British Journal of Plastic Surgery (1987), **40**, 377–383

The effect of tongue position on division of airflow in the presence of velopharyngeal defects*

W. G. SELLEY, M-C. ZANANIRI, R. E. ELLIS and F. C. FLACK
Medical Physics Group, Department of Physics, University of Exeter

Summary—Results of studies of nasal to oral airflow ratios are reported using simple and accurate anatomical models to record the effect of differing positions of lips, tongue and soft palate, with particular reference to the effect of the position of the dorsum of the tongue and various sizes of velopharyngeal defect.

The resistances to airflow produced by the labial, palatolingual, velopharyngeal and naral valves were found to be interdependent. Variations in tongue position alone could allow the same nasal airflow during a more than three-fold variation in the size of velopharyngeal defects.

The degree of nasal escape of air which is responsible for the typical "cleft palate" type of speech cannot be assessed by observation of the size of the velopharyngeal defect alone.

Cleft palate type speech is characterised by excessive abnormal nasal escape of air and by an abnormal resonance compared to local speakers and may be treated by plastic surgery or speech therapy (or a combination of the two). The more accurate the assessment the better will be the choice of treatment. Although recognition of nasal airflow is simple, quantitative interpretation of the findings is complex. It is generally recognised that defective velopharyngeal valving produces modifications in speech sounds (Subtelny *et al.*, 1961; Rolnik and Hoops, 1971; Warren *et al.*, 1985), but there is often an apparent lack of correlation between the size of the defect and the amount of abnormal nasal escape of air. Nasendoscopy and video-radiography permit accurate assessment of a velopharyngeal defect and it is desirable to discover more about the relationship between these findings and the degree of nasal escape.

This research was undertaken in an attempt to quantify airflow in models representing patients with velopharyngeal defects.

Much of the quantitative research on vocal tract airflow has been performed by Warren *et al.* (1985) in studies on a "semi-anatomical" perspex model. This consists of a large tube to represent a mouth and two smaller tubes to represent the nasal airway, connected to a yet larger tube representing the pharynx. The tubes were constructed so as to offer resistances to airflow comparable to known values in normal individuals. The sizes of the velar and oral ports could be changed (Warren and Devereux, 1966). The results were interpreted as showing that, in the presence of velopharyngeal incompetence, the effects of oral port constriction, nasal pathway resistance and respiratory effort can mask defects of velopharyngeal function (Warren and Ryon, 1967).

Due to the fundamental importance of Warren's work, it was felt necessary to repeat it and to extend it by constructing and using a more accurate anatomical model based on the anatomy of one of the authors, which could be checked for accuracy against him. In this way both the copy of Warren's model and the anatomical model could be compared, the recording equipment tested and the work extended by including a model tongue of variable conformation.

We attempted to answer the questions:

(i) What are the factors which control direction of airflow?
(ii) What is the relationship between airflow and the size of the velopharyngeal defect—"hole size"?

We also wished to test the hypothesis that a part of the success in treating abnormal nasal escape with a Palatal Training Appliance (Selley, 1979) is due to the loop of the appliance modifying tongue humping and permitting an improved oral airflow.

---

*Supported by a grant from The Northcott Devon Medical Foundation.

Material and method

A perspex copy of Warren's model was made using the dimensions quoted in his paper (Warren and Devereux, 1966). Our anatomical model was based on a CAT scan of the head of one of the authors (Fig. 1). Sixteen horizontal CAT scan "slices" each of 3 mm thickness were obtained from the larynx to the orbits. Further radiation was thought to be unwise, so these films were supplemented by 40 vertical CAT scan "slices" taken at 2 mm intervals from a recently deceased person of the same age, to show the minute details of the nasal cavity.

Images from the CAT scan transparencies were enlarged to life size and projected on to perspex sheets the same thickness as the slices; the cavities were marked and then carefully cut out. The dental arches were represented by acrylic models cast from dental impressions. Different tongue shapes were modelled in plasticine. A separate block of perspex was used to model the nares in the same way and when these sheets of perspex and the dental models were assembled they produced an airtight model of the upper respiratory tract, which was demountable to test the nasal and oro-pharyngeal sections separately, and to allow alterations to be made (Fig. 2).

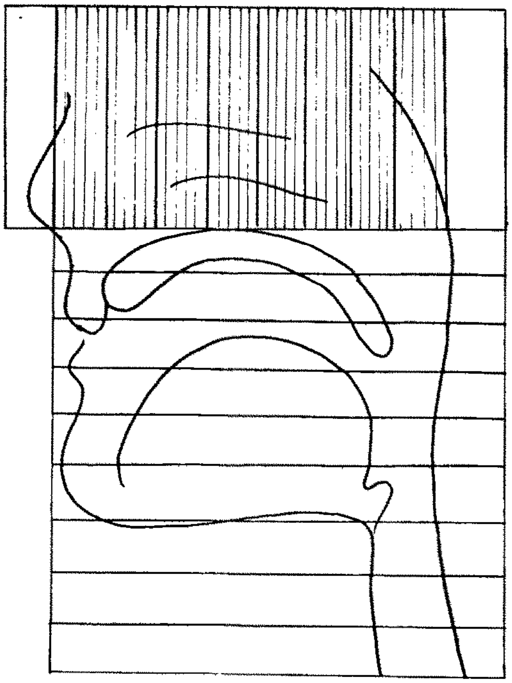


Fig. 1

Figure 1—Sketch illustrating division of model of upper respiratory tract into 9 horizontal sheets for the oral part and 40 vertical sheets for the nasal part, together with a separate block for the nasal region.

The dental models were positioned with the teeth separated and the anterior oral port was produced by adding one of five plates drilled with holes increasing from 5.0 to 18.5 mm in diameter, giving port areas of 20 to 270 mm^2. The soft palate position on the model was permanently in the relaxed position and the velopharyngeal orifice could be varied by means of three plates with holes of 16, 55 and 122 mm^2. Three plasticine "tongues" produced *effective* lingual port sizes of 30, 370 and greater than 400 sq mm, *i.e.* although the actual shape and size of the hole varied with tongue model, a calculation was made to derive the value of the diameter of a single, circular, sharp-edged hole which would offer the same resistance to flow, known as the "effective hole size". A circular mid-line "oro-nasal fistula" with an area of 78.5 mm^2 was also inserted into the model for further tests.

Through the "trachea" steady total airflows from 0 to 25 litres per minute were introduced and the corresponding nasal airflow was measured by two thermistors (Ellis *et al.*, 1978) at the nostrils. Pharyngeal air pressure was measured by a catheter connected, through the perspex, to a pressure transducer. The presence of the measuring equipment added no discernible extra resistance to the airflow.

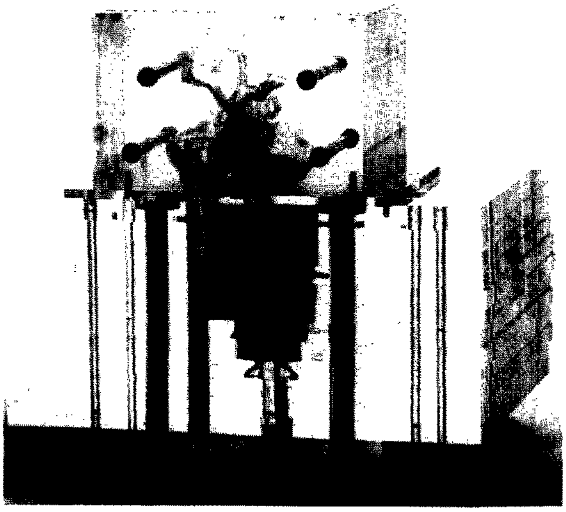


Fig. 2

Figure 2—Photograph of assembled anatomical model in perspex of upper respiratory tract.

Results

Figure 3 shows the results from our simple model compared to those obtained by Warren, the flow being 15 litres per minute in each case. The graphs have similar shapes, but our pressure readings were consistently, significantly lower.

All subsequent figures relate to steady total flows of 25 litres per minute (0.42 litres per second) for two reasons, (a) to enable comparison with Warren's data obtained at the same flowrate and (b) because it is a value in the mid-range of flows commonly seen in cases of severe nasal escape.

On separating the nasal portion from the oral portion of the anatomical model and testing the resistance offered by the nasal airway alone, it was found to be equivalent to an effective hole size of 45 sq mm, 65% of which was produced by the two nares, where constrictions caused a narrowing from a nasal cavity with an effective hole size of 160 mm^2 to 35 mm^2 on each side. These findings compare well with those of Proctor (1982), *viz.* 130 mm^2 and 30 to 40 mm^2 respectively (35 mm^2 has a 6.7 mm diameter hole). The turbulent nature of the flow was investigated using the equation: pressure = HR × (Flow)^n where HR, the hydraulic resistance, depends on the properties of the hole and where n = 2 for fully turbulent flow and n = 1 for fully laminar flow. (The authors have produced a more comprehensive discussion of these matters, which is available on request.) Intermediate values suggest a mixture of both types of flow. Without the naral portion, airflow was found to be not fully turbulent in the nasal cavity, giving a value of n = 1.5, but when that portion, with its 90° bend, was replaced the flow became completely turbulent (n = 2).

The model was reassembled and pressure and flow measurements were taken with different velar port sizes. When the effective velar orifice exceeded 45 mm^2 (*i.e.* that of the nasal passages), little increase in nasal flow was recorded.

Comparison between the author and his anatomical model fitted with normal ports showed agreement within 5% for both resistance to airflow and oropharyngeal pressures.

Further flow tests were carried out with different oral and velar port sizes and different tongue positions. Figure 4 shows the four "ports" where resistance to airflow can be varied. Some of the results are illustrated on schematic anatomical diagrams in Figures 5 to 9 in order to demonstrate features of clinical interest.

Analysis of the individual figures shows that tongue position had no effect on nasal airflow if the anterior oral port was small, whether the velopharyngeal port was small or large (Figs 5 and 7). However, humping of the tongue caused a significant increase in nasal airflow with both small and large velopharyngeal ports when the anterior oral port was large (Figs 6 and 8).

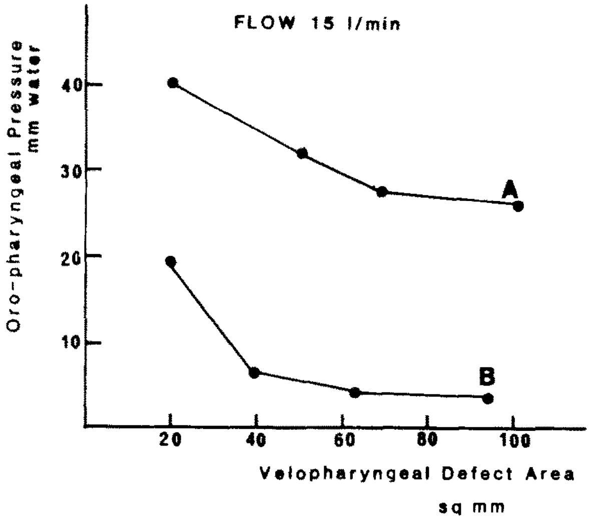


Fig. 3

Figure 3—Oropharyngeal pressure versus velopharyngeal area in (A) Warren's model and (B) our "simple" model.

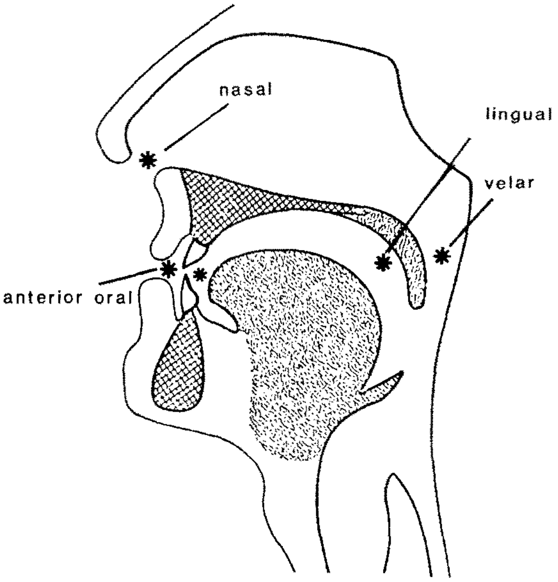
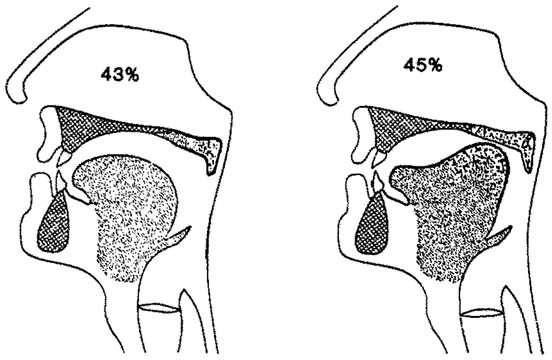


Fig. 4

Figure 4—Sketch showing the four "ports" discussed in the text.

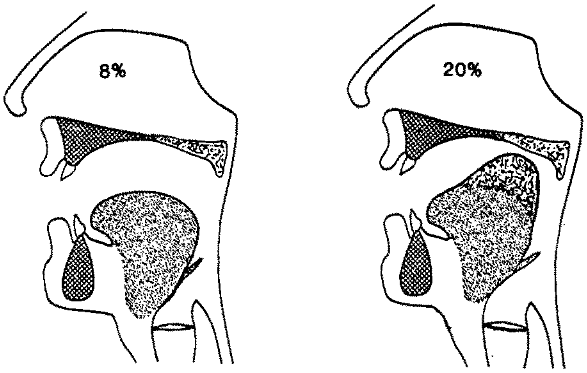


Tongue	*Normal*	*Humped*
Lingual	>400	30
Nasal	45	45
Velar	16	16
Ant. oral	18.5	18.5

(Effective port sizes in square mm.)

Fig. 5

Figure 5—Comparison of nasal airflows as percentages of tracheal flows for small anterior oral and velar ports with (left) normal tongue and (right) humped tongue.

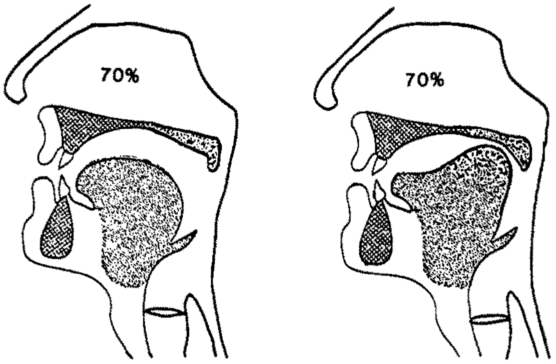


Tongue	*Normal*	*Humped*
Lingual	>400	30
Nasal	45	45
Velar	55	16
Ant. oral	217	217

(Effective port sizes in square mm.)

Fig. 6

Figure 6—Comparison of nasal airflows as percentages of tracheal flows for large anterior oral ports and the same velar defects as Figure 5. (left) normal tongue and (right) humped tongue.

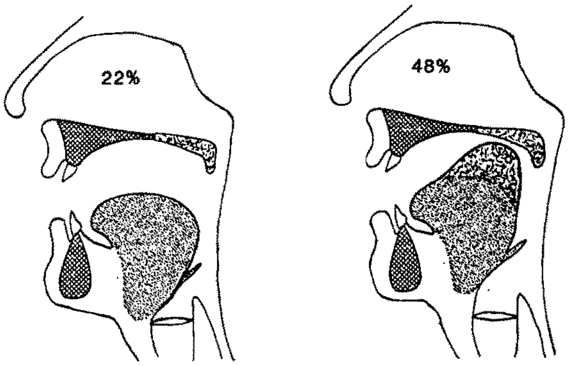


Tongue	*Normal*	*Humped*
Lingual	>400	30
Nasal	45	45
Velar	55	55
Ant. oral	18.5	18.5

(Effective port sizes in square mm.)

Fig. 7

Figure 7—Comparison of nasal airflows as percentages of tracheal flows for small anterior oral ports and large velar ports with (left) normal tongue and (right) humped tongue.

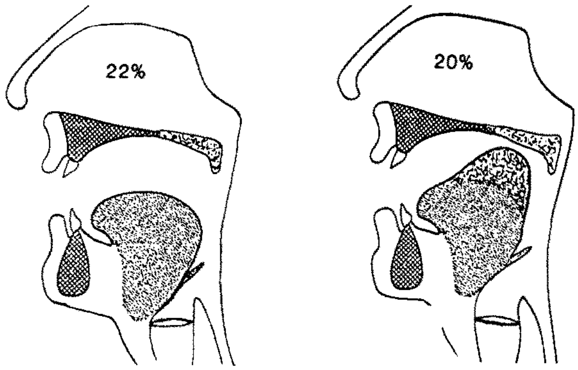


Tongue	*Normal*	*Humped*
Lingual	>400	30
Nasal	45	45
Velar	55	55
Ant. oral	217	217

(Effective port sizes in square mm.)

Fig. 8

Figure 8—Comparison of nasal airflows as percentages of tracheal flows for large anterior oral ports and the same velar defects as Fig. 7. (left) normal tongue and (right) humped tongue.



Tongue	*Normal*	*Humped*
Lingual	>400	30
Nasal	45	45
Velar	55	16
Ant. oral	217	217

(Effective port sizes in square mm.)

Fig. 9

Figure 9—Comparison of nasal airflows as percentages of tracheal flows, (left) normal tongue with large velar and large anterior oral ports and (right) humped tongue with small velar and the same anterior oral port.

If these results are examined another way, referring to Figure 9 it will be noted that nasal airflow is roughly the same at 20% with two different effective velar defects of 55 and 16 mm^2, but with a flat tongue for the large defect and a humped tongue for the small defect.

In tests with the fistula, it behaved like another port, and because oral pressures generally were greater than nasal pressures, air flowed from the mouth into the nose. There was one exception when, with an incompetent velar port and tongue humping, an effective lingual port size of approximately 30 mm^2 was produced and air flowed from the nose into the mouth. This flow was reversed when the tongue humping was corrected and nasal airflow therefore increased.

Discussion

The simple "semi-anatomical" model described by Warren is useful to study some aspects of airflow in the vocal tract but does not produce the same degree of turbulence seen in the fully anatomical model and in the human subject, and cannot be modified to reproduce all anatomical variations. We were unable to reproduce Warren's own early measurements, possibly because they were not corrected for the pressure drop across the pneumotachograph he used to measure the flow.

Measurements on our fully anatomical model showed that a velopharyngeal orifice of effective area, 40 to 60 mm^2, produces the same resistance to flow as the nasal passages complete with the nares. This, therefore, represents the critical velopharyngeal aperture; with larger values the nose controls the flow.

Nasal resistance depends on the size of the nares and it is important to realise that a small intra-nasal catheter or any other item passed into the nose during investigations will constrict this opening still further and will have a marked effect on any calculations of nasal airflow. For example, halving the effective hole size of the nares halves the nasal airflow for the same oropharyngeal driving pressure. Conversely, enlarged nostrils and absence of the right-angled bend at the nares will remove control of large nasal flows from the nose to the velopharyngeal defect.

The question of major interest is: how small a velopharyngeal defect will still produce cleft palate type speech? A figure of 20 sq mm has been suggested based on model studies (Warren and Ryon, 1967) and also on some clinical studies (Warren, 1964a and b).

Our study has shown that steady nasal airflow, in our complete anatomical model, is fully turbulent, unlike Warren's simple model in which nasal flow is only partially turbulent. In view of the relationship: pressure drop is proportional to (flow)^n, the effective resistance will rise more rapidly as airflow increases in the anatomical model than in Warren's model.

Our results are presented pictorially rather than in graph form, as a guide to illustrate the factors responsible for the "wastage" of airflow nasally. Since total flow varies during speech, the actual amount of nasal flow, even for a fixed anatomical situation, will vary in a complicated manner. For this reason our nasal flow data have been presented as a percentage of total flow.

The effect that a velopharyngeal defect will have on nasal airflow and intra-oral pressure will depend on the resistances produced by the nasal, lingual and anterior oral ports, together with the turbulence produced. A change in tongue position alone has been shown in these experiments to allow more than a three-fold variation in the effective size of such a defect and yet produce the same amount of nasal airflow. It can also cause dramatic changes in nasal airflow when the velopharyngeal defect stays constant. These findings confirm the suggestion of Machida (1967) that the position and shape of the tongue should not be neglected in studying the aerodynamic relationship between articulatory movements and airflow rates. To predict nasal airflow from the size of a velopharyngeal defect, it would be necessary to measure the resistances of all the other ports described, determine the flow at the appropriate time and assess the degree of nasal turbulence. Furthermore, an effective hole size cannot be calculated from measurement (*e.g.* by nasendoscopy) of an observed area of velopharyngeal defect due to the complex shapes of the air passages. Due to the speed of speech, data deduced from relatively static investigations such as nasendoscopy and video-radiography may alter transiently, for example with a sudden brief humping of the tongue or an incorrectly timed movement of the soft palate. The resistance of the nasal passages varies in individuals due to congestion and reciprocal cyclic changes in each side (Eccles, 1978).

There is therefore no direct relationship between an observed velar defect and nasal airflow and it would be difficult to estimate accurately the effect the defect would have on flow, even taking into consideration the other controlling factors.

The third aspect of the investigation was to consider a possible mode of action of the Palatal Training Appliance. The appliance consists of an acrylic base plate carrying a metal wire loop bent into the shape of a "U" and adjusted *just* to touch the soft palate at rest in the region of the maximum lift. We have found it to be an effective form of treatment for dysarthria presenting as hypernasal speech. One possible mode of action by which the appliance helps to reduce nasal escape is that the presence of the loop reduces tongue humping, perhaps because the palato-glossal region becomes "re-sensitized" (Selley, 1979). Our findings have shown that for open mouth sounds, where the lingual port is the dominant factor, lowering a humped tongue will reduce the resistance and increase the oral flow, so decreasing nasal flow. It is likely that the anterior oral port would dominate any lingual resistance for fricatives and plosives. However, if tongue humping was present during the build-up for these sounds then there might be a significant pressure drop across the tongue, reducing the pressure available for plosive action.

Conclusion

In this project, which was primarily concerned with the position and action of the dorsum of the tongue and its relationship to nasal airflow in the presence of a velopharyngeal defect, the following observations have been made.

The airflow recordings of the anatomical model coincided closely with those recorded of the subject on whom the model was based.

The model demonstrated clearly the interdependence of the four major resistances (ports) in influencing flow.

In the presence of velopharyngeal incompetence, the amount of nasal airflow (escape) was directly influenced by the resistance due to the lips, tongue and nares.

A velopharyngeal defect which was larger than the effective area of the normal constriction, including the right-angled bend, of the nares had its flow properties dominated by the latter.

Dorsal tongue humping has a marked effect on the amount of nasal airflow (escape) for oral (open mouth) sounds. It is likely that it may prevent the generation of adequate intra-oral air pressure for plosives, fricatives and affricates in rapid speech, especially where there is a co-existing velopharyngeal defect.

The hypothesis that the Palatal Training Appliance by reducing tongue humping decreases nasal escape has been shown to be reasonable.

The dynamics of turbulent airflow in the vocal tract are shown to be of a highly complex nature and the amount of nasal escape cannot be related to observed size of velopharyngeal defects.

The mathematical calculation of the "effective hole size", while a useful guide to clinical appraisal, may not coincide with the defect endoscopically observed.

We believe that this study has important implications for those concerned with the surgical treatment of hypernasal speech.

Acknowledgements

The authors are very grateful for the grant awarded by The Northcott Devon Medical Foundation, which made this research possible.

References

Eccles, R. (1978). The central rhythm of the nasal cycle. *Acta Otolaryngology*, **68**, 464.

Ellis, R. E., Flack, F. C., Curle, H. J. and Selley, W.G. (1978). A system for the assessment of nasal airflow during speech. *British Journal of Disorders of Communication*, **13**, 31.

Machida, J. (1967). Airflow rate and articulatory movement during speech. *Cleft Palate Journal*, **4**, 240.

Proctor, D. F. (1982). *Upper Airway Physiology and the Atmospheric Environment, The Nose*. Amsterdam: Elsevier Biomedical Press.

Rolnik, M. I. and Hoops, H. R. (1971) Plosive phoneme duration as a function of palato-pharyngeal adequacy. *Cleft Palate Journal*, **8**, 65.

Selley, W. G. (1979). Dental and technical aids for the treatment of patients suffering from velo-pharyngeal disorders. In *Diagnosis and Treatment of Palato-glossal Malfunction*. R. E. Ellis and F. C. Flack (Eds). London: College of Speech Therapists.

Subtelny, J. D., Koepp-Baker, H. and Subtelny, J. D. (1961). Palatal function and cleft palate speech. *Journal of Speech and Hearing Disorders*, **26**, 213.

Warren, D. W. (1964a). Velopharyngeal orifice size and upper pharyngeal pressure flow patterns in normal speech. *Cleft Palate Journal*, **33**, 148.

Warren, D. W. (1964b). Velopharyngeal orifice size and upper pharyngeal pressure flow patterns in cleft palate speech: a preliminary study. *Cleft Palate Journal*, **34**, 15.

Warren, D. W. and Devereux, J. L. (1966). An analog study of cleft palate speech. *Cleft Palate Journal*, **3**, 103.

Warren, D. W. and Ryon, W. E. (1967). Oral port constriction, nasal resistance and respiratory aspects of cleft palate speech; an analog study. *Cleft Palate Journal*, **4**, 38.

Warren, D. W., Dalston, R. M., Tarier, W. C. and Holder, M. B. (1985). A pressure-flow technique for quantifying temporal patterns of palatopharyngeal closure. *Cleft Palate Journal*, **22**, 11.

The Authors

W. G. Selley FDS, RCS(Eng), Dental Surgeon, Royal Devon and Exeter Hospitals; Hon. Research Fellow, University of Exeter.
M-C. Zananiri, BSc, MPhil, Research Assistant
R. E. Ellis, MPhil, Senior Experimental Officer
F. C. Flack, BSc, PhD, Reader in Medical Physics

Medical Physics Group, Department of Physics, University of Exeter.

Requests for reprints to: W. G. Selley, Medical Physics Group, Department of Physics, University of Exeter, Exeter EX4 4QL.