

ON THE NASAL CYCLE

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Introduction.

Occasionally a patient comes to see us because he complains about a one-sided obstruction of his nose, which in the course of a few hours will shift to the other side, while the previously-obstructed side opens up. His ability to breathe through his nose remains normal however, and he does not have to resort to mouth breathing. The history and examination being otherwise within normal limits, this patient's attention has been drawn toward a normally occurring mechanism which is the **nasal cycle**.

This normal phenomenon practically always goes on unnoticed, even doctors have been noted to be unaware of its existence in their own noses. This unfamiliarity may explain the often apparently misunderstood implications of the finding, at subsequent routine nasal examinations, of grossly swollen turbinates on different sides. It also may explain why this phenomenon, which was described for the first time in 1889 by Kayser still is waiting for an explanation. After Heetderks in 1927, in recent years only Stoksted and Flottes studied the nasal cycle.

The gross demonstration of the obstruction of one nasal side as compared to the other, is easy, even the layman can do it, by alternately compressing the nares and expiring forcefully, whereby the more obstructed side emits a sound with a higher pitch. For a more sophisticated demonstration of the degree of patency of both nasal sides artificial devices (rhinometry) are indispensable. A discussion of these, among which the one developed by the author, will be found in the following pages, as well as the study of several aspects of the nasal cycle, which still is a not well understood phenomenon.

CHAPTER I.

§ 1. Literature on the nasal cycle.

Kayser (1895) found alternating changes in patency in both nasal cavities, which he called the nasal cycle. He ascribed these to a continuously changing blood volume in the nasal mucosa, which he felt was due to a continuous shifting in tonus between the two body-halves. He stated that the nasal cycle did not influence the total nasal patency. **Lillie** (1923) described how patients often complain of obstruction on alternate sides, but that this is normal, and that there appears to be a cycle of reaction: while the mucous membrane of one nostril is filling to a point approaching obstruction, the other nostril is opening and throwing off its secretion. **Heetderks** (1927), in a rhinoscopic investigation on the influence of various climatic conditions on the nasal mucous membranes, found a definite cycle of reaction in 80% of his test-persons. In general, damp, cold atmosphere brought about the greatest swelling of the turbinates, and dry warm air a little less, while optimal atmospheric conditions (humidity 50—60% temperature 13°—18°) caused cycles of least degree.

The cycle is most active during adolescence which is explained by the hormonal activity at this age.

In older persons a rather superficial cycle was observed. The same nose responded differently at various times under apparently the same conditions. These cycles, including both the phase of filling as well as the phase of emptying on the same side, occurred over periods from fifty minutes to four hours. In general more time was required under favourable conditions, when the cycle was of less degree. Heetderks explains these reactions of the turbinates by the great amount of airconditioning the nose has to perform and also as a reflex mechanism in preventing too free an admission of the air. The average time under all atmospheric conditions for a complete cycle was two and a half hours.

He also examined the effect of sleeping postures, and found that the lower nasal side filled to its maximum size of swelling in an average of twenty five minutes. By turning the patients over, the other side could be made to swell. He concluded that the distribution of the nasal vascular contents must be largely controlled by gravitation.

Stoksted (1952) also found a regular cycle in 80% of his normal test persons. He was the first to study the cycle rhinomanometrically. He was also able to calculate the total nasal resistance and to show experimentally that this figure remained practically constant, notwithstanding the bilaterally changing conchal volumes. This was an important addition to rhinologic examination, the total nasal passage being ultimately the most important. He showed and

stressed the importance of the total resistance along with the cyclical curves in pathological cases (**Stoksted 1953, Stoksted and Nielsen, 1957**). As to the nervous regulation of the cycle Stoksted assumes that the rhythmic cycle is maintained through the peripheral vegetative centers, sphenopalatine and stellate ganglion, with eventual connections through which an increase in tonus in one set of centers may give a decrease in tonus in the other two. The two peripheral centers must be regulated by a central sympathetic center possibly situated in the hypothalamus. By increasing or decreasing the tonus this center will give increased or decreased nasal passage, so that it becomes possible to regulate the total nasal volume according to the temperature and moisture of the external atmosphere, in keeping with the requirement of the organism for intake of oxygen or discharge of carbon dioxide. This is probably a reflex arising in the respiratory centers which, by acting on the central center in the hypothalamus, elicits vasomotor changes in the nasal mucosa.

Ogura and Stoksted (1958) investigated the nasal cycle in pathological noses. When one nasal cavity is anatomically wide and the other one narrow the contribution of the narrow side to the total nasal respiration is small, so that the respiration through the wide side runs parallel to the total nasal respiration. Due to the cyclical changes of the turbinates of the wide nasal cavity the total nasal passage shows the same cyclical fluctuations, and during the swollen phase the patient may complain about obstruction of his wide side, which might seem improbable to the ill informed examiner who sees the patient during the shrunken phase. In the case of an important septum deviation, one side is usually fairly normal and shows regular cyclical changes; the narrower side will show cyclical changes due to the contact which occurs between the turbinates and the septum during the swelling-phase of the turbinates. In vasomotor rhinitis both nasal cavities react synchronously to external influences to an exaggerated degree, so that the cyclical pattern is disturbed, and the total nasal patency is decreased. It is pointed out, that in the normal nose these bilateral synchronous conchal reactions take place to a limited degree and during a relatively short time of adaptation so that the cyclical rhythm is not obscured. In ozena only very limited turbinal swelling was observed, along with a very low total nasal resistance.

Flottes e.a., (1961) studied the nasal cycle in 25 persons aged 20—30 years, having normal noses. Patency measurements were done every 10 minutes.

They found a cycle duration of

2 hours	in 4 persons
3 hours	in 7 ,,
4 hours	in 6 ,,
5 hours	in 3 ,,
6 hours	in 1 ,,
7 hours	in 2 ,,
8 hours	in 2 ,,

So 80% of the testpersons had a cycle duration between 2 and 5 hours, with an average of $3\frac{1}{2}$ hours. 20% had a cycle duration of up to 8 hours. The duration of the cycle seemed to be constant in the same person at repeated examinations. The total patency was not determined; no subjective sensation of nasal obstruction had been experienced by the testpersons. The amplitude

of the cycles in normal noses was identical on both sides and the cycle evolved in a regular and harmonious way. In persons having subacute or chronic sinusitis or an important septum deviation the cycle was irregular; the amplitudes differed on both sides while the maximal amplitudes exceeded those of cycles in normal noses.

Grobler (1966) found a nasal cycle in 5 normal testpersons while in 5 patients "with nasal and chest complaints" no alternating cycle was observed.

Factors influencing the nasal cycle by their action on the nasal mucosa as a whole are manifold and diverse. Some have already been mentioned, such as the influence of posture.

Rundcrantz (1964) studied the influence of increased hydrostatic pressure of the blood in the head when lying down in a head-high position (15° angle with the floor) and a head-low position (also in a 15° angle with the floor). In normal persons he found no change in the total nasal patency but in 17 out of 25 cases having atopic vasomotor rhinitis he found a considerable decrease in total nasal patency in the head-low position which rapidly increased again in the head-high position. The rapidity with which this happens points to vasodilatation, and not to extravascular escape of fluid as the cause for the decreased nasal patency. As an explanation vascular hypotonicity due to parasympathetic preponderance, as is believed to be existing in vasomotor rhinitis, is mentioned. The same mechanism may be responsible for the well-known side-effect of sympatholytic and ganglionblocking agents and of Reserpine, e.g. nasal obstruction especially on lying down (**Connor e.a.**, 1957). In these cases a parasympathetic predominance exists, as is also the case in Horner's syndrome. (**Milloning e.a.**, 1950).

Emotional influences were studied by **Holmes e.a.** (1950) who found vasoconstriction, a sympathetic reaction, in sudden fright situations, and vasodilatation, a parasympathetic reaction, in anxiety situations.

Drettner (1963 a.) beautifully demonstrated the vasoconstriction in the nasal mucosa of a patient upon the entrance of the surgeon in the operating room. An extensive study on the effect of cooling was undertaken by **Drettner** (1961). Cooling of the back, cooling of the feet, cooling of the inspiratory air, general cooling of the skin with or without cooling of the inspiratory air, all were followed by a narrowing of the nasal passage. Local warming of the nasal mucosa by infra-red rays with long wavelengths causes a swelling of the mucosa while shorter wave-lengths cause an initial widening of the nasal passage (nose opening and nose shutting rays, **Hill**, 1933, **v. Dishoeck**, 1935, **Drettner**, 1963 b).

Local skin warming causes an initial widening of the nasal passage followed by mucosal swelling (**Ralston and Kerr**, 1945). General warming causes mucosal swelling (**Drettner**, 1963, b, **Ralston and Kerr**). Hyperventilation causes swelling of the nasal mucous membranes and dyspnoea a widening of the nasal airway (**Tatum**, 1925).

Stoksted (1954, 1960) and **Stoksted and Nielsen** (1957) studied the influence of nasal stenosis on the respiration. According to them an important function of the nose besides its airconditioning function is to offer a resistance to breathing in correlation to the function of the lung. This rheostatic effect influences the exchange of air in the alveoli and contributes to building up a sufficiently great oxygen partial-pressure in the alveoli as well as to en-

courage the diffusion of oxygen in the blood, and the venous reflux to the right chamber of the heart. According to **Kilik** (1935) an increased respiratory resistance gives rise to an increase in the carbon-dioxide content of the blood which, by its action on the respiratory center, effects a deeper and slower breathing rate; at this level the patient can again breathe without difficulty. Beyond a certain resistance this compensation fails. So, according to **Stoksted**, oral breathing will supervene when lung-compensation of a nasal stenosis fails. The ability of the individual patient to compensate for a nasal stenosis is varied. In some patients the type of respiration changes, while others completely or partially lack this ability, and quickly resort to mouth-breathing even in the presence of a very moderate stenosis.

Ogura and Stoksted (1958) found that a patient may have to resort to mouth-breathing while having a normal nose, when, because of a bad pulmonary function, the normal nasal resistance cannot be overcome by the lungs. So normal rhinometric values may give a suspicion in a patient with mouth-breathing of an underlying pulmonary or cardiac deficiency, and also of a nasal neurosis.

Ogura e.a., (1964) made a detailed study on the effect of varying degrees of nasal stenosis on several lungfunctions.

Compression of the jugular vein causes swelling of the homolateral turbinates, which does not occur in sigmoid sinus thrombosis (**v. Dishoeck**, 1957).

Drettner (1963 a) found vasoconstriction in the nasal mucous membranes after the ingestion of either cold or warm food, even after tube-feeding.

Flottes e.a. (1961) studied the action of nasal vasoconstrictor drops. They found that the cycle stopped after the application, to resume its activity when the drug's activity was over. So they found a precise means to test the duration of activity of the several nasal vasoconstrictor agents. They also stated that the so called rebound-effect of nose-drops, a period of nasal stuffiness after the vasoconstriction phase, is nothing but the resumption of cyclical activity. The influence of the sympathetic and parasympathetic innervation will be discussed later.

What actually goes on in the turbinates during the cycle has not received much attention. The matter was discussed by **Drettner**, (1960). A swelling of the mucous membranes may be due to arterial hyperemia, venous engorgement, edema or to combinations of these. In measuring the mucosal temperature simultaneously with the resistance of the nasal air passage he found that the temperature of the mucosa of the swollen side usually is higher than on the shrunken side, but he adds that this does not necessarily mean that the swelling is caused by arterial hyperemia. The nasal mucosa is cooled by the inspiratory air, and the cooling is less on the swollen side. That the turbinal volume changes are due to changes in blood volume, at least at the moment of reversal, seems to be beyond doubt because of the rapidity with which this occurs. In all, clarity does not exist regarding this essential point.

§ 2. Anatomy.

The nasal cavity proper is covered by its mucous membrane, consisting of respiratory epithelium, e.g. columnar ciliated epithelium. The nasal cavity is vascularised for the greatest part by the branches of the sphenopalatine artery, which run close to the periosteum or perichondrium. From here

arteriolae branch off at right angles towards the mucosa. In the submucosa periglandular and mucosal capillary networks branch off, which collect into venules which empty into the veins running in the periosteal layer. **In the turbinates** quite specialized structures are encountered, pseudo-erectile tissue or swell-bodies, where large sinusoids have muscular walls.

Wustrow (1959) studied the turbinates in embryo's and describes how in the last fetal months the cavernous tissue is already discernible, and that post-natally the swell-bodies proper develop.

Their classical localisation is according to **Zuckerkindl** (1885): along the inferior-interior side of the inferior turbinate from head to tail, along the underside of the middle turbinate from head to tail, on the posterior part of the superior turbinate, and on the tuberculum septi. The latter swell body was described in more detail by **Wustrow** (1951) who states that its extent depends on the size of the inferior and middle turbinates, and that in extreme states of swelling the lateral and medial walls of the nasal cavity fit exactly into each other. Sphincters are present at many points in this vascular network, in the arteriolae and at the in- and outlets of the sinusoids. Furthermore, direct arterio-venous anastomoses are present, so that a great variety of vascular reactions is possible.

Negus (1958) gives four possible modes of reaction:

1. Hyperemia, as a result of arteriolar dilatation, with an increase of the mucosal temperature and a swelling of the cavernous spaces, in order to warm and humidify cold dry air.
2. Ischemia, as a result of arteriolar constriction and shrinkage of the cavernous spaces, with a decrease of the mucosal temperature, in order to respire warm moist air.
3. Swelling, with a decrease in mucosal temperature, and congestion of the cavernous spaces and constriction of the arteriolae, in order to humidify warm air.
4. Increased mucosal temperature with arteriolar dilatation and shrinking of the cavernous spaces, in order to warm moist cold air.

The sensory innervation of the nasal cavity is supplied by the trigeminal nerve. Its nuclei in the pons are connected with the vasomotor centers, and the motor-nuclei of the nn. III, IV, VII, IX, X and XII, thus constituting many possible reflex-arcs (**Mitchell**, 1954).

The vasomotor regulation is carried out by the sympathetic and parasympathetic systems. The sympathetic fibers arise in the first two thoracic segments of the spinal cord, they synapse in the superior cervical ganglion, and reach the nose via the internal carotid plexus and the n. petrosus profundus through the sphenopalatine ganglion. The parasympathetic fibers arise in the nucleus lacrymo-nasalis and reach the nose via the intermedial nerve portion of the facial nerve, the n. petrosus superficialis maior, the vidian nerve and the sphenopalatine ganglion where they synapse, and then are further distributed to the nasal cavity (**Malcolmson**, 1959). The exact termination of the nerve fibers is uncertain. (**Naumann**, 1961).

Terracol (1958) and **Temesrekasi** (1959) described nervous structures in the nasal mucosa to which a receptor-action may be ascribed.

The erectile vessels have a very rich innervation, to such a degree that it seems that each smooth muscle fiber receives its own innervation (**Arduin** and **Maillet**, 1964). **Dahlström**, (1965) could demonstrate a very rich sympathetic innervation of the wide veins of the erectile tissue, in contrast to the sparse adrenergic innervation in most other tissues. She concludes that this unusually high developed innervation apparatus must be of great importance in regulating the blood flow. So the nose, and especially the turbinate apparatus, appears as an organ having an abundant and intricate blood-supply as well as nervous supply.

§ 3 RHINOMANOMETRY

Determination of nasal patency.

Terminology.

P_{ph} = **nasopharynx pressure** = the pressure occurring in the nasopharynx by breathing through the nose. This pressure depends upon the respiratory Volume (V), the Time (T), and the Resistance (R) according to the formula $R = \frac{P \times T}{V}$

(**Zwaardemaker**) and so, when T and V are constant c.q. known, upon the degree of patency of the nose.

Anterior rhinometry

The nasopharynx pressure occurring by breathing through one nasal cavity can be measured through the other nasal cavity by connecting the latter via a tube to a manometer, so that this nasal cavity becomes a closed system with the manometer, and an elongation so to speak, of the tube from the manometer, now reaching down to the nasopharynx.

Posterior rhinometry.

The nasopharynx pressure may also be measured through the mouth by way of a tube, which is tightly enclosed by the lips, reaching the pharynx. In this way it is possible to measure the nasopharynx-pressure created by breathing through the whole nose.

P_n = **prenasal pressure** = the pressure occurring at the naris c.q. ostium internum by breathing through the nose. In expiration P_n is smaller than P_{ph} , and in inspiration P_n is higher than P_{ph} . P_n can be measured by way of a side-tube from the **nozzle** = nose piece which connects the naris with the tube to the manometer.

$$\text{Nasal pressure-drop} = \Delta P = \left| P_n - P_{ph} \right|$$

As the literature abounds with methods to measure the patency of the nose, and practically every rhinologist uses his own, it will be clear that the ideal method has not yet been found, but at the same time that an obvious need exists to be more or less objectively informed about the actual patency of the

nasal airway, either for physiological purposes, or for the evaluation of complaints about nasal obstruction and of the pre- and postoperative state of the nose.

The following description is an attempt to classify different methods.

A. Direct study of intranasal-volume.

1. **Inspection** (anterior rhinoscopy). The state of swelling of the turbinates is noted. (**Heetderks**, 1927). **Holmes** e.a. (1950) gave five stages in the evolution from the wide open to the obstructed nasal cavity.

By inspection only the gross changes in turbinal volume can be determined. We found that in the wide nose, with constricted turbinates, the method is quite exact, because even small volume changes can be noted against the background of a practically always present small septal irregularity.

2. **Plethysmography**. The nose is converted into a closed chamber (**Tschalusow**, 1913) filled with saline (**Aviado**, 1959), or with an inflated balloon (**v. Dischoeck**, 1957) and the volume changes of the nasal cavity are transmitted to a measuring device.

B. Indirect study of intranasal volume.

(= Rhinometry)

I. Indirect rhinometry.

Various sequelae of a certain degree of patency of the nose are estimated.

a. Hygrometric methods.

Expiration through both nares onto a cold mirror gives rise to two **breathing spots** the sizes of which depend on the amount of vapour being exhaled through the nares, which in turn is dependent on the respective patencies (**Zwaardemaker**, 1894 and modifications). The size of the breathing spots depends on many more parameters, so that it can only be used in a qualitative sense. As such it is very simple and useful and we have used it in children, and during sleep.

b. Varia.

1. **Sound-test** (**Bruch**, 1901). The pitch of the sound caused by forced expiration through the nostrils changes with the patency. It is still often used routinely as a quick orientation.

II. Direct rhinometry.

In direct rhinometry in the formula $R = c \frac{P \times T}{V}$, two parameters can be made known or are constant, so that measuring the third one gives a direct relation to the resistance.

a. **T measured**, P and V known. **Kayser** (1895) measured the time needed to force a known volume of air through the nose at a given pressure.

b. **V measured**, T and P known. **Jacobson** (1891) used a kind of spirometer out of which an amount of water is forced, in a given time and at a given pressure, during expiration, which corresponds to the volume of air.

c. **P measured**, T and V constant or known. This is the usual procedure.

1. V and T constant.
(= rhinomanometry).

In rhinomanometry it is assumed that when basal conditions prevail during the examination and the patient is at rest, V and T remain constant. Usually P_{ph} is measured anteriorly with a water manometer. More modern is to register P_{ph} electronically so that a PT curve is obtained in which P forms

the absciss and T the ordinate (**Stoksted**, 1953; **Cottle**, 1963).

Stoksted elaborated on this, and had R computed electronically. The problem in rhinomanometry is to keep V constant. In a well informed testperson the respirations can be made fairly constant by making the same breathing excursions with the respiratory muscles and the chest-wall (proprioceptive memory). This is possible to a certain degree when both nasal cavities do not differ greatly in patency. But rhinomanometry fails especially when one nasal side is greatly obstructed, because here it is simply impossible to breathe the same volume as through the wider side, so quantitative comparison becomes impossible.

To ascribe far reaching conclusions to the breathing pattern of a PT curve from an obstructed nasal cavity seems illogical, because a patient breathes through the unobstructed side (**Ogura** and **Stoksted**, 1958; **v. Dishoeck**, 1965) and in bilateral obstruction through the mouth (**Uddströmer**, 1940; **Stoksted**, 1954).

2. V and T known

(= rhino-rheo-manometry).

Here P is measured at a known flow ($PE\Omega =$ to flow) (= V/T), so that all the parameters can be known exactly, and all determinations become reproducible and comparable.

2a. **Passive rhino-rheo-manometry.**

Here the airflow is caused artificially, the patient does not breathe himself. — insufflation, anteriorly (**v. Dishoeck**, 1957; **Malcomson**, 1959). The air is insufflated into one naris; the soft palate must be flaccid and the patient must stop breathing. It is done either with the mouth open and the other nostril closed, or with the mouth closed, so that the air leaves by way of the other nostril. Thus the inflow-resistance of one side and the outflow-resistance of the other side are measured. P_n is measured via a side-tube from the nose piece.

— insufflation, posteriorly (**Seebohm** and **Hamilton**, 1958). Two tubes are introduced in the mouth, one to insufflate a known flow, the other to measure P_{ph} .

— aspiration, posteriorly (**Scheideler**, 1940).

2b. **Active rhino-rheo-manometry.**

Here the patient breathes himself at a known flow rate.

2.b. 1. Measuring P_{ph} .

- **Zwaardemaker's** rhinodromometer (1909). In a glass tube, connected to the nasal side to be measured, a small aluminum plate is hung between two coil-springs. The respiratory air can pass along the plate, and moves it in proportion to the flow rate. P_{ph} is measured through the other nostril.
- **Semarak (1958)** used as a flowmeter a large number of tubes of small internal diameter, so that within the flowmeter the flow regime was always laminar. The flow is measured, after calibration, proportionate to the pressure drop along the tubes. P_{ph} is measured via the other nostril. Flow and pressure are registered photo-kymographically.

2.b. 2. Measuring ΔP

- **Cottle (1957)** introduced this principle. The flow is measured via a rotatory flow meter, which is connected to the nasal side to be measured. P_n is transmitted via a side tube near the nozzle to one end of an U-type oilmanometer. P_{ph} is led to the other end of the manometer via the other nostril. The disadvantage of the actual apparatus is its very high internal resistance which is very often higher than that of a greatly obstructed nasal cavity (**Spoor, 1965**).
- **Guillerm (1961) a.o.**
The flowmeter consists of a tube which is partly closed by a diaphragm. The flow-pressure is led via a side tube to a marey-capsule bearing a long needle which directly indicates the flow on an calibrated scale. ΔP is measured by means of two marey-capsules enclosing a common membrane. P_n is led to one side of the membrane via a side tube near the nostril of the side to be measured; P_{ph} is led to the other side of the membrane via the other nostril. The differential pressure is indicated on a calibrated scale by a long needle which is borne by the membrane. A gadget the importance of which is stressed is the triple **blocking switch**, which obviates the need for an intricate electronic registering device (**Stoksted, 1953; Semarak, 1958; Drettner, 1961**) which is used because it is very difficult to keep the respiratory flow constant, so that normally oscillations of the flow meter and the differential manometer are observed. The blocking switch can be turned at the exact moment the desired flow is reached, and then the connections between the circuit and the capsules are interrupted simultaneously so that the needles also stop at the position they occupied at the moment the switch was turned, ΔP can now be read at leisure.
- **Drettner (1961)** who was interested in the patency of the nose as a whole, used a nasal mask to allow for free breathing through both nostrils. The flow was determined by measuring the pressure-drop across a plate orifice situated in an open tube leading from the mask, and through which respiration takes place. P_n is transmitted from the mask via a tube, and P_{ph} via a tube in the mouth, both to a differential manometer.

— **Guillerm a.o.** (1967) also developed a method utilizing a nasal mask, about similar to Drettner's method.

Generally speaking a nasal mask is ideal for rhinometric purposes because no manipulations within the nose are necessary, and there are no problems with nozzles, which always have to be used with diligence and intelligence. Besides the need to have a mask which fits to every face, the difficulty in using a mask is the fact that, in order to measure the nasopharynx-pressure, an oral tube is necessary and for its functioning properly, the palate must be kept flaccid, and this is sometimes hard to achieve.

Yet for practical use the nasal mask is to become the method of choice, because it is easier to use in the average patient.

— **Spoor** (1965). Flow- and ΔP curves are obtained in a way resembling Drettner's method, only nozzles are used. Moreover the **nasal conductivity** can be read directly from a separate tracing. Spoor defines nasal conductivity as the discharged flow in cc per second at a ΔP of 1 cm. of water; it is computed electronically.

This is a very valuable addition because now the nasal patency can be obtained at any prevailing V and ΔP . The apparatus is intricate and expensive.

Keuning (1963). Author's method.

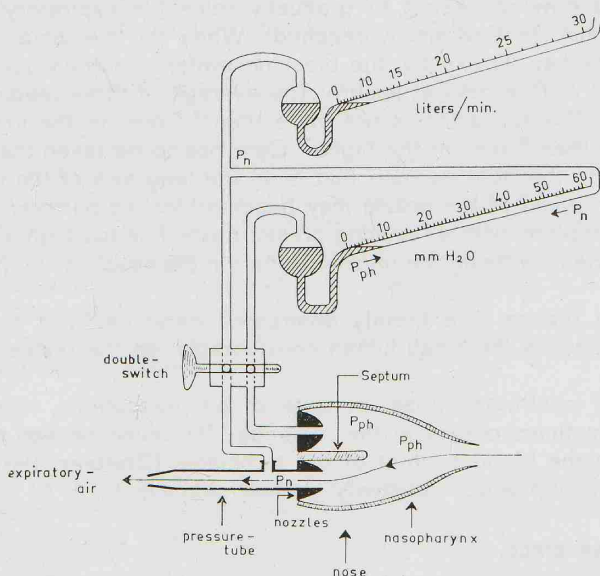


Figure 1. Author's rhinorheomanometer, schematically. The nasal cavity to be measured is connected via a nozzle, to a pressure tube. P_n is transmitted via a side tube to one end of an oblique water manometer, P_{ph} via the other nostril to the other end, so that ΔP ($P_{ph} - P_n$) can be read on the manometer. P_n serves also as the flow-pressure, and is led to another oblique water manometer, which after calibration serves as a flow meter. The double switch is turned when a desired flow is reached, the menisci in the flow meter and differential manometer then stop instantly, so that the pressure at the desired flow can be read at the precise moment and at leisure.

A copper tube, length $9\frac{1}{2}$ cm., internal diameter 11 mm., with a smaller open tube at the end, length 5 cm., internal diameter $4\frac{1}{2}$ mm., serves as a flow meter. The flow pressure caused by expiring through the tube, is led via a side tube to an oblique watermanometer, with a scale reading in L/min. ("flow-manometer"). At the same time this side tube transmits P_n to one end

of another oblique watermanometer. P_{ph} is led to the other end of the latter manometer, thus enabling to read ΔP in mm. H_2O ("pressure-manometer"). A double **blocking switch** adapted from **Guillerm** (1961) is interposed in the circuit. Perspex nozzles fit onto the tubes for introduction into the nares. A concomitant advantage of using two nozzles is the fact that the nostril through which the flow occurs is not distorted by having one nozzle in the other nostril (**Grobler**, 1966).

Calibration of the "flow-manometer" was done by leading known flows of up to 30 L/min. through the pressure tube and marking the menisci corresponding with certain flows. Calibration of the "pressure-manometer" was done by making a parallel connection with an upright watermanometer.

The actual measurement is carried out as follows: a suitable nozzle is selected for each nostril, and mounted on to the tubes. The nozzles are lubricated with a little vaseline to prevent leaking, and to avoid irritation of the skin after repeated introductions, and introduced into the vestibule.

The testee is now requested to gradually raise his expiratory flow up to around 20 L/min. (a flow easily reached). When the meniscus in the flow-manometer reaches 20 exactly, the blocking switch is turned quickly and the corresponding ΔP is read at leisure. The average of three readings is taken as the result. The spreading is not more than $\frac{1}{2}$ mm. in the lower regions, and not more than 2 mm. in the higher. Care has to be taken that the nozzle is introduced in the right manner, that is, in the long axis of the nasal cavity, otherwise the lumen of the nozzle may become blocked partially by the nose wings or the septum, with a resulting pressure which is too high. (The partially occluded nozzle now being the narrowest part in the nasal cavity. (**v. Dishoeck**, 1957).

For the same reason in a greatly obstructed nasal cavity a small drop of mucous may narrow the small lumen considerably, so the testee has to sniff this out.

In the greatly obstructed nose, in spite of all precautions, relatively large spreading sometimes occurs in the readings. This must be due to the small fluctuations in the blood content of the turbinates (**Drettner**, 1961) which for the same reason produce relatively marked changes in ΔP .

§ 4. The nose piece.

An essential part in the rhinomanometric examination is the nose-piece or nozzle, which is the snugly-fitting connection between the tubing of the manometer and the nose. A brief discussion of the structure of the nasal vestibule is necessary, because it has a direct bearing on rhinometric methodology. As described by **Cottle** (1960) the frame work of the vestibule is formed by the lower lateral or lobular cartilage, the cartilaginous and membranous septum and the upper lateral or roof-cartilage. The lower lateral cartilages later-

ally and medially encircle the naris and thus uphold the lobule. The cartilaginous septum stands in the midline. The upper lateral cartilages form the roof of the vestibule; they are attached cephalically underneath the nasal bones, medially in their cephalic two thirds they are cartilaginously fused with the septal dorsum, while in the caudal third the attachment to the septum is fibrous, allowing for a certain amount of movement, and the formation of a valve-mechanism (Mink, 1920). The caudal ends of the upper lateral cartilages extend into the vestibule, forming the lateral wall of the ostium internum. The caudal tip extends underneath the lower lateral cartilage from which it is suspended by a dense aponeurosis.

Thus pressure exerted by a nozzle upon the naris will diminish the valve-area because the upper lateral cartilage will be inverted by the pressure of the overriding lower lateral cartilage. This will result in a measured nasal resistance which is higher than it actually is.

The most commonly used nozzle is the nasal olive, which has a round blunt end, which can be applied to, or slightly introduced into the naris. Always a compromise has to be sought between a too light contact with the naris (causing leakage) and too much pressure against the naris (causing distortion). It would seem more logical to use a nozzle which is more adapted to the oval shape of the naris, because in that way a better contact with less pressure may be obtained. Therefore we use oval - shaped nozzles, tapering slightly towards the end, the lumina of which are only slightly wider than the ostium internum. For noses of different dimensions nozzles of different sizes are available.

The lumen of the nozzle transmitting the nasopharyngeal pressure is of no importance, but the lumen of the second nozzle, as used in rhino-rheo-mano-metric determinations is of great importance especially in an anatomically or cyclically wide nose. As was shown by Van Dishoeck (1957) the resistance of the nose is caused in its narrowest area, which in a wide nose is the ostium internum. When a nozzle having a lumen of considerably smaller dimensions than that of the ostium internum is used a considerable resistance is measured, which is only caused by the nozzle (according to Spoor, 1965 up to a conductivity of 60, which corresponds to a greatly blocked nose).

Two types of nozzles have to be distinguished, because they serve different purposes. (The actual difference is only in size).

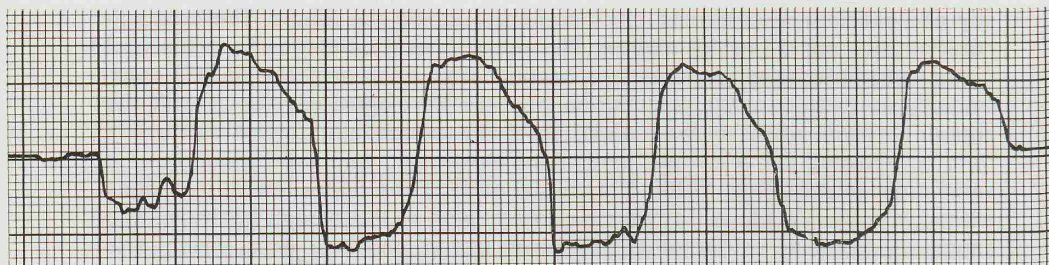
a. The naris nozzle.

This is applied onto the naris in rhino-rheo-mano-metric determinations of the resistance of one whole nasal half (vestibule + nasal cavity). We use an oval-shaped nozzle which is slightly larger than the naris.

b. The vestibule nozzle.

This nozzle is slightly smaller than the naris, and is introduced up to the ostium for rhinorheometric determinations of the resistance in the nasal cavity only (e.g. cycle-determinations).

The differences that are found in using the different nozzles are illustrated by Fig. 2. The curves are nasopharyngeal pressures, as measured through



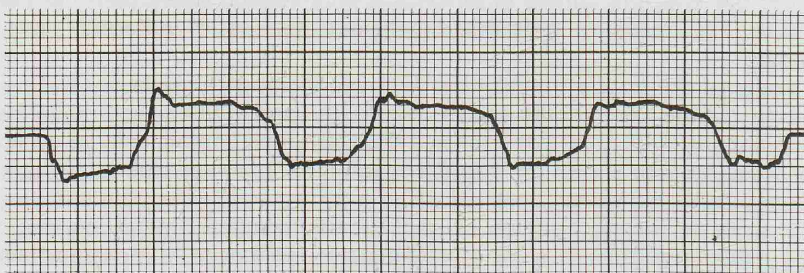
A

free breathing.



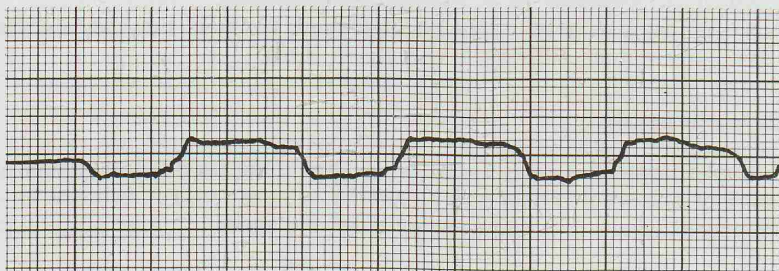
B

naris nozzle.



C

vestibule nozzle.



D

speculum-test.

Figure 2. a. Free breathing. b. Breathing through naris nozzle. There exists only a slight difference between a and b. c. Breathing through vestibule nozzle. d. Breathing through spread speculum (speculum-test, **Van Dishoeck**).

The difference between c and d exists because the speculum dilates the ostium internum slightly. The difference between a, b and c, d exists, because in c and d the vestibule has been eliminated.

one nostril, and caused by breathing through the nasal side to be measured, which was in the cyclically decongested phase.

Van Dishoeck (1957) uses the speculum-test, to differentiate between a turbinal resistance and a vestibular (ostial) resistance. When after introduction of the speculum the resistance does not decrease, the resistance of the nasal cavity is due to turbinal swelling; when it does decrease, the resistance is due to the vestibular (ostial) resistance.

As follows from **v. Dishoeck's** observations and experiments on cadaver material and with the artificial nose, the upper limit of patency of the nasal cavity which it is possible to measure rhinorheomanometrically is when the nasal cavity has a hydraulic diameter not exceeding the diameter of the ostium internum c.q. that of the nozzle.

§ 5. Calculation of total nasal patency.

Stoksted (1957) stressed the importance and demonstrated the clinical value of the total nasal resistance in correlation with the resistance of the left and right side, and he demonstrated specifically that in the normal nose the total resistance remains constant in the face of the everchanging bilateral resistances. **Spoor** (1965 b) states that the total conductivity can be calculated simply as the sum of the left and right conductivities.

We express the total nasal patency as the pressure-drop ΔP which would occur if the flow V would pass through both nasal sides simultaneously, and is calculated from the following formula:

$$P_{\text{tot}} = \frac{P_R}{\left(\sqrt{\frac{P_R}{P_L}} + 1 \right)^2} \quad \text{or} \quad \frac{P_L}{\left(\sqrt{\frac{P_L}{P_R}} + 1 \right)^2}$$

which has been arrived at as follows:

R side	cross-section	C_R
	flow	V
	pressure-drop	P_R
L side	cross-section	C_L
	flow	V
	pressure-drop	P_L
R + L side	cross-section	$C_{\text{tot}} = C_R + C_L$ (the total cross-section).
	flow	V
	pressure-drop	P_{tot} (the total pressure-drop, over the nose as a whole).

At the flow rates used, the flow-regime always is turbulent (Spoor, 1965), so according to Venturi's law for turbulent regimes

$$P = 10^{-6} \frac{V^2}{C^2}, \text{ and } P_L = 10^{-6} \frac{V^2}{C_L^2} \quad P_R = 10^{-6} \frac{V^2}{C_R^2}$$

$$\text{So: } \frac{P_R}{P_L} = \frac{C_L^2}{C_R^2}$$

$$\text{and } \frac{P_R}{P_{\text{tot}}} = \frac{C_{\text{tot}}^2}{C_R^2} = \frac{(C_R + C_L)^2}{C_R^2} \quad \dots$$

$$\begin{aligned} \text{So: } P_{\text{tot}} &= \frac{P_R C_R^2}{(C_R + C_L)^2} = \frac{P_R C_R^2}{C_R^2 + 2C_R C_L + C_L^2} = \left(: C_R^2 \right) \\ &= \frac{P_R}{1 + 2\frac{C_L}{C_R} + \frac{C_L^2}{C_R^2}} \\ &= \frac{P_R}{\left(1 + \frac{C_L}{C_R}\right)^2} \\ &= \frac{P_R}{\left(1 + \sqrt{\frac{P_R}{P_L}}\right)^2} \\ &= \frac{P_R}{\left(\sqrt{\frac{P_R}{P_L}} + 1\right)^2} \end{aligned}$$

As can be seen from the formula the more P_L increases, the more P_{tot} approximates P_R .

CHAPTER II.

Own investigations on the Nasal Cycle.

§ 1. Minor fluctuations.

Drettner (1961) described spontaneous and relatively regular variations of the blood flow in the nasal mucosa with a time interval of between $\frac{1}{2}$ and 2 minutes. In order to investigate whether small fluctuations in nasal patency occurred we did patency determinations twice every minute.

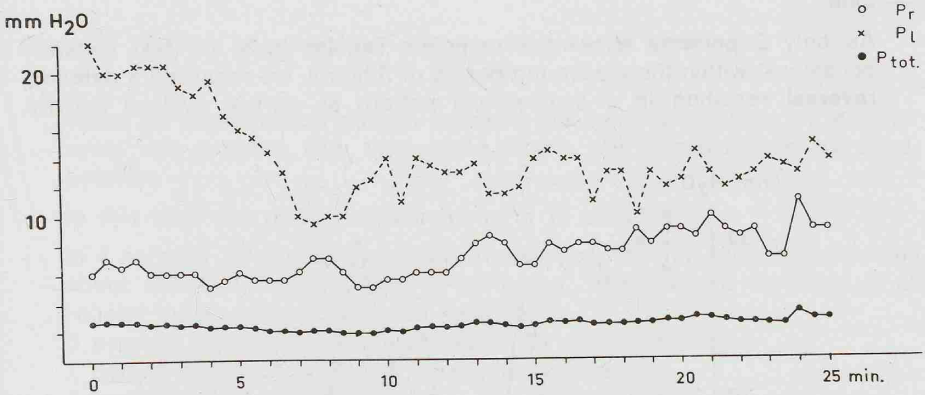


Figure 3. Minor patency-fluctuations. P_R and P_1 determined with the author's rhinomanometer; P_{tot} computed from the foregoing formula.

It appears that small fluctuations take place, especially on the more swollen side at about every 1—3 minutes. They tend to be more or less antagonistic in phase, and therefore practically do not alter the total patency. The cyclical pattern can be seen to continue independent of these smaller fluctuations. As these fluctuations are greater than the measuring error, it seems likely that they are due to spontaneous turbinal volume changes caused by changes in blood flow as described by **Drettner**.

§ 2. Normal nasal cycle.

According to **Heetderks** (1927), **Stoksted** (1953) and **Flottes** (1961) 80% of their cases showed an average cycle-length of about 3 hours. In the remaining 20% **Flottes** found cycle lengths of up to 8 hours. **Heetderks** states that in

about 20% of the cases there was fluctuation without a cycle of reaction, that is, one side of the nose was swelling and emptying without the opposite phase on the other side. Heetderks (1927) states that in the same individual the cycle was not the same under apparently the same conditions, while Flottes (1961) found the cycle-lengths about the same at repeated examinations. We did cycle determination in 17 test-persons having nasal cavities within normal morphological limits and no history of allergy or recurring sinusitis. They were all males and in their twenties. They were chosen, because according to Heetderks in this agegroup active turbinal movements may be expected. This we wanted for further experiments, so that these would not be too time-consuming. After a period of acclimatization of half an hour, we tested during 3 hours, a measurement was done every 15 minutes. We did cycle determinations twice in all testpersons, and three times in a few cases, on separate occasions. Examples of a normal and a long cycle are found in Fig. 4 and 5.

Results.

— time.

As only 2 persons showed a complete regular cycle on two separate occasions, within the measuring period of 3 hours, we regarded a **patency-reversal** resulting in a symmetrical pattern as normal cyclical activity.

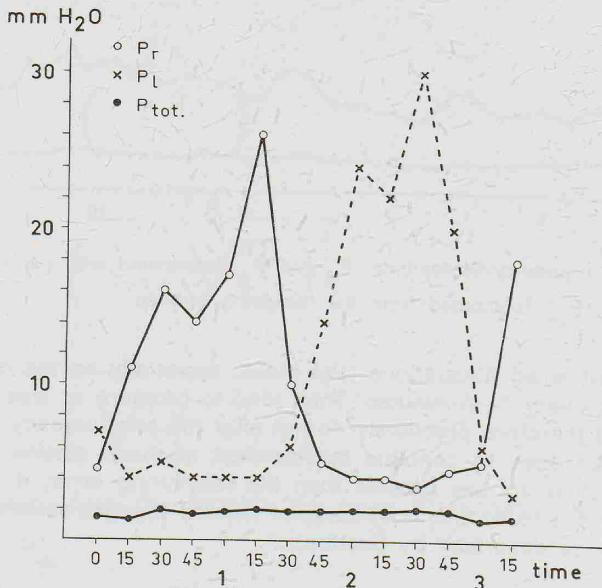


Figure 4. Example of a normal nasal cycle. The phases of turbinal swelling and constriction last about the same. The amplitudes in the swelling phase are about the same on both sides. The total patency, as expressed by P_{tot} remains practically the same throughout the cycle.

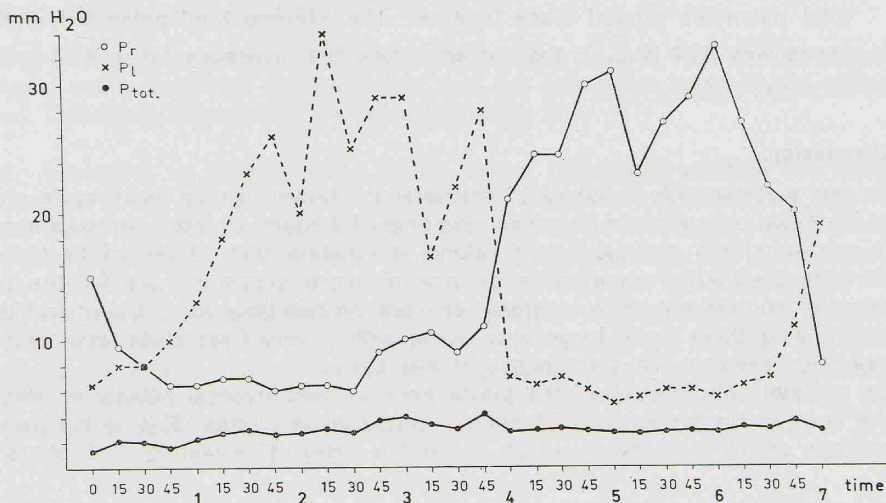


Figure 5. Example of a long, but otherwise fairly normal nasal cycle.

When an asymmetrical pattern followed the patency-reversal, an irregular cycle was present. The total cycle length was approximated by extrapolation.

In this way, out of 37 determinations in 17 persons:

In 7 persons 15 regular cycles were observed; the individual duration was about constant, ranging from 2—7 hours. The average duration of all regular cycles was 4 hours 20 minutes.

6 persons had no patency reversal in 12 determinations.

1 person had 2 regular and 1 irregular cycles.

1 person had 2 irregular cycles.

1 person had 2 irregular cycles and once no reversal.

1 person had 1 irregular cycle and once no reversal.

So out of 37 determinations came 17 regular cycles, 6 irregular cycles and 14 times no patency reversal.

— Amplitudes.

The amplitude of the curves, whether they showed nice cyclical movements, reversals or no reversals was generally remarkably constant for the same person.

Among the testpersons the maximum amplitudes ranged from around 25 mm H₂O at V₂₀ to around 80 mm H₂O at V₂₀.

— Total patency.

The total patency was practically constant in all cases, whether they showed nice cyclical movements, reversals or no reversals. The individual

total patencies ranged close together. The average total patency for all cases was 3.24 (P_{tot}^{20}); the extreme individual averages being 4.82 and 2.12 mm H₂O.

Discussion.

To our surprise only 7 out of 17 testpersons showed a nice nasal cycle on at least two occasions in the measuring time of 3 hours, or after extrapolation. If one considers cyclical activity alone, it appears that 12 out of 17 testpersons showed a nasal cycle on one or more occasions. As for the 6 persons who did not show a patency-reversal on two occasions, it is probable that among them there have been some with a very long cycle who were examined twice in the same phase of that cycle.

So probably about 80% of the testpersons showed cyclical activity as was the case in Heetderks' and Stoksted's series while Flottes' 80% is the percentage of his testpersons which showed a nasal cycle lasting 2—5 hours.

§ 3. The nasal cycle in children.

In order to investigate a possible cyclical activity in children, which has not been done before, we did cycle determinations in 19 children, aged 4—10 years with apparently normal noses. The children were either sitting up in bed or playing quietly at a table. Zwaardemaker's mirror was used, and the appearing breathing spots delighted the children, so they cooperated easily. A test was done every 15 minutes for 2 hours.

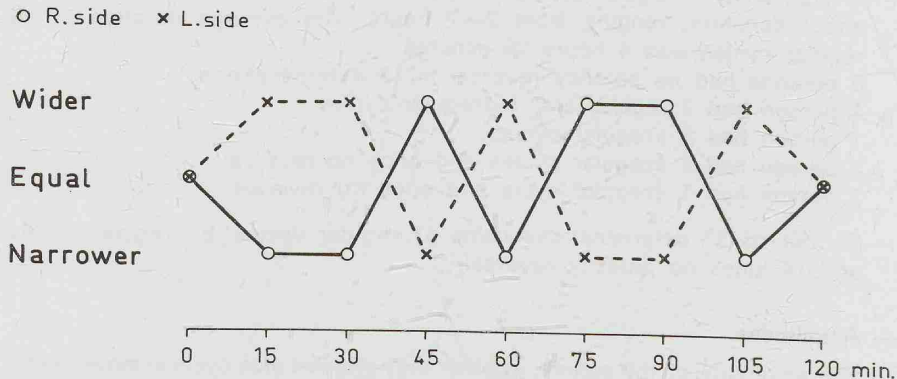


Figure 6. Qualitative patency-determinations in children with Zwaardemaker's test, as judged from the two breathing spots. Four patency reversals can be seen.

It appeared that patency-reversals appeared very rapidly, so that cycle lengths as short as 30 minutes were observed. Only one child showed one reversal, all other children had at least one full cycle during 2 hours, 6 children had two cycles in 2 hours.

§ 4. Posture and nasal cycle.

It is known (**Heetderks**, 1927) that in lying down on one side the lowermost nasal cavity becomes engorged and that the uppermost nasal cavity opens up. Gravity is assumed to be responsible for this.

a. According to **Winslow** e.a., (1939) in the **recumbent position** the nasal mucosa swells up, and does not alter after changing from the head high to the head low position (**Rundcrantz**, 1964). He states that before lying down the nasal patency was determined in the sitting position, but unfortunately no mention is made of that value, so we assume that it did not change.

b. we studied the **influence of the side position** as in sleeping. In the just developed phase of the cycle the testperson was laid on the side to which the opposite phase could be expected. It appeared that a reversal in turbinate filling occurred within a few minutes and could be reserved as quickly, by turning the testperson over on the other side.

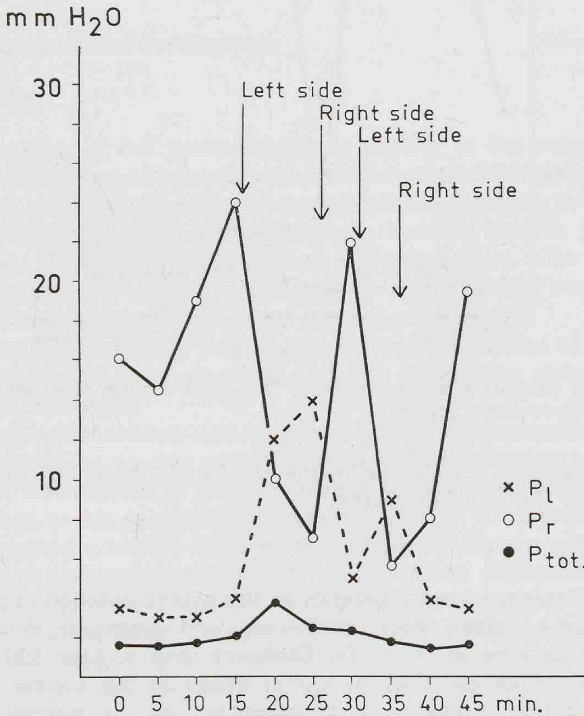


Figure 7. Influence of the side positions when lying down. The right side is the cyclically swollen side. Turning over on the other side produces a patency-reversal.

c. In the **up-right position** inclining the head sideways, as it would be in the side position, did not affect the turbinate filling.

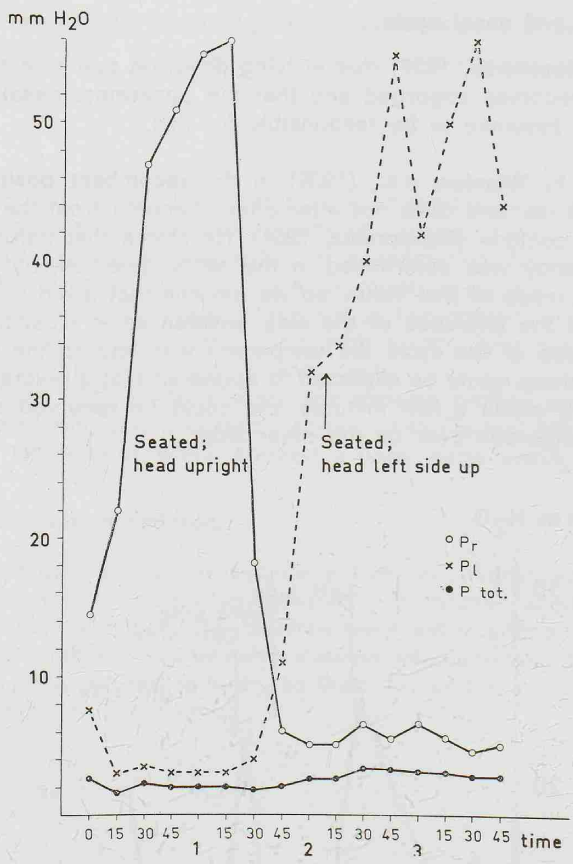


Figure 8. In the vertical position, inclining the head to the side with the swollen side up, no patency-reversal occurs.

In the supine position no change in the total nasal patency could be found nor a change in the cyclical rhythm.

d. Discussion.

The finding of decreased nasal patency in the supine position in patients with Horner's syndrome, getting reserpine therapy, and vasomotor rhinitis, in which the vasomotor balance is upset (v. Dishoeck and v. Lier 1960) suggests, according to our findings, that in normal noses in the supine position the increased hydrostatic pressure is counteracted by the normal local vasomotor tonus, because here no change in patency results. In the side position the cyclical pattern becomes rapidly deranged. The speed with which volume changes of the turbinates occur suggests that these are due to changes in blood content (Drettner). The hydrostatic pressure differences between the upper and lower nasal cavities now cannot be overcome either by the local vasomotor tonus, or by the cyclical mechanism. This view is supported by the

fact that in the upright position, where the hydrostatic pressure differences are relatively much smaller, a side position of the head is not able to change the cyclical pattern. Many complaints about nasal obstruction in the sleeping position in noses without vasomotor troubles thus are not likely to be due directly to turbinate factors but to anatomical deviations in the nose. A valvular defect is accentuated by gravity when one lies on the side opposite to it (it is surprising how many deficient valves there are, when the valve area is inspected, not with a nasal speculum, but with a wide earspeculum or special retractor when one does not efface the valvular pathology by the examining act (Cottle, 1962)) or, in the case of an important septum deviation in which the other side is anatomically wide, when one lies on the side of the anatomically wide nasal cavity, the ensuing turbinate swelling will soon block the nasal respiration where it would be anatomically possible. So it is a good advice — besides operation — to sleep on the side of the anatomically least patent nasal cavity, c.q. the least optimal valve, because often the patient has no important reason for sleeping on the rhinologically wrong side!

§ 5. Parasympathetic denervation.

a. Introduction.

After interruption of the parasympathetic supply to the nose the turbinates become shrunken due to sympathetic predominance (Milloning, 1947; Golding-Wood, 1963). After interruption of the sympathetic supply to the nose, as in Horner's Syndrome, the turbinates become swollen, due to parasympathetic predominance. Stoksted (1953) studied the nasal cycle after blocking of the stellate ganglion. The cycle stopped rapidly and a strong swelling appeared. The cycle continued again after the block had worn off.

So it is obvious to assume, that during the swelling phase of the nasal cycle the parasympathetic is dominating, and that during the shrunken phase the sympathetic dominates.

Cycle determinations have not been done before in cases with a parasympathetic denervation on one side. We investigated 5 patients with an anatomically certain interruption of the n. petrosus superficialis major. In one patient the nerve had to be sacrificed at operation for an extensive cholesteatoma, in another patient a middle ear carcinoma had completely infiltrated the facial nerve beyond the geniculate ganglion and in three patients the facial nerve had to be sacrificed at operation for a pontine angle tumor.

Two patients had a facial paralysis (Bell's palsy) with a strongly decreased lacrimation as shown by the Schirmer test, so that at least a decreased function of the n. petrosus superficialis major might be assumed.

In the Schirmer test a strip of filtrating-paper is hung in both lower conjunctival sacs and after 5 minutes the lengths of the wetted areas are compared.

b. Results:

In all cases practically no turbinal changes were present on the affected side, while the heterolateral side showed cyclical fluctuations.

R SIDED PARASYMPATHETIC DENERVATION

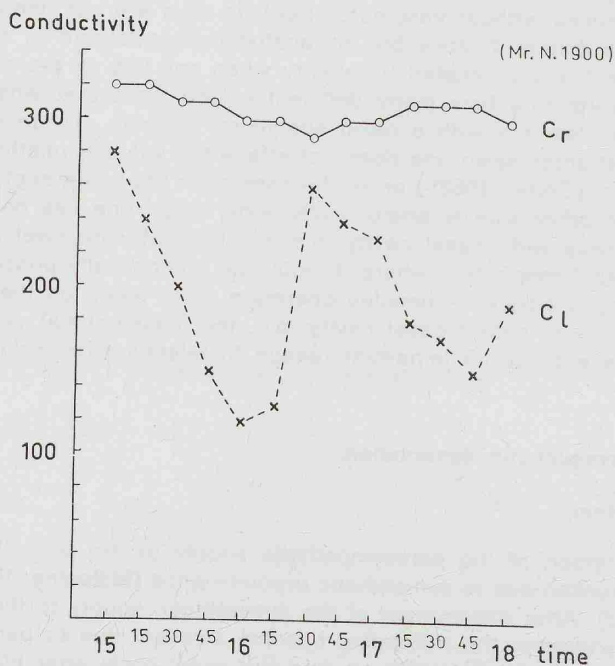


Figure 9. Right sided parasympathetic denervation after the n. petr. sup. maj. had been avulsed during operation for extensive cholesteatoma. This determination has been done with **Spoor's** conductivity-meter. Note that a lower conductivity means a lesser patency, and a higher conductivity means a greater patency.

At repeated anterior rhinoscopies no turbinal changes could be detected. The turbinates appeared shrunken and pale, the mucous membrane appeared rather dry. In only one case more pronounced dryness and some crust-formation was encountered. This was also the only patient who after operation for pontine angle tumor spontaneously had observed this symptom.

We had an interesting experience regarding the Schirmer test: a patient came to the out-patients department one morning with a Bell's palsy on the left side. The Schirmer test was strongly decreased on the same side and so was the size of the turbinates. We wanted her for rhinometric studies, but the apparatus being out of order, she came back the next day. In the meantime the rhinoscopic picture was checked regularly during several hours, the turbinates remaining shrunken on the affected side. At the cycle determination the next morning the turbinates on the left side were shrunken as before, but after 75 minutes a patency-reversal occurred. This did not fit into the picture, but now the Schirmer test was repeated and found to be the same on both sides. Consequently a functional recovery must be assumed to have taken place in the n. petrosus superficialis major.

c. Discussion.

Golding-Wood (1963) performed section of the n. petrosus superficialis, in cases of therapy resistant vasomotor rhinitis. The mucosa became shrunken, but no undue dryness was observed. The cases described by **Gardner** and **Stowell** (1947), who performed section of the n. petrosus superficialis major for migraine, and the case described by **Malcomson** (1959) with a Bell's palsy showed dryness and crust-formation. **Milloning, Harris** and **Gardner** (1947) also described shrunken dry mucosae after section of the nerve. Our own observations tend to fall within the range of these observations.

As regards the nasal cycle, it appears that in the parasympathetically denervated nasal cavity, where now the sympathetic acts alone, cyclical activity is absent. This means that the sympathetic alone cannot effect the cyclical mechanism.

The nasal cycle seems to be brought about by a mechanism where a sympathetic predominance exists on one side and parasympathetic predominance on the other side, while the predominance alternates regularly from one side on the other.

§ 6. Nasal cycle during oral breathing and after mucosal anesthesia.

a. Introduction.

It has been stated by most investigators that the function of the nasal cycle is the air-conditioning of the respiratory air. On the shrunken side the turbinates give off their secretions, while on the swollen side the air is warmed. It must be remarked, however, that these suppositions have not been proved, by examination of the humidity and temperature of the air of each nasal cavity during a cycle. In view of the rhinoscopic findings (dry mucosa in the swollen phase, moist mucosa in the shrunken phase) and of the important part apparently played by the nose in the air-conditioning of the air (e.g. **Ingelstedt** and **Toremalm**, 1960) however, these suppositions seem likely to be true.

In view of the important function of the cycle for the respiration, most authors place the origin of the nasal cycle in the respiratory center.

The sensory endings of the trigeminal nerve in the nose might be the triggering mechanism as well (**Soubeyrand**, 1963; **Melon**, 1964; **Golding-Wood**, 1966).

In order to further investigate the role of nose-breathing in the mechanism of the nasal cycle, we carried out two experiments in both of which irritation of the mucosa by the airstream was absent: (1) The nasal cycle was determined while the nasal mucous membranes were anesthetized with Stovaine — a local anesthetic which has no vasomotor activity according to **Flottes** (1961) — and (2) the cycle was determined during oral breathing only, nasal breathing being prevented, in persons whose cyclical patterns were known from previous determinations.

The noses were sealed off with plastic tape, as this was found to be the surest way of blocking all nasal air passage, without any irritative procedures within the nose. After initial values had been obtained, patency determinations

were done every 15 minutes. The tape was removed, and only one expiration was allowed, during which the measuring was done. Then the nose was sealed off again.

b. Results.

1. 4 determinations were done.

2. 7 determinations were done.

It appeared from all cases that:

- the total patency did not change from the previously known normal values.
- the cyclical patterns did not change materially, and that persons who had a nice cycle beforehand also showed the same cycle during nasal obturation.

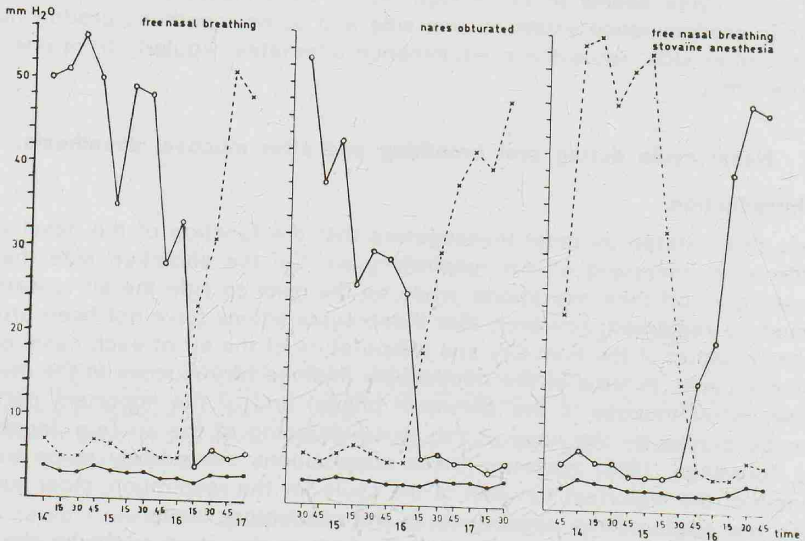


Figure 10. During normal nose-breathing, during mouth-breathing, and during nose-breathing while the mucosa was anesthetized with Stovaine, nasal cycles in the same person are practically identical.

c. The influence of the larynx on the nose.

Several studies have been done on the influence of the nose on the larynx (naso-laryngeal reflexes) but the influence of the larynx on the nose has received scant attention. **Sternberg** (1924, 1925), from experiments on dogs, concluded that the larynx has an inhibiting influence on the nasal vasoconstrictors. In man he found the nasal mucosa a little anemic after tracheotomy and pale and shrunken after laryngectomy, as a sign of atrophy by inactivity. We could confirm this in 4 laryngectomees. At repeated rhinoscopies not a sign of cyclical activity could be found.

So it seems that breathing through a tracheostoma after laryngectomy abolishes the turbinal activity.

To see whether the sensory innervation of the larynx has any direct influence on the turbinates, we measured the nasal patency after local anesthesia of the larynx (at a suggestion by prof. **Luyendyk**).

Rhinorheomanometric determinations were done on 6 persons *) having apparently normal noses. After initial patency values were obtained the larynx was anesthetized with a $\frac{1}{2}$ % Tetracain-solution. Patency determinations were made directly and then every 5 minutes, as well as anterior rhinoscopy. The examination lasted from 15—30 minutes, which is about the duration of the local anesthesia as testified by the testpersons.

Results.

In all cases it appeared that the cyclical curves did not change nor the total patencies. The rhinoscopic picture did not change.

d. Discussion.

It appeared that the nasal cycle is neither influenced directly by mouth-breathing, nor by anesthetizing the nasal mucosa. Moreover, local anesthesia of the larynx had no direct influence on the turbinates. These are arguments against the cycle originating in the nose, either by irritation by the airstream of the trigeminal nerve endings or in some other way, whereas a direct influence from the larynx seems improbable. Possibly some mechanism is present in the bronchi which could be directing the nasal cycle, although it would seem more probable that at this level in the airway this mechanism would have more to do with the general nasal resistance to breathing (see chapter 1, § 1).

On the other hand, the cycle is absent after laryngectomy. Studies of the nasal mucous membranes after laryngectomy have to be done after the nose has been in disuse for some time. Examination right after the operation is quite impracticable, because the patient has to be fed by way of an indwelling nasal tube for at least a week or two, after which it takes some time for the nose to recover from the traumatization by the tube. So, judging from the results of the immediate stopping of nasal respiration upon the mucosa, it appears that the nonfunctional state of the mucosa must be of longer duration for all turbinal activity to become absent, and we assume that the cyclical mechanism peters out in that time.

Judging from the fact that the cycle continues after local anesthesia of the nasal as well as laryngeal mucous membranes, and during temporary prevention of nose breathing, it must be concluded that, apart from a possible bronchial factor, the initiating mechanism of the nasal cycle has not been located. The possibility must be kept in mind, however, that some unknown factor may be present in the larynx, and especially in the nose — e.g. the "receptor"-structures in the nasal mucosa (**Temesrekasi**, 1959), — which was not put out of action by local anesthesia, or which, in the case of nasal obturation, may have been kept going behind the obturation in the nasal vestibule by the pressure changes in the nasal cavity caused by mouth-breathing.

*) My thanks are due to the nurses of the Juliana Hospital, Apeldoorn, who so willingly underwent this experiment.

§ 7. Bilateral effects of homolateral influences.

a. Adrenaline - histamine experiments.

The impression that some balancing mechanism must be in existence when one looks at nasal cycles, is strong. Indeed, the French term for the nasal cycle, *rhinite à bascule*, seems to be aptly chosen. **Beickert** (1950) investigated this. He applied adrenalin to one nasal cavity, and observed homolateral shrinking and heterolateral swelling of the turbinates, histamine effected the reverse. On repeating this, we could confirm these findings. The heterolateral reaction was less intense, and of shorter duration than the homolateral reaction.

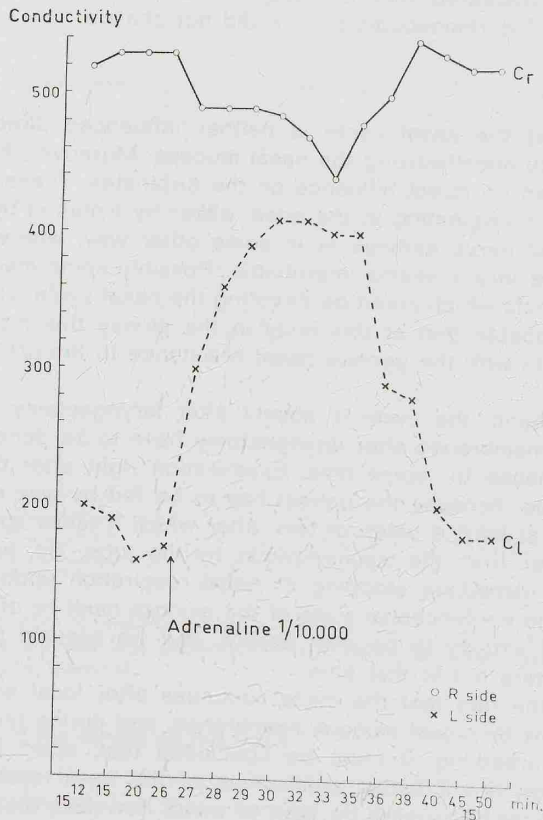


Figure 11. Spraying Adrenalin $1/10.000$ on the swollen side produces a marked decongestion homolaterally, and a slight relative congestion heterolaterally.

b. Horner syndrome and interruption of the *n. petrosus superficialis maior*.

In order to try to gain insight into this balancing-mechanism, we did experiments on patients having a one-sided interruption of either the sympathetic or the parasympathetic innervation of the nose (Horner syndrome and interruption of the *n. petrosus superficialis maior* respectively).

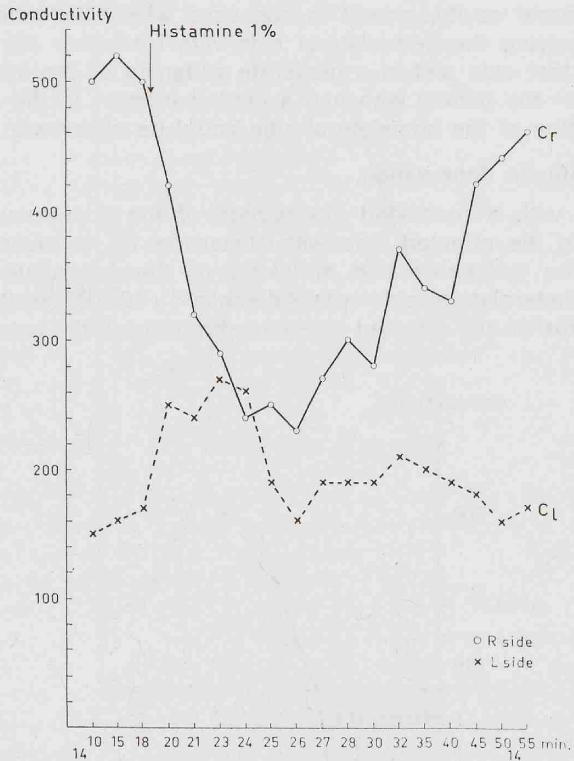


Figure 12. Spraying the decongested side with Histamine 1% produces a marked drop in conductivity homolaterally, and a slight decongestion heterolaterally.

1. Horner syndrome. *)

6 patients having a Horner syndrome from various causes — one patient had a central Horner, two had a cervical trauma, one had had a resection of a Zenker diverticulum, two patients were seen after a stellate ganglion block — were examined. Only 3 patients had complete rhinorheomanometric as well as rhinoscopic examinations, in the others rhinorheomanometry was felt to be unreliable, so only anterior rhinoscopy was done. At anterior rhinoscopy it appeared that the mucosa on the affected side was swollen, but that the cases of longer duration showed less swelling than the recent cases, e.g. after stellate ganglion block.

Results.

In all patients spraying the homolateral side with Adrenalin gave rise to a substantial widening of the nasal cavity and to a moderate narrowing of the

*) My thanks are due to Prof. W. Luyendyk, Leyden, and A. J. Froeling, M.D., J. S. F. Oud, M.D., and J. Th. Voorpool, M.D., Apeldoorn, for their interest in this study and their consent to examine their patients.

heterolateral nasal cavity, except in one case where the patency remained the same. Spraying the heterolateral side with Histamine 1% resulted in a narrowing of that side and in a moderate widening of the homolateral side in one case — the patient who had a central Horner; in the other patients no clear reaction of the homolateral side could be observed.

2. Parasympathetic denervation.

In 2 patients with a one-sided interruption of the n. petrosus superficialis maior spraying the affected side with Histamine 1% produced homolateral swelling of the turbinates and shrinking of the heterolateral turbinates. Spraying the heterolateral side with Adrenalin 1 : 10.000 resulted in swelling of the turbinates on the affected side and shrinking on the heterolateral side.

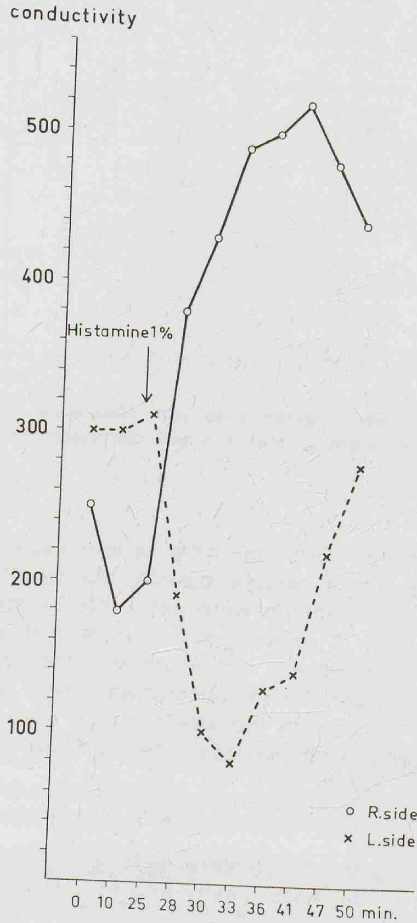


Figure 13. Left sided parasympathetic denervation after operation for pontine angle tumour. Spraying Histamine 1% on the left side is followed by congestion homolaterally and decongestion heterolaterally.

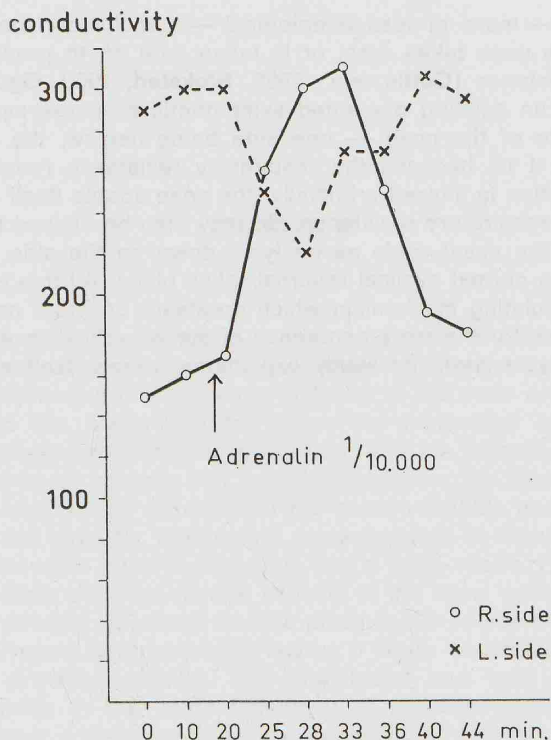


Figure 14. Left sided parasympathetic denervation after operation for pontine angle tumour. Spraying Adrenalin $1/10.000$ on the right, congested, side is followed by decongestion homolaterally and a, slighter, congestion heterolaterally.

c. Discussion

These findings suggest again that some balancing mechanism, in other words probably the mechanism which effects the nasal cycle, must exist. However, the heterolateral reaction above described is not the same as the patency-reversal which is the essence of the normal nasal cycle. But this may only be a quantitative difference because in normal noses after spraying with doses of Histamine 1% or Adrenaline $1/000$ higher than those previously used on respectively the cyclically shrunken and swollen sides it is possible to obtain a complete patency reversal. From the experiments on the one-sided parasympathetically or sympathetically denervated nasal cavities it appears that a reaction on the affected side may be obtained in the parasympathetically denervated nasal cavity but not on the sympathetically denervated side. This seems to imply that an intact sympathetic is essential for this reaction. The importance of the sympathetic in mediating vascular reactions in the nasal mucosa was also demonstrated by **Drettner** (1961) who found that the initial reaction in the nasal mucosa on local skin-cooling, e.g. a decrease in the bloodflow, was absent on the side where a stellate ganglion block had been performed.

As a general — more or less teleological — conclusion, the view may be offered that the nose takes care, or is taken care of, to present a constant respiratory resistance (Cottle, e.a., 1958, Stoksted, 1960, Ogura e.a., 1964). In the case of an existing one-sided sympathetic or parasympathetic denervation this state of the nose — one side being narrow, the other wide — can remain as it is, because the respiratory resistance remains the same. When this situation is altered artificially, the nose adapts itself so as to again offer the same respiratory resistance. So may also be viewed the involuntary disturbance of the nasal cycle as on lying down on the side, and even the situation when a normal cyclical reversal takes place. All this is possibly due to a central regulating mechanism which creates a constant nasal resistance to breathing. But, for the mere occurrence of these cyclical reversals or rather for the nasal cycle itself, no ready explanation offers itself at the moment.

CHAPTER III

Summary.

After an introduction, the literature on the nasal cycle, and factors influencing it, are discussed. A description of the pertaining anatomy is given.

Methods for measuring the degree of patency of the nose are systematically presented, and the preference for rhino-rheo-manometry is stressed. The author's rhino-rheo-manometer is described, and his formula presented with which the total nasal patency can be calculated from the patency of the right and left nasal cavity. The structure of the nasal vestibule and its importance when nozzles are used for rhinometric measurements is discussed. A distinction is made between a vestibule-nozzle and a naris-nozzle. Rhino-rheometric investigations were done on several aspects of the nasal cycle. Minor fluctuations in nasal patency appeared to take place in the course of a few minutes. From repeated cycle determinations lasting 3 hours we obtained less **regular** cycles than previous authors. The amplitudes and total patencies were remarkably similar in the same individual at repeated occasions. Children appeared to have a very lively cyclical activity.

On lying down in the side position a patency reversal occurred in a few minutes. This could be reversed as quickly by turning the testperson over on the other side. In the upright position inclining the head to the side appeared not to affect the patency of either nasal cavity. In the recumbent position no change could be found either in cyclical rhythm or in the total patency as compared with the upright position.

Cycle determinations done in patients having a one-sided interruption of the parasympathetic nerve supply to the nose revealed that cyclical activity was absent on the affected side, the turbinates being constricted. The absence of cyclical activity could be used in topodiagnosis of facial nerve lesions. During temporary occlusion of the nose, the cyclical activity remained the same as during nose-breathing. This was also the case during nose-breathing when the mucous membranes were anesthetized. After laryngectomy no cyclical activity was apparent on either side.

After local anesthesia of the larynx the state of the turbinates was not altered. In the normal nose it was possible by applying unilateral vasoconstriction or vasodilatation, to elicit the reverse reaction in the other nasal cavity.

In the unilaterally parasympathetically denervated nose it was possible to elicit this opposite reaction on the affected side. In the unilaterally sympathetically denervated nose it was not possible to elicit this opposite reaction on the affected side. The possibility is mentioned that the sympathetic is responsible for the heterolateral reaction.

CONCLUSIONS

1. In a measuring time of 3 hours less regular nasal cycles are found than by previous authors.
2. Minor fluctuations in nasal patency take place.
3. Children have a lively cyclical activity.
4. In the recumbent position no change in cyclical pattern occurs.
5. On lying down on the side a patency reversal occurs rapidly, and is caused by gravity.
6. In a nasal cavity without parasympathetic innervation cyclical activity is absent, the turbinates remaining constricted. This is an aid in the topodiagnosis of facial nerve lesions.
7. The nasal cycle is not affected by temporary occlusion of the nares, or by anesthesia of the mucosa or by anesthesia of the larynx.
8. In laryngectomies no nasal cycle was found.
9. Reversing the state of patency in one nasal cavity by vasoconstrictor or vasodilating agents effects the opposite state in the other nasal cavity. This "hetero lateral reaction" may be dependent on an intact sympathetic innervation.
10. The mechanism of the nasal cycle is probably, that in the swollen phase the parasympathetic dominates and in the constricted phase the sympathetic.

RÉSUMÉ

Après l'introduction suit une discussion de la bibliographie concernant le cycle nasal, ainsi que les facteurs qui influencent le cycle nasal. L'anatomie concernant ce sujet est présentée brièvement. Les méthodes pour mesurer la perméabilité nasale sont décrites et la préférence fut accordée à la rhinorhéomanométrie.

La méthode propre de la rhinorhéomanométrie est décrite. On y trouve la formule qui permet de calculer la perméabilité nasale totale par la perméabilité nasale gauche et droite. La différence entre l'olive narinaire et l'olive vestibulaire y est indiquée.

Les propres recherches sur les aspects différents du cycle nasal sont décrites.

De légères fluctuations dans la perméabilité nasale semblent apparaître pendant quelques minutes.

Des examens du cycle nasal furent établis sur plusieurs sujets et refaits au moins une fois. Comparé avec les autres auteurs on observa un nombre inférieur de cycles réguliers, quoique le pourcentage total où l'activité cyclique fut constaté était probablement à peu près pareil c'est à dire 80%.

L'amplitude du cycle et la perméabilité nasale totale chez les mêmes personnes coordinaient d'une façon surprenante avec les valeurs acquises après un second examen. Des enfants semblaient avoir une activité cyclique vive. La position couchée sur le coté amena un renversement de la perméabilité en quelques minutes; cet renversement fut observé aussi rapidement en sens inverse, dès que la position sur l'autre coté fut prise. Assis une inclinaison de la tête vers l'épaule ne semblait guère influencer la perméabilité d'aucune des deux cavités nasales. Couché sur le dos aucune modification dans l'acti-

tivité cyclique ou la perméabilité nasale totale, par rapport à la position assise, ne fut constatée.

Les examens du cycle chez les malades ayant une interruption unilatérale de l'innervation parasymphatique du nez ont prouvé que l'activité cyclique était absente du coté atteint; les cornets furent dégonflés. Cette absence de l'activité cyclique peut être utile pour la topodiagnostic des affections du nerf facial.

En supprimant temporairement la respiration nasale, l'activité cyclique resta la même que pendant la respiration nasale. Le même résultat fut observé avec la respiration nasale lors d'une anesthésie locale de la muqueuse nasale.

Après une laryngectomie l'activité cyclique était absente. L'anesthésie locale du larynx ne semblait guère influencer les cornets. Une vasoconstriction ou une vasodilatation unilatérale dans un nez normal provoqua une réaction inverse dans l'autre cavité nasale. Cette réaction inverse pouvait être provoquée du coté atteint lors de la dénervation parasymphatique unilatérale mais pas lors de la dénervation sympathique. On suggère la possibilité que le sympathique soit la cause de la réaction hétéro-latérale.

SAMENVATTING

Na een inleiding volgt een literatuuroverzicht betreffende de neuscyclus, en factoren die de neuscyclus beïnvloeden. In het kort wordt de van belang zijnde anatomie besproken.

De methoden om de doorgankelijkheid van de neus te meten worden besproken waarbij de voorkeur wordt uitgesproken voor rhinorheomanometrie. De eigen methode van rhinorheomanometrie wordt beschreven. De formule wordt gegeven, waaruit de totale neus-doorgankelijkheid berekend kan worden uit de doorgankelijkheid van de linker en de rechter neusholte. Het onderscheid tussen naris-neusstuk en vestibulum-neusstuk wordt aangegeven.

Eigen onderzoekingen worden beschreven over verschillende aspecten van de neuscyclus. Kleinere fluctuaties in neus-doorgankelijkheid bleken voor te komen in het verloop van enige minuten. Neuscyclusbepalingen werden verricht bij een aantal proefpersonen, en bij allen tenminste eenmaal herhaald. Er werd een kleiner aantal regelmatige cycli gevonden dan bij andere auteurs, hoewel het totale percentage waarin cyclische activiteit werd gevonden waarschijnlijk ongeveer gelijk was, namelijk 80%. De amplitudo van de cycli en de totale neus-doorgankelijkheid bij dezelfde proefpersonen bleken opvallend gelijk te zijn aan de waarden die bij een herhaald onderzoek werden gevonden. Kinderen bleken een levendige cyclische activiteit te hebben.

In zijligging bleek een omslag in doorgankelijkheid op te treden binnen enige minuten; deze omslag kon even snel omgekeerd worden door zijligging op de andere zijde. In zittende houding bleek zijwaarts neigen van het hoofd de doorgankelijkheid van geen van beide neusholten te beïnvloeden. In rugligging kon geen verandering in cyclische activiteit of totale neus-doorgankelijkheid ten opzichte van de zittende houding worden aangetoond.

Cyclusbepalingen bij patiënten met een eenzijdige onderbreking van de parasymphatische innervatie van de neus lieten zien dat cyclische activiteit afwezig was aan de aangedane kant; de conchae waren hier ontzwollen. Deze

afwezigheid van cyclische afwezigheid kan van nut zijn bij de topodiagnostiek van n.facialis-aandoeningen.

Bij tijdelijk uitgeschakelde neusademhaling bleef de cyclische activiteit dezelfde als bij neusademhaling. Dit bleek ook het geval bij neusademhaling, terwijl het neusslijmvlies plaatselijk verdoofd was.

Na laryngectomie was cyclische activiteit afwezig. Plaatselijke verdoving van de larynx bleek geen invloed te hebben op de conchae.

In de normale neus bleek eenzijdige vasoconstrictie of vasodilatatie in de andere neusholte de tegengestelde reactie teweeg te brengen. Deze tegengestelde reactie bleek opgewekt te kunnen worden aan de aangedane kant bij eenzijdige parasymphatische denervatie van de neus, maar niet bij eenzijdige sympathische denervatie van de neus. De mogelijkheid wordt genoemd dat de sympathicus verantwoordelijk is voor de heterolaterale reactie.

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