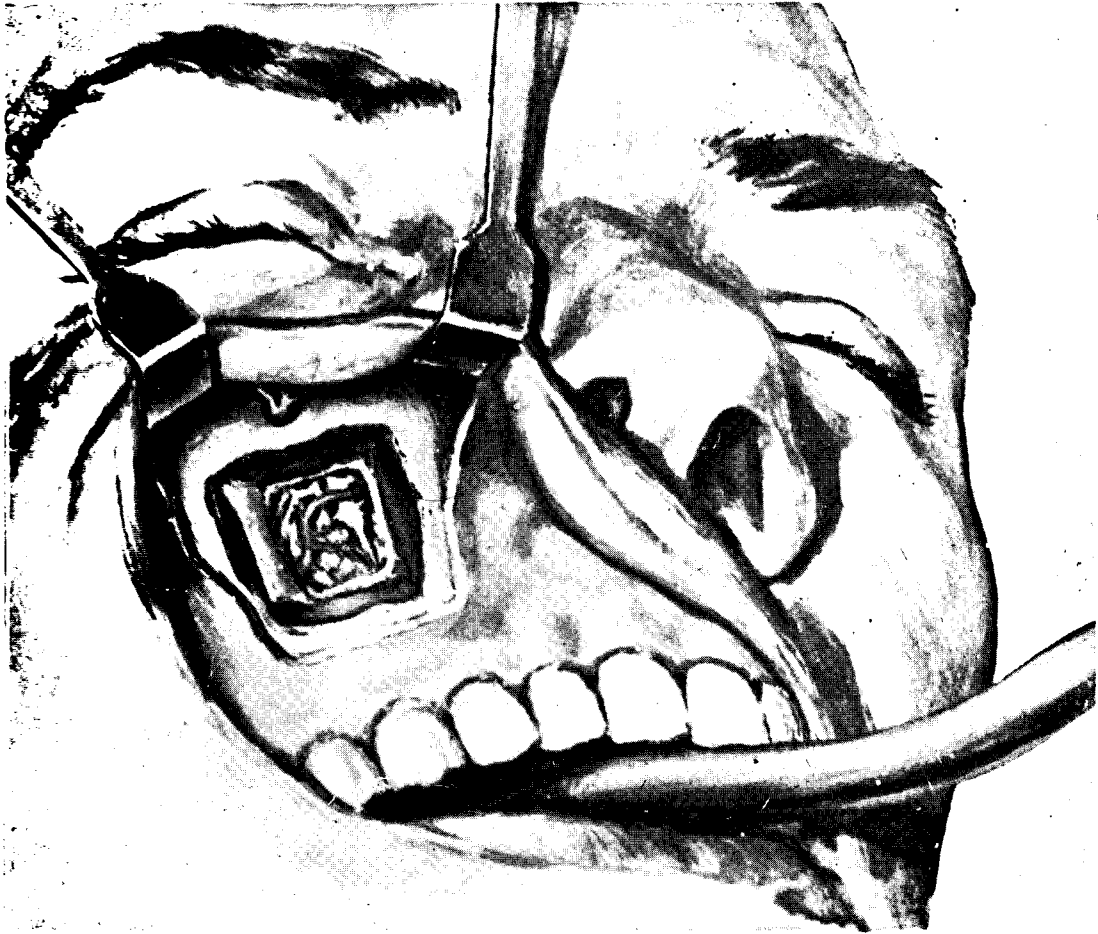


# Vidian neurectomy in vasomotor rhinitis experience in Suriname



by

RAMMOHAN TIWARI

COVER

OPERATIVE FIELD — VIDIAN NEURECTOMY — RIGHT SIDE.

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**VIDIAN NEURECTOMY IN VASOMOTOR RHINITIS  
EXPERIENCE IN SURINAME**

**PROEFSCHRIFT**

TER VERKRIJGING VAN DE GRAAD VAN DOCTOR IN DE MEDISCHE WETENSCHAPPEN AAN DE UNIVERSITEIT VAN SURINAME, OP GEZAG VAN DE RECTOR MAGNIFICUS DR. B.F.J. OOSTBURG, HOOGLEERAAR IN DE FACULTEIT DER MEDISCHE WETENSCHAPPEN, VOLGENS BESLUIT VAN DE SENAAAT IN HET OPENBAAR TE VERDEDIGEN OP WOENSDAG 16 NOVEMBER 1977 OM 19.00 UUR PRECIJS IN DE AULA VAN DE UNIVERSITEIT VAN SURINAME AAN HET KERKPLEIN TE PARAMARIBO

DOOR

**RAMMOHAN TIWARI**

Geboren in het distrikt DRUG, INDIA in 1934.

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PROMOTOR : PROF. DR. F. JESSUREN  
University of Suriname

CO PROMOTORS : PROF. DR. W. LUYENDIJK  
University of Leiden

and

PROF. DR. J.L. SHEEHY  
University of Southern California

To,

**Ma, Sanjay, Anjali and Ina.**

Dit proefschrift werd bewerkt op de afdeling Keel-Neus-Oorkunde  
van het Academisch Ziekenhuis te Paramaribo.

योगो ज्ञानं तथा सार्व्व्यं विद्याः शिल्पादिकर्म च  
वेदाः शास्त्राणि विज्ञानमेतत्सर्व्वं जनार्दनात् ॥

All knowledge such as Yoga, Sankhya, sculpture and other forms of arts, Vedas, Shastras and all Sciences have their origin in Him.

नता ययोः श्रीपतितां समीयुः  
कदाचिदप्याशु द्रिद्रवर्याः  
चशकमू वाचस्पतितां हि ताभ्यां  
नमो नमः श्री गुरु पादुकाभ्याम् ॥

I offer my humble salutations to Gurudev by whose  
blessings even the downtrodden can rise to great heights  
and even the dumb becomes wise.

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## Introduction

Nasal obstruction is a common complaint for which medical advice is sought, and vasomotor rhinitis seems certainly to be one of the commonest cause for this. Although its frequency may vary from country to country and perhaps in various parts of the same country, the condition is encountered pretty well everywhere. Coming to Suriname in Augustus 1972, I was struck by the large number of patients seeking medical advice for nasal complaints of which chronic vasomotor rhinitis formed an important part. Although the term rhinitis conveys inflammation, the condition is essentially noninflammatory. Many alternative terms such as vasomotor nasal disharmony, vasomotor rhinopathy and neuro-vegetative vasomotor rhinitis have been suggested and are in use. For the purpose of this investigation, I have adhered strictly to this term i.e. vasomotor rhinitis. In several texts one also encounters several different conditions such as allergic rhinitis included under this heading. I have not done so.

Inability to breath through the nasalairways is annoying and reduces the ability of the person to concentrate. Not uncommonly these patients start treating themselves by local decongestants and first seek medical advice when the condition of rhinitis medicamentosa is already well established.

Until the late fifties the condition was thought to be due to allergy, although no clearcut association with known allergens was established. The observations of Goldnig Wood and the successful performance of vidian neurectomy with marked alleviation of symptoms in these patients has opened new ways for the chronically ill patients in whom medical treatment has failed.

There have been reports from various parts of the world notably East Europe, Egypt, India, England and the United States. The opportunity to study this condition in Suriname is of interest in many ways. In the first place, much has been written about vasomotor rhinitis in cold countries, in the geographical temperate zone and subtropical countries, but never in the tropics. Secondly, some reference has been made to the increased incidence of this condition in the East Indians, but no conclusive statistics have been provided. The community in Suriname is cosmopolitan with Chinese, Lebanese, Indonesians, Europeans, Negroes, Creoles and East Indians all living together. This therefore provides an excellent situation for such a study.

The object of this investigation was :

1. to assess the frequency of the condition of vasomotor rhinitis in Suriname

- and any relationship with the changes in weather conditions.
2. to study the effect of conservative therapeutic measures.
  3. to determine the role of vidian neurectomy in our patients.

## Historical

The first description of the condition of vasomotor rhinitis was by JULES TOQUET in 1821. The earliest description of vidian nerve was by GUIDO-GUIDI, an Italian anatomist, in 1555. Surgical approach to pterygopalatine fossa was first made more than a century ago and reported in 1858 by CARNOCHAN for the treatment of trigeminal neuralgia. The condition of vidian neuralgia and its treatment was described by VAIL in 1932. However, the possibility of vidian neurectomy as a possible mode of treatment for vasomotor rhinitis was realized much later. For years it was believed that the condition was an allergic phenomenon, an atypical reaction. It was in 1944 that CODE demonstrated that no antigen-antibody reaction appears to play a part in this condition. He thought that this was an example of "overaction of a physiological mechanism". This view was later substantiated by many workers notably LUSCHER (1954) and ROBERTS (1957). Later several others notably WOLFF (1950) and GEERHOM suggested the role of emotional factors in this condition.

In 1943 FOWLER described a case where Stellate ganglionectomy which was carried out for vidian nerve causalgia also produced stuffiness of the nose accompanied by sneezing and watering, but only on the left side i.e. the side on which the Stellate ganglionectomy was carried out. Golding Wood, who took note of the above mentioned case, first performed petrosal neurectomy for vasomotor rhinitis in 1956 with successful results.

This operation, however, had earlier been performed by MURRAY FALCONER in 1954. MALCOMSON in 1957 was the first to perform vidian neurectomy for vasomotor rhinitis with success. His approach to the vidian nerve was transseptal. Encouraged by this report Golding Wood first performed and popularized the transantral approach to vidian nerve. This approach was, however, first devised by SEWELL in 1926. He had advocated this for sphenopalatine ganglionectomy. SLUDER published several papers in the early part of this century dealing with anatomy of sphenopalatine ganglion. The credit, however, for the refinement of his approach through the maxillary antrum to the pterygopalatine fossa as applied to the operation of vidian neurectomy goes to Golding Wood. It is however of interest that several years passed before his work was generally accepted in England itself. In recent years the clinical and surgical aspects of vidian neurectomy have been studied by many authors including CHANDRA, CHASIN and LOFFEREN, MUFTEY and KAHLEY.

HIRANANDANI was the first to advocate vidian neurectomy for recurrent nasal polyposis. In recent years MORGENSTEIN has done some excellent work on the sur-



gical anatomy of pterygopalatine fossa and has been the main proponent of this treatment in the United States. So far as is known vidian neurectomy was never carried out before in Suriname.

## Methods and materials

This study was carried out in Suriname over the period of more than four years. The cases that are included are those seen personally in the Academic Hospital Paramaribo and at the St. Vincentius Hospital Paramaribo. Cases seen earlier than 1973 were not included because the records kept prior to this date were considered inadequate. In order to assess the frequency of the disease all the cases in one year i.e. in 1976 were reviewed. During this period 3507 patients were seen in the outpatients departments personally. Out of these 462 patients had symptoms pertaining to their nose. Of these 77 patients were diagnosed as cases of chronic vasomotor rhinitis.

### *Prevalence of chronic vasomotor rhinitis.*

Total number of patients in 1976	3507
Patients with nasal complaints	462
Patients diagnosed as chronic vasomotor rhinitis	77 or nearly 2.2% of the total number of patients.

TABLE I

These patients were worked up thoroughly. A complete record of their name, age, sex, occupation; was first made. The investigation then proceeded with:

1. History taking
2. Clinical examination
3. Routine laboratory examination of bloodpicture and examination of faeces for intestinal parasites.
4. X-ray examination
5. Special Tests.

### HISTORY

A proper history taking is one of the most essential step of diagnosis. Many of these patients have a long history ranging many years. They have often visited so many physicians already, consumed so many capsules, tablets and nosedrops and are so much depressed by their illness that repeated questioning was necessary and perhaps at more than one sitting only, could all the facts be properly assembled. The patient was questioned about his leading symptoms. These were usually in the form of sneezing attacks, watery rhinorrhea, nasalobstruction and headaches. Occasionally during acute exacerbation patients complained of some pain by the side of the nose. Itching in the region of the hard palate was complained by some. Nasal obstruction was usually bilateral, intermittent, and worse at night or early in the mornings. Rhinorrhea was watery and followed a bout of sneezing.

Occasionally, however, patients complain of a dripping nose where watery nasal-discharge occurred without any sneezing.

Sometimes in the presence of a process of secondary infection the discharge changed its character and one had to wait for a proper diagnosis of the underlying primary condition until the secondary infection had been treated.

Any variations in the nature of symptoms with regard to the time or surroundings or any other associated factor such as emotional stress was enquired into. In most cases the symptoms were worse early in the mornings, at night, in the rainy seasons, or if the patient remained outside the house, in the open, late at night e.g. at a party etc. Some patients complained that their symptoms were worst if they were in airconditioned atmosphere such as exists in many of our offices.

There is a small group of patients whose leading symptom is nasal obstruction and who do not have significant rhinorrhea or sneezing. Otolaryngologists in the United States are of the opinion that they see many more such patients with vasomotor rhinitis, primarily with nasal obstruction than with sneezing and rhinorrhea as well, while Golding Wood in England seems to see mostly patients with the other group of symptoms. This was my observation too. Out of the 77 patients diagnosed as chronic vasomotor rhinitis only 5 had nasal obstruction as the only leading symptom.

A history of trauma was asked. Personal history of the patient with regard to his personal habits such as smoking and intake of alcohol (amount and frequency), were all taken into account.

Especially patients were questioned if they were dependant on any drugs such as marihuana, cocaine, heroine, or peppills etc. Women were asked as regards the use of any contraceptive pills. A large number of our patients complained of associated headaches which seem to be related to the nasal blockage. They described it mostly as heaviness around the frontal area ("spanning").

Where there was the slightest suggestion of an allergic basis, patients were enquired about the presence of any generalized itching or dyspnoea. A family history was taken and the presence or otherwise of similar symptoms in any other member of the family was enquired. The living and working conditions of the patients such as dry-, damp- or dusty surroundings, country- or town residence, presence or otherwise of pets (animal and or birds) were all taken into account. Detailed history of any other associated illness for which patient may have been under treatment elsewhere, was taken.

Clinical examination of the ear, nose and throat was then made. If a satisfactory view of the nasal cavity was not possible, two percent pontocaine was

applied into the nose and the examination was repeated after ten minutes.

This enabled to exclude the presence of any coexistent pathology, such as polyp, deviation or septal spurrs. The general condition of the patient was taken into account and any associated findings such as pallor or signs of endocrine dysfunction such as hypothyroidism were noted.

In chronic vasomotor rhinitis, usually, the nasal mucosa especially over the inferior turbinates is blueish and boggy. The turbinate is swollen. There may be thin mucoid discharge. Where the patient presented with associated secondary infection, allergy or established rhinitis medicamentosa, these secondary conditions were first treated. Cases with rhinitis medicamentosa were "deprogrammed". They were explained about their condition and advised to stop nasal decongestants. Since many of them were unable to breath through the nose normally, adults were given a single injection of cortisone such as depomedrone or dexamethasone intramuscular along with systemic nasal decongestants and or antihistaminics. These patients were seen regularly to prevent them falling prey to their nose drops again, until the nasal mucosa had returned to its original state. Only then could a diagnosis of the underlying condition be made with certainty.

The clinical diagnosis could be made with reasonable certainty.

#### SPECIAL INVESTIGATIONS

An X-ray examination of the paranasal sinuses was done in all cases. The following special tests were carried out where necessary.

*Examination of the nasal smear for eosinophils.* This test, however, is no more regarded as typical for allergy since eosinophils are found in the nasal discharge in many nonallergic conditions as well.

*Tests for allergy.* Patients were examined by scratch tests and/or intradermal tests. (The antigens used were from BENCARD, England). And if any positive responses were obtained, these patients were advised about exclusion of these allergens wherever possible. If a diagnosis of allergic rhinitis was made then these patients were first treated for their allergy. The following cases will serve as good examples.

A young man R.P., 25 years of age, came with the history of nasal obstruction, sneezing and watery rhinorrhea. Investigations showed that he had markedly positive responses to shellfish (Krobia). He was advised to exclude this item from his diet with the result that he was almost completely free of his symptom

within three weeks and did not need any antiallergic therapy anymore. He has since been regularly followed up for a period of two years.

A similar case was of a young boy, who was found to be sensitive to antigen prepared from rice. Exclusion of this substance from the diet at first seemed very difficult since rice is a staple food of the Surinamers. However, with gentle persuasion this was achieved with considerable improvement in the boy's symptoms.

Other tests such as Nasal Provocation Tests, Provocative Food Testing and the cytotoxic Food Tests were not done. These tests have been used in diagnosis of allergic rhinitis, but their validity is not unquestionable.

#### TEST FOR ENDOCRINE FUNCTIONS.

Endocrine deficiency such as of thyroid gland or pituitary hypofunction sometimes produces changes in the nasal mucosa which are rather similar to those seen in local nasal malfunction such as allergic rhinitis or vasomotor rhinitis. The nasal mucosa in these endocrine deficiency states appears pale, blueish and boggy. Although the symptomatology is significantly different, however, such patients need thorough endocrine work up and in such situations intercollegial consults with the internist was the rule in this series.

In the same way examination of the nasal discharge for glucose to differentiate cerebrospinal rhinorrhea was done only as a measure to differentiate the condition, if this was deemed necessary.

Although a clinical diagnosis of chronic vasomotor rhinitis can be arrived at quite simply by the above mentioned procedures, the condition has to be differentiated from the following:

1. Allergic rhinitis — This term signifies the specific IgE mediated immunological reaction of the nasal mucosa to aeroallergens. Symptoms which are similar to those of acute vasomotor rhinitis or common cold
2. Coryza or common cold
3. Sinusitis associated with rhinitis
4. Cerebro spinal rhinorrhea
5. Endocrine disturbances
6. An after effect of drug therapy.

## Structural and Physiological considerations

Some basic structural details must be considered before detailed physiological considerations. The nasal cavity is divided into two areas by the nasal septum.

Each side of the nasal cavity consists of an anterior part lined by squamous epithelium which is in fact extension of the skin of the face and contains many sebaceous or vibrissae. These have a protective and filtering action. In addition it contains sebaceous and sweat glands. Bone, cartilage and fibrous tissues provide necessary rigidity to the nasal cavity and keep it patent. Behind the vestibule area is the large respiratory portion of the nasal cavity, except the upper part which is the olfactory area. The lateral wall of the nasal cavity contains bony like bony prominences or turbinates which are covered with mucous membrane and help to increase the area of the mucous membrane.

The epithelium of the vestibule is squamous but becomes less and less keratinized inwards and changes into pseudostratified ciliated columnar epithelium with goblet cells. This kind of epithelium lines the rest of the nasal cavity except the olfactory area which contains the neuro-receptors for olfaction.

The histological structure of the respiratory epithelium is of importance to the present discussion. The mucous membrane as mentioned above is pseudostratified ciliated columnar with goblet cells and lamina propria that contains mucous and serous glands and which is adherent to the periosteum of the bone or the perichondrium of the cartilage beneath it. The basement membrane which separates the respiratory epithelium from the lamina propria is thicker than for most other kinds of epithelium. The epithelium cells excluding the goblet cells have cilia, ten to twenty each, about 7 micron long which help to keep the mucous blanket moving. The mucous is produced in turn by the cells themselves and by the mucous glands. The lamina propria contains both collagenic and elastic fibres and also various cells such as lymphocytes, plasma cells, macrophages and small lymphocytes. The lamina propria is a very vascular membrane and contains arterioles, capillaries and veins. This is especially well marked in the middle and inferior conchae where in addition to the normal quota of blood vessels, are also present a large number of venous spaces which under normal conditions are collapsed and under certain abnormal states become distended with blood, thus increasing the size of turbinate. This distention may be so pronounced as to produce the symptoms of nasal obstruction. The structural peculiarity of the nose is likened to erectile tissue and is often referred to in the texts as spongy tissue. There are however important differences. The first is that the venous sinuses in the nasal venous spaces and network do not contain muscle. More-

over these cavernous spaces receive blood through intervening capillary bed instead of directly from arterioles as in the genital tissues.

Certain areas of the cavernous tissue of the nose react as physiological units. The cavernous tissue of the inferior turbinate is divided into three areas, namely the anterior two fifths, middle one fifth and the posterior two fifths. It has been observed experimentally that these areas do not react simultaneously to stimuli. Under normal circumstances reactions in the two anterior segments i.e. in the anterior three fifths take place without any appreciable influence or change in the posterior two fifths. But when the first area of the anterior tip of the inferior turbinate is subject to excessive stress, changes occur in the posterior segment as well. In practice it is observed that removal of the anterior tip of the inferior turbinate produces much subsequent discomfort to the patient such as excessive watering of the nose. Removal of the posterior segment on the other hand does not produce such changes. The inferior turbinate contains most of the cavernous tissue of the nose. This can be clinically seen in patients where the slightest vasomotor reaction presents a marked contrast in the appearance, colour and size of the inferior and middle turbinates. The former being blueish, boggy and turgid while the latter retains much of its natural pink colour without gross enlargement.

The blood supply of the nose is derived from both the external and internal carotid systems. The internal carotid supplies, the anterior and posterior ethmoidal arteries, which are branches of the ophthalmic artery and enter the nasal cavity through foramina in the medial wall of the orbit. In addition a terminal branch of the ophthalmic artery called dorsal nasal artery enters the nose along its dorsum to anastomose with angular and lateral nasal branches of the facial artery. The main arterial supply to the nasal cavities and its related parts on both sides comes from the sphenopalatine arteries. This vessel is the terminal part of the maxillary artery and enters the nasal fossa through the sphenopalatine foramen located on the lateral wall of the nasal cavity close to the superior meatus. The maxillary artery supplies ninety percent of the blood supply of the nose through its sphenopalatine and posterior nasal branch.

The nose provides a rigid airway through which the incoming and the outgoing air may pass. The rigidity of the airways ensures prevention of any collapse of the nasal passage during deep inspiration when intranasal pressure falls. The direction in which normally inspired air travels, depends upon the directing effect of the anterior nares, shape of the nasal vault and the fact that the anterior nares are invariably smaller than the posterior choanae. This means, relative ob-

struction at the anterior nares and thus alteration in the intranasal pressure takes place. Normal intranasal pressure varies from 6 to — 6 mm of water. In case of resistance to air flow in the nasal cavity such as happens in gross turgidity of the nasal conchae in vasomotor rhinitis alters this situation. There is also evidence to suggest, as shown by the recent researches of Ogura, that the change in intranasal pressure and resistance to nasal airpassage is accompanied by changes in intranasal temperature.

When inspiration is commenced the narrow point of the nose at the nares presents obstruction to the free entry of the air. Thus a relatively lower pressure is maintained beyond the nares and within the nose. On expiration the obstruction at the nares blocks the outgoing air and causes a positive pressure. The greater the relative obstruction at the nares, the greater will be the fluctuation in intranasal pressure during respiration. If the choanae are reduced as by adenoidectomy there will be little or no variation in pressure, or if choanae are smaller than the nares the above changes are reversed.

#### NASAL CYCLE

Another interesting observation is the fact that both nasal airways do not function simultaneously. It is a well supported observation of LILLIE that in a normal nose one nasal airway opens up with secretion of serous and mucous discharge, while the opposite airway closes down with almost complete cessation of secretory activity and that passage of respiratory air is carried out in its entirety through the open side. The phenomenon has been termed nasal cycle by KAYSAR. A satisfactory explanation of this phenomenon has been presented but it seems probable that the nasal mucous membrane which is constantly and heavily worked up in its multifarious activities such as cleansing, warming, humidifying amongst others, should be allowed time to recover. It is of historical interest that the ancient Hindus had knowledge of this fact and it is mentioned in the yogic literature.

The presence of a cycle of congestion and decongestion of the cavernous tissue of the nasal conchae in normal individuals was observed by KAYSAR, who suggested that there is a continuing shift in the autonomic balance between the two halves of the nose. This in turn, he thought, causes a change in the amount of blood circulating in the erectile tissues of the turbinates and septum. He also related the complaint of alternating nasal obstruction of some patients' to the nasal cycle. Experiments of HEETDERK showed that damp and cold atmosphere favoured the maximum mucosal congestion. Such atmosphere is very often encountered in Suriname in the rainy seasons. It is also a fact that more patients seek relief for their nasal complaints in these months of rainy seasons in Suriname.

	Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sept.	Oct.	Nov.	Dec.
Mean temperature in degrees centigrade	25.7	26.0	26.6	26.4	26.1	26.8	28.0	28.2	29	27.8	26.3	
Percentage of average humidity	84	81	84	85	85	81	76	74	71	76	75	
Number of new patients with chronic vasomotor rhinitis seen	10	7	11	12	6	4	6	7	7	2	5	

Table 2.

Monthly break up of a number of cases of chronic vasomotor rhinitis along with the temperature and humidity in Paramaribo in the year 1976. No figures have been mentioned in March since I was away on vacation.

It is clear from the above facts that high humidity does play a role here. Even without March figures nearly 60% of the cases were first seen in the first half of the year when the humidity was higher.

Eighty percent of normal individuals exhibit nasal cycle (STOKESTED 1953). Rhinometric studies of the nasal cycle has shown that the alternating passage of air through the nasal airways does not alter the total resistance offered by the nose, and so the nasal physiology basically remains the same. Rhinometric tests conducted by OGURA and STOKESTED in cases of nasal obstruction with anatomical deformities and vasomotor rhinitis showed interruption and disturbance of normal cycle. In the normal nose, they found that bilateral synchronous turbinal reaction to extreme irritants took place to a limited degree and required but a short time for adaption, so that cyclical rhythm is not disturbed. These observations are of great importance and explain the presence of certain symptoms in conditions such as vasomotor rhinitis.

The duration of these cyclical changes in the nose varies from two to seven hours. Experiments of KEUNING (1968) on the working of the nasal cycle throw light on the autonomic control of the nasal cycle.

#### NERVE SUPPLY AND NERVOUS PATHWAYS

*The nerve supply of the nose comes from three different sources:*

1. The general sensation i.e. touch, temperature and pain is mediated through the branches of the fifth cranial nerve.

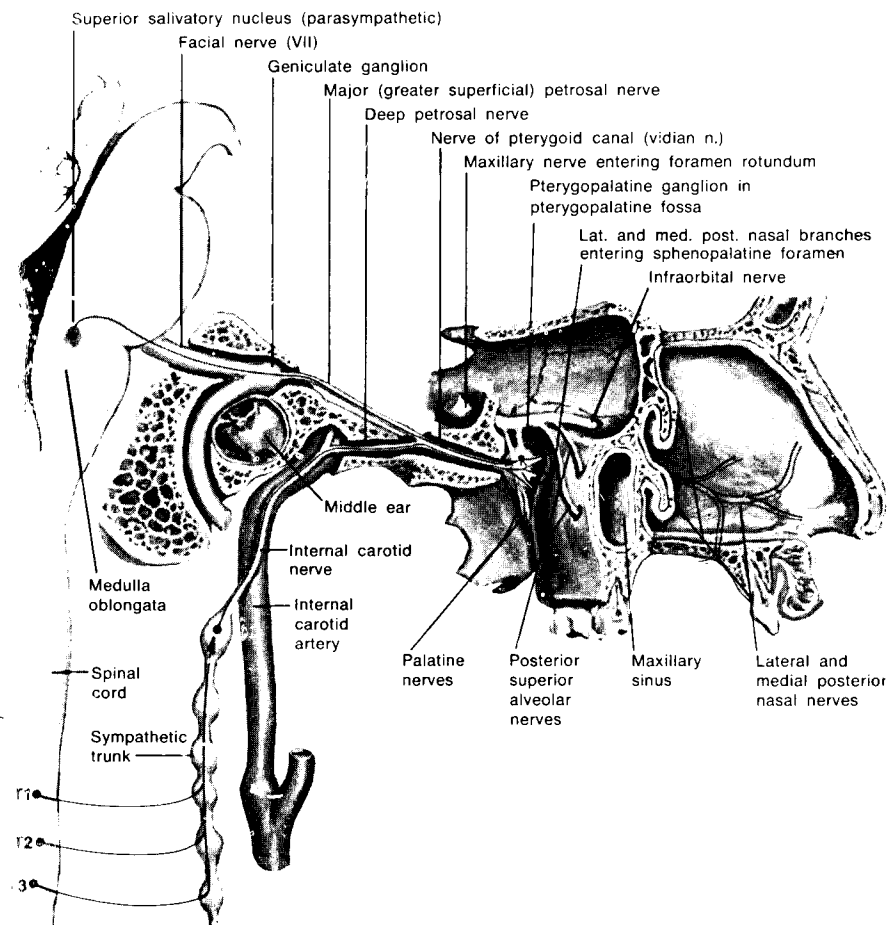


Figure 1

Diagram showing formation and distribution of vidian nerve. With kind permission of Medical Education Division, CIBA Geigy Pharmaceutical Company Basle.

Motor supply to the nasal respiratory muscles comes from the seventh cranial nerve. Integration of their respiratory rhythm is carried through the vagus.

The physiologically important control of the circulation to the nasal airway is mediated by autonomic nervous system.

Both sympathetic and parasympathetic nerves are in abundance and their actions balance each other in health to ensure a satisfactory nasal airway.

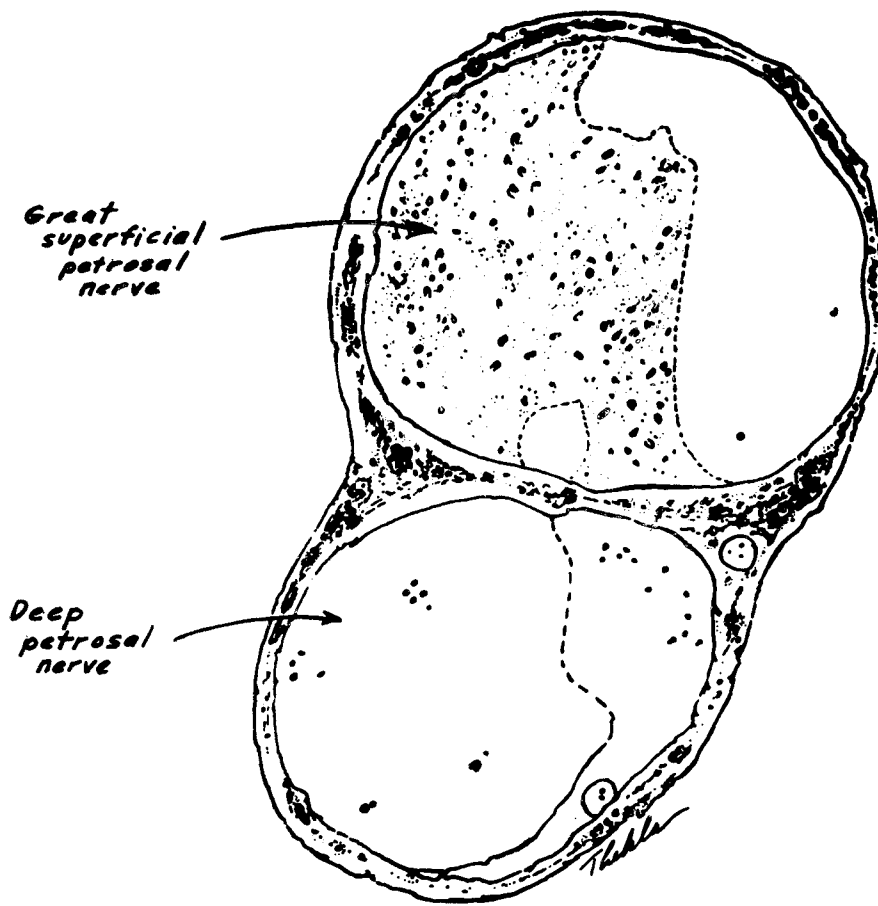


Figure 2

*A cross section of nerves in vidian canal.  
Adopted from Otolaryngology Vol. III Chapt. 21a. With kind permission of Harper & Row, Publishers Maryland U.S.A.*

The sympathetic innervation of the head and neck originates in the pre-ganglionic neurones of the upper thoracic segments which terminate in the stellate and superior cervical ganglion. Postganglionic fibers extend into the head by forming plexuses along the walls of the great blood vessels. The internal carotid plexus gives a branch while the internal carotid artery is passing through the carotid canal. This branch is termed the deep petrosal nerve. Extensive sym-

ple supply also reaches the nasal cavity through the plexuses around the carotid artery and its branches.

Parasympathetic innervation to the nasal cavity has its beginning in the salivatory nucleus (part of the general visceral efferent column). The fibres emerge with the nervus intermedius at the lower border of the pons and pass through the geniculate ganglion of the facial nerve after their passage through the internal auditory meatus. They pass through the ganglion and leave in the great superficial petrosal nerve in a short bony canal in the petrous bone, through which journey they receive a twig from the tympanic plexus. This nerve carries secretomotor supply to the mucous glands of the nasal cavity and also to the lacrimal gland. The nerve runs on the anterior surface of the petrous bone and the route of the carotid canal is joined by the deep petrosal nerve carrying sympathetic supply. This union of nerves is known as the nerve of pterygoid or vidian nerve. At this junction a small parasympathetic ganglion exists.

The vidian nerve is thus a composite nerve carrying the autonomic supply to the nasal cavity. The two sets of fibres i.e. the postganglionic sympathetic and preganglionic parasympathetic do not intermingle but remain in separate compartments within the same nerve sheath. (Fig. 2). The vidian nerve runs through the pterygoid canal and leaves the canal at its anterior exit on the anterior surface of the sphenoid.

It arrives in the retro-maxillary space where it enters the deep surface of the sphenopalatine ganglion soon after its exit from the pterygoid canal. During its passage through the sphenoid bone the nerve lies in close relation to the floor of the sphenoid sinus. Should the bony floor of the sphenoid sinus be dehiscent, the vidian nerve could be irritated by an inflammatory process in the mucous membrane of the sphenoid sinus and could lead to vidian neuralgia. The sympathetic fibres in the vidian nerve do not relay in the sphenopalatine ganglion but the parasympathetic fibres do. The autonomic supply to the nasal cavity then reaches the nose through the branches of the sphenopalatine ganglion. The lacrimal fibres run the same course to the sphenopalatine ganglion. The postganglionic sympathetic fibres travel through the maxillary nerve to its zygomatic branch and join the zygomatic nerve.

The experiments of ECCLES and WILSON, MALM, ROEKER, ANGGARD and those of GADLAGE have thrown light on the manner in which the vidian nerve functions. It would appear from their experiments that there is considerable variation especially in the laboratory animals. The composition of the vidian nerve is still not known for certain. However, from these experiments which consisted of monitoring the results after direct stimulation and timing of the responses by parasympathomimetic drugs, it was found that elec-

trical stimulation of the vidian nerve in dogs induces nasal secretion and produces vasodilatation. They could block the secretory effect but not the vasodilatation, by parasympathomimetic drugs and thus conclude that the vasodilatation is not a cholinergic effect.

Mediation of the impulses in the peripheral receptors is chemical i.e. via sympathin at the sympathetic nerve endings and acetyl choline at the parasympathetic nerve endings respectively.

The stimulation of the sympathetic produces vaso-constriction of the turbinates and increase in nasal airways. The secretion of mucous glands is grossly diminished. The response is basically protective allowing maximum air intake for olfaction and detection of the source of danger. Parasympathetic activity on the other hand produces vasodilatation and slowing of circulation with resultant congestion and diminished air entry. The secretion of mucous is abundant. In health, there exists a balance between the two i.e. sympathetic and parasympathetic deposits energy and the sympathetic spends it.

Golding Wood rightly mentioned that the vidian nerve also carries some afferent fibres too. This is borne by clinical experience as well. Patients who undergo vidian neurectomy have reduced pain sensation on the side of operation.

The autonomic nerve supply of the nasal cavity is subject to control by higher centers. The division of the autonomic system into peripheral and central portions is actually for the sake of description.

There are levels in the spine medulla and pons where reflex centers exist but all are coordinated and subjected to higher integrative controls which are found in the hypothalamus and cerebral cortex. The hypothalamus receives impulses from the nuclei governing the peripheral autonomic nervous system. It is also connected closely to the cerebral cortex and the hypophysis. It is a diamond shaped area in the floor and walls of the third ventricle bounded by the optic chiasma. Impulses are received by the hypothalamus from internal and external sources, through hormones circulating in the blood and the organs of sense of perception. By integrating these with the requirements of the body, reflexes are established which innervate both sympathetic and parasympathetic divisions at various levels. The hypothalamus is also a centre for emotional expression. The cerebral cortex which exercises an inhibitory control is connected to the hypothalamus. In 1875 SCHIFF first described areas of autonomic regulation in the cerebral cortex. Since that time numerous workers have reported that all autonomic activity is represented in cerebral cortex. There are autonomic mechanisms in primary motor areas of precentral gyrus (area 4), premotor cortex (area 6) in the frontal lobe (area 8) and in occipital lobe (area 18 and 19).

## Pathology of vasomotor rhinitis

and about twenty years ago chronic vasomotor rhinitis was described in literature as "a condition characterized by recurring colds accompanied by a watery nasal discharge, sneezing and nasal obstruction. The condition is sometimes also accompanied by lachrimation and itching of the ear, nose and throat."

It was also believed that the condition was allergic in origin but no relationship with any known antigen was ever clearly demonstrated. The idea of autonomic imbalance was put forward by WAELSH, MALCOMSON and FOX, and later substantiated by GOLDING WOOD.

The symptomatology of vasomotor rhinitis being very similar to that of allergic rhinitis, that is the first major condition from which a clearcut differentiation is essential before a diagnosis of vasomotor rhinitis can be established. For this purpose a detailed careful history as mentioned under the heading of Allergies and Materials has first got to be obtained. In patients with allergic rhinitis an association with a particular food, inhalant or contact should be sought. Seasonal allergy such as hayfever is very typical and begins suddenly and ends dramatically at the end of the season. There are now a host of tests for allergy such as scratch test, intradermal test and patch test available. In addition to tests of food allergy, deliberate intake under supervision or abstinence is used as a test of allergy.

Demonstration of eosinophils in nasal secretion or bronchial mucous was first suggested and consisted by many authorities such as GAY, HARRIS, HANSEL, GLASER and FOX-JENSEN, to be diagnostic of an allergic process. It has now however been shown that eosinophils may be present in nasal secretions in many non-allergic states and may be absent or scarce in a typical case of known allergy. There is evidence that eosinophilia in nasal secretions can occur with shifts in autonomic balance. FOWLER'S case where a typical picture of vasomotor rhinitis developed unilaterally after a stellate ganglionectomy on the same side showed eosinophilia on the effected side of the nose only. WOLFF induced eosinophilia in the peripheral blood and nasal secretion by purely psychological means. It is also known that the eosinophils present in the nasal secretions of patients with vasomotor rhinitis disappear after vidian neurectomy.

Vasomotor rhinitis on the other hand can be induced by a variety of physical factors such as cold, heat, moisture, light or local irritants and sometimes by hormonal or endocrine factors as well. From the standpoint of immunobiology these factors are neutral and provoke no antibody reaction. The condition is not immunologic or IgE mediated. The history as for allergy, is essentially negative and reactions by the tests mentioned above are also negative. An attack of vaso-

motor rhinitis occurs typically early in the morning when the patient wakes up with a bout of sneezing. His nose is blocked and has watery discharge. The condition is worst if the patient exposes him- or herself to cold such as the winter mornings in Europe or the nights in Suriname. Night temperatures in Suriname range from 16 to 18° centigrade. Typically at times the patient complains that the attacks come on Saturday evenings after a party (which are usually outdoors in Suriname) and when he has had a few cold drinks. There is no seasonal variation as such but the frequency of these attacks is more in the rainy season in Suriname when the temperatures are lower and the humidity is high. The therapeutic response to antihistaminics is no conclusive proof of allergy. Antihistaminics also have anticholinergic effect and also effect capillary permeability. It has been known that antigen-antibody reaction release not only histamine but also heparin and 5- hydroxy — tryptamine. In the respiratory mucous membrane the indicated structures remain to be mucous glands, smooth muscle and capillaries. All these structures are under autonomic control. It is known that parasympathetic stimulation induces muscle contraction and secretion of mucous and that the parasympathetic mediator of acetylcholine is pharmacologically similar to histamine.

Many workers thought the condition of vasomotor rhinitis to be similar to the syndrome of vagotonia and basically a product of sympathetic and parasympathetic imbalance. The restricted distribution of parasympathetic ganglion facilitates greater central nervous system effect upon a particular organ in comparison to the wide spread arrangement and influence of the sympathetic system. This is evident in vasomotor rhinitis where impulses are fired exclusively to the nasal cavity. This also facilitates surgical interference, in cases where parasympathetic overactivity leads to pathological symptoms and sectioning of parasympathetic effect fully or partly in an anatomic accessible situation provides relief or complete cure in some cases. Vidian nerve section in chronic vasomotor rhinitis and vagotomy in cases of peptic ulcer are striking examples. Sympathetic and parasympathetic normally maintain the ballance which seems to be disturbed in vasomotor rhinitis. The data accumulated by HIGBEE shows that in a normal person the threshold of stimulation of the parasympathetic nerves is relatively low and the dominant effect on the nasal mucous membrane is vaso constriction. Adjustments through atmospheric conditions and constitutional states are being continually made through secretory and vasomotor nerves to maintain the nasal mucosa in a normal state. In chronic vasomotor rhinitis the parasympathetic takes the upper hand with resultant vasodilatation and increased nasal secretion.

Vasomotor rhinitis has been compared to non-allergic bronchial asthma. The possible role of acetylcholine was emphasized by NAKAMURO.

Other known etiological factors have been related to vasomotor rhinitis. They have been mentioned earlier but are being included here for the sake of completeness.

*Emotional stimuli.* This is perhaps one of the most important factors. Many patients have a positive history of emotional maladjustment. Others give no positive history but in some cases the symptoms could be traced to a psychological trauma. There are yet others who live in a continued state of stress but it is not always easy to elicit this information.

*Hormonal changes.* The work of HOLMES, GOODELL, WOLF and WOLFF have drawn attention to changes in nasal mucous membrane and function accompanying sexual excitement, menstruation and pregnancy.

*Physical agents.* Cold, heat, sunlight and moisture have been mentioned earlier. Mention must be made here of some patients whose symptoms seem to worsen in airconditioned atmosphere.

*Air pollution* has been mentioned as a possible causative factor. It certainly may act as an irritant to the nasal mucous membrane. Since no statistics about the incidence of the condition of chronic vasomotor rhinitis in other parts of the world are available it is difficult to draw any conclusion. However nasal polyposis has been mentioned as a possible sequel of vasomotor rhinitis or its accompaniment at any rate, by HIRANANDANI. Etiology of nasal polyposis is uncertain but it would appear that chronic irritation of the nasal mucosa plays a significant role.

I had the opportunity of working for several years in England where the incidence of nasal polyposis is fairly high. My records of operations performed in 10 centres in England from 1963 until 1968 show an average of 43 nasal polyps removed per year in an otolaryngological department. In the last 5 years in my clinic I have had to perform this procedure only on seven patients.

*Reflex activity.* This is seen in cases where simple nasal stimuli such as exposure to dust, may produce violent vasomotor reactions. Many patients attribute the onset of sneezing attacks to exposure to house dust.

*Racial factors.* There appears to be a positive marked difference in the incidence of vasomotor rhinitis in different races. In Suriname where all races (Europeans, Indians, Negroes, Chinese, Indonesians and Amerindians) are



encountered, vasomotor rhinitis is seen mostly in the East Indian population. The Chinese and mixed races are also effected but the negroes are the least effected. (see table 3).

All these factors seem to indicate that vasomotor rhinitis is a localized manifestation of parasympathetic overactivity.

Various Ethnic groups living in Suriname:	Percentage of population of special racial origin in total population of Suriname	Percentage of population of special racial origin having chronic vasomotor rhinitis
East Indians	35	49.3
Indonesians	15	6.5
Bush Negroes	10	nil
Chinese	2	9.1
Amer Indians	2	nil
Europeans	1	3.9
Creoles (Mixed)	35	31.2

Table 3

Incidence of chronic vasomotor rhinitis in the various ethnic groups in Suriname. There was only one patient of Lebanese origin. There total number is less than 0.5%.

The possible explanation for the almost total absence of chronic vasomotor rhinitis in the Bush Negroes and Amer-Indians could be the lack of stress in these groups because of their life style.

Golding Wood (1962) offered what he described as a neural basis to explain the mechanism of vasomotor rhinitis. He postulated that the beginning of parasympathetic activities which leads to the changes in the nasal mucosa in vasomotor rhinitis occurs in the hypothalamus. The afferent pathways from the nasal mucosa are through the somatic trigeminal fibres. Purely autonomic pathways may also be important.

The vidian nerve in human being carries also some afferent fibres. Central factors such as emotions endocrine factors and also afferents through the trigeminal arc affect the hypothalamus via the cerebral cortex. The endocrine factors may also effect the hypothalamus directly because of the hypophyseal connection. The hypothalamic centers in turn stimulate the superior salivary nucleus. Impulses then travel via the greater superficial petrosal and vidian nerve to the sphenopalatine ganglion and after being relayed postganglionic parasympathetic fibres reach the nasal mucous membrane and exert their mucosal effects by release of acetylcholine.

The role of racial constitutional and genetic factors is to create the conditions for the onset or otherwise of the trigger mechanism but basically the mechanism remains the same.

Very little has been said with regard to this theory, ever since Golding Wood wrote about it but over the years it has been tested and proved to be sound and correct.

The study of histopathology of chronic vasomotor rhinitis has been diligently done by Miranandani. The nasal epithelium in these cases shows changes from normal ciliated, columnar epithelium, transitional epithelium and changes of metaplasia occurring in different sections or in different parts of the same section. In some areas ciliated columnar cells are seen with elongated nuclei placed at variable distances between the base of the cell and free border. The free border contained cilia. In other areas transitional cells are polygonal and flattened with small rounded nuclei and faint cytoplasm. Basal layers of cells with their long axis at right angles to free surface is found.

Chronic metaplasia may be present in scattered unconnected areas. Broadening of the basement membrane is a constant finding. Oedema of the stroma is the most constant feature. Oedema is seen in the midst of interlacing network of connective tissue. Dilated bloodvessels are seen which are sometimes engorged with blood. Large numbers of serous and mucinous glands of varying sizes are present.

In many sections subepithelial infiltration with eosinophils is seen. This is however not a conspicuous feature. On the other hand infiltration with mononuclear and plasma cells is a constant feature present due to coexisting inflammatory changes in many cases.

In this connection it is interesting to recall the concept of autonomic dysfunction as enunciated by HENRY L. WILLIAMS in 1951. Basically his concept is similar to what has been just described.

Williams related it physical allergy and he stated that the state of autonomic imbalance specifically of the cholinergic fibres could be enunciated by physical stress, emotional perturbation, endocrine imbalance, air pollution and weather changes. In the area of autonomic dysfunction a stereotyped reaction of the peripheral vasculature takes place. It consists of arteriolar spasm with atonic dilatation of the capillaries and venules. Because of the arteriolar spasm there exist a block in arterial circulation and a relative anoxic state of the capillaries. This leads to cellular damage and release of histamine with resultant capillary permeability and oedema. This reaction is the basis of the physical changes seen in the nasal

mucosa in vasomotor rhinitis. Some observers and specialists, however, are of the opinion that vasomotor rhinitis could be explained as a phenomenon of the delayed sensitivity type of allergic reaction. This however, is an assumption and there is no evidence to substantiate this.

## Evolution of therapeutic measures

The treatment of chronic vasomotor rhinitis has undergone considerable change in the last 25 years especially since there has been greater understanding of the mechanism and etiological factors involved. In the days when the condition was confused with allergic rhinitis treatment was generally symptomatic. Decongestants were the mainstay of treatment. Long before this, therapy was symptomatic. Oral administration of ephedrine, belladonna and zinc ions were considered to be the standard method of treatment in most clinics. Since the mechanism was illunderstood, many intranasal procedures were carried out in the hope that these will stop the trigger mechanism and will improve the condition. Amongst these turbinectomy, septal resection and diathermy cauterisation were most frequently done. Sometimes the procedures produced temporary relief for a variable period of time but invariably the relief is incomplete and symptoms tend to return.

Patients were studied for allergy and were tested for specific allergies. However, in almost the case with these patients, the symptoms have no definite seasonal or particular time of the year and conclusive association with any allergen is almost never found. When patients were tested for allergens and if any responses were obtained, specific desensitisation was usually tried out.

Experience with such therapy was disheartening since in most cases the positive response to skin tests occurred to common factors such as house dust etc. After a prolonged period of desensitisation therapy, symptoms recurred within a few days at the most. Seasonal allergy where allergic symptoms can be directly related to the onset of changes in atmospheric conditions at a particular time of the year also show definite positive allergic responses to skin tests. These patients respond well to desensitisation therapy and the treatment has become relatively simpler since the evolution of special vaccines which can be administered in two doses. In Suriname specific seasonal allergy is uncommon while local allergy is more common. It could be due to inhalants such as hair, dust, cosmetics etc. or ingestants such as various food factors like eggs, shellfish, fish, chocolate, tomatoes etc. These are relatively easy to avoid if a trigger factor is involved.

Drugs such as reserpine and many other antihypertensive agents as well as tranquilizers produce nasal stuffiness. These drugs may inhibit hypothalamic sympathetic activity thereby leading to the predominance of parasympathetic activity with resultant vasodilatation. The clinical appearance of nasal mucous membrane in these patients looks similar to vasomotor rhinitis. After prolonged treatment with antihypertensives permanent changes may take place due to exten-

sive fibrous hyperplasia. Such patients may require surgical help such as turbinal diathermy to improve the nasal airway.

The first principle of treatment is to locate the irritating and precipitating factor and advise the patient to avoid them. Specific hyposensitisation with vaccines is advisable. However, this has not been found necessary in any of my patients. Nonspecific hyposensitisation is mentioned here only for completion. It has been used in many clinics all over the world before the present concept of vasomotor rhinitis was developed. Procedures such as injections of peptone and autohaemotherapy were frequently practised. Injections of peptone occasionally led to complications in the form of jaundice.

Histamine desensitisation was practised all over the world at the time that vasomotor rhinitis was regarded as an allergic condition. Since the release of Histamine in the tissues was found to be a result of allergic reaction, this procedure gained considerable popularity but has been discarded. The procedure consisted of administering increasingly higher doses of histamine subcutaneously, over a long period of time.

Before the advent of antihistaminics the systemic or oral administration of ephedrine and belladonna were practised with considerable symptomatic relief. The regression of symptoms was attributable to the sympathomimetic effects of these compounds. They produced dryness of the nose and improved the nasal airway but the sneezing attacks were unaltered. These drugs also produced systemic side effects such as palpitation and dryness of the mouth.

Injections of Calcium salts such as Calcium gluconate, and administration of Vitamin C and P were at one time highly recommended and widely used. But these are also being abandoned and I have never prescribed them to my patients. Their use was empirical. It was based on the finding that Vitamin C played a role in the normal maintenance of collagen and intercellular ground substance. It is now ascertained that the use of Vitamin C is only indicated in the prevention and treatment of scurvy.

The discovery of antihistaminics in 1937 and in subsequent years has completely revolutionized the conservative management of vasomotor rhinitis especially for the symptomatic relief of acute attacks. The drugs are safe and effective. Antergan and Neo-antergan (pyrilamine malleate) were the first antihistaminic drugs to be used. Traditionally antihistaminics were classified into:

- a. Ethylendiamine derivatives such as antergan, neo-antergan, histadyl.
- b. Ethanolamine derivatives such as Benadryl, Decapryn and Clistin.

amines such as Chlortrimeton and Termenton.

amines such as Marezine and Bonine.

benzines such as Phenergan.

Antihistaminics are effective against many responses to histamine which are mediated by a distinct class of histamine receptors, known as the receptors of ASH and SCHILD. The new class of histamine antagonists blocked histamine on receptors of a different type termed histamine H<sub>2</sub>-receptors. These have been referred to as H<sub>2</sub>-receptors antagonists by BLACK et al, in 1972. The extent of the therapeutic value of the H<sub>2</sub>-receptor antagonists has not fully realized. Examples of this class of antihistaminics are :  
1. Metiamide.

The mode of action of antihistaminics in vasomotor rhinitis is :

1. Local sedative action.

2. Anticholinergic effect.

3. Reduction of the permeability of the capillary wall.

4. They especially the H<sub>1</sub> blocking agents inhibit the vasodilator effects of histamine. The action on capillaries reduces the oedema of the tissues and the local sedative action tends to calm such a patient. These drugs are however, not without side effects and patients sometimes complain of drowsiness, dizziness and dryness of mouth and throat as well as respiratory passages leading to gastrointestinal disturbances, etc. Long acting antihistaminics are preferable chemotherapy of vasomotor rhinitis.

PREPARATIONS AND DOSAGE OF REPRESENTATIVE OFFICIAL  
H<sub>1</sub>-BLOCKING ANTIHISTAMINES

CLASS AND NONPROPRIETARY NAME	STATUS TRADE NAME	DURATION OF ACTION (HOURS)	USUAL PREPARATION	OTHER PREPARATIONS AVAILABLE	SINGLE DOSE (ADULT)
<i>Ethanolamines</i> Diphenhydramine Hydrochloride	U.S.P. BENADRYL	4—6	Capsules, 25 and 50 mg	Injection (Syringes and ampuls); elixir	50 mg
Dimenhydrinate	U.S.P. DRAMAMINE	4—6	Tablets, 50 mg	Injection suppositories; Syrup	50 mg
Carbinoxamine Maleate	N.F. CLISTIN	3—4	Tablets, 4 mg; repeat-action tablets, 8 and 12 mg	Elixir	4 mg
<i>Ethylenediamines</i> Triptenamine Hydrochloride	U.S.P. PYRIBENZAMINE	4—6	Tablets, 25 and 50 mg; delayed-action tablets 50 and 100 mg	Cream (topical), 2%; ointment (topical 2%)	50 mg
Triptenamine Citrate	U.S.P. FYRIBENZAMINE		Elixir, 37.5 mg/5 ml		5 mg
Pyrilamine Maleate	N.F. HISTALON, NEO-ANTERGAN,	4—6	Tablets, 25	Various (in combinations)	25-50 mg
<i>Alkylamines</i> Chlorpheniramine	U.S.P. CHLOR- TRIMETON and many others	4—6	Tablets, 4 repeat-action tablets, 8 and 12 mg	Injection	2-4 mg
Brompheniramine Maleate	N.F. DIMETANE, DISOMER	4—6	Tablets, 4 mg	Elixir	4-8 mg
<i>Piperazines</i> Cyclizine Hydrochloride	U.S.P. U.S.P. MAREZINE	4—6	Tablets, 50 mg	Suppositories, 50 and 100 mg; injection	50 mg
Cyclizine Lactate	N.F. MAREZINE	4—6	Injection 50 mg/ml in 1-ml ampul		50 mg
Meclizine Hydrochloride	U.S.P. BONINE	12—24	Tablets (chewing), 25 mg		25-50 mg
<i>Phenothiazines</i> Promethazine Hydrochloride	U.S.P. PHENERGAN	4—6	Tablets, 12.5 25, and 50 mg	Injection; suppositories syrup	25-50 mg

Adapted from *The Pharmacological basis of Therapeutics. Goodman and Gilman. Fifth Edition.*

In chronic vasomotor rhinitis however, antihistaminics are of limited value. The choice of drug sometimes lies on the patient's individual reaction. On the whole the patient has to use the drug for about a week and assess the improvement with his treating physician until he finds the right drug for himself. Prolonged therapy with one particular drug, besides its side effects, also produces adaptation and diminished response, both in duration and degree of relief.

The next important group of drug effective in the medical treatment of acute and sometimes chronic vasomotor rhinitis are the corticosteroids. Although the importance of adrenocorticotrophic hormone in maintenance and control of salt- and sugar metabolism and the production of sexhormones was known for sometimes since 1920, it was HANS SELYE in 1946 who first pointed out the close relationship between the production of this hormone and stress. It was in 1957 that BORDLEY as quoted by TREYNOR was among the first to report the effect of A.C.T.H. and cortisone on nasal mucosa. He reported thinning of the nasal mucosa which developed a slate pink colour and disappearance of nasal polypi. However, he found that both these agents lowered the resistance of nasal mucosa to infections.

A.C.T.H. stimulates the human adrenal cortex to secrete cortisol corticosterone, aldosterone and a number of other adrogenic substances. The presence of adeno-hypophysis is essential to the function of adrenal cortex. Corticosteroid administration suppresses A.C.T.H. release and produces functional impairment of adeno-hypophysis, A.C.T.H. is destroyed by proteolytic enzymes of the gastrointestinal tract. Therefore the hormone is ineffective when given orally. From cholesterol, the adrenal cortex synthesizes two classes of steroids: the corticosteroids, and the adrenal androgens. The adrenal corticosteroids have a wide number of synthetic analoges such as desoxycorticosterone, fludrocortisone, hydrocortisone, betamethasone, dexamethasone, prednisone, prednisolone, methylprednisolone, paramethasone, triamcinolone, flumethasone, fluocinolone, fluocinonide, fluormethalone, flurandrenolide and medryson. Depending upon the potency of these compounds on sodium retention and on liverglycogen deposition, the corticosteroids have been classified into: Mineralocorticoids and Glucocorticoids. Corticosteroids like other steroid hormones are thought to act by controlling the rate of synthesis of proteins. Corticosteroids have a wide variety of physiological actions such as on metabolism, electrolyte- and water ballance, cardiovascular system, skeletal muscle, central nervous system, haemopoïtic system, lymphoid tissue, immune responses and growth and cell division. They also have antiinflammatory properties and suppress the development of signs of inflammation. In clinical terms the admini-

stration of corticosteroids for their antiinflammatory effect is palliative therapy. The underlying cause of the disease remains unaltered. It is the suppression of inflammation that has made corticosteroids such valuable therapeutic agents.

In the treatment of vasomotor rhinitis corticosteroids help by their antiinflammatory effect. However, the treating physician has to bear in mind that the same drug may suppress symptoms of underlying inflammatory process such as acute sinusitis. Corticosteroids also help by the reducing capillary permeability. They are of great value in treating an acute attack, however, in view of the variety of actions, which in the long run could be detrimental, prolonged use is inadvisable. I have used methylprednisolone acetate in a single dose of 10-80 mg in cases of acute attacks, successfully and with great relief of the patient. This is in keeping with the recommendations of wellknown authorities on this subject such as GOODMAN and GILMAN.

In the last decade many pharmaceutical companies have brought out preparations which are a combination of a corticosteroid with a long acting antihistaminic. This is an excellent combination as applied to the treatment of chronic vasomotor rhinitis and are useful for medical therapy over a long period of time.

Another group of drugs which offer symptomatic relief when used alone or in combination with antihistaminics and/or corticosteroids, is the pseudoephedrine. They act by their local effect on the nasal mucous membrane which is similar to adrenaline and at the same time, in therapeutic doses they do not have any systemic side effects. However, they should be used with caution in patients with hypertension. They sometimes produce gastrointestinal upset especially in children. Drowsiness is also occasionally complained of.

#### TREATMENT

The most commonly prescribed and perhaps misused local treatment are the vasoconstrictors. The eagerness of the patient to relieve his/her symptoms by the quickest possible way, and perhaps of the physician to satisfy his patient more quickly, often leads to the use of local nasal vasoconstrictors. This eagerness is well matched by the enthusiasm of the pharmaceutical industry which in the last 25 years flooded the market with a wide range of preparations for local application. These are mostly available in the form of drops and sprays and many of them contain also agents such as antihistaminics cortisone, etc. Vasoconstrictors applied locally to the nasal mucosa cause shrinkage of the mucous membrane tissue and provide temporary symptomatic relief. The temporary relief encourages the patient to instill more and more of the drug repeatedly. The duration of relief from nasal obstruction becomes shorter and shorter until the

PREPARATIONS OF ADRENOCORTICAL STEROIDS AND THEIR SYNTHETIC ANALOGS

NONPROPRIETARY NAME	DERIVATIVE	TRADE NAMES	ORAL FORMS		INJECTABLE FORMS	TOPICAL FORM	TOPICAL OPHTHALMIC PREPARATIONS †
			Tablets	Liquids			
Desoxycorticosterone	Acetate, U.S.P.	DOCA ACETATE, PERCORTEN ACETATE	2.5 mg (buccal)		5 mg/ml (oil) ▲, 125 mg (pellets) ‡		
	Pivalate, N.F.	PERCORTEN PIVALATE			25 mg/ml (susp.) ‡		
	Acetate, U.S.P.	FLORINEF ACETATE	0.1 mg ▲				
Hydrocortisone, U.S.P.		CORTEF, HYDROCORTONE, and others	5, 10, 20 mg ▲		25, 50 mg/ml (susp.)	0.125-2% cream ▲, 1% ointment ▲, 0.25% lotion ▲, 100 mg/60 ml enema ▲	0.2% suspension
	Acetate, U.S.P.	CORTEF ACETATE, HYDROCORTONE ACETATE, and others			25, 50 mg/5 ml (susp.) ▲	1, 2.5% ointment ▲, 1%, 2% mg suppositories, 10% rectal foam	2.5% suspension ▲, 1.5% ointment ▲
	Cypionate, N.F.	CORTEF FLUID		2 mg/ml (susp.)			
	Sodium Phosphate, U.S.P.	HYDROCORTONE PHOSPHATE			50 mg/ml ▲		
	Sodium Succinate	SOLU-CORTEF			100, 250, 500 mg		
Cortisone	Benzoate	BENISONE GEL, FLUROBATE GEL				0.025% cream or gel	
	Acetate U.S.P.	CORTONE ACETATE	5, 25 mg ▲		25, 50 mg/ml (susp.) ▲		1.5, 2.5% suspension
		DECADRON, GAMMACORTEN, and others	0.25-1.5 mg ▲	0.5 mg/5 ml (elixir) ▲		0.04% cream; 0.1% gel; 0.011% aerosol (topical) ‡	0.1% suspension
Dexamethasone, U.S.P.	Sodium Phosphate, U.S.P.	DECADRON PHOSPHATE, HEXADROL PHOSPHATE			4 mg/ml ▲	0.1% cream ▲, 18 mg/12.6 g aerosol (inhalation)	0.1% solution ▲, 0.05% ointment ▲
	Acetate	DECADRON-L.A.			8 mg/ml		
Prednisolone, U.S.P.		DELTA-CORTEF and others	1, 2.5, 5 mg ▲			16.6 mg/50 g aerosol	
	Acetate, U.S.P.	METICORTEONE ACETATE and others			25, 50, 100 mg/ml (susp.) ▲		0.12, 1% suspension

Adapted from *The Pharmacological basis of Therapeutic. Goodman and Gilman (fifth Edition).*

resultant tissue anoxia and irritation leads to rebound vasodilatation over a period of months.

A state of rhinitis medicamentosa is reached and such patients often seek a specialist opinion with the complaint that the nosedrops originally prescribed to them and which they have diligently used for months or years has now failed to relieve the symptoms. Often the patients has already tried every brand of available nosedrops and was ill for the last 45 years. Unfortunately in most of the free world nosedrops can be purchased from drugstores or chemists without a prescription. The nasal mucous membrane especially at the anterior end of the conchae is red, granular and angry looking. Hiranandani has described that the mucous membrane of such patients becomes hypertrophic and "cauliflower like", requiring surgical removal of all or part of the inferior turbinate. I have never encountered this in my patients.

These patients have to be "deprogrammed" by first stopping the use of vasoconstrictor agents. I have found that at this stage administration of a single dose of intramuscular Methylprednisolone accompanied by oral administration of long acting antihistaminics helps to stabilize these patients. At the outset of the treatment a full and frank discussion of the condition is undertaken of prolonged local nasal decongestant therapy and the role of the medical treatment he or she is just likely to undergo.

Patients are advised to get rid off the bottles of nosedrops that they have at home and I usually persuade them to leave the one they have in their bag, with me. Usually one to three weeks later the nasal airway returns to normal. Sometimes local surgical procedures such as submucosal turbinal diathermy is necessary.

I have never advocated the use of nosedrops except as a temporary measure for 3 to 4 days. Sometimes it is better to prescribe corticosteroids as spray or drops in place of vasoconstrictor agents.

#### AUTOHAEMOTHERAPY

Autohaemotherapy has been practised in certain clinics. Hiranandani mentioned the use of 1 ml of patients own blood with a small amount of sodium citrate 0.5 ml injected submucosally in the inferior turbinate after suitable local anaesthesia. The procedure is repeated once a week for about 6 weeks. In the series published by Hiranandani he claimed partial relief for 1—3 years in 50% of patients. The procedure, however, has not been advocated generally by other authorities.

*Injection of hydrocortisone* or other corticosteroids such as Methylprednisolone has been practised. Half a millilitre is injected in each inferior turbinate once a week for 5—6 weeks.

About 30% of patients showed temporary relief. The relief is certainly due to the absorption of corticosteroid and its central action. I have not found this procedure particularly helpful in vasomotor rhinitis.

*Ganglionic ganglion injection* has been recommended. The local anaesthetic such as 3—5 cc of 2% xylocaine or pontocaine is injected in the inferior turbinate. The resultant improvement in nasal symptoms is said to be due to the blockade of parasympathetic impulses and is transient. The procedure is practised by me. It has certain pitfalls. In the first place the concentration of local anaesthetic agent reaching the ganglion is uncertain. There is a possibility of occasional bleeding from the maxillary artery and vein in its branches. It is a blind procedure.

*Local intranasal operations* have been practised but very few of them have any specific role to play. For instance procedures such as turbinectomy and resection of the septum, have been mentioned. While it is indeed possible to improve the nasal airway, these procedures have no role to play in the treatment of the disease itself. Besides, modern approach to intranasal surgery is to avoid as far as possible surgical excision of the nasal mucous membrane. Therefore intranasal structures, is not recommended. I have personally performed this operation only when necessary to perform turbinectomy.

When the nasal septum is deformed I prefer septoplastic procedures rather than resection. Turbinectomy, an intranasal surgical procedure however, is useful and this is submucosal diathermy or electrocoagulation of the inferior turbinate. This procedure was first described by BECK in 1930. I have practiced this with 70% temporary relief. The procedure is especially helpful in those patients where obstruction is the leading symptom. The improvement is temporary and lasts from 6—12 months.

There are however, a number of patients, who are still chronic sufferers of rhinitis and for these surgical treatment in the form of section of the trigeminal nerves supply to the nasal cavity has produced very gratifying results. There are various surgical operations which would achieve the desired result.

#### *Trigeminal neurectomy*

It was first described by ZIEGELMAN and performed by MURRAY FAL-

CONER in 1954. GOLDING WOOD performed this procedure on a few patients in 1956. The procedure is carried out through a vertical incision above the zygoma one inch in front of the external auditory meatus. After splitting the temporalis muscle a burrhole is made to enter the middle cranial fossa, the dura is elevated and the middle meningeal artery is coagulated and cut. The dura is further stripped to reveal the petrous portion of the temporal bone. The greater superficial petrosal nerve is indentified as a shining strand emerging from the hiatus fallopii and running forward deep to the mandibular nerve and sectioned.

b. *Sphenopalatine ganglionectomy*

This includes section of sensory somatic roots as well as the parasympathetic supply. The approach to the sphenopalatine ganglion is transnasal similar to the one used for approaching the vidian nerve.

c. *Vidian nerve section*

This consists of section of predominantly parasympathetic fibres. The procedure can be carried out transantrally through the maxillary sinus, through transpalatal approach or through the nasal septum (transseptal approach).

In this series all patients were operated through the transantral approach. This approach has the advantage that the surgeon works in a familiar area, and the exposure of all the structures in the pterygopalatine fossa make proper identification of the nerve easier. This approach seems certainly more popular and is recommended by various authorities on this subject such as GOLDING WOOD, MONTGOMERY and HIRANANDANI.

## indications and selection of cases for vidian neurectomy

For vidian neurectomy has been recommended not only for rhinitis but also other conditions. The procedure is recommended for :  
1. Chronic rhinorrhea of cholinergic type not responding to conservative treatment. This is by far the most common indication for this procedure.  
2. Chronic nasal obstruction alone as their major symptom and without rhinorrhea. Patients with grossly engorged turbinates and minimal rhinorrhea are considered suitable for this procedure. It must be emphasized that in such cases where medical treatment has failed to produce results and do not lead a normal life because of their illness, are advised for this procedure.

3. Chronic rhinitis where tympanic neurectomy is either not feasible or has failed.

4. Chronic nasal drip.

5. Chronic nasal polyposis, together with thorough removal of the polyps and functional ethmoidectomy.

6. It is primarily an elective operation. There are no absolute contraindications to this operation but severe diabetes, gross cardiac disease, severe renal and other systemic illness are relative contraindications. The procedure is not recommended in the presence of gross nasal or sinus infection.



## Surgical anatomy

Recently our knowledge of the surgical anatomy of pterygopalatine fossa and on most standard textbooks of anatomy which gave a very brief description of this area. Our present knowledge has been greatly widened by the work of HARRIS and GOLDING WOOD. The pterygopalatine fossa is also known as maxillary fossa, sphenopalatine fossa, retramaxillary space and retro-orbital

pyramidal space situated below the apex of the orbit enclosed by the surface of the greater wing and the pterygoid process of the sphenoid

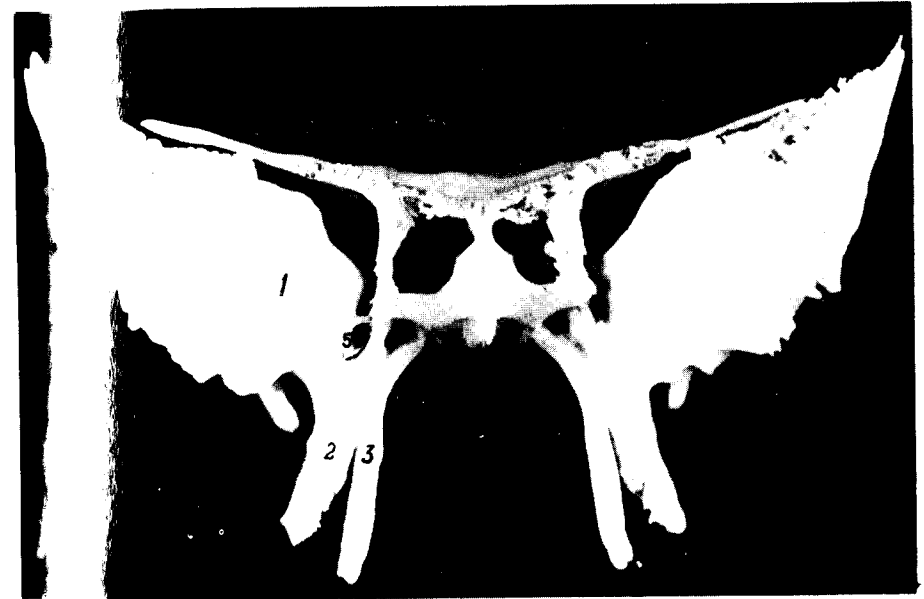
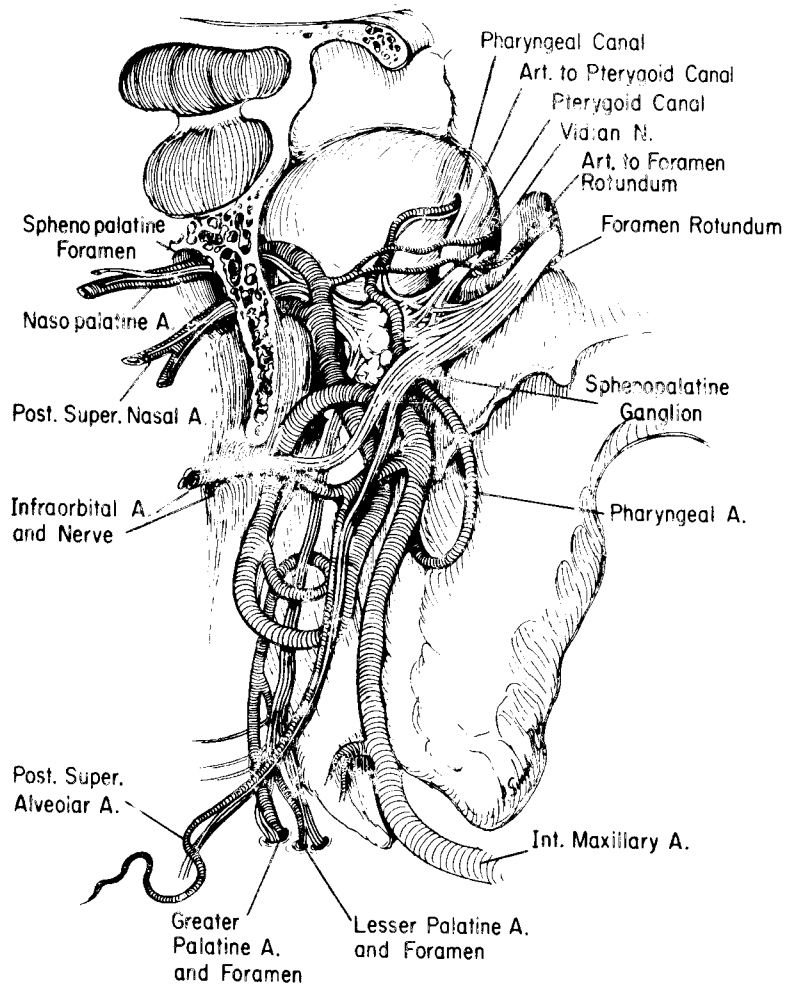


Figure 3.

aspect of the sphenoid bone. The largest portion of the bone is the greater wing (1), which is separated from the body by the inferior orbital fissure. Below the fissure is a vertical bar of bone which separates the foramen rotundum (5) from the pterygoid canal (6). The pharyngeal canal (7) lies more laterally. The relationship between the sphenoidal sinus (4) and the pterygoid canal (6) suggests the relationship between the vidian nerve and the floor of the pterygopalatine fossa. The lateral (2) and medial (3) pterygoid plates have a common origin, i.e. the floor of the pterygopalatine fossa.

From *Otolaryngology* Vol. III Chapt. 39. With kind permission of H. B. Saunders, Publishers.

bone posteriorly, the palatine bone and pterygoid process of the sphenoid medially and the posterior surface of the maxilla and the orbital process of the palatine bone in front. Medially the pterygopalatine fossa is bounded by the vertical plate of the palatine bone as it bridges the gap between maxilla and medial pterygoid lamina.



**Anatomy of the pterygopalatine fossa, based on serial sections of Morgenstein.**

Figure 4.

*Adopted from Otolaryngology Vol. III Chapt. 39. With kind permission of Harper & Row, Publishers.*

It communicates with the infratemporal fossa through the pterygoid canal. The fossa communicates medially with the nasal cavity through the palatine foramen situated at the upper limit of the vertical plate of the palatine bone as it bifurcates into its sphenoidal and orbital processes. The foramen opens into the posterior end of the superior meatus.

The foramen is closed by mucous membrane of the nose, and transmits the maxillary and nerves from the fossa into the nasal cavity. On the posterior wall of the fossa and laterally is an opening in the greater wing of the sphenoid. This is the foramen rotundum which begins as a depression in the floor of the middle meatus and actually becomes a short canal through the greater wing of the sphenoid. It transmits the maxillary nerve. About 10 mm postero-medial and inferior to the foramen rotundum is the opening of the pterygoid canal. It is about the same size in diameter and transmits the vidian nerve. A vertical plate separates these two orifices. More medially about 4 mm from the posterior wall of the pterygoid canal is the opening of the pharyngeal canal which is a sort of semicanal between the body and the greater wing of the sphenoid. It transmits the nerves and vessels of the same name.

Superiorly the fossa is open into the apex of the orbit via the inferior orbital foramen. Inferiorly it is closed by the pyramidal process of the palatine bone which projects laterally, fusing with the maxilla in front and the pterygoid process of the sphenoid behind. The pterygopalatine canal, which transmits the maxillary palatine nerves and artery, is situated inferiorly vertically below the pterygoid canal on a slightly anterior plane.

The anteroposterior dimension of the pterygopalatine fossa is quite variable with the degree of pneumatization of the sinuses and with the variable development of the bones. On an average this depth is approximately 10 mm. This fact is of importance while choosing the method of surgical approach to the fossa.

The contents of the pterygopalatine fossa are :

- 1. Maxillary artery and its branches namely
  - a. Superior superior alveolar artery.
  - b. Infraorbital artery.
  - c. Inferior palatine artery.
  - d. Greater palatine artery, which may be sometimes absent.
  - e. Sphenopalatine artery.
  - f. Pharyngeal artery.

2. Various unnamed branches to the pterygoid canal, the foramen rotundum and soft tissues.

2. The maxillary vein and other tributaries. There is no plexus of veins here, as encountered in the infratemporal fossa over the pterygoid muscles.
3. The maxillary nerve and it's branches.
4. The vidian nerve or the nerve of the pterygoid canal.
5. The sphenopalatine ganglion and it's branches.
6. All the structures are enclosed in a pad of fat, which is loose and can be readily pulled away. Golding Wood has drawn attention to another pad of fat (bichat's) situated close to the posterolateral wall of the maxillary antrum. This fat may encroach the operative field if the posterior antral window is extended too far laterally at operation. It can not be pulled away and any such attempt merely drags it into the pterygopalatine fossa and makes the operation more difficult. Those who have encountered this, know how true this statement is.

The studies of Morgenstein demonstrated clearly the inverted conical shape of the pterygopalatine fossa, (Fig. 5) the base of which is above upon the antero-inferior aspect of the sphenoid bone and the apex is directed downwards into the pterygopalatine canal. These studies also demonstrated the two compartment concept of the fossa. The vessels lie more anteriorly right behind the maxilla and most of the fat is spent around them. The neural structures are deeper, close to the sphenoid and on a relatively higher plane.

The maxillary artery runs a rather tortuous course through the fossa, arching upwards and forwards as it passes medially. It terminates by bifurcating into the sphenopalatine- and descending palatine arteries. Just as it enters the pterygopalatine fossa the maxillary artery gives a small posterior superior alveolar branch that runs downwards and outwards. Soon after, the infraorbital branch arises and runs laterally and upwards to enter the infraorbital canal through the inferior orbital fissure. While in the fossa itself the sphenopalatine artery may divide into its terminal branches namely nasopalatine (to the nasal septum) and posterior nasal artery (to the lateral wall of the nasal cavity). The sphenopalatine artery forms an important anatomic relationship as it crosses the sphenopalatine ganglion superficially in it's course to the sphenopalatine foramen.

The origin of the pharyngeal and vidian arteries which are very small vessels by themselves, is of importance to the surgeon. Usually these vessels arise from the sphenopalatine or posterior nasal arteries but may frequently originate from the maxillary artery itself just as it enters the fossa. In this event either



Figure 5.

*though pterygopalatine fossa (after Morgenstein) showing inverted cone of the fossa. The two compartment concept namely the anterior (containing neural elements) and posterior (containing the vessels) is clearly seen from Otolaryngology Vol. III Chapter 39. With kind permission of H. & W. Publications Maryland U.S.A.*

or both vessels run medially over the surface of the sphenoid bone and reach their destination by passing deep to the nerves.

The maxillary nerve, which is the second division of the trigeminal nerve, leaves the semilunar ganglion in the middle cranial fossa and enters the pterygopalatine fossa through the foramen rotundum. It consists primarily of two nerve bundles each about 2 mm thick. Upon entering the fossa, it gives a distinct thick branch to the sphenopalatine ganglion. Golding Wood has rightly referred to this branch as the sphenopalatine bundle. This nerve passes downwards and medially while the rest of the main trunk runs laterally and slightly upwards before turning forward through the inferior orbital fissure to continue as the infraorbital nerve.

The Sphenopalatine ganglion lies immediately in front of the funnelled opening of the pterygoid canal. It has been described as being fusiform in structure, greyish in colour and is so depicted in most anatomical and otolaryngological texts. However, it is important to know that the ganglion is laterally compressed and is therefore not seen as a distinct fusiform structure through the transantral approach. The vidian nerve enters the posterior surface of the ganglion. As the sphenopalatine bundle is traced medially it bifurcates into a descending palatine nerve and a number of other branches that enter the sphenopalatine foramen. The sphenopalatine ganglion is the distribution centre for sensory fibres from the maxillary nerve reaching it via the sphenopalatine bundle, and autonomic fibres from the vidian nerve. It is a rather diffuse collection of ganglion cells and measures approximately 5 mm in diameter. Its so called branches are distributed through the sphenopalatine nerve, to the mucous membrane of the nasal cavity and its peripheral fibres also accompany the branches of the internal maxillary artery.

The vidian nerve supplies the autonomic root to the sphenopalatine ganglion and contains both sympathetic and parasympathetic fibres. It has been shown that the sympathetic fibres in the vidian nerve are in fact cholinergic in action. This is in keeping with the clinical experience that vidian neurectomy is predominantly parasympathetic in its effects.

The autonomic fibres which are secretomotor to the glands of the nasal mucosa are distributed through the branches of the sphenopalatine ganglion. The secretomotor fibres to the lacrimal gland which also are in the vidian nerve, join the maxillary nerve via the sphenopalatine bundle and pass with the zygomatic branch to the lacrimal nerve.

## Surgical Procedures

Different approaches to vidian nerve have been described.

Transantral approach.

Palatal approach.

Labial approach.

Transantral approach is the one most commonly used. It has the advantage that if the surgeon is more familiar with this area, the exposure is larger than with the other approaches and one works through a relatively clean area provided of course there is no antral infection in which case the operation would not be advisable. Besides, through this approach one can visualize all the structures in the depths of the pterygopalatine fossa. This is essential in my opinion if the vidian nerve is to be avoided.

The patient is placed in supine position with his shoulder slightly elevated 15 degrees and the table tilted antitrendelenburg, 30—40 degrees.

ANESTHESIA

This procedure can be done under local anesthetic as well especially if no indication exists to the use of general anesthetic, I have preferred to use general anesthesia for my patients. This utilizes the use of intravenous and endotracheal techniques. A combination of pentothal sodium, succinyl hydrochloride, nitrous oxide and oxygen supplemented by fluothane, is used. The intratracheal intubation through the nasal root from the opposite nostril is preferred. Before the skin incision in the gingivobuccal sulcus, about 5 cc of 2% lignocaine with 1:100,000 adrenaline is injected.

A skin incision beginning at the root of the canine tooth and terminating at the first molar, and with its concavity towards the alveolus, is made. The soft tissue is elevated along with the mucoperiosteum over the antero-lateral wall of the maxilla. The upper limit of this procedure is the infraorbital foramen. Injury to the infraorbital nerve is carefully avoided. The labial tissues are retracted. A selfretaining retractor is ideal but in the absence of these, the Jackson's retractors can be used.

The anterior wall of the maxillary antrum is then removed with a high speed burr and an as large an opening as possible is made. Once again the infraorbital nerve is respectfully preserved. Damage to the upper alveolars and roots of teeth is avoided. In the absence of a drill, hammer and gouge can be used.

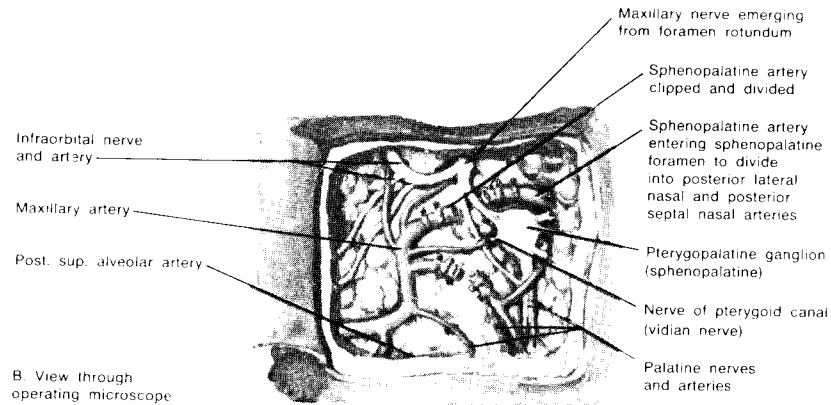


Figure 6

*Contents of the Pterygopalatine fossa as seen through the operating microscope. Adopted from Clinical Symposia CIBA Vol. 26 no. 1 1974. With kind permission of Medical Education Division, CIBA Pharmaceutical Company.*

The cavity of the antrum is inspected and a flap of the mucoperiosteum from the posterior wall of the antrum, based inferiorly or laterally, is elevated. If a small pack soaked in the local anesthetic combination mentioned earlier, is packed for about three minutes in the antrum before elevating the mucoperiosteal flap, there is no significant bleeding.

Again using the drill or the mallet and chisel, a large opening is made in the posterior wall of the antrum. I find the drill sharper, precise and less traumatic. The removal of bone from the posterior superior angle is sometimes difficult and special rongeurs (Kerrison) are useful. Whatever the instrument used at this stage they should not be allowed to penetrate beyond the periosteum on the posterior surface of the bone, since all the important blood vessels and nerves especially the maxillary artery and its branches lie close to it. Most of the bony posterior wall except at the superomedial angle is thin.

The operating microscope with 300 mm objective is now brought into focus and the periosteum is opened and a U-shape flap is made. I find it easier to lift the periosteum with a tiny rightangle hook near the upper margin of the exposure, avoiding carefully the underlying bloodvessels and making a slit incision. An angled elevator of the type such as (Rosen's elevator for lifting the tympano meatal flap) is then passed to elevate the periosteum with one hand and incise with a pair of scissors or a sickle knife, with the other.

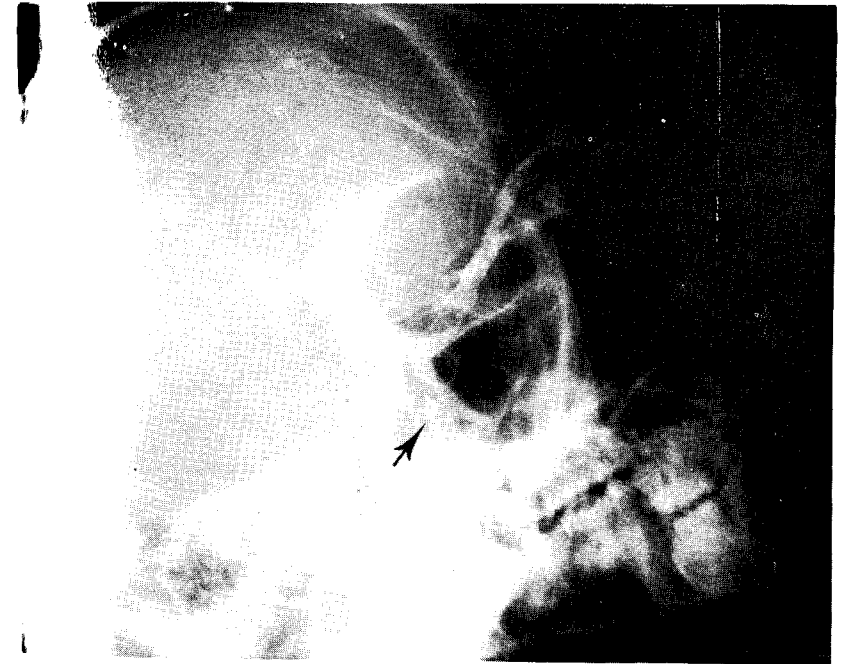


Fig. 7

*Pre Röntgen photograph of Pterygopalatine fossa showing clips on the artery.*

Various micro-surgical instruments for this procedure are described and are available from various companies such as Morgenstein's, Montgomery's and Robbett's. Wood has devised a special hook, clips and applicator for the maxillary artery. It is used over the sphenoid bone and his special diathermy applicator is used in the vidian canal. In practice a set of rightangle stapedectomy hooks is adequate but the clips must be at hand. Mc Dermott selflocking clips are handy.

The next step is to look for the maxillary nerve which lies transversely across the superior surface of the sphenoid above the maxillary artery and slightly at a deeper level. The nerve is followed laterally to its emergence from the foramen rotundum. This is the superior limit of the operation. Immediately after it leaves the foramen rotundum the maxillary nerve gives the sphenopalatine bundle which is

actually the nerve which is traced transversely across the sphenoid. The main continuation of the maxillary nerve into the inferior orbital fissure is not seen in this exposure. As the sphenopalatine bundle is traced medially, the loose fat around it is tethered away. About 1 cm medially and inferiorly is a vertical ridge of bone and close to this is the opening of the vidian canal. To expose the vidian nerve fully the sphenopalatine bundle is picked up on a rightangle hook and rotated forwards and downwards. The vidian nerve can be seen stretched and its canal also clearly visualized. The nerve is now sectioned with a sickle knife.

It may be necessary to divide the sphenopalatine artery before sectioning the vidian nerve as it crosses the sphenopalatine bundle. The artery is coagulated before section. It is not always necessary to divide the maxillary artery, but if it lies in the way of the exposure then this can be done after application of double clips, and before the vidian nerve is cut.

Clips are applied :

- a. right where the artery enters the pterygopalatine fossa i.e. at the lateral limit of the exposure.
- b. a set of clips is placed on the infraorbital artery and another set of clips is placed on the maxillary artery before it's division into the sphenopalatine and descending palatine and sphenopalatine arteries themselves.

The terminal end of the vidian nerve is coagulated with diathermy. Golding Wood recommends application of a probe in the vidian canal. Others use gelfoam, surgical or bonewax to plug the vidian canal. The object of all these procedures is the prevention of regeneration of the nerve endings.

Having accomplished vidian neurectomy the periosteal and mucoperiosteal flaps are replaced.

Morgenstein recommends the creation of a nasoantral window. Golding Wood does not find it necessary. In all the cases operated in Suriname I have not created a nasal antrostomy in these cases. The gingivobuccal incision is then closed with interrupted 3—0 chronic catgut.

## Postoperative care

Patients are prescribed analgesic usually tablets Novalgin 3—4 times a day.

Preparations such as Varidase or Tanderil 3—4 times a day, help to prevent the inevitable swelling of the buccal soft tissues within a week. Antibiotics are provided and continued for one week postoperatively. I have used 200 mgs four times a day for 5 days. The patient is allowed fluid diet for the first 24-hours, thereafter soft diet is permitted. After the third postoperative day patients are allowed to eat normally. They are instructed to rinse their mouth everytime after they have had a meal. They are encouraged early to get up and all patients in this series were up and about next day after the operation.

## COMPLICATIONS

Complications are not usually encountered with this kind of surgery. The following complications have been mentioned by all those who have operated on a large number of patients.

### *Lacrimation.*

There may be a relative dryness of the eye on the operative side for a few days after the operation. However, local goblet and mucous cells are sufficient to give adequate moisture to protect the cornea and conjunctiva. If the dryness cause any problems it can be relieved by artificial tears. BIRMER'S test is a useful measure of the absence of lacrimation. In this series none complained of this symptom postoperatively. Neither did any patients complain of any loss of emotional tearing.

### *Swelling.*

A moderate degree of swelling of the soft tissues of the cheek as a result of traction is inevitable but clears up within a week or two.

*Paresthesia* of the face may follow this procedure and is also due to prolonged traction of the soft tissues of the cheek. It usually lasts from a few days to a few weeks, but clears up by itself and no special treatment is necessary.

### *Blindness.*

Excessive diathermy probe in the pterygoid canal is allowed to penetrate deep, this complication is likely to occur. Golding Wood recommends a special probe for this purpose. His probe has a specially designed shoulder which prevents penetration of the diathermy needle beyond 2—3 mm in the vidian

5. *Infection of the maxillary antrum may occur.*

In the first place this is best avoided by meticulous attention to asepsis, and careful hemostasis. Should postoperative sinusitis occur, it is treated in the usual way with antibiotics and or antral lavage.

6. Secondary hemorrhage has been described and is usually due to secondary infection or if the hemostasis has been inadequate.

7. Persistent swelling and pain on the operative side of the cheek has been described, where a piece of spongostan was left behind in the antrum. Hirandani described a case where he had to operate to remove this material.

8. *Infraorbital neuralgia.*

This is due to overtraction of the infraorbital nerve and can be avoided by not overstretching the soft tissues especially in the region of the infraorbital nerve.

In the small number of patients that I have so far operated except for transient swelling of the soft tissues of the cheek, no other complication has been encountered.

#### ALTERNATIVE APPROACHES

The trans-septal approach to vidian nerve is described by Malcolmson. In this approach a submucous resection of the nasal septum is first performed and the front of the sphenoid sinus is reached. The rostrum is then followed laterally until the pterygoid plates are encountered and at the junction of the pterygoid plates with the body of the sphenoid the pterygoid canal is located. The approach has the singular advantage that both pterygoid canals can be exposed simultaneously by this route. The only structure which sometimes creates a problem in this procedure is the pharyngeal artery.

CHANDRA (1969) described the transpalatal approach. This approach is through a transveere incision between the hard and the soft palate. It gives access to the roof of the nasopharynx. The inferior aspect of the sphenoid bone overlying the pterygoid canal is removed and vidian nerve is destroyed in the pterygoid canal. This approach allows access to the vidian nerve without entering the pterygopalatine fossa.

The transpalatal approach has also been used by AVERBUKH with a slight variation. In his operation the posterior nasal fossa was exposed via the transpalatal route. The thin vertical portion of the palatine bone was then removed and the sphenopalatine ganglion was indentified.

## STELLINGEN

behorend bij het proefschrift  
van R. TIWARI

### I

Chroniche vasomotorische rhinitis kan vergeleken worden met niet-allergische astma bronchiale.

### II

Infra-orbitaire neurectomie bij chronische vasomotorische rhinitis is in fysiologisch opzicht hetzelfde als vagotomie bij ulcus pepticum.

### III

De otolaryngoloog-oor arts is de aangewezen specialist die het eerste geconsulteerd moet worden voor een tumor, die ervan verdacht wordt een metastase van een goetrukkend neoplasm in de hals te zijn.

### IV

De oorzaak van exophthalmus kan in het oor, de neus of de keel zijn.

### V

Elk medisch ziekenhuis moet een stal met proefdieren hebben met het oog op wetenschappelijk onderzoek.

### VI

De chirurg weet wanneer hij niet moet opereren.

### VII

De belang van onze studenten aan de Universiteit van Suriname is het aan te raden een systeem van technisch onderwijs in de Engelse taal in te voeren.

### VIII

De gezondheidszorg verdient in Suriname meer aandacht dan tot nu toe het geval is.

IX

Het is de taak van de Faculteit der Medische wetenschappen aan de Surinaamse bevolking meer voorlichting te geven over het ter beschikking stellen van hun lichaam na hun dood ten behoeve van het medisch onderwijs.

X

Het verdient aanbeveling te onderzoeken waarom diagnose neurolyues praktisch niet meer gesteld wordt in Suriname.

XI

Een betere motivatie voor de prompte aangifte van besmettelijke ziekten bij de artsen in Suriname is noodzakelijk voor de ontwikkeling van de epidemiologie.

XII

Een studie van moderne ontwikkelingen en toekomstige mogelijkheden is niet volledig zonder een grondige kennis van het verleden.

XIII

Het is van belang dat er meer aandacht besteed wordt aan de export van bloemen uit Suriname

PERSONAL OBSERVATIONS

The first vidian neurectomy in Suriname was performed two years ago. It is my policy to treat these patients medically at first on the lines elaborated elsewhere. Where the treatment failed to produce results and the patients had to return on medicine everyday and the patient had been under my observation for a year, I discussed the condition once again with the patient and acquainted him with the possibility of permanent relief by surgery and it was left to the patient to make up his/her mind. In the last two years eleven patients have undergone the unilateral transantral vidian neurectomy. In these patients the shortest duration of symptoms was three years and the longest thirteen years. No major operative complication has been encountered. The small number of patients operated is due to :

1. The fact that mine is a single handed general otolaryngological practice where patients are operated strictly on first come first serve basis except of course in emergencies.

2. The number of available hospital beds is limited and

3. It is so important, the strict criteria of selection of cases for surgery.

In the operated patients I have proceeded to record the intranasal temperature before and after surgery on the operated side of the nose. The results compared slightly with individual patients but generally the intranasal temperature on the operated side was lower.

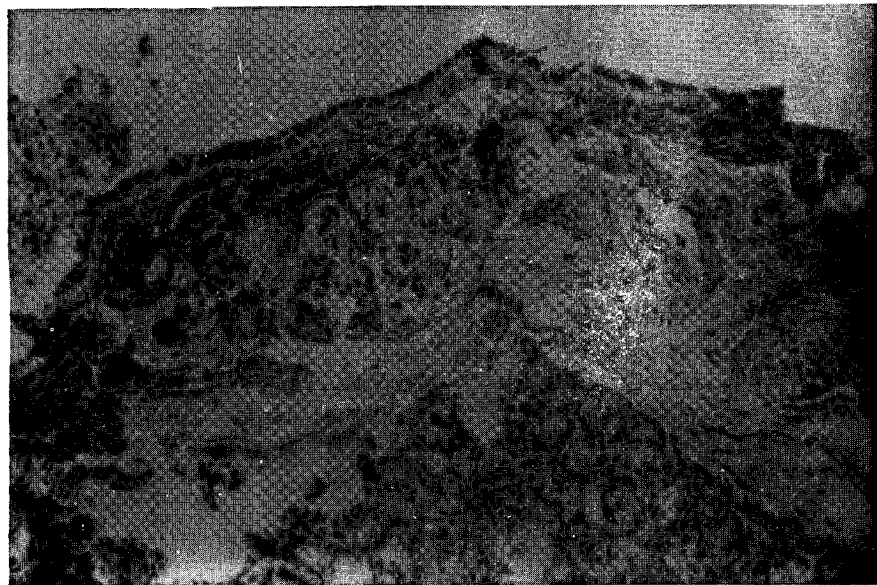
Body Temperature in degree centigrade	Intranasal Temperature on operated side in degree centigrade	Intranasal Temperature on unoperated side in degree centigrade
37.4	35.6	35.8
36.8	35.6	35.8
36.8	36.2	36.2
37.2	35.4	35.8
36.6	36	36
37.2	36.2	36.4
36.6	35.6	35.8
37.2	35.8	36.2

Table 6

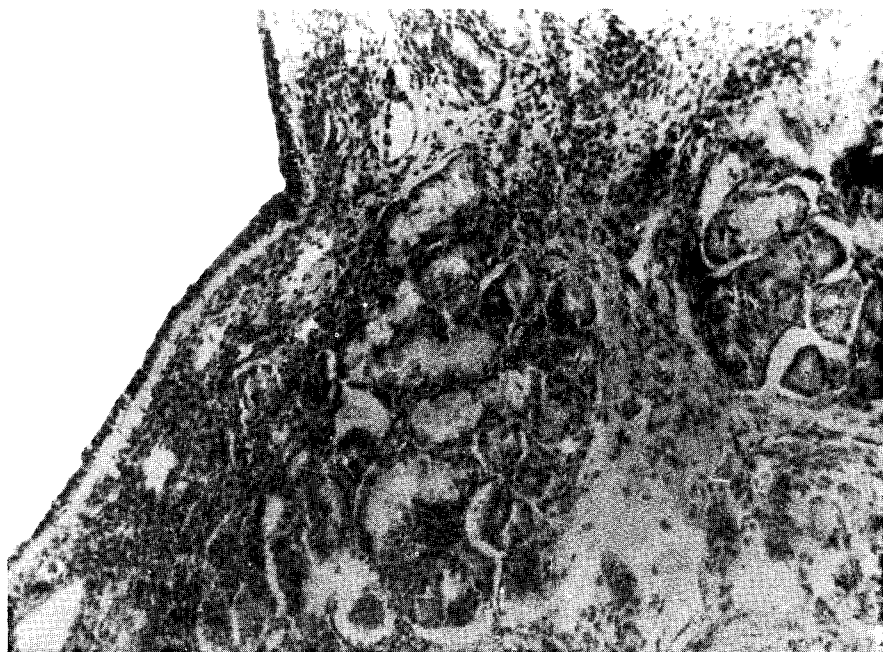
In the first three patients this observation was not recorded.

I have also taken biopsies from both inferior turbinates one centimeter from the anterior end, to compare the histopathological changes on the operated and unoperated side of the nose. It was interesting that no gross difference was visible in the tissues on microscopic examination on either side.





*Histopathology of nasal mucosa on operated (no. 8) and unoperated side (no. 9). Areas of maximum dis-similarity were chosen for this illustration. Fig. 8 shows some fibrosis after operation while fig. 9 on the unoperated side shows slightly more cellularity. Actually in the overall picture there was little different between the two sides.*



## Case reports

A few interesting case histories are presented :

Patient E.H., an eighteen year old young man, was referred by the Neurologist on December 21st, 1976 with complaints of chronic bilatered nasal obstruction, slight watery rhinorrhea and minimal sneezing for the last three years.

He had been seen 18 months ago with history of headaches for which no cause except vasomotor rhinitis was detected. The Neurologist's opinion was sought, but after thorough investigation the Neurologist came to the conclusion that the condition was due to stress. He had also been seen by the Internist and Otolaryngologist. All necessary investigations regarding to his nasal condition such as X-ray sinuses, allergic tests etc. were negative. He was put on a medical therapy of antihistaminics but the relief was not noticeable. On January 15th, right vidian neurectomy was done through the transantral approach. The maxillary artery was not ligated. A full discussion of the condition and the details of the operation preceded. Following surgery the patient has shown marked improvement in his nasal condition and his general state also improved considerably.

Patient R.S., a thirty year old female, who is employed at one of the local hospitals, had been suffering from nasal obstruction slightly more on the right side, sneezing attacks, watery rhinorrhea, anosmia and heaviness of the head for the last five years.

She was first seen on the 17th October, 1975. The clinical picture was suggestive of vasomotor rhinitis and on further investigation she was found to have had positive responses to house dust and tobacco. The sinus X-rays showed marginal thickening of the antral mucosa but on antral lavage no infection was found. She was treated medically and advised to avoid dust and smoke. She herself is a nonsmoker. Two years of medical treatment produced no permanent relief in her symptoms. Therefore after prior discussion on July 15th, 1977, right vidian neurectomy was done. Her postoperative course was eventful and when reviewed after a month she stated that she was 100% better as regards her complaints on the right side and had 80% improvement on the left.

Patient I.B., a twenty two years old young girl, who works in a local departmental store had been suffering from sneezing attacks, watery rhinorrhea, headaches and earaches, since the last few years. Before she was referred for treatment on December 1st, 1976, she had already received considerable medical treatment by her family physician. The clinical diagnosis of vasomotor rhinitis was made. Special investigations revealed no abnormalities but a

general check up showed that she was anaemic with a Hemoglobin of 5,8 and also had ascariasis. She was referred for treatment of these conditions to an Internist and in the meantime received medical treatment for vasomotor rhinitis.

When her general health improved and an o.k. was received from the Internist her condition was reviewed. Despite energetic medical treatment for the nasal condition, there was no change whatsoever. It was therefore decided to proceed with surgical treatment and right vidian neurectomy was done. She had postoperatively, a transient swelling of the cheek, but no other complication was encountered. The maxillary artery was not ligated. She is much happier since surgery and has 75% improvement in her symptoms.

4. Patient S.H., a twenty five year old young lady, was first referred on November 30th, 1976 with the history of nasal obstruction, occasional headaches and rhinorrhea for the last three years. She had some sneezing attacks, but these were not excessive. She had received medical treatment inclusive of antihistaminics over the past three years without relief. On clinical examination and special investigations she was diagnosed as a case of vasomotor rhinitis. The marked swelling of inferior turbinates of her nose prevented satisfactory examination and the examination had to be repeated after treatment with local anesthetic. There was no other intranasal pathology. She was put on a clinical trial of antihistaminics and cortisone but without much relief. On January 3rd, 1977, after prior discussion she was operated and right vidian neurectomy was done. The improvement in her condition that followed surgery, was significant and she has maintained this improvement ever since.

## Summary and conclusions

In the preceding pages an attempt has been made to discuss the incidence, aetiology and pathology of vasomotor rhinitis. Etiological factors with a special reference to the conditions in Suriname have been discussed. The histopathological changes and the treatment medical and surgical have been described and the clinical observations have been recorded.

This study and the facts presented herein must be considered with one fact in mind and it is, that these cases have been seen and studied in general otorhinolaryngological clinics namely at the Academic Hospital Paramaribo and St. Vincent Hospital Paramaribo. This work has been part of a singlehanded practice and it would be fair to draw the following conclusion from this work :

Vasomotor rhinitis is a common malady seen in Suriname and most of our patients present with symptoms of sneezing, watery rhinorrhea, nasal obstructions and other associated symptoms such as headaches which have been mentioned earlier. This is similar to the symptomatology as described by Golding Wood in England and Hiranandani in India, but is in contrast to the observation of most otolaryngologists in the United States where nasal obstruction is the main presenting symptom.

Chronic vasomotor rhinitis is more common in conditions of high humidity and is observed in the rainy seasons in Suriname.

There seems to be a definite higher incidence of chronic vasomotor rhinitis in the people of East-Indian origin (Hindustanians). The mixed races are also prone to it but the pure negroid races and Amer-Indians seems to suffer least.

While many cases are relieved by medical treatment, there is a distinct group of patients who fail to get permanent benefit and where vidian neurectomy is indicated.

When chronic vasomotor rhinitis calls for continued and prolonged medical treatment it is better to consider surgical operation rather than prolonged medical therapy.

Vidian neurectomy is a much more physiological operation than any other intranasal operation for this purpose.

Economically speaking the cost to the patient and or to the Health Service in a patient with chronic vasomotor rhinitis is significantly less when surgery is carried out rather than indefinitely long medical treatment.

8. It would appear that there is a very slight but definite fall in the intranasal temperature on the side of the operation after vidian neurectomy. This may be due to the vascular changes accompanying this operation.
9. From the histopathological studies carried out, it seems that unilateral vidian neurectomy does produce bilateral changes in the nasal mucosa. As to whether the changes especially on the unoperated side are permanent, can only be said after a long term follow up.
- 10 Although it is a general experience that patients with nasal obstruction as the only symptom do not respond well to vidian neurectomy it would appear that patients who have other symptoms such as sneezing, watery rhinorrhea etc. as well, but in whom nasal obstruction is the leading symptom, do respond to this procedure. The two cases included in the preceding case reports are illustrative.

#### SAMENVATTING

In de inleiding wordt aandacht besteed aan het voorkomen van Vasomotor rhinitis in Suriname en aan de verschillende verslagen over deze ziekte afkomstig uit verschillende delen van de wereld. Het doel van dit onderzoek wordt uiteengezet.

In het tweede hoofdstuk wordt een historisch overzicht over deze ziekte gegeven met verwijzing naar de verschillende belangrijke bijdragen van inzicht in de anatomie en de ontwikkeling van de chirurgie van de pterygopalatine fossa.

Het derde hoofdstuk is gewijd aan de methode en het materiaal, die worden gebruikt met betrekking tot dit onderzoek.

In het vierde hoofdstuk, Physiologische consideraties, wordt de nadruk gelegd op de fundamentele physiologische aspecten, die betrekking hebben op een beter inzicht in de functies van de neus in gezondheid en ziekte.

Het vijfde hoofdstuk is gewijd aan de Etiologie en Pathologie van Vasomotor rhinitis met verwijzing naar de omstandigheden in Suriname.

In het zesde hoofdstuk wordt de Evolutie van de Therapeutische Methoden in de behandeling van vasomotor rhinitis besproken.

Hierna worden de indicaties en selectie van gevallen voor vidian neurectomy genoemd.

De chirurgische anatomie van de pterygopalatine fossa wordt hierna gedeeltelijk uiteengezet.

Vershillende chirurgische benaderingen worden in het volgende hoofdstuk genoemd met nadruk op de transantral vidian neurectomie.

Postoperatieve behandeling en complicaties worden hierna vermeld.

Vier illustratieve gevallen en persoonlijke observaties met speciale verwijzing naar de intranasale temperatuur en histopathologische veranderingen aan de opereerde zijde van de neus na unilaterale vidian neurectomy worden besproken.

In het laatste hoofdstuk volgt de slotbeschouwing).

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