

Velopharyngeal insufficiency following adenoidectomy¹

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Velopharyngeal insufficiency (VPI) is a well recognized but rare complication of adenoidectomy. Twenty children with this condition were seen and assessed at Great Ormond Street Hospital between 1993 and 2000. The commonest aetiology was occult submucous cleft palate ($n = 5$) but there was a wide range of other causes. Two children with severe behavioural disorders and normal palates developed mild symptoms, an aetiology not previously reported. Only two children had a classical submucous cleft palate. Nine children required surgical intervention and three revision procedures. Of the 15 treated children for whom follow-up data was available, 13 regained normal or near-normal speech. Many cases of postadenoidectomy VPI was not foreseeable. Following referral to a specialist cleft unit, normal or near-normal speech can be achieved in the majority with a combination of surgery and speech therapy.

Keywords *velopharyngeal insufficiency adenoidectomy hypernasality cleft palate*

Velopharyngeal insufficiency (VPI) is a well-recognized complication of adenoidectomy. It is a condition characterized by hypernasal speech, nasal emission and turbulence, and in some cases nasal regurgitation of fluids. Its true incidence is difficult to establish but has been estimated between one in 1500 and one in 10 000 adenoidectomies.^{1,2} It is often because of the unmasking of a pre-existing palatal problem by removal of the tissue against which a poorly functioning palate was achieving nasopharyngeal closure. Its treatment is multidisciplinary, involving specialist speech therapy input into assessment and correction, and in some cases surgical intervention.

Previous studies have examined the aetiology of this condition but there has been little information regarding the treatment and outcomes of affected patients.

Patients and Methods

Great Ormond Street Hospital provides a tertiary referral multidisciplinary cleft palate service. Patients were identified from a prospectively compiled database of referrals to this service, held by the Speech and Language Therapy department. Patients had been referred over a 7-year period from 1993 to

2000. All patients had undergone perceptual speech assessment, and where possible, nasendoscopic and videofluoroscopic examination. All assessments had been video-recorded and archived. Data were collected by case note review and, where necessary, the video recordings were re-examined.

Results

Twenty patients were identified, 13 boys and 11 girls. The mean age at adenoidectomy was 4 years (range 17 months to 7 years) and the mean age at referral for assessment was 6 years (range 4–8 years). Eleven had undergone adenotonsillectomy and nine adenoidectomy alone.

Perceptual assessment rated their hypernasality as minimal in two cases, mild or mild to moderate in eight, moderate or moderate to severe in seven and severe in three. Eleven children underwent full investigation with nasendoscopy and videofluoroscopy, two had nasendoscopy alone and three had videofluoroscopy alone.

The aetiologies of the VPI are given in Table 1 and compared with the findings of Croft *et al.* in 1981.³

The treatment modalities employed are summarized in Table 2. Of the three children not treated, in two the treatment was not felt to be indicated because of the minimal nature of their symptoms and in one child, surgery was considered but not felt appropriate because of severe underlying neurological problems.

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Table 1. Aetiologies of velopharyngeal insufficiency

Aetiology	Number of patients (%)	
	Saunders <i>et al.</i> (2003)	Croft <i>et al.</i> (1981)
Occult submucous cleft palate	5 (25)	20 (17)
Neuromuscular disorder	3 (15)	17 (14)
Residual adenoid tissue	2 (10)	–
Classical submucous cleft palate	2 (10)	35 (29)
Poor palatal mobility	2 (10)	8 (7)
Behavioural disorder	2 (10)	–
Normal palate, 22q11 deletion	2 (10)	–
Postoperative nasopharyngitis	1 (5)	–
Scarring from tonsillectomy	1 (5)	4 (3)
Unknown	–	36 (30)

Table 2. Summary of treatment

Treatment	Number of patients (%)
Surgery (<i>n</i> = 9)	
Pharyngoplasty	4 (20)
Submucous cleft palate repair	4 (20)
Combined	1 (5)
Speech therapy alone	7 (35)
Prosthetic palatal lift device	1 (5)
Nil	3 (15)

In the surgical group, six (67%) achieved normal speech after one procedure. Three required revision procedures, resulting in normal speech in two and some improvement in one. Of the speech therapy group, four achieved normal speech, one had some improvement and two were lost to follow-up. The fitting of the single palatal lift device resulted in near normal speech.

Discussion

Submucous cleft palate is a condition that is well recognized by ENT surgeons, with a typical appearance of a bifid uvula, a midline lucency of the soft palate and notching of the hard palate. The palatal hypofunction that results from the associated muscular abnormalities is well known to carry a high risk of VPI should an adenoidectomy be performed. The difference between the numbers of children with a classical submucous cleft in our study and that of Croft *et al.* 20 years ago (10% compared with 29%) suggests that better preoperative assessment may be leading to adenoidectomy either being avoided altogether in these children or being undertaken much more judiciously.

An occult submucous cleft is a less well-recognized anatomical anomaly. It too involves abnormality of the structure and function of the palatal musculature, but is not detectable on oral examination. On endoscopic examination of the nasopharynx, there is loss of the usual midline convexity of the superior

surface of the soft palate with either flattening or a midline groove, consistent with the absence of musculus uvulae. This is sometimes known as the 'seagull sign'.⁴

The presence of an irregular mass of residual adenoid tissue can lead to VPI if it prevents the soft palate from moving to close against the posterior pharyngeal wall and there is space for air to escape around the residual tissue.⁵ Careful visualization of the postnasal space during or after adenoidectomy should minimize this problem. The precise effect of any remaining tissue depends on the pre-existing pattern of velopharyngeal closure (coronal, circular or sagittal). If deliberate partial adenoidectomy is considered in an attempt to avoid VPI in a patient with known palatal problems, this should be borne in mind and careful preoperative evaluation undertaken.

In three patients with neurological or neuromuscular disorders, adenoidectomy resulted in VPI. In two of these, this represented an exacerbation of mild pre-existing hypernasality. Adenoidectomy should be performed with caution in such children.

Two children with severe behavioural difficulties developed mild self-limiting VPI after adenoidectomy. This is a phenomenon not previously reported. The exact reason for the VPI in these cases is unclear.

Two children in the group had apparently normal palates but on review were noted to have the characteristic facies of velocardiofacial syndrome (prominent nose, long philtrum, malar flattening, retrognathia). On subsequent genetic testing they were confirmed to have the 22q11 deletion diagnostic of the condition. Although children with velocardiofacial syndrome may have associated overt or submucous cleft palate, those with apparently normal palates may have subtle functional and structural abnormalities predisposing to VPI.

One child had developed a fever and mucopurulent nasal discharge shortly after adenoidectomy. This had been diagnosed as nasopharyngitis and successfully treated with antibiotics, but the child was noted to have hypernasal speech 1 week postoperatively, after the infection had settled. This was in contrast to the other children, in whom the changes in speech were noted in the immediate postoperative period, and suggests that the infection rather than the surgery itself was the cause.

Morris *et al.*⁶ suggested a number of preoperative factors that could alert the surgeon to the possibility of postadenoidectomy VPI. These include submucous cleft palate, anterior dimpling of the soft palate, a history of nasal regurgitation of fluids, neurological disorders, pre-existing hypernasality of speech or a family history of cleft palate or VPI. Witzel *et al.*⁷ found that over 30% of patients with postadenoidectomy VPI had such identifiable risk factors. In our series there were two children with a classical submucous cleft palate, two with pre-existing hypernasality as a result of neuromuscular disease and two with a history of nasal vomiting or regurgitation as a baby, one of who also had a brother with a bifid uvula. Thus

30% of the children had features that might have suggested they were at higher risk of developing VPI. We also propose that facies consistent with velocardiofacial syndrome be added to the list of potential risk factors.

It should be noted that of the cases reported by Croft *et al.*,³ in 30% the cause of VPI remained obscure. The authors of that paper point out that the majority of these children were seen before they introduced endoscopic visualization of the nasopharynx into their diagnostic protocol. This, and the high percentage of patients in our series in whom a diagnosis was made, reinforce the need for these children to be assessed as fully as possible, ideally in the context of a specialist multidisciplinary clinic. Treatment options can then be discussed and individualized management plans formulated, implemented and followed up.

In this series, nine patients (45%) required corrective surgery. This is a remarkably similar figure compared with those of Croft *et al.*³ (52%) and Witzel *et al.*⁷ (50%). However, in both these series, surgery consisted mainly of pharyngoplasty or posterior pharyngeal wall augmentation, whereas over half of our patients underwent corrective palatal surgery. Surgery should generally be reserved for those with identifiable anatomical abnormalities or those who comply with but fail to respond to speech therapy. It should be remembered that even in those who undergo surgery, speech therapy forms an integral part of their postoperative rehabilitation.

Conclusion

Velopharyngeal insufficiency remains a rare complication of adenoidectomy. Some cases are potentially avoidable, but

many are not or are avoidable only with hindsight. If there is any doubt as to the structure or function of the palate preoperatively, the child should be referred to a multidisciplinary cleft palate team for full assessment before any decision is made regarding surgery. If VPI is suspected following adenoidectomy, the child should initially be assessed by an appropriately experienced speech therapist and then referred as indicated. Approximately 50% of such children will require some form of surgical intervention, which is successful in the majority of cases.

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