Obstructive Sleep Apnea Following Treatment of Velopharyngeal Incompetence by Teflon Injection

LEONARD T. FURLOW JR., M.D.
A. JAY BLOCK, M.D.
WILLIAM N. WILLIAMS, PH.D.

From 1967 to 1974 a clinical trial of Teflon injection into the posterior pharyngeal wall for correction of velopharyngeal incompetence (VPI) was conducted in thirty-six patients. Six years after Teflon injection, one of the patients reported the onset of severe snoring punctuated by silences when he seemed not to be breathing, daytime hypersomnolence, and tiredness severe enough to interfere with work and studies. The diagnosis of obstructive sleep apnea (OSA) was confirmed by polysomnographic sleep monitoring, and the dynamics of the obstruction elucidated by cinefluoroscopy performed with the patient asleep. Resection of the lower ¼ of the Teflon pad, leaving the upper rim to avoid recurrence of his VPI, has eliminated the symptoms of OSA and produced an improvement in his polysomnographic findings.

The injection of Teflon into the posterior pharyngeal wall of selected patients with velopharyngeal incompetence (VPI) has shown promise as an effective treatment for restoring functional velopharyngeal closure. This technique has received considerable attention because of its relative simplicity. Since 1967, at least seven different studies have reported on the efficacy of the Teflon injection procedure in treating VPI in over 250 patients (Blockma and Braley, 1969; Bluestone et al, 1968; Furlow et al, 1982; Kuehn and Van Demark, 1978; Smith and McCabe, 1977; Sturim and Jacob, 1972; Ward et al, 1966). Despite these studies, FDA approval for Teflon injection into the posterior pharyngeal wall has not been obtained.

The authors are all affiliated with the University of Florida in Gainesville. Dr. Furlow is in the private practice of Plastic and Reconstructive Surgery, and is Clinical Professor, Department of Surgery, College of Medicine. Dr. Block is Professor in the Department of Medicine and Anesthesiology in the College of Medicine. Dr. Williams is Associate Professor, Department of Basic Dental Sciences in the College of Dentistry.

This paper was presented at the Florida Cleft Palate Association meeting January 1984, Lake Buena Vista, Florida.

The Teflon paste consisted of Teflon particles 50 to 100 microns in diameter suspended in glycerine and was supplied by the Ethicon Company of Somerville, New Jersey.

From 1967 to 1974 two of us (L.T.F., W.N.W.) took part in a clinical trial of Teflon injection for VPI in 36 patients. The results of this investigation have been previously reported (Furlow et al, 1982). In summary, only patients who had good palatal motion, good articulation, and a residual velopharyngeal gap of 10 mm or less (as measured from lateral cinefluoroscopy during the individual’s best attempt at closure) were selected for treatment. Teflon paste1 was injected into the posterior pharyngeal wall to produce a bulge or pad against which the velum could abut for closure. Twenty-six of the 35 patients followed (74%) were successfully treated by one or two injections. Success was defined as total elimination of hypernasality and inappropriate nasal air emission. In this group there were no deaths or acute or chronic postoperative infections. However, one of our patients developed obstructive sleep apnea (OSA) more than 2 years after receiving the Teflon injection. To our knowledge, OSA has not been previously
reported as a complication following Teflon injection. This paper is to report the complication and its treatment in this individual.

SLEEP APNEA

Sleep apnea is a problem most frequently seen in middle-age overweight males. It is characterized by heavy snoring punctuated by momentary cessation of respiration. Each snoring cycle is initiated by a gasping inspiration. The patient's sleep is restless, as he in essence must partially arouse to breathe. Blood oxygen desaturation, sometimes to remarkably low levels, occurs during the apneic periods. Cardiac arrhythmias may occur during desaturation. The patients have hypersomnia that interferes with daytime activities. Morning headaches and hypertension are common, and death has been reported (Chaudhary and Spier, 1982).

Sleep apnea in children is most often related to enlarged tonsils and adenoids. A snoring pattern similar to that in adults is seen. Additional symptoms common in children with obstructive sleep apnea include nocturnal enuresis, decreased school performance, personality changes, and failure to thrive (Guilleminault et al, 1976). Chronic upper respiratory obstruction has long been identified as a cause of pulmonary hypertension and cor pulmonale in children (Cox et al, 1965).

Sleep apnea is defined as the occurrence of at least 30 apneic episodes 10 or more seconds in length during a 7-hour sleep cycle. When respiratory movements are attempted during the periods of apnea, the disorder is classified as obstructive sleep apnea. If there is neither air flow nor respiratory motion it is defined as central sleep apnea. Mixed sleep apnea is characterized by episodes with initial cessation of respiration and air flow, with resumption of respiratory efforts later in the apneic period (Chaudhary and Spier, 1982).

Case Report

In 1974 we examined a 24-year-old white male who had a degree of dysarthria secondary to mild cerebral palsy. A cinefluoroscopic assessment of his velopharyngeal port revealed that he could achieve closure in single words or short speech samples containing only oral consonants. However, in words with alternating oral and nasal consonants and with longer speech samples, velopharyngeal competence diminished. A residual gap appeared on cinefluoroscopy, and by cul-de-sac resonance tests and testing for nasal air flow (Bzoch, 1979) hypernasality and inappropriate nasal air emission were detected. His symptoms of VPI became especially noticeable when he was tired or stressed. Figure 1 illustrates from a cinefluoroscopic lateral view the width of the patient's velopharyngeal gap or airway space with the velum in its physiological rest position.

Treatment of the patient's VPI by Teflon injection was selected because it presumably would be less detrimental to an already faulty neuromuscular mechanism than would a pharyngeal flap. Approximately 10 cc of Teflon paste was injected above the level of the atlas across his posterior pharyngeal wall to produce an anteriorly displaced transverse bulge against which his soft palate could more easily make contact. Six months after the Teflon injection, speech evaluation demonstrated normal resonance quality and no detectable nasal air emission.

Although his VPI had been eliminated, during the 2 years that he was followed postoperatively, he complained of severe snoring and of the feeling that he had a lump in the back of his throat which gave him the sensation of choking. At that time he moved and was lost to follow-up.

He returned to our clinic 4 years later (6 years after the Teflon injection) at which time he reported that he snored so loudly that it interfered with his social life. The snores were punctuated by moments of silence when he seemed not to be breathing. He described severe daytime hypersomnia and fatigue that made it difficult for him to hold a job or study. He was quite concerned and severely depressed be-
cause of these changes, which he attributed to the Teflon injection.

A diagnosis of OSA due to narrowing of his airway by the Teflon pad was made. A cinefluoroscopic study was conducted with the patient lying on his side. He quickly fell asleep and immediately began snoring. The snoring was interrupted by periodic cessations of respiration. Cinefluoroscopic analysis revealed that the cessation of breathing resulted from an obstruction in the pharynx created as the base of the tongue came into contact with the thickened posterior pharyngeal wall. The posterior pharyngeal wall thickness (anterior border of atlas to mucosal surface) was 10 mm. However, his velar gap or airspace during physiological rest was only 3 to 5 mm in width. Figure 2 illustrates the reduced depth of the nasopharynx following the Teflon injection produced by the bulging of the posterior pharyngeal wall which extended as low as the fourth cervical vertebra.

A polysomnographic sleep study, which was conducted in the supine position for 157 minutes, showed a regular pattern of hundreds of episodes of cyclic upper airway OSA. The apnea and hypopnea index was 48 episodes per hour of sleep. These episodes resulted in severe cyclic oxygen desaturation. The nadir of each desaturation episode was consistently between 50 and 60 percent and after 2 hours of sleep fell even lower. Because the Hewlett-Packard 47201A ear oximeter may not be accurate at levels below 50 percent saturated, the very low levels of 16 to 25 percent that were recorded could be spurious. Cyclic bradycardia occurred concomitant with oxygen desaturation, but no serious cardiac arrhythmias were noted. Only 5 minutes of rapid eye movement (REM) sleep was noted.

After detailed analysis of the cinefluoroscopic records, it was determined that the lower portion of the Teflon pad should be resected, retaining that portion of the pad above the occlusal plane in order to avoid recreating the problem of VPI. Under general anesthesia 13.5 cc of tough, granular, fibrotic material was excised from the posterior pharyngeal wall, leaving the portion above the palatal plane intact. Microscopic examination of the resected tissue showed the Teflon particles to be encased in dense fibrous tissue (Fig. 3).

Two months later the patient reported that his snoring had almost stopped and that he was no longer tired during the day. However, by 8 months his symptoms had recurred to a considerable extent. Ten months after resection of the Teflon pad a second polysomnographic sleep study was conducted which revealed marked improvement. The patient slept for 388 minutes and the apnea and hypopnea index fell to 26 per hour. In addition, there were long periods, nearly half of his sleeping time, when he had no apneic episodes. This study included 60 minutes of REM sleep. During the time apneic episodes were occurring, however, his oxygen saturation fell as low as 44 percent.

A cinefluoroscopic study was also repeated with the patient asleep on his side. Although the lower portion of the Teflon pad was thinner as a result of the surgical resection, an obstructive seal between the base of the tongue and the posterior pharyngeal wall was still occurring intermittently during sleep, resulting in apneic periods. Figure 4 illustrates the reduction in Teflon pad thickness below the level of the atlas without a significant increase in the velar-pharyngeal air space.

Following this analysis a further resection of the Teflon pad was done. Only that portion of the pad at and above the upper margin of the atlas was retained. Four and one-half cc of tissue were removed, making a total volume of 18 cc of tissue removed during the two resections.

Following the second resection the patient’s symptoms were alleviated. One and one-half years after the second resection a polysomnographic study showed considerable improvement, although some apneic episodes still occurred. During these apneic periods the patient’s blood oxygen saturation dropped, but not lower than 66 percent. A cinefluoroscopic assessment was conducted, but the patient was not able to fall asleep. Although the Teflon pad was identifiably smaller, velopharyngeal closure remained functional. Figure 5 illustrates the reduction in thickness of the posterior pharyngeal wall from the level of the palatal plane inferiorly and a doubling of the width of the velar-pharyngeal wall air space.

In the 3 years following the second resection of the Teflon pad, the patient’s speech has shown no evidence of VPI and he has had no more daytime sleepiness. He has resumed postgraduate studies and is gainfully employed.
FIGURE 3  Histologic examination of resected tissue revealed Teflon particles to be encased in dense fibrous tissue.

FIGURE 4  Velopharyngeal airway space 10 months after initial partial resectioning of the Teflon pad. The velum is at physiological rest. Although the thickness of the posterior pharyngeal wall below the level of the atlas has been reduced, there has been little or no increase in the velopharyngeal air space.

FIGURE 5  Velopharyngeal airway width and configuration 18 months after a second and final partial resection of the Teflon pad. The velum is at physiological rest. The posterior pharyngeal wall has been reduced in thickness from the level of the palatal plane inferiorly, doubling the velopharyngeal air space.
DISCUSSION

Following Teflon injection nearly all patients experience a sore throat and neck stiffness for several days. Complications have been few and minor (Bluestone et al., 1968; Furlow et al., 1982; Kuehn and Van Demark, 1978). The complication of OSA that we are reporting is serious because of its detrimental physical, mental, social, and economic effects upon the patient.

Adult patients with OSA studied in the waking state by computed tomography (CT) have demonstrated narrowing of the upper airway because of diffuse hypertrophy of the tissues (Bohman et al., 1983). Fluoroscopic and CT studies during sleep of patients with OSA have revealed that the soft palate and uvula, the tongue base, and the posterior pharynx come together to occlude the airway (Suratt et al., 1983).

Although OSA is most common in obese middle-aged males, it has been reported in children with enlarged tonsils or adenoids (Guilleminault et al., 1976; Mauer et al., 1983) and with various craniofacial malformations (Johnston et al., 1981; Schafer, 1982). Kravath et al. (1980) reported OSA following surgical correction of VPI by pharyngeal flap in three children; in two children the symptoms cleared spontaneously sometime after surgery, but one child died 4 weeks following surgery, presumably from the additive effects of OSA and mild bronchitis.

In the classic middle-aged obese patient with OSA, permanent tracheostomy is the most reliable treatment, but resection of the excess tissue by uvulopalatopharyngoplasty has been reported to be effective in decreasing snoring, and in some patients, improving or clearing the symptoms associated with OSA. Interestingly, improvement in the symptoms frequently has not been commensurate improvement in polysomnographic findings (Fujita et al., 1981; Simmons et al., 1983). This is similar to the findings in our patient, whose symptoms were ameliorated by resection of most of the Teflon pad, but who still demonstrated intermittent moderate reduction in $P_{O_2}$ on polysomnographic studies.

Unlike OSA in adults, OSA in children is usually due to a specific identifiable anatomic abnormality of the oronasopharyngeal airway, and its signs and symptoms usually clear following surgical correction of the responsible abnormality. Mauer et al. (1983) reported a study of children with markedly enlarged tonsils and adenoids whose obstructive symptoms cleared following T&A. Johnston et al. (1981) reported improvement in the symptoms of OSA in an 8-year-old patient with Treacher Collins syndrome following mandibular advancement. Puckett et al. (1982) reported amelioration of OSA symptoms in a 5½-year-old child with mandibular hypoplasia by means of a fascia lata sling from the base of the tongue to the mandible. Robson et al. (1977) described a 4-year-old child who subsequent to a palatal pushback and pharyngeal flap at age 2 developed gasping respiration during sleep, failure to thrive, and cor pulmonale. These signs and symptoms cleared when the completely obstructing flap was divided. In three children with Pierre Robin syndrome, Jackson et al. (1976) described symptoms consistent with OSA following cleft palate repair with pharyngeal flap. In one child a tongue-gingival adhesion resolved the problem. In a second, division of the flap did not help, but after excision of the redundant pharyngeal flap tissue the girl’s symptoms cleared. The third child died of airway obstruction three nights following flap division.

In our patient, the narrowing of the pharyngeal airway by the Teflon pad was clearly the cause of his OSA, and partial resection of the pad, which seemed to be the most logical treatment, proved effective.

In treating our patients, we injected the Teflon paste through 3 to 5 sites across the pharyngeal wall above the level of the atlas. During injection, fascial attachments limit the superior extension of the Teflon bulge. In order to produce an adequate pad at the appropriate level for velopharyngeal closure, the injection pressure may force material inferiorly through the muscle fibers and in the submucosal plane, narrowing the airway below the level of velopharyngeal closure. We have found the use of a tongue blade as a barrier to block inferior migration of the Teflon paste (Bluestone et al., 1968; Ward et al., 1966) to be only partially effective. Downward displacement of the material probably occurs during or very shortly after the injection. Serial postoperative cinefluoroscopic studies (Furlow et al., 1982) showed that after 3 months no significant change occurred in posterior pharyngeal wall thickness or vertical position of the Teflon pad.

In this case study the downward displacement
of the Teflon particles produced enough narrowing of the patient's airway so that with relaxation of the base of the tongue and velum during sleep, obstruction of the airway occurred. Resection of all but the upper rim of the Teflon-fibrous tissue pad has relieved the symptoms of OSA.

The relationship between the volume of Teflon paste injected to the volume of tissue resected is worth noting. Teflon paste is by volume 50 percent Teflon particles and 50 percent glycerine. After injection the glycerine is absorbed. In this case, 10 cc of Teflon paste was injected; absorption of the glycerine presumably left a 5 cc volume of Teflon particles. However, a total volume of 18 cc of tissue containing the Teflon particles was removed in the two resections. On microscopic examination of the resected material (Fig. 3) one sees that much of the bulk is made up of fibrous tissue which encapsulates the individual Teflon particles, explaining why more material was resected than was injected.

Our original assessment of the 36 patients whose VPI had been treated by Teflon injection did not identify any patients with OSA, including the patient we have reported above (Furlow et al, 1982). In this earlier study the 26 patients successfully treated had been followed postoperatively for a mean of 36 months and a median of 27.5 months. We are currently reevaluating these patients, who are now 10 to 17 years post Teflon injection, for permanency of speech results and stability of the injected material. We are including a thorough evaluation for OSA. In the 15 patients reevaluated to date, none have been found to manifest signs of OSA.

References


