Rhinology, 38, 7–12, 2000

The nasal valves: changes in anatomy and physiology in normal subjects*

A.M. Shaida[1] and G.S. Kenyon[2]

[1] Department of Asthma and Respiratory Medicine, The London Chest Hospital, London, UK
[2] Department of Otolaryngology, St Bartholomews Hospital, West Smithfield, London, UK

SUMMARY

There is confusion in the literature concerning the physiology and pathology of the nasal valve, and some debate as to whether there is one valve or two. In an attempt to clarify these uncertainties we have measured nasal function by assessing nasal minimum cross-sectional area, inspiratory resistance and peak inspiratory flow under baseline conditions and after the application of a topical vasoconstrictor. These measurements were then repeated following the application of external and internal nasal splints. Whatever test was employed the results showed that vasoconstriction tended to be the most potent stimulus which changed nasal function producing significant expansion of the minimum cross-sectional area, a decrease in inspiratory resistance and an increase in peak inspiratory flow. External splints also increased the minimal cross sectional area but they had no effect on inspiratory resistance or on the tendency of the vestibular rim to collapse at high inspiratory flow rates. The tendency for lower lateral cartilage collapse was, however, prevented by internal splintage using alar dilators.
The results of this study suggest that there is an internal valve at the nasal isthmus where the principal alterations in airway patency follow changes in mucosal congestion, and a mobile external valve where airflow is limited by the tendency of the alar cartilages to collapse. These should be considered as separate entities with differing pathophysiology and these differences should be taken into account when treating patients with airway obstruction due to pathology at these sites.

Key words: Nose, Pathophysiology, Nasal Obstruction, Ventilation Tests, Nasal Valve

INTRODUCTION

The word valve derives from the Latin "valva" and was originally used to describe one leaf of a folding door. A structure which controls flow is therefore implicit in the concept of a valve, and a controlling function is also stressed in modern dictionary definitions which define a valve as a regulatory device which acts to control the flow of liquids or gases (Oxford English Dictionary, 1971).

The concept of a valve in the nasal airway was first suggested by Mink who used it to describe the region of maximal nasal resistance (Mink, 1920). Initially it was thought that this zone lay in a coronal plane at the junction of the upper and lower lateral cartilages and studies based on radiological findings seemed to confirm this hypothesis (Van Dishoeck, 1965). However luminal impressions of the nose suggest that the narrowest segment is oblique to the septum and is bound by the limen nasi laterally, the septum medially and the rim of the piriform aperture inferiorly (Bachman and Legler, 1972). More recently, acoustic rhinometry has shown that the site of minimal cross sectional area occurs approximately 2.3 cm from the nostril at the level of the anterior end of the inferior turbinate (Roithmann et al, 1995, Grymer 1991).

However an isthmus, or area of minimal cross section, is not necessarily valvular. Moreover implicit in these anatomical studies is the assumption that the minimum cross sectional area produces a zone of maximum nasal resistance. This is true for simple structures with laminar flow, such as pipes, but is not necessarily true for complex and irregular shapes such as the nose where flow patterns are turbulent and the internal surface is irregular. Some workers have therefore looked for the site of maximum resistance by measuring pressure changes along the length of the nose. In one such study dilating the nasal alae was found to reduce the total nasal resistance by only one third, which was taken to imply that the majority of resistance lies within the bony cavum at the piriform aperture (Haight and Cole, 1983). A computer aided simulation of nasal airflow has

* Received for publication March 4, 1999; accepted July 12, 1999

appeared to verify this conclusion suggesting that the highest resistance segment is bound inferiorly by the rim of the piriform aperture and is limited laterally by the caudal edge of the upper lateral cartilage and medially by the septum (Tarabichi and Fanous, 1993).

There are also dynamic components to nasal airflow. Mucosal congestion is the most obvious of these for it is widely known that vasoconstriction increases the minimum cross-sectional area and reduces nasal resistance. But limitation of airflow may also occur due to collapse of the cartilaginous lateral wall of the nose (Fomon et al, 1950) and this collapse has been reported to occur in the region of the caudal end of the upper lateral cartilages (Bridger and Proctor, 1970). Movement of the alar cartilages with respiration has also been documented (Cole et al, 1985) and stenting of the alae is known to prevent collapse and to increase peak inspiratory nasal flow (Pertuze et al, 1991). That the alar rim has a probable valvular function has also been emphasised by the finding that this area may collapse in patients with facial palsy (May et al, 1977), and it is also known that increased electrical activity in the alar muscles occurs during inspiration (Haight and Cole, 1983). A single site for the nasal valve therefore seems unlikely on empirical grounds.

In order to characterise some of these components we have measured various parameters of the nasal airway and airflow in normal volunteers to ascertain the position of the valve or valves. Changes in nasal architecture have also been measured following external and internal splinting and following the use of decongestants. In making these measurements we were conscious that any one single clinical test tends simultaneously to assess several parameters of nasal function. However we made the assumption that measurement of the minimal cross sectional area acts as a proxy for the degree of airway obstruction imposed by the nasal isthmus and that peak inspiratory flow demonstrates the tendency of the lateral nasal wall to collapse. We also included measurements of nasal resistance as these give an overall impression of nasal airway performance and because resistance is a parameter that has been measured in previous work pertaining to the nasal valve.

MATERIALS AND METHODS

Ten healthy subjects with no nasal symptoms or abnormalities on nasal examination were included in the study. All volunteers were subject to the same test battery. This consisted of measurements of the minimum nasal cross sectional area, the nasal inspiratory resistance and the nasal peak inspiratory flow before and after the use of a topical decongestant and the application of external ("Breathe Right" Nasal Strips, 3M Healthcare, UK) and internal nasal splints (Francis alar dilators). In all cases patients were tested in a controlled environment at an ambient temperature of 26°C and a relative humidity of 30%.

Measurements of minimal cross-sectional area were undertaken using an A1 acoustic rhinometer (GM Instruments). To effect this a 7cm tapered probe was used with a coupling gel to ensure a good seal in the external nares while avoiding vestibular distortion. Ten measurement curves were recorded for each nostril and the mean curve used to determine the minimum cross-sectional area. Nasal inspiratory resistance and flow were measured by active anterior rhinomanometry at a trans-nasal pressure of 150 Pascals using a NR8 Rhinomanometer (GM Instruments). A soft facemask was used to ensure a good seal and to avoid distortion of the nose. Peak nasal inspiratory flow was measured using a mask attached to an Autolink spirometer (PK Morgan Ltd). Four consecutive flow-volume loops were recorded and the maximum peak inspiratory flow determined.

Measurements were made in the resting state and were then repeated following the application of external and internal splints. For external splintage Breathe Right Nasal Strips were placed across the caudal border of the upper lateral cartilage, and for internal splintage Francis alar dilators of an appropriate size were inserted into the external nose to prevent collapse of the vestibular rim. To assess the effects of vascular decongestion all measurements were repeated 15 minutes after the application of three sprays of 0.1% xylometazoline to each nostril.

For the purposes of statistical analysis, the sum of the left and right minimum cross-sectional areas was used to calculate the total nasal cross-sectional area. Overall nasal inspiratory resistance and flow were automatically calculated by the software built in to the rhinomanometer based on the individual values for each nostril. The data was then assessed by statistical tests of normality to determine its distribution. If this was normal (Gaussian) then a related samples t-test was used to for comparison; if a normal distribution was not found then a Wilcoxon signed rank test was used for analysis. All calculations were performed after the data had been entered to a computer based statistics package (SPSS Stats, SPSS Inc., USA).

RESULTS

There were 7 males and 3 females included in the study. Their mean age was 33.8 years (range 22-50 years). All were in good health with no complaint of nasal obstruction or rhinosinusitis. None had a personal history of atopy or aspirin sensitivity. No subject included was a smoker.

Cross-sectional area:

Measurements of the minimal cross sectional area and changes following decongestants and external splintage are tabulated in Table 1 and are shown in Figure 1. From this data it can be seen that the application of an external Breathe-Right nasal splint produced a 14.2% increase in the minimal cross sectional area – a change which was significant ($p=0.002$).

Topical vasoconstriction produced a much more marked increase in cross sectional area with a 54% increase over baseline recordings ($p=0.001$). When a splint was applied after vasoconstriction the additional increase due to the splint (14.6%) was similar to the effect of the splint when applied to the undecongested nose and was, again, statistically significant ($p=0.005$).

From this it can be concluded that the application of external splints increases minimum cross sectional area by approximately 14 % and that this effect is seen in both the normal and the decongested nose. This increase is statistically significant but is small in comparison to the effect of a topical vasoconstrictor. The minimum cross-sectional area occurred at a mean distance

Table 1. Minimum cross-sectional area.

Area	Mean (cm^2)	S.D.		Mean Difference	95% C.I. of difference	Change	Sig. (2-tail t-test)
Normal (TAN)	1.262	0.333					
External Splint (TAS)	1.441	0.328	TAN-TAS	0.179	0.085 -0.273	**14.2%**	**p=0.002**
Vasoconstricted (TAV)	1.943	0.581	TAN-TAV	0.681	0.384 -0.978	**54%**	**p=0.001**
Vasoconstricted + External Splint (TAVS)	2.227	0.406	TAV-TAVS	0.284	0.112 -0.456	**14.6%**	**p=0.005**

Table 2. Mean distance inside the nose of minimum cross-sectional area.

	Mean (cm)	S.D.	Difference of means	95% Confidence Interval of difference	Significance (2 tail t-test)
Normal	2.28	0.349			
Vasoconstricted	2.12	0.338	0.16	0.071 – 0.249	**p=0.001**

Table 3. Inspiratory resistance.

	Mean (Pa/cc/s)	S.D.		Mean Difference	95% C.I. of difference	Change	Sig. (2-tail t-test)
Normal (IRN)	0.250	0.085					
External Splint (IRS)	0.255	0.064	IRN-IRS	0.006	-0.06 - 0.07	**2%**	**p=0.846 (t-test)**
Vasoconstricted (IRV)	0.182	0.052	IRN-IRV	–0.068	*	**-27.2%**	**p=0.059***
Vasoconstricted + External Splint (IRVS)	0.184	0.061	IRV-IRVS	0.002	*	**1%**	**p=0.878***

* Wilcoxon signed rank test

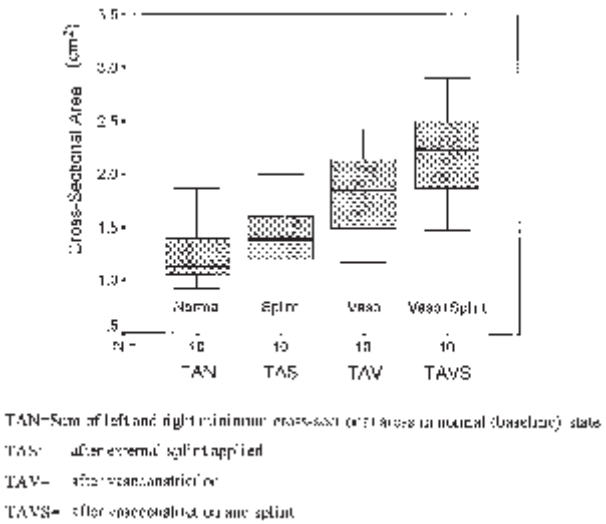

Figure 1. Minimum Cross-Sectional Area.

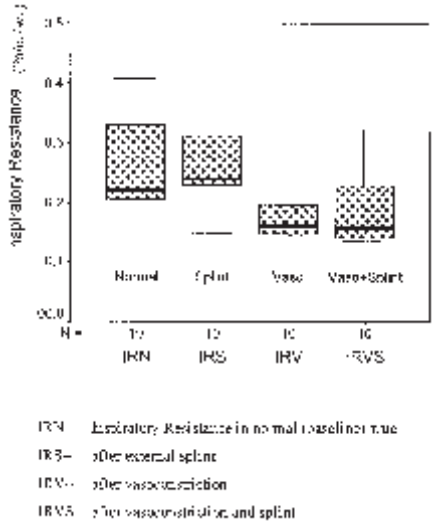

Figure 2. Inspiratory Resistance.

of 2.28 cm inside the nose before vasoconstriction and 2.12 cm after vasoconstriction, a 0.16 cm change, which was again statistically significant (Table 2). Similar changes have been reported previously in other studies (Roithmann et al, 1995).

Inspiratory Resistance:

The effects of the decongestant on inspiratory resistance were variable. The majority of subjects showed the expected fall in resistance but in one case the resistance appeared to rise, which may have been due to an abnormal reaction to the xylometazoline.

Table 4. Peak inspiratory flow.

	Mean (L/s)	S.D.		Mean Difference	95% C.I. of difference	Change	Sig. (2-tail t-test)
Normal (PIFN)	3.083	1.201					
External Splint (PIFS)	3.141	1.198	PIFN-PIFS	0.058	*	**1.9%**	**p=0.646***
Vasoconstricted (PIFV)	4.361	1.237	PIFN-PIFV	1.278	*	**41.5%**	**p=0.013***
Vasoconstricted + External Splint (PIFVS)	4.31	1.489	PIFV-PIFVS	–0.051	–0.3 - 0.198	**-1.2%**	**p=0.654**

* Wilcoxon signed rank test

Table 5. Effect of alar dilator on peak inspiratory flow.

	Mean	S.D.		Mean Difference	95% C.I. of difference	Change	Sig. (2-tail t-test)
Normal (PIFN2)	3.101	0.853					
Alar Dilator (PIFN2Sp)	3.872	0.863	PIFN2 – PIFN2Sp	0.771	0.499 -1.043	**24.9%**	**p<0.001**
Vasoconstricted (PIFV2)	4.144	1.446					
Vasoconstricted + Alar Dilator (PIFV2Sp)	5.370	1.470	PIFV2 – PIFV2Sp	1.226	0.769 -1.682	**29.6%**	**p<0.001**

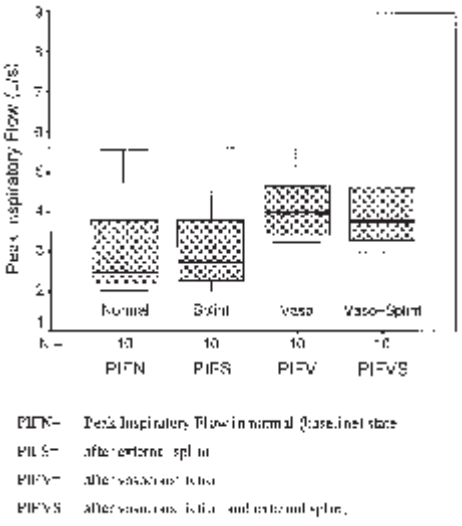

Figure 3. Peak Inspiratory Flow.

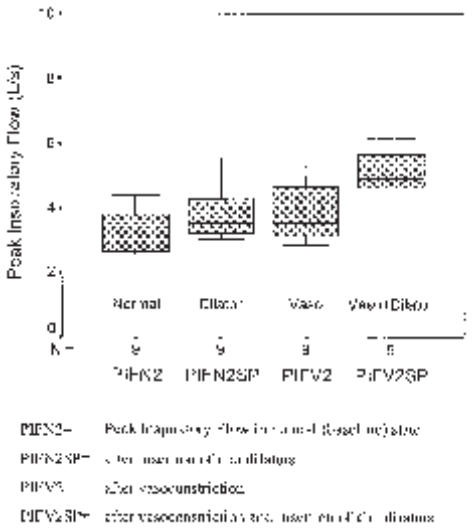

Figure 4. Peak Inspiratory Flow after Alardilators.

As would be expected the overall effects of vasoconstriction was to produce a marked change in inspiratory resistance (27.2%) although this failed to attain statistical significance. From the same study it was shown that the application of external splints produced a minimal effect on mean resistance whether applied before or after vasoconstriction (Figure 2 and Table 3).

Peak Inspiratory Flow:

The application of an external splint did not produce a significant effect on peak nasal inspiratory flow either before or after vasoconstriction (Figure 3 and Table 4). Once again the use of a topical vasoconstrictor was capable of provoking large and statistically significant increases in peak flow (41.5%: Table 4).

During these experiments with peak flow and external splints it was apparent that flow was being limited by the collapse of the lower lateral cartilages against the septum. The measurements were therefore repeated following insertion of Francis alar nasal dilators. The use of such dilators produced an increase of 24.9% in peak inspiratory flow in the normal nose and an increase of 29.6% when the same experiment was repeated in the decongested nose (Figure 4 and Table 5). The overall effects of alar dilatation on peak flow were therefore similar in magnitude whether measurements were made with or without vasoconstriction.

DISCUSSION

Since the original description of a nasal valve there has been much confusion as to its location and purpose. The original descriptions concentrated on anatomy and suggested that the area of the isthmus, and hence of maximal resistance, constitut-

ed the valve (Mink, 1920; Van Dishoeck, 1965; Bachman and Legler, 1972). The most recent technique available to measure this area - acoustic rhinometry - has shown that this plane lies within the nose at a level which corresponds to the level of the anterior end of the inferior turbinate (Roithmann et al, 1995; Grymer 1991).

However this area does not appear, at first sight, to be valvular. Airflow may be limited by collapse of the cartilaginous lateral walls of the nose, as has been suggested by some authors (Fomon et al, 1950). However even if that is so it is not clear whether this inward movement principally occurs at the level of the upper lateral cartilages or at the vestibular rim (Bridger and Proctor, 1970; Cole et al, 1985). Internal stenting of the alae to prevent collapse has been shown to increase peak inspiratory nasal flow in some studies (Pertuze et al, 1991) but the alar rim is not synonymous with the valve as previously described. There is therefore some doubt as to whether the lateral walls in the region of the nasal valve move with respiration in order to contribute to valvular function.

Our results demonstrated that the site of minimum cross sectional area is located approximately 2.3 cm from the external nares as measured by acoustic rhinometry and this area in the normal nose measures 0.63 cm^2 . These findings are consistent with other studies (Roithmann et al, 1995; Grymer et al, 1991). Our results also show that the upper lateral cartilages that overlie this area can be stented by external splintage and, if this is done, the minimal cross sectional area will passively increase. However this increase does not act to change the overall resistance to airflow and is not sufficient to resist alterations in peak flow produced by a maximal inspiratory effort. Moreover the effect of an external splint is small when compared to alterations in cross sectional area produced by vasoconstriction. It therefore seems likely that the state of mucosal engorgement is the principal driver of airway patency at the isthmus of the normal nose and that skeletal collapse only contributes significantly if there is some pathology of the lateral nasal wall. Scarring of the angle between the upper lateral and septum with weakening of the upper laterals may follow rhinoplasty if the upper laterals are dissected free of the dorsal septum. Such pathology may be corrected by spreader grafts as described by Sheen (Sheen, 1984). Alternatively, if the inferior rim of the piriform aperture is narrowing the airway then removal of the bony rim will serve to increase the surface area of the valve (Tarabichi and Fanous, 1993: Woodhead, 1995).

However in the normal nose valvular activity at the isthmus seems to be principally driven by the state of vascular engorgement of the head of the inferior turbinate. This conclusion is corroborated by other work which has reported that shrinkage of the anterior end of the inferior turbinate has the effect of moving the minimal cross sectional area anteriorly and posteriorly by up to 5mm (Haight and Cole, 1983). Moreover it has also been reported that profound changes in cross sectional area follow turbinate trimming (Jones et al, 1988). It therefore seems that valvular activity at the isthmus is primarily due to the inferior turbinate and to the engorgement of its overlying mucosa. Movement of the lateral wall, in the normal nose, would therefore appear to have a relatively minor role in determining airway patency at this site.

If this area acts as a valve then it would also appear that there is valvular activity at the level of the lower lateral cartilages. In the course of our experiments we observed a tendency to collapse at the vestibular rim and this was not inhibited by external splintage of the lower lateral cartilages. However this tendency to collapse was inhibited by internal splints, in the form of alar dilators, and these produced significant increases in peak flow – both in the normal and decongested nose. Factors which cause narrowing of the airway, such as septal deviation, may result in higher airflow velocity and provide an increased tendency to alar collapse (Bridger and Proctor 1970; Elwany and Thabet 1996). Moreover when a patient has a facial nerve palsy (May et al, 1977), a developmental anomaly such as malpositioning of the lateral crura or weakening due to over resection (Bridger, 1981), the rigidity of the alar rim might be so reduced that alar collapse tends to occur at physiological flow rates. Although the nose is supposed to act as a Starling resistor, and display increased resistance with increased flow, this tendency to collapse will be enhanced if the transmural pressure gradient is raised and structural rigidity is compromised (Bridger and Proctor, 1970).

It would therefore seem that there are two areas in the nose that can display valvular activity. In the normal nose the isthmus, or internal valve, primarily acts as a valve due to alterations in the state of mucosal engorgement of the inferior turbinate. By contrast movement of the lower lateral cartilages, the external valve, acts to regulate airflow at the vestibular rim. In the normal nose these areas are inter-linked through the fibrous connections that join the caudal ends of the upper lateral and cephalic rims of the lower lateral cartilages. Presumably these connections act so that alar flaring from the dilator muscles around the vestibular rim prevent lower lateral cartilage collapse and that such flaring serves also to stent the caudal ends of the upper lateral cartilages. However there are clinical cases where these two areas should be viewed separately.

In conclusion there seem to be two valvular mechanisms in the nose. One of these is at the level of the nasal isthmus and is principally altered following changes in vascular congestion of the mucosa. The other is located at the vestibular rim and displays a tendency to collapse at high flow rates in normal subjects. Our study suggests that these two areas should be considered as distinct entities and confirms the idea, expounded by others, that nasal obstruction may well occur due to pathology at these sites (Constantian and Clardy, 1996).

REFERENCES

1. Bachmann, W. and Legler, U. (1972) Studies of the structure and function of the anterior section of the nose by means of luminal impressions. Acta. Otolaryngologica (Stock). 73: 433-442.
2. Bridger, G.P. (1981) Split rib graft for alar collapse. Archives of Otolaryngology, Head and Neck Surgery, 107:110-113.
3. Bridger, G.P., Proctor, D.F. (1970) Maximum nasal inspiratory flow and nasal resistance. Annals of Otology, 79:481-488.
4. Cole, P., Haight, S.J., Love, L., Oprysk, D. (1985) Dynamic components of nasal resistance. American Review of Respiratory Diseases, 132:1229-1232.

5. Cole, P., Chaban, R., Naito, K., Oprysk, D. (1988) The obstructive nasal septum. 114:410-412.
6. Constantian, M.B., Clardy, R.B. (1996) The relative importance of septal and nasal valvular surgery in correcting airway obstruction in primary and secondary rhinoplasty. Plastic and Reconstructive Surgery, 98:38-58.
7. Van Dishoeck, H.A.E. (1965) The part of the valve and turbinates in total nasal resistance. International Rhinology, 3:19-26.
8. Elwany, S., Thabet, H. (1996) Obstruction of the nasal valve. Journal of Laryngology and Otology, 110:221-224.
9. Fomon, S., Gilbert, J.G., Caron, A.L., Segal, S. (1950) Collapsed ala. Archives of Otolaryngology, Head and Neck Surgery, 51:465-484.
10. Grymer, L.F., Hilberg, O., Pederson, O.F. et al. (1991) Acoustic rhinometry: values from adults with subjective normal nasal patency. Rhinology 29:35-47.
11. Haight, J.S.J., Cole, P. (1983) The site and function of the nasal valve. Laryngoscope, 93:49-55.
12. Jones, A.S., Wight, R.G., Stevens, J.C., Beckingham, E. (1988) The nasal valve: a physiological and clinical study. Journal of Laryngology and Otology 102: 1089-1094.
13. May, M., West, J.W., Hinderer, K.H. (1977) Nasal obstruction from facial palsy. Archives of Otolaryngology, Head and Neck Surgery, 103:389-91.
14. Mink P.J. (1920) Physiologie der oberen Luftwege, Leipzig. Cited by Uddstromer M. (1940) Acta Otolaryngologica, Stock (Suppl 42).
15. Pertuze, J., Watson, A., Pride, N.B. (1991) Maximum airflow through the nose in humans. Journal of Applied Physiology, 70(3): 1369-1376.
16. Roithmann, R., Cole, P., Chapnik, J., Shpirer, I., Hoffstein, V., Zamel, N. (1995) Acoustic rhinometry in the evaluation of nasal obstruction. Laryngoscope 105:275-281.
17. Sheen, J.H. (1984) Spreader graft: A method of reconstructing the roof of the middle nasal vault following rhinoplasty. Plastic and Reconstructive Surgery, 73(2):230-237.
18. Tarabichi, M. Fanous, N. (1993) Finite element analysis of airflow in the nasal valve. Archives of Otolaryngology, Head and Neck Surgery, 119:638-642 .
19. The Oxford English Dictionary. 3rd Edition (1971) Oxford University Press.
20. Woodhead, C.J. (1995) Piriform aperture surgery for alar collapse. Clinical Otolaryngology. 20: 74-79.

G S Kenyon, MD FRCS
Department of Otolaryngology,
St Bartholomews Hospital,
West Smithfield, London EC1A 7BE
United Kingdom.