

Risk factors for permanent hypernasality after adenoidectomy

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Objectives. To investigate the causes of persistent, apparently permanent hypernasal speech following adenoidectomy in 10 subjects without overt cleft palates, and to establish a protocol to be followed before this operation is performed.

Design. Retrospective and descriptive design.

Participants. Ten subjects, fulfilling the following criteria, were included: (i) subjects had undergone adenoidectomy which resulted in hypernasal speech that persisted for longer than 3 months (and was therefore considered to be permanent); (ii) subjects did not have a cleft lip or overt cleft palate; (iii) there was no hearing loss of sufficient magnitude to account for the hypernasal speech; and (iv) the hypernasality was rated as severe by a speech therapist, could not be remedied by speech therapy alone and required further management by a plastic surgeon through pharyngoplasty. Ten subjects were found through the clinical records of speech therapists and plastic surgeons working in hospitals and private practice. The following information was obtained through interviews or by reading the case files: (i) identifying information; (ii) the presence of any of the factors reported in the literature to be associated with the permanent hypernasality or nasal emission, as well as the method of identification; and (iii) whether these factors had been identified before or after the adenoidectomy.

Results. Nine out of a total of 10 subjects showed pre-operative perceptual and structural characteristics and/or case history factors that have been documented to constitute risk factors for the development of nasal speech, should an adenoidectomy be performed. The methods used to investigate these factors pre-operatively appear to have been inadequate.

Conclusion. This undesirable sequel to surgery can be prevented if certain case history and speech factors are investigated and followed up with radiographic procedures if necessary.

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Hypernasal speech, typical of individuals with cleft palate, is characterised by too much nasal resonance on the vowels, with nasal escape of air on the fricative sounds (e.g. /f/, /sh/, /s/) as well as on the plosive sounds (e.g. /p/, /b/, /d/, /t/).¹ Since it draws attention to itself, such a speech disorder has serious emotional, social, educational and vocational impact.

The highly complex velopharyngeal mechanism, responsible for sealing off the oral from the nasal cavity during speech, creates a balance in the oral/nasal resonance for normal speakers. A sphincteric movement pattern involving the elevation of the velum (soft palate) and inward movement of the lateral pharyngeal walls occurs to achieve velopharyngeal closure.²

Although hypernasal speech is associated with cleft palate, this condition may occur in children or adults without cleft palates. There are many factors that could cause such a speech disorder, one of which is surgical removal of the adenoids in at-risk cases. A review of the literature reveals that researchers have found a number of 'at-risk' factors for permanent hypernasal speech and nasal escape in patients without overt cleft palates, should an adenoidectomy be performed. These factors are: a submucous cleft palate;³⁻⁶ a bifid uvula;^{4,6} a short or thin velum;^{4,5,7} an occult submucous cleft palate;^{3,6} decreased palatal mobility;^{4,5} a deep nasopharynx;³ extensive movement of the pharyngeal wall musculature or, conversely, inadequate movement of the pharyngeal wall;⁹ neurological abnormalities;³ delayed speech and language development;⁶ difficulty sucking in infancy⁶ (symptomatic of velopharyngeal incompetency which creates an inability to impound negative intra-oral pressure⁹); nasal regurgitation of liquids during infancy;⁶ and a family history of velopharyngeal incompetency.⁶

The incidence of such a disorder has been reported to range between 1:1 500 and 1:3 000 in the American literature. While incidence figures are not available for South Africa, the researchers were able to identify 10 subjects in South Africa over a period of 4 months, suggesting that the disorder is prevalent in this country.

Aims

The aims of this study were to investigate the causes of permanent hypernasal speech following adenoidectomy, and to establish a protocol to be followed before an adenoidectomy is performed.

Method

A *post hoc* study was performed on 10 subjects who had developed severe permanent hypernasality following adenotonsillectomy. Their hypernasality was considered permanent because it had persisted for longer than 3 months.¹⁰ Five subjects were reported by their parents to have had slightly hypernasal speech pre-operatively. The condition was, however, mild and none of the patients had sought treatment for the speech problem. Rather, the reason for consultation with the practitioners concerned was chronic upper respiratory tract infections.

The case histories of subjects were obtained from the files

of plastic surgeons and speech therapists who had assessed and treated them postoperatively, and had considered their speech to be severely hypernasal.

Where possible, the ENT specialists and general practitioners who had performed the adenotonsillectomies were interviewed personally. Partially structured interviews were used. This allowed the researchers to ask the questions required in order to fulfil the aims of the research, but did not limit them to specific opening, closing and 'bridging' remarks. The questions were designed to elicit the following information: the age of the subject; the date of the adenotonsillectomy and the reason for the adenotonsillectomy; the interval between the adenotonsillectomy and the treatment for hypernasality; the type of treatment following the adenotonsillectomy; the presence of any 'risk factors' associated with permanent hypernasality; the method of identification of these factors (where applicable); and whether these factors were identified before or after the adenotonsillectomy.

Subjects had been aged between 2 and 6 years when the adenotonsillectomy was performed, and had been operated on between 2 and 10 years previously.

Discussion

As is evident from Table I, which is a summary of the results, a combination of anatomical, physiological, perceptual and/or case history factors documented to contribute to hypernasality following adenotonsillectomy were identified in 9 of the 10 subjects. In the other case, the problem was caused by scarring of the velum during the surgery. This subject was then discounted from further analysis of the data, since the problem had not existed pre-operatively.

Table I. Number of subjects in which at-risk factors were present pre-operatively and time of identification of risk factors

Risk factors	No. of subjects	Pre-operative identification	Postoperative identification
Anatomical deviations			
Submucous cleft palate	3	1	1
Bifid uvula	1	1	
Thin velum	1		1
Short soft palate	5		5
Physiological deviations			
Decreased palatal mobility	1		1
Inadequate pharyngeal wall mobility	1		1
Neurological abnormalities			
1	1		1
Case history factors			
Delayed speech and language development	2	1	1
Difficulty sucking in infancy	2		2
Nasal regurgitation of fluid in infancy	1		1
Family history of cleft palate	1		
Hypernasality present before adenoidectomy			
5	5	1	4
Palatal scarring			
1	1		

Anatomical deviations

Three of the remaining 9 subjects had evidence of submucous cleft palates. Despite reports of digital palpation and intra-oral inspection of the hard and soft palate on 1 of the subjects pre-operatively, the submucous cleft palate was not identified. A submucous cleft palate with a bifid uvula was identified in 1 subject, but the adenotonsillectomy was considered to be unavoidable.

Postoperative examination revealed a short soft palate in 5 subjects. In 2 of these, it was diagnosed by the use of multiview videofluoroscopy, in 1 by multiview videofluoroscopy and a static X-ray, and in 1 by a static X-ray only. In 3 out of the 5 cases, palatal length was assessed through intra-oral inspection pre-operatively, but the length was not identified as a problem. In the other 2 cases, the operating ENT specialist could not be interviewed, and it therefore could not be determined whether or not palatal length had been assessed, and if so, what the method of investigation had been.

One subject was found through postoperative videofluoroscopy to have a thin soft palate. This was not detected on intra-oral inspection pre-operatively.

Physiological deviations

Inadequate soft palate and pharyngeal wall movement were diagnosed in 1 subject postoperatively through the use of videofluoroscopy. In this case the gag reflex was elicited pre-operatively, but clearly did not reveal inadequate velopharyngeal movement.

Anatomical and physiological abnormalities may not be apparent on intra-oral inspection or on elicitation of the gag reflex and may require more sophisticated instrumentation or techniques of investigation. For example, naso-endoscopy may be the only way of detecting a deficiency in the musculus uvulus for diagnosing an occult submucous cleft palate, as it allows visualisation of the velopharyngeal valve during speech¹¹ and does not require surgical dissection of the palate to inspect muscle orientation.

Lateral radiography or videofluoroscopy may be used to detect a thin and short palate, which is also suggestive of an occult submucous cleft palate.³ Williams,¹¹ while not negating the value of intra-oral examination in determining submucosal clefts and bifid uvulas, states that 'it is not possible to determine from intra-oral inspection alone whether the soft palate, when it elevates while the patient produces an /ah/ sound, is or is not making contact with the posterior pharyngeal wall'. Shprintzen¹² advocates the use of both naso-endoscopy (to observe velopharyngeal structure) and videofluoroscopy (to observe velopharyngeal wall movement) as part of every pre-operative examination.

Neurological abnormality

One subject, a spastic diplegic, presented with a spastic velum. Digital palpation and intra-oral inspection of the hard and soft palate and elicitation of the gag reflex were performed pre-operatively. No abnormality in structure or function was noted. Case history factors were not probed, and persistent nasal emission was therefore not expected to result from the adenotonsillectomy.

Case history factors

A delay in speech and language development was present in 2 out of 9 patients. This was determined pre-operatively in 1 patient, but was not thought to present a risk for persistent postoperative hypernasality. In the other, the delay was only established postoperatively. Difficulty in sucking during infant feeding and fluid regurgitation through the nose were experienced by 1 subject, while another subject only experienced difficulty in sucking. In both of these cases, questions were not directed at such case history factors pre-operatively; neither were any structural or physiological abnormalities noted in either subject pre-operatively.

Delayed speech and language development, sucking difficulties and nasal regurgitation of fluid may be indicative of velopharyngeal incompetence⁶ and in some cases, the patient may not be hypernasal before the adenotonsillectomy is performed because the adenoid pad facilitates velopharyngeal closure for many borderline cases of velopharyngeal incompetence.¹³ Therefore removal of adenoid tissue may result in velopharyngeal insufficiency.

A family history of velopharyngeal incompetence was present in 1 subject. The ENT specialist who performed the adenotonsillectomy could not be interviewed, so it could not be established whether or not he was aware of this.

These case history factors discussed above may alert one to the possibility of persistent hypernasality should an adenoidectomy be performed, and could be established through careful questioning. None of the ENT specialists interviewed investigated the presence of these case history factors.

Hypernasal speech before adenoidectomy

Hypernasal speech was present pre-operatively in 5 of the 9 patients. In every case, the doctor performing the adenotonsillectomy had listened to the subject's speech. In 1 case no resonance problem was detected and in 3 cases speech was incorrectly perceived to be denasal (as opposed to hypernasal). While the adenoid pad most often assists in maintaining velopharyngeal adequacy for speech, it may hypertrophy to the extent of blocking the nasopharynx causing denasality (the opposite of hypernasality), with reduced nasal resonance¹⁴ and possible obstruction of the eustachian tubes. According to Bzoch:¹⁵ 'The conditions of hypernasal distortion of voice quality versus denasal distortion related to adenoid hypertrophy are often confused by medical specialists.' In fact, the performance of adenoidectomies on subjects with pre-operative hypernasal speech has been documented by Witzel *et al.*⁶ Bzoch¹⁵ and Witzel *et al.*⁶ recommend that patients with resonance disorders be referred to a speech therapist for evaluation before an adenoidectomy is performed.

In 1 of the patients in whom speech was considered to be 'denasal', an absent nasal spine was identified, but it was not considered to be a definitive indication of a submucous cleft palate. Shprintzen, as cited by Peterson-Falzone,¹⁶ states: 'The posterior nasal spine is not a consistently found landmark in normal patients nor is its absence found with very high consistency in patients with submucous cleft palates.' Therefore, while there may not have been

conclusive anatomical evidence of a submucous cleft palate, the presence of hypernasality may have suggested it in this subject. In 1 case, the ENT specialist did perceive the speech to be hypernasal and was aware that this carried a risk of persistent hypernasality should an adenotonsillectomy be performed. However, he considered the operation unavoidable because of a hearing loss he attributed to eustachian tube malfunction and hypertrophied tonsils. He also identified a submucous cleft palate pre-operatively. However, according to Saad¹⁷ and Witzel *et al.*,⁶ the malpositioning of the tensor palati muscles found in a submucous cleft palate results in abnormal functioning of the eustachian tubes, and removal of the adenoids would therefore be unlikely to resolve middle ear problems.

The findings above suggest that hypernasality following adenoidectomy can be prevented if the necessary case history factors are checked and followed up with further investigations pre-operatively.

We suggest the use of a flow chart (Fig. 1) to guide the pre-operative assessment of adenoidectomy candidates. Where the adenoidectomy is considered unavoidable, the doctrine of informed consent and the increasing threat of medicolegal action require that patients be warned of the possibility of hypernasality and of the required corrective surgery (pharyngoplasty), should it occur.

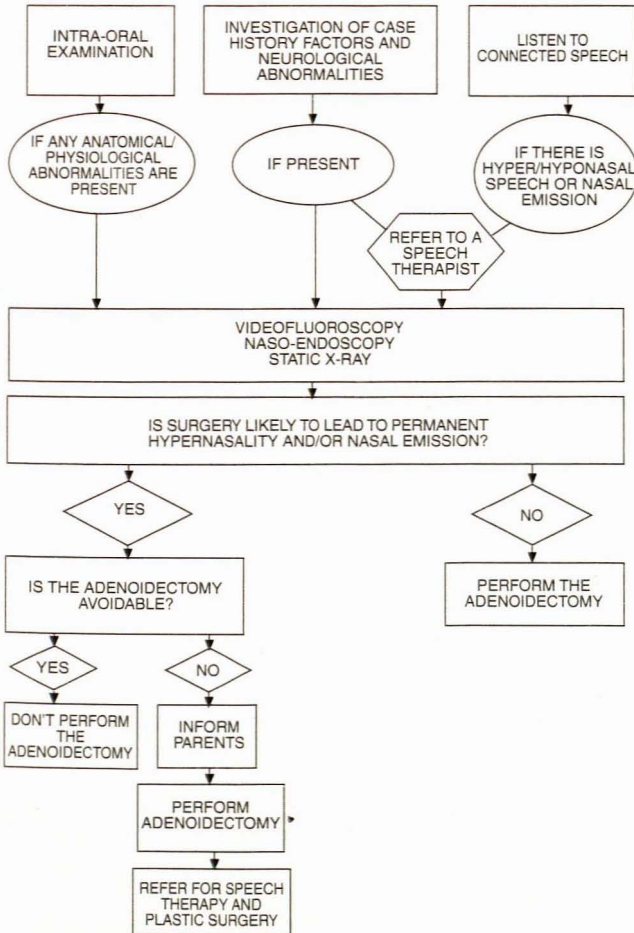


Fig. 1. Pre-operative assessment procedure for adenoidectomy candidates.

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