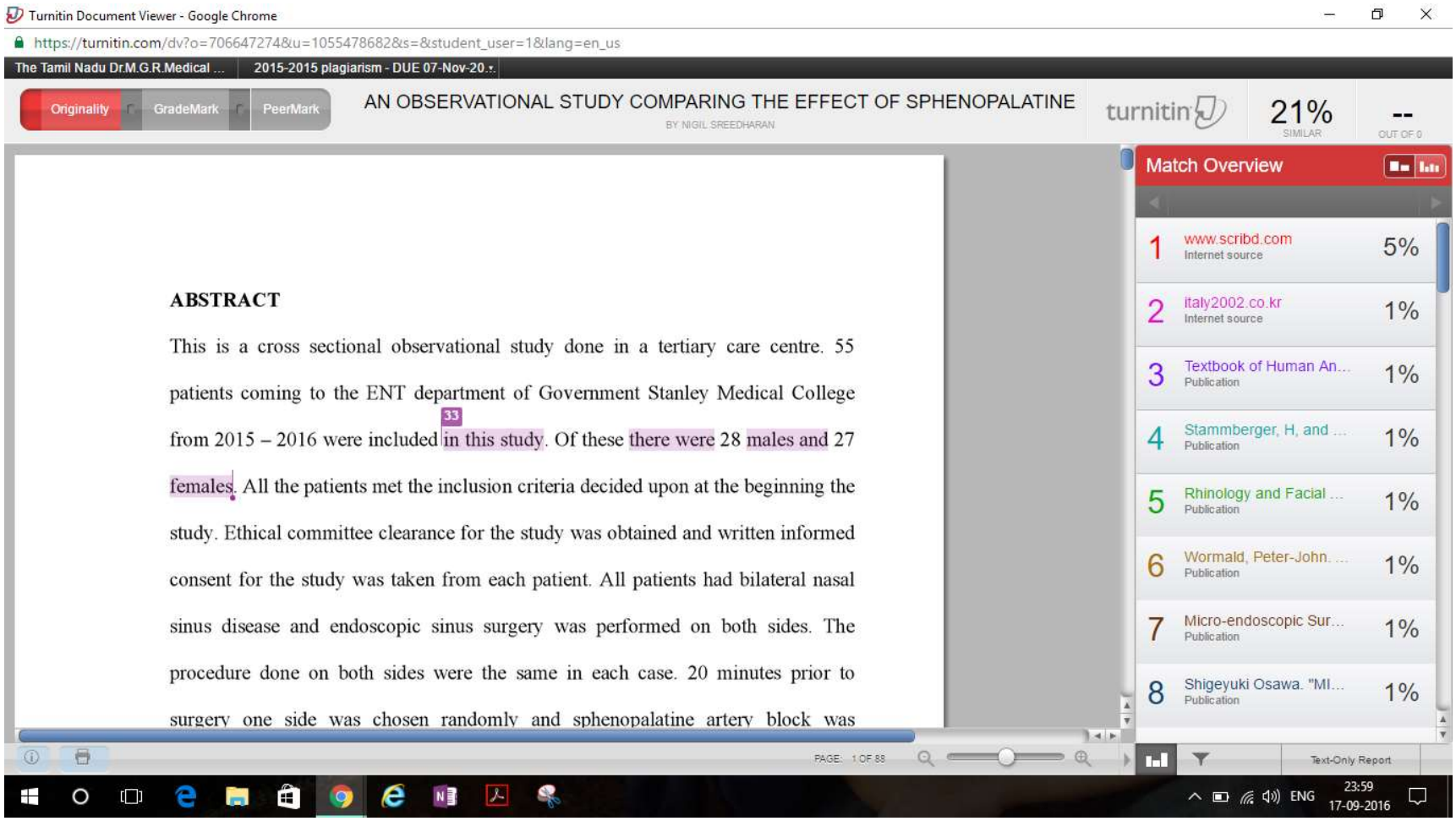

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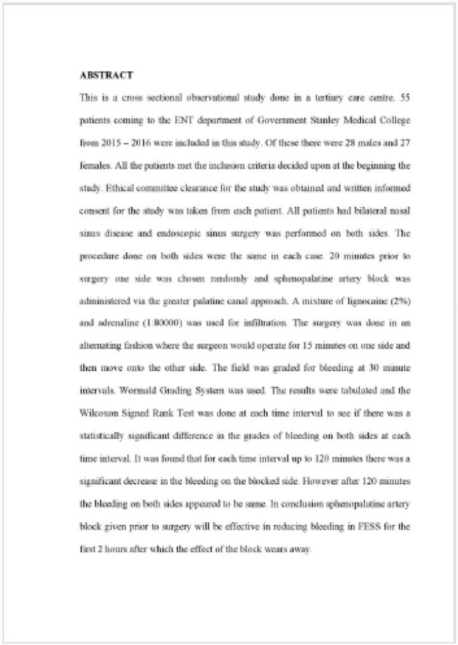

ABSTRACT

This is a cross sectional observational study done in a tertiary care centre. 55 patients coming to the ENT department of Government Stanley Medical College from 2015 – 2016 were included in this study. Of these there were 28 males and 27 females. All the patients met the inclusion criteria decided upon at the beginning the study. Ethical committee clearance for the study was obtained and written informed consent for the study was taken from each patient. All patients had bilateral nasal sinus disease and endoscopic sinus surgery was performed on both sides. The procedure done on both sides were the same in each case. 20 minutes prior to surgery one side was chosen randomly and sphenopalatine artery block was administered via the greater palatine canal approach. A mixture of lignocaine (2%) and adrenaline (1:80000) was used for infiltration. The surgery was done in an alternating fashion where the surgeon would operate for 15 minutes on one side and then move onto the other side. The field was graded for bleeding at 30 minute intervals. Wormald Grading System was used. The results were tabulated and the Wilcoxon Signed Rank Test was done at each time interval to see if there was a statistically significant difference in the grades of bleeding on both sides at each time interval. It was found that for each time interval up to 120 minutes there was a significant decrease in the bleeding on the blocked side. However after 120 minutes the bleeding on both sides appeared to be same. In conclusion sphenopalatine artery block given prior to surgery will be effective in reducing bleeding in FESS for the first 2 hours after which the effect of the block wears away.