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Pressures generated during nose blowing in patients with nasal complaints and normal test subjects*

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SUMMARY

The authors compared nasal resistance and pressures generated during breathing and nose blowing in patients with chronic sinusitis, septal deviations and a control group consisting of normal test subjects.

The chronic sinusitis group generated pressures during nose blowing that were significantly higher (898 daPa for the left side and 913 daPa for the right side) than in the other two groups. The decongestion didn't change the generated pressures very much. Pressures generated during nose blowing with both nostrils closed are much higher than pressures generated during nose blowing with one nostril open. These very high pressures could have an important role in the pathophysiology of chronic sinusitis.

Key words: nose blowing, pressures, chronic rhinosinusitis

INTRODUCTION

Not much is known about the physics of nose blowing. Nose blowing in its most typical way consists in a manoeuvre during which in a first step the nostrils are closed by digital pressure (pinched nostrils), in a second step a high intranasal pressure is built up by contracting the thoracal muscles and diaphragm, keeping the cheek muscles tight and retracted, and making an attempt to blow out the closed nostrils. Finally, in a third step this high intranasal pressure is suddenly released by removing the digital pressure on the nostrils. This manoeuvre, similar to a Valsalva manoeuvre, is meant to clear the upper airways from an excess secretion and is very similar to the coughing manoeuvre that clears the lower airways. The only difference, however, is that nose blowing is quite unphysiologic as it moves the secretions against the direction of the normal mucociliary clearance which is directed towards the nasopharynx.

Nose blowing in the above-described way is in fact a most typical human manoeuvre, as animals are not able to close their nostrils by nostril pinching. When closing both nostrils unphysiological high pressures are built up, that can result in pressure-induced expansion of the maxillary sinus (pneumosinus maxillaris dilatans: Wolfensberger, 1984; Sobin et al., 1986; Wolfensberger et al., 1987; Juhl et al., 2001), propelling viscous fluid into the maxillary sinus (Gwaltney et al., 2000), and in extreme cases it can even cause an orbital floor fracture (Oluwole et al., 1996), orbital emphysema (Brown et al., 1995), or even induce an acute epidural hematoma (Omana et al., 1995).

Not much is known about the pressures generated during this manoeuvre. The highest pressure Wolfensberger (1984) measured after a Valsalva manoeuvre was 400 mm H_2O (400 daPa), Sobin et al. (1986) found a pressure of 14 cm H_2O (= 140 daPa) and Gwaltney et al. (2000) mention a mean maximal pressure during 35 nose blows of 66 mm Hg (= 858 daPa).

When looking at CT scans of patients with chronic sinusitis the author sometimes found some typical isolated features such as: an isolated ethmoidal cell that looked hyperinflated, compressing the surrounding narrow ethmoidal passages, or even a hyperinflation of the whole ethmoid (ethmoidal emphysema - Figure 1) leading to chronic inflammation of the sinuses draining via these compressed passages, or an ethmoidal cell invading the frontal sinus via its aperture, sealing off completely the drainage and ventilation of this frontal sinus and finally leading to chronic frontal sinusitis. All these findings suggest that pressure changes might be responsible for these phenomena. The "uncapping the egg" (Stammberger, 1991) manoeuvre during FESS surgery to restore the frontal sinus drainage and ventilation is an illustration of this phenomenon, where an ethmoidal cell invades the apertura of the frontal sinus.

The aim of this study was to compare nasal resistance and

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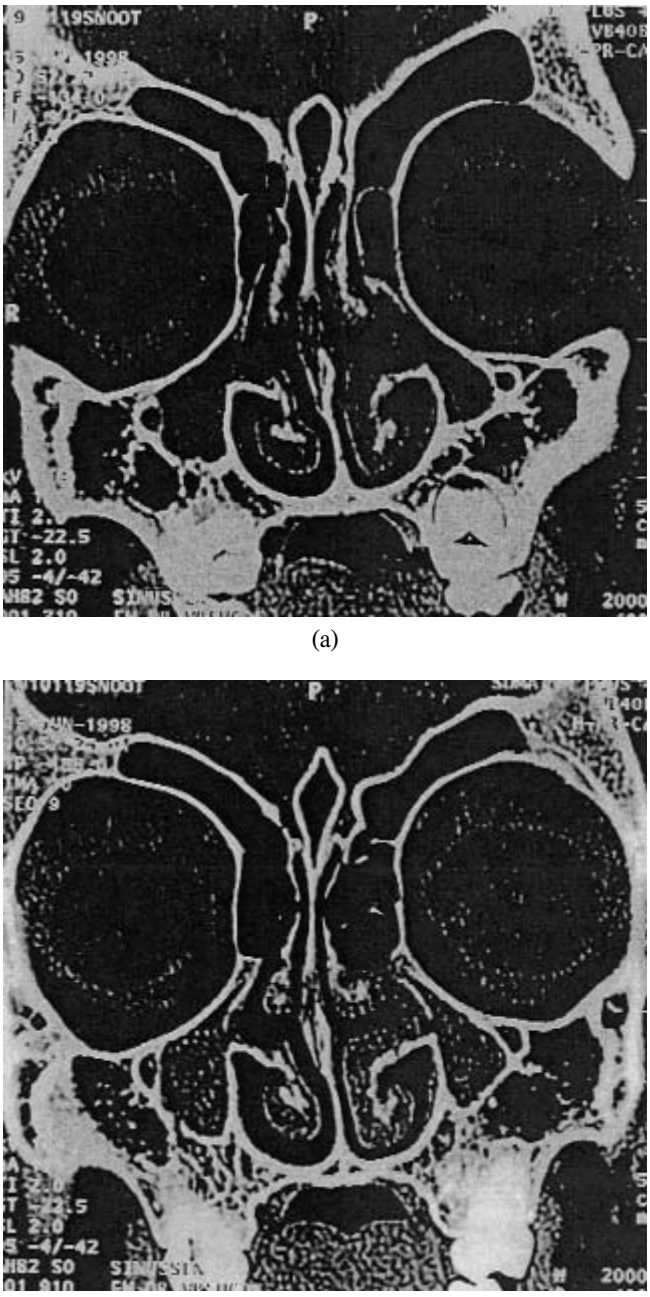


Figure 1. Bilateral hyperinflated uncinate process (a) and ethmoid complex (b) ("ethmoidal emphysema") impeding drainage and ventilation of the maxillary sinus in a child of 7 years of age (measured pressure 650 daPa).

pressures generated during normal breathing and nose blowing in patients with chronic sinusitis, patients with septal deviations, and in a group of normal test subjects.

MATERIAL AND METHODS

Patients and normal subjects

The first group included 14 patients with chronic sinusitis who were scheduled for FESS surgery after failure of conservative medical treatment. Five patients had massive nasal polyposis (polyps extending from the middle meatus but not obstructing the nasal airway medial of the inferior turbinate because otherwise rhinomanometry could not be performed: Lund et al. - score 2) and 9 patients had chronic sinusitis without massive nasal polyposis (polyps not protruding beyond the middle meatus: Lund et al. - score 1). The mean age of this group was 46 years (34-59 y), and the male/female ratio was 6 (12/2).

The second group included patients with nasal complaints and septal anomalies (with or without mucosal hyperreactivity – Graamans, 1980), scheduled for septoplasty and/or turbinoplasty and no CT scan evidence of sinus involvement. The mean age of this group was 33 years (21-67 y) with a male/female ratio of 3 (15/5).

The control group consisted of 18 normal test subjects without any nasal complaints, no obvious septal deviation at rhinoscopy, no history of allergy or recent URI and no use of drugs. The average age was 28 (20-47 y) with a male/female ratio of 1.25 (10/8).

Pressure measurements

In all three groups the following measurements were performed:

1. **Rhinomanometry:** a rhinomanometer Atmos 800 was used to measure nasal resistance in $Pacm^{-3}s$. The inspiratory nasal resistance was measured at a pressure of 75 Pascal because pressures of 150 Pa could not be reached in all test subjects (International Committee on Standardization of the Objective Assessment of the Nose, Clement, 1984). Two measurements without decongestions were performed, the first without decongestion, and the second 15 minutes after nasal decongestion (administration of 1% xylometazoline: Otrivine®).

2. **Nose blowing:** the patients placed a metal Politzer nasal olive tightly in one nasal vestibulum, sealing off this nasal cavity. The patients were asked to increase the pressure in the nasal cavity while closing the other nostril, to rule out any leakage. The metal olive was connected via a plastic tube to a pressure transducer (Siemens Pressure Transducer 74) and linked to a mingograph 43 (Siemens-Elema) that recorded the pressure changes in time.
   In this way the nasopharyngeal pressure could be measured in decapascal (daPa). The following pressures were measured:
   a. Positive nasal pharyngeal pressure during normal breathing through the non-occluded nasal cavity (left and right side separately).
   b. Maximum positive pressure generated during nose blowing with the remaining nostril not occluded, before and after decongestion, left and right side
   c. Maximum positive pressure generated during nose blowing, with the remaining nostril occluded, before and after decongestion, left and right side

(Every measurement was performed 3 times and the average pressure was calculated)

So the following variables were recorded

a. Resistance
   1. Inspiratory nasal resistance at 75 Pa before decongestion left side
   2. Inspiratory nasal resistance at 75 Pa before decongestion right side
   3. Inspiratory nasal resistance at 75 Pa after decongestion left side
   4. Inspiratory nasal resistance at 75 Pa after decongestion right side

b. Pressure measurements
   1. Maximum pressure generated during quiet breathing through the left side
   2. Maximum pressure generated during quite breathing through the right side
   3. Nose blowing maximum pressure generated before decongestion left side with right side open
   4. Nose blowing maximum pressure generated before decongestion right side with left side open
   5. Nose blowing maximum pressure generated after decongestion left side with right side open
   6. Nose blowing maximum pressure generated after decongestion right side with left side open
   7. Nose blowing maximum pressure generated before decongestion left side with right side closed
   8. Nose blowing maximum pressure generated before decongestion right side, left side closed
   9. Nose blowing maximum pressure generated after decongestion left side, right side closed
   10. Nose blowing maximum pressure generated after decongestion right side, with left side closed.

Statistics

The following parametric tests were used: to compare the variables between the 3 groups (two patient groups and one test subject group) a one-way Anova was used. For these variables where a global significant difference existed ($p<0.05$) the Scheffe Post Hoc test was used to know between which groups a significant difference existed.

To test a homogeneous distribution of the gender between groups, a Pearson Chi-square test was used. To test if a significant difference existed between the different parameters before and after decongestion for the 3 groups separately, a paired t-test was used. In the control group the authors looked for a significant difference between left and right side.

RESULTS

Gender

From Table 1 follows that the groups showed no significant difference between the male/female ratios. The Pearson Chi-square test showed a p-value of 0.155.

Table 1. Gender group cross tabulation.

Gender	Group 1	Group 2	Group 3	Total
Male				
Count	12	15	10	37
Female				
Count	2	5	8	15
Total				
Count	14	20	18	52

Inspiratory nasal resistance at 75 Pa during quiet breathing

From Table 2 it appears that the mean unilateral inspiratory nasal resistance at 75 Pa between the 3 groups does not show any significant difference (One-Way Anova), although there exists a tendency in the septal deviation group to generate higher resistances (before and after decongestion).

Table 2. Inspiratory nasal resistance at 75 Pa (R) during quiet breathing before and after decongestion in Pa $cm^{-3}s$.

Groups	R before decongestion		R after decongestion	
Chronic	**Left**	**Right**	**Left**	**Right**
Mean R	0.427	0.250	0.250*	0.197
N	13	13	12	12
s.d.	0.221	0.239	0.339	0.224
Septal deviation				
Mean R	1.359	0.465	0.334**	0.281**
N	20	20	18	18
s.d.	2.760	0.320	0.307	0.354
Control				
Mean R	0.226	0.506	0.182**	0.191**
N	18	18	17	17
s.d.	2.070	0.365	0.137	0.075

s.d. standard deviation

* Significant difference between the values before after decongestion

** Very significant difference between the values before and after decongestion

In the group of chronic sinusitis the difference in left nasal resistance before and after decongestion was significant (a resistance of 0.250 $Pacm^{-3}s$ after decongestion, compared to 0.427 $Pacm^{-3}s$ before decongestion: $p=0.015$) and the differences in right and left nasal resistances before and after decongestion in the test subject group were very significantly different (for the left side 0.182 $Pacm^{-3}s$ after decongestion versus 0.226 $Pacm^{-3}s$ before decongestion ($p<0.001$) and for the right side 0.91 $Pacm^{-3}s$ after decongestion versus 0.506 $Pacm^{-3}s$ before decongestion ($p=0.002$)). For the group with a septal deviation because a non normality of distribution was observed, a Wilcoxon Matched-Pairs Signed-Rank test was used, and a very significant difference was shown for the resistant values of the left side (0.334 $Pacm^{-3}s$ before decongestion versus 1.359 $Pacm^{-3}s$ after decongestion ($p=0.007$)) and right

side (0.281 $Pacm^{-3}s$ versus 0.465 $Pacm^{-3}s$ (p=0.0022) before and after decongestion).

So, in general one can state that the differences in nasal resistance before and after decongestion in the septal deviation and control group are particularly significant and this both for the left and right side.

For the chronic sinusitis group, the differences in nasal resistance before and after decongestion are less prominent and only significant for the left side.

Mean maximum pressure in daPa (decapascal) generated during normal breathing

Table 3. Mean maximum pressure in daPa generated during normal breathing.

Groups		
Chronic sinusitis	**Left side**	**Right side**
Mean p	68.571*	75.714
N 14	14	
s.d.	60.259	75.825
Septal deviation		
Mean p	57.000	60.750
N 20	20	
s.d.	33.103	23.524
Control		
Mean p	31.667*	45.556
N 18	18	
s.d.	8.575	15.424

* significant difference

Using the Scheffe Post Hoc test there existed a significant difference (p=0.0028) in maximum pressure generated during quiet breathing between the chronic sinusitis group (68.571 daPa) and the control group (31.667 daPa) and this only at the left side (Table 3).

So it seems that during normal breathing, chronic sinusitis patients need to generate continuously higher pressures than patients with septal deviations or the control group. These differences in maximum pressure, however, are not important, and reach the significance level only between the left side of the chronic sinusitis group and the control group.

Maximum pressure generated during nose blowing, one nostril open, and both nostrils closed before and after decongestion.

Using the Scheffe Post Hoc test, the following could be determined:

- Blowing the nose left side, *one nostril open*, after decongestion showed a significant difference in pressure between on the one hand the sinusitis group (496.240 daPa) and on the other hand the septal deviaton group (178.000 daPa, p=0.028) and the normal group (180.000 daPa, p=0.032) (Table 4a). There is no significant difference between the septal deviation group and the normals.

Table 4a. Pressure (daPa) generated during nose blowing, one *nostril open*, before and after decongestion.

Groups	**Before decongestion**		**After decongestion**	
Chronic	**Left**	**Right**	**Left**	**Right**
Mean	331.818	438.182*	496.240*	512.250*
N	11	11	8	8
s.d.	285.441	513.319	633.628	697.433
Septal deviation				
Mean	201.000	210.500	178.000	172.000
N	20	20	20	20
s.d.	83.281	97.575	83.136	87.154
Control				
Mean	204.444	195.000	180.000	188.889
N	18	18	18	18
s.d.	118.928	90.894	119.312	113.183

* significant p=0.006

Table 4b. Pressure (daPa) generated during nose blowing, *both nostrils closed*, before and after decongestion.

GROUPS	**Before decongestion**		**After decongestion**	
Chronic	**Left**	**Right**	**Left**	**Right**
Mean	860.000	955.714*	898.750*	913.750*
N	14	14	8	8
s.d.	495.068	571.620	615.620	742.216
Septal deviation				
Mean	620.000	573.000	461.250	477.500
N	20	20	20	20
s.d.	248.956	183.621	214.572	253.645
Control				
Mean	620.000	533.333	532.500	541.667
N	18	18	12	12
s.d.	208.891	183.143	219.509	190.064

* significant p=0.015

- Blowing the nose right side, *one nostril open*, after decongestion again showed a similar pattern of a significant pressure difference between on the one hand chronic sinusitis group (512.250 daPa) and on the other hand the septal deviation group (172.000 daPa, p=0.026) and the control group (188.889 daPa, p=0.039) (Table 4a).
- Blowing the nose right side, *one nostril open*, before decongestion showed a significant difference in pressures generated between on the one hand the chronic sinusitis group (438.182 daPa) and on the other hand the septal deviation group (210.500 daPa, p=0.067) and normals (195.000 daPa, p=0.053) (Table 4a).
- Finally, blowing the nose right side, *both nostrils closed*, before decongestion, showed a significant difference in pressures generated between on the one hand the chronic sinusitis group (955.714, daPa) and on the other hand the septal deviation group (573.000, p=00.8) and the normals (533.3 daPa, p=0.004) (Table 4b).

Between the septal deviation group and the normals no statisti-

cally significant difference in pressure was found. For the variables before and after decongestion there existed only a significant difference in pressures generated during nose blowing in the septal deviation group, one nostril open, right side (Table 4a, p=0.006) and in the chronic sinusitis group where there even existed an increase in left side pressure after decongestion generated with both nostrils closed (Table 4b, p=0.015).
The differences, however, were rather small: 210.500 versus 172.000 (Table 4a) daPa in the septal deviation group and 860.000 versus 898.750 in the chronic sinusitis group (Table 4b).

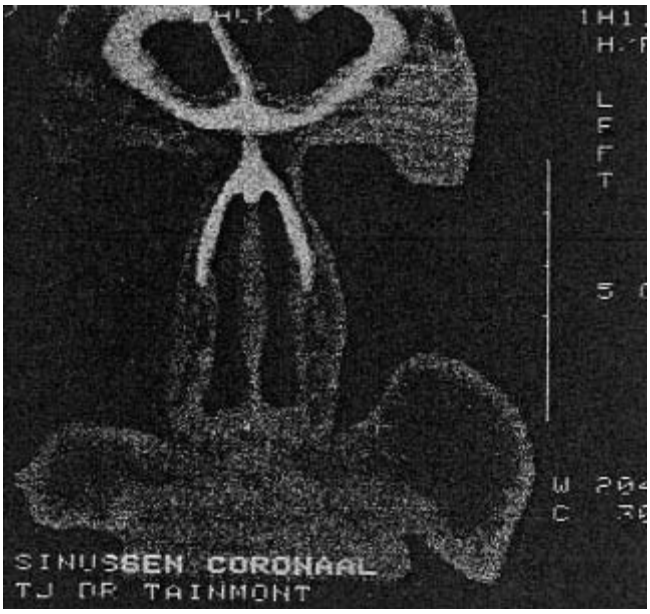
In conclusion, one can state that in the chronic sinusitis group pressures generated during nose blowing are higher than in the other two groups (septal deviation and control group). These differences reach significant levels for the pressures one nostril open after decongestion, both sides, before decongestion right side, and both nostrils closed before decongestion right side. Decongestion itself does not change the generated pressures very much (only a factor 1.1-1.2) and there are no major differences in pressures between the left and right side.
Pressures generated with both nostrils closed are much higher than the pressures generated during nose blowing with one nostril left open (factor 2-3).

DISCUSSION

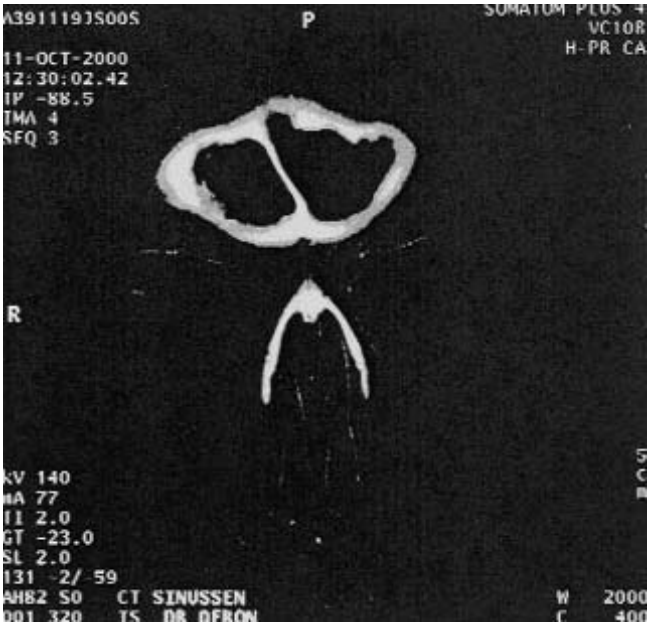
Although the Valsalva manoeuvre is similar to nose blowing, it is not completely the same. During a Valsalva manoeuvre the pressure in the pharynx and nasal cavity is also increased but released by relaxing the activity of the diaphragm and the intrathoracal muscles, before the release of the pinched nostrils. During nose blowing, however, the pressure in the nose and the pharynx is forcefully released by removing the digital pressure on the nostrils while the diaphragm and intrathoracal muscles keep up their activity.

Rhinomanometry was included in this study because the authors wanted to know if there existed a correlation between nasal resistance, the maximum pressure reached during normal breathing, and the pressures generated during nose blowing. From the data it appeared that there was no clear-cut correlation between the nasal resistances of the different groups and the high pressures generated during nose blowing. Interesting is the fact that the maximum pressures reached during normal breathing were higher in the septal and rhinosinusitis group, although they only reached significant levels in the sinusitis group and only for the left side. In comparison to the pressures generated during nose blowing, those maximum pressures generated during normal breathing still remain very low (31 to 75 daPa versus 533 to 955 daPa).
It might be that all patients with nasal complaints needing surgery generate very high pressures. Therefore the authors included a group of patients with nasal complaints needing septal surgery and no signs of sinusitis on the CT scan. It showed that these very high pressures during nose blowing were significantly more related to the rhinosinusitis group. One might also wonder why this group of patients needing septal surgery did not show significantly different nasal resistance compared to the control group. This can be explained by the fact that in this study the mean resistances were computed for the right side versus the left side and not for the most blocked side versus the non-blocked side (Postema et al., 1980). For statistical reasons one cannot compare variables between different groups if they are not ranked the same way. Furthermore the septal surgery group included both patients with anterior deviations inducing high nasal resistances and patients with posterior spurs of the nasal septum. It is a well-known fact that posterior septal deviations interfere much less with nasal resistance than anterior deviations (Szücs et al., 1998).

From this study it appears that rhinomanometric data (inspiratory nasal resistance at 75 Pa) and the maximum pressure generated during quiet breathing will not allow to do any meaningful predictions on the pressures that will be generated during nose blowing.
The study, however, shows that nose blowing can generate extremely high pressures, especially with both nostrils closed, compared to the pressures generated during normal breathing. Our champion nose blower reached an amazing maximum pressure of 2420 daPa.
It also shows that pressures generated during nose blowing in chronic sinusitis patients are often significantly higher than in patients with septal deviation and normal controls. These high pressures might be an important factor in the pathophysiology of chronic sinusitis. The authors found on a CT scan investigation with a 4-year interval (Figure 2) a clear-cut increase in the size of a type III frontal sinus cell (Bent et al., 1994). It is highly improbable that in this case the increase in size of this ethmoidal cell extending into the frontal sinus would be genetically determined. It looks more likely that continuous high pressure nose blowing pushes this cell deeper and deeper into the frontal recess until it blocks entirely the aperture of the frontal sinus. Once the frontal aperture is completely sealed off, the air in the frontal sinus will be resolved by the mucus lining and the initiating mechanism is even increased due to the negative pressure that will exist in the frontal sinus and the repetitive high positive pressures that are induced during nose blowing.
Also in other cases, it is possible that pre-existing anatomical variations (concha bullosa, oversized ethmoidal bulla or even the whole ethmoid) are increased in size by the pressures generated during forced nose blowing. The increase in size of these structures will impede the drainage and ventilation of the surrounding sinuses via the compressed ethmoidal clefts, leading to a permanent obstruction and irreversible chronic sinusitis.
The mechanism of a pneumosinus dilatans is probably different from that of inflating an isolated ethmoidal cell or the whole ethmoid. In a pneumosinus dilatans the pressure



(a)



(b)

Figure 2. (a) Type 3 cell (ethmoidal cell) invading richt frontal sinus, showing a similar picture as in Figure 3a (patient generated 600 daPa during nose blowing). (b) Same patient as in Figure 4a but 4 years later. Note that this type 3 cell has grown considerably in this 4 years occupying now the whole frontal sinus.

remains high (350 mm H_2O= 350 daPa, Wolfensberger, 1984; Wolfensberger et al., 1987; Juhl, 2001) because of air trapping in the sinus due to a valve mechanism at the level of the ostium. If one accepts that these high pressures can induce a pneumosinus dilatans or the complication mentioned in the introduction, then it must also be possible that these repetitive high pressures influence the size of expanding ethmoidal cells.

During surgery the author has observed several times in the living individual that the bony structures of the ethmoid are not hard and rigid, but often very soft showing a high elasticity index. It might be that chronic inflammation of the bone, with findings similar to osteomyolitis (Kennedy et al., 1998; Jang et al., 2002) makes the bony lamellae of the ethmoid softer and easier to be displaced by high pressures. The author does not imply that these high pressures during nose blowing observed in patients are the only mechanism responsible for chronic sinusitis, but it can be an important contributor in maintaining the disease. It might also partially explain the good results seen after FESS. The aim of endoscopic sinus surgery is to remove all these ethmoidal cells and lamellae, restoring ventilation and drainages of the blocked sinuses.

Nose blowing with both nostrils closed is a typical human manoeuvre. Generating these extremely high pressures is very unphysiological. Patients should be instructed that nose blowing is only needed when secretions that are not adequately removed by the mucociliary clearance block the nose. One should only blow the nose when it is productive. When nasal obstruction is caused by mucosal congestion, nose blowing will not improve the nasal patency, on the contrary, these unphysiological high pressures generating the sound during nose blowing will only increase the mucosal swelling. Patients should also be instructed only to blow their nose with one nostril open. After nose blowing, repeated sniffing should be advocated inducing negative pressures (up to 700 daPa), in order to compensate for the positive pressures generated before.

According to Stammberger (1991) one of the causes of polyp formation is the intimate contact of opposing mucosal surfaces. Temporary touching mucosae because of inflammation, also present during a common cold, will rarely lead to polyp formation. On the contrary a permanent touching mucosa around a bony anatomical variation induced by an increase in size of this anatomical variation, will induce a “bottle neck phenomenon”. An increased pressure will follow this on the opposing mucosal surfaces, leading to a total stop of the coordinated ciliary beat, ventilation and drainage of the involved sinus. Finally this will lead to chronic inflammation and polyp formation resulting in chronic sinusitis.

Summarizing, one could assume that high pressures generated during nose blowing may contribute to the etiology of chronic rhinosinusitis by two possible mechanisms:

1. Blowing pathological secretions in the sinuses (Gwaltney et al., 2000) or
2. By increasing the size of a pre-existing anatomical anomaly in the ethmoid, resulting in permanent stenosis of the narrow sinus drainage pathways.

This is the first time in the literature that pressures generated by nose blowing in normal subjects and patients have been studied. Recently, the frequency of nose blowing was studied in a normal population (Hansen et al., 2002). So, in the future, it could be interesting to study more often these 2 parameters determining nose blowing (frequency and pressures) in patients with nasal complaints, signs and symptoms.

In conclusion, one can state that nose blowing is a typical human manoeuvre, if non-productive it is highly unphysiological, because it can induce extremely high pressures, especially when both nostrils are kept closed during this manoeuvre, and it could have an important role in the pathophysiology of chronic rhinosinusitis.

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REFERENCES

1. Bent JP, Cuilt-Siller, Kuhn FA (1994) The frontal cell as a cause of frontal sinus obstruction. Am J Rhinology 8: 185-194.
2. Brown SM, Lissner G (1995) Orbital emphysema. Ophtal Plast Reconstr Surg 11: 142-143.
3. Clement PAR (1994) Committee report on standardization of rhinomanometry. Rhinology 22: 151-155.
4. Graamans K (1980) Neus en luchtweg. Plethysmografische meting van luchtweerstanden bij klachten over neusobstructie. Thesis Amsterdam 1-106.
5. Gwaltney JM, Hendley JO, Phillips CD, Bass CM, Mygind N, Winther B (2000) Nose blowing propels nasal fluid in paranasal sinuses. Clinc Infect Dis 30: 387-391.
6. Hansen B, Mygind N (2002) How often do normal persons sneeze and blow the nose. Rhinology 40: 10-12.
7. Jang YJ, Koo TW, Chung SY, Park SG (2002). Bone involvement in chronic rhinosinusitis assessed by 99mTc-MDP bone SPECT. Clin Otolaryngol 27: 156-161.
8. Juhl HJ, Buchwald C, Bollinger B (2001). An extensive maxillary pneumosinus dilatans. Rhinology 39: 236-238.
9. Kennedy DW, Senior BA, Gannon F.H., Montone KT, Hwang P, Lanza DC (1998) Histology and histomorphometry of ethmoid bone in chronic rhinosinusitis. Laryngoscope 108: 502-507.
10. Lund VJ (2002) Clinical aspects of nasal polyposis - staging. In Nasal polyposis, edited by RS Mladina, Croatian Medical Association, Zagreb, Croatia, Varteks d.d., 107-113.
11. Oluwole M, White P (1996) Orbital floor fracture. ENT Journal 75: 169-170.
12. Omama S, Sugawara T, Oku T, Arai H, Niimura K (1995) Acute epidural haematoma. Neurol Surg 23: 639-642.
13. Postema CA, Huygen PLM, Le Cluse RGM, Wentges RThR (1980) The lateralisation percentage as a measure of nasal flow asymmetry in active anterior rhinomanometry. Clin Otolaryngol: 165-170.
14. Sobin A, Carenfelt C, Haverling M, Anggard A (1986) Pressure-induced expansion of the maxillary sinus. A rare entity. Rhinology 24: 283-286.
15. Stammberger H (1991) Secretion transportation. Role of ethmoidal prechambers. Chapter 2 in Functional Endoscopic Sinus Surgery. The Messerklinger Technique. B.C. Decker, Philadephia: 35-44.
16. Szücs E, Clement PAR (1998) Acoustic rhinometry and rhinomanometry in the evaluation of nasal patency of patients with nasal septal deviations. Am J Rhinology 12: 364-352.
17. Wolfensberger M (1984) Zur pathogenese des pneumosinus maxillaris dilatans. HNO 32: 518-520.
18. Wolfensberger M, Herrman P (1987) Maxillary pneumocele. Arch Otolaryngol Head Neck Surg 113: 184-186.

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