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Management of Oropharyngeal and Tracheobronchial Secretions in Patients with Neurologic Disease

LAUREN B. ELMAN, M.D.,[1] RANDY M. DUBIN, M.A. CCC-SLP,[2]
MARY KELLEY, R.N., M.S.N., and LEO McCLUSKEY, M.D., M.B.E.[1]

ABSTRACT

***Background*: Neurologic disorders may impair the normal clearance of secretions. Effective palliation requires the management of excessive oral, pharyngeal and/or tracheobronchial secretions. This requires an understanding of underlying mechanisms and familiarity with the many available medical and surgical treatment options.**

***Objectives*: The authors intend to review the relevant anatomy and physiology along with the available medical, surgical and physical therapies available to treat this commonly encountered problem.**

***Design*: A review of current management and the supporting literature.**

***Conclusions*: Clinicians have many effective therapeutic options to choose from when managing the excessive oral, pharyngeal and/or tracheobronchial secretions caused by neurologic disorders. Treatment choices that are predicated upon pathophysiologic causes and patient status are the most likely to succeed.**

INTRODUCTION

Acute, chronic stable, and chronic progressive neurologic disorders may impair the normal clearance of secretions. Effective palliation of these patients requires management of excessive oral, pharyngeal and/or tracheobronchial secretions that can be embarrassing, socially isolating, uncomfortable, debilitating and potentially life threatening. Successful therapy requires an understanding of underlying mechanisms and familiarity with the many available medical and surgical treatment options.

PATHOPHYSIOLOGY

Neurologic disorders

Neurologic disorders do not result in excessive production of oropharyngeal secretions. Rather, disorders of the central (CNS) or peripheral nervous system (PNS) may result in weakness or incoordination of voluntary skeletal muscles or may disrupt sensory function necessary for swallow and cough. The normal daily volume of saliva produced by the major and minor salivary glands, approximately 1.5 L, and the volume of mucus produced by oropharyngeal and tracheobronchial mucus glands, approximately 2 L, must be swallowed.[1–3] To accomplish this a healthy adult who is awake and not eating swallows approximately once per minute.

Saliva is produced by the major and minor salivary glands. The parotid, submandibular, and sublingual glands are paired structures that comprise the major salivary glands, which are responsible for 90% of normal saliva production. The submandibular and sublingual glands are responsible for approximately 70% of basal saliva production; the contribution of the parotid glands increases with stimulation, such as eating. The

[1]Department of Neurology,
[2]Department of Otorhinolaryngology, Division of Speech and Language Pathology, Philadelphia, Pennsylvania.

salivary glands are innervated for secretory function by parasympathetic fibers that originate in brain stem nuclei. Preganglionic fibers destined for the parotid glands originate in the inferior salivatory nucleus and travel with the glossopharyngeal nerve (IX) to the otic ganglia to synapse with postganglionic fibers. Preganglionic parasympathetic fibers from the superior salivatory nucleus travel with the facial nerve (VII) to synapse with postganglionic fibers in the submandibular ganglia that terminate in the submandibular and sublingual glands. Secretory innervation of the glands is mediated by muscarinic cholinergic receptors.

Voluntary and reflex control of swallowing and coughing requires an intact sensory feedback and motor outflow system. The motor system is comprised of the upper motor neurons (UMN), lower motor neurons (LMN), neuromuscular junctions (NMJ), and muscles. The UMNs reside in the frontal lobe and their axons traverse the hemispheric white matter and corticospinal tracts to reach the brainstem and spinal cord LMNs. Brain stem LMNs are organized within motor nuclei. The axons of brainstem LMNs travel to target muscles via cranial nerves V (trigeminal), VII (facial), IX (glossopharyngeal), X (vagus), XI (spinal accessory), and XII (hypoglossal). Diaphragmatic LMNs, located within the anterior horn of the upper cervical spinal cord (C3,4,5), project their axons via the phrenic nerve and thoracoabdominal muscles receive LMN innervation via thoracic roots. Voluntary control is subconsciously modulated and coordinated by the basal ganglia (extrapyramidal system) and cerebellum.[4–14] Sensory afferents from the oral cavity travel in the trigeminal (V) and glossopharyngeal (IX) nerves while those from the larynx and tracheobronchial tree traverse the vagus (X) nerve. Each provides sensory input necessary for reflex motor control of swallow and cough by local brainstem centers. Sensory afferent input is also relayed to the thalamus and then to the primary sensory cortex where it can be consciously perceived and used to control voluntary motor output.

Dysphagia

Swallowing requires the orderly coordination of over 30 muscles and is divided into oral, pharyngeal, and esophageal phases, based on the location of the bolus. The oral phase is under voluntary control, while the pharyngeal and esophageal phases are involuntary. Neurologic disorders that produce weakness or incoordination of the swallowing musculature are the primary cause of oropharyngeal dysphagia,[15] which is often accompanied by difficulty managing the constant flow of secretions. Neurologic disorders do not produce esophageal dysphagia, which is most commonly caused by mechanical obstruction or esophageal dysmotility.

The prevalence of dysphagia among individuals older than 50 years may be as high as 22%.[16,17] Perhaps 12% to 13% of patients in acute care hospitals and up to 60% of nursing home occupants have dysphagia.[18,19] Dysphagia is present in 29% to 64% of patients after an acute stroke; this decrease to approximately 17% by 2 to 4 months poststroke.[20–26] Dysphagia is also a major symptomatic issue in cerebral palsy, traumatic and hypoxic brain injury and in neurodegenerative disorders such as amyotrophic lateral sclerosis, Parkinson's disease and Parkinsonian syndromes, Alzheimer's disease, and Huntington's disease. Neuromuscular disorders including myasthenia gravis, inflammatory muscle diseases, post polio muscular atrophy syndrome, and muscular dystrophies may also be associated with dysphagia.[27–29]

Oral dysphagia

During the oral phase solid food is masticated to a size, shape, and consistency that will easily pass through the pharynx. Solids and liquids are then transferred into the upper pharynx when the anterior portion of the tongue lifts up, contacts the hard palate, and retracts posteriorly. Weakness or incoordination of the tongue results in oral dysphagia. Patients with oral dysphagia experience difficulty propelling a solid bolus or controlling the flow of a liquid bolus into the pharynx. This may result in aspiration prior to the swallow response. They exhibit a prolonged oral phase of swallowing as well as pocketing of food in the cheek, under the tongue, or on the hard palate.[3,30]

Sialorrhea

Patients with liquid oral dysphagia can develop drooling (sialorrhea). Sialorrhea results when saliva that is not efficiently propelled into the pharynx pools in the mouth and escapes through the lips. When mild, sialorrhea may only occur at night. When most severe, the constant

drooling prompts the patient to frequently mop the lips or even to place an absorbent towel into the mouth while protecting the shirt and lap with a bib or a towel. The reflex increase in the production of saliva that accompanies eating or drinking usually worsens sialorrhea. Sialorrhea may also be exacerbated by mechanical factors. Reduced labial and buccal strength can result in food or liquid falling from the mouth. Neck extension weakness with a resultant head drop can worsen sialorrhea. A similar phenomenon can occur with the stooped posture that accompanies parkinsonism or with weakness of truncal extensor muscles. Reduced sensation of the lips and oral cavity may also worsen sialorrhea.[9]

Pharyngeal dysphagia

The pharyngeal phase of swallowing is triggered by the presence of liquids or solids in the upper pharynx. Contraction of the tensor palatini and levator palatini muscles elevates the soft palate, sealing the nasopharynx and preventing nasal regurgitation. Closure of the larynx occurs in a specific sequence to prevent aspiration. True vocal fold adduction is followed by adduction of the false vocal cords and aryepiglottic folds. As the larynx is elevated and pulled forward there is retroversion of the epiglottis to assist in closing off the laryngeal vestibule and opening up the upper esophageal sphincter. Tongue base retraction and orderly rostral to caudal contraction of the upper, middle and then lower pharyngeal constrictors propels the bolus towards the open upper esophageal sphincter.

Dysfunction of pharyngeal muscular function may produce dysphagia for liquids and solids. In general it is more difficult to swallow liquids than solids. Even mild pharyngeal muscle dysfunction may result in liquid dysphagia. More significant pharyngeal muscle dysfunction is necessary to produce solid dysphagia. Patients may initially describe the need for multiple liquid swallows and a new inability to rapidly swallow or "chug" liquids. They may also note that thicker consistency liquids are more easily swallowed than thin liquids. Patients with pharyngeal dysphagia may exhibit frequent throat clearing, a wet or gurgly sounding voice, nasal regurgitation of liquids or solids and symptoms of aspiration such as coughing, choking, tearing, sneezing, strained speech, difficulty breathing, stridor, and increased heart rate. Aspiration may lead to symptoms of pneumonia such as dyspnea, coughing, and fever. In patients with disorders of the CNS aspiration pneumonia may present clinically with reduced levels of consciousness. Aspiration can also occur silently without symptoms.[3,4,6,9,30,31]

Impaired clearance of pharyngeal secretions

Difficulty swallowing liquids may result in the pooling of saliva and mucus in the pharynx, especially in the valleculae (wedge-shaped space formed by the base of the tongue and the epiglottis) and the pyriform sinuses (space formed on each side between the inferior pharyngeal constrictor and the thyroid cartilage). This results in the perception of excessive pharyngeal secretions, similar to postnasal drip. Minor symptoms cause the patient to frequently dry swallow or clear the throat. When most severe the patient describes a constant sense that "something is in there" and experiences spontaneous coughing or choking upon their own secretions. This may be accompanied by symptoms of laryngeal aspiration resulting in transient tearing, straining of the voice, stridor, and dyspnea. These episodes may be induced by a change in body position such as laying flat or head turning. Patients with such significant difficulty swallowing liquids have an increased risk of pulmonary aspiration leading to pneumonia.[9,32]

Hypersecretion

Comorbid disorders that increase the production of saliva and mucus may contribute to difficulty with secretion management in neurologic disorders. Dental caries, oral infections and gastroesophageal reflux increase production of saliva. Medications such as acetylcholinesterase inhibitors (e.g., pyridostigmine) and muscarinic cholinergic agonists (e.g., bethanechol) directly stimulate production of saliva and mucus. Smoking may increase the production of saliva and mucus while use of chewing tobacco increases the production of saliva. Comorbid chronic bronchitis, emphysema, and bronchiectasis may increase the production of tracheobronchial secretions.[32]

Weakened cough

The three phases of the normal cough include inspiration, forced expiration against a closed glottis with assistance from the abdominal mus-

cles, and glottic opening with expulsion of air, particulates and secretions from the tracheobronchial tree and pharynx. During vigorous coughing, intrathoracic pressures may reach 300 mm Hg and expiratory velocities approach 500 miles per hour.[33] Neurologic disorders may compromise the effectiveness of the cough by producing weakness or incoordination of the involved skeletal muscles. A weakened cough leads to inability to clear secretions and places patients at risk for atelectasis, mucus plugging, and pneumonia.

MANAGEMENT

Medications

There are multiple medications that reduce the production of saliva that may benefit patients with sialorrhea and/or excessive pharyngeal secretions (Table 1). Anticholinergic medications or medications with anticholinergic side effects reduce the production of saliva and mucus to amounts that may be more manageable for the patient. Anticholinergic side effects such as constipation, blurred vision, and difficulty urinating may limit dosing. Various routes of administration are available including oral, transcutaneous, and nebulized.

Botulinum toxin is active at both nicotinic and muscarinic cholinergic terminals where it blocks presynaptic release of acetylcholine. Injection of botulinum toxin produces local functional denervation. When injected into the salivary glands botulinum toxin can reduce the production of saliva while minimizing systemic side effects. Botulinum toxin injection has been used successfully to treat sialorrhea related to cerebral palsy, stroke, Parkinsonism and amyotrophic lateral sclerosis (ALS)[34–40] and objective measurements have demonstrated a reduction in salivary flow. Both botulinum toxin A and B have been used. The parotid glands alone or the parotid and submandibular glands are most commonly selected. Ultrasonographic guidance may improve results.[41–45] Patients usually experience relief within days and the therapeutic effect most often lasts for approximately 3 to 4 months, when reinjection may be required. Side effects are usually minimal. Diffusion of the toxin into neighboring muscles such as the tongue, pharyngeal constrictors and masseter may produce temporary weakness that can further complicate secretion management.[34]

Anticholinergic medications and botulinum toxin may cause thickening of the secretions, thereby making them more tenacious and difficult to mobilize in the setting of compromised musculature. This can be minimized by maintenance of adequate hydration and may also be ameliorated by the coadministration of medications that thin secretions such as guaifenesin or N-acetyl-cysteine.[6,29,32,46–50]

PHYSICAL MODALITIES

Manual suction

Secretions within the oral cavity can be aspirated with a hand-held bedside or portable suction device. The use of this device by the patient may be limited by coexistent arm or hand weakness.

Respiratory physical therapy

Various techniques for respiratory physical therapy may be used for patients with neurologic disorders that weaken the cough, compromise airway clearance and increase the risk of mucus plugging, atelectasis, and pneumonia. Such therapy is particularly important for patients with comorbid pulmonary disorders that result in excessive production of tracheobronchial secretions. Currently there are insufficient data to suggest one or more of these techniques over any other.[51] The techniques can be combined and used in conjunction with the other physical modalities. The regimen selected should be time efficient, comfortable, effective, and able to be implemented by the patient or with the aid of one assistant.[51]

Postural drainage. Classic postural drainage consists of the mobilization of tracheobronchial secretions with the use of gravity-assist positioning, deep breathing with or without chest clapping, vibration, or shaking. When secretions reach the upper airway they are expelled via coughing. Postural drainage has been demonstrated to be more effective than coughing alone in patients with excessive bronchial secretions. Its role in the management of other disorders, including neurologic disorders, is less clear. It has been largely supplanted by the more effective and more easily applied techniques reviewed below.[51,52]

TABLE 1. MEDICATIONS FOR SECRETION MANAGEMENT[82]

Drug	*How supplied*	*Dosage*	*Specific side effects*
Anticholinergics for thin secretions (saliva)			
Glycopyrrolate (Robinul)	1 mg tablets, can be crushed	Start at 1 mg 1 to 4 times daily. Titrate to symptom control and side effects. Max 8 mg/24 hrs	See below[a]
	0.2 mg/mL	0.1–0.2 mg IV or IM 1 to 4 times daily. Titrate to symptom control and side effects	
Hyoscyamine (Levsin)	0.125 mg tablets, 0.125 mg/cc and 0.125 mg/5 cc elixir	Start at 0.125 mg 1 to 4 times daily. Titrate to symptom control and side effects. Max 12 tabs/24 hrs.	See below[a]
Atropine	0.4 mg tablets	Start at 0.2 mg 1 to 4 times daily. Titrate to symptom control and side effects.	See below[a]
Atropine sulfate	1 mg/10 cc injectable	0.025 mg/kg in 3 cc saline 3 to 4 times daily via nebulizer. Titrate to symptom control and side effects.	See below[a]
Atropine eye drops	1% solution	1–2 drops SL or PO Every 4–6 hours. Titrate to symptom control and side effects.	See below[a]
Ipratropium	500 mcg/0.02% in 2.5 cc saline unit dose vial	250 to 500 mcg 1 to 4 times daily via nebulizer. Titrate to symptom control and side effects.	See below[a]
Scopolamine	Transdermal 1.5 mg	Start at 1 patch every 3 days. Titrate to symptom control and side effects.	Skin irritation and see below[a]
Tricyclic antidepressants (TCAs) for thin secretions (saliva)			
Amitriptyline (Elavil)	10, 25, 50, 75, 100, 150 mg tablets. 10 mg/5 cc oral suspension.	Start at 10 mg hs Titrate to symptom and side effects. Max 150 mg/24 hrs	Drowsiness, low BP quinidine like effect on the heart, bone marrow depression, rash. See below[b]
Desipramine (Norpramin)	10, 25,50, 75, 100, 150 mg tablets.	Start at 10 mg hs Titrate to symptom and side effects. Max 300 mg/24 hrs.	Same as amitriptyline See below[b]
Nortriptyline (Pamelor)	10, 25, 50 75 mg tablets	Start at 10 mg hs Titrate to symptom and side effects. Max 150 mg/24 hrs.	Same as amitriptyline. See below[b]
Medications for thick secretions (mucus)			
Guaifenesin (Robitussin) (Mucinex)	Elixir 100 mg/5 cc 600 mg tablets	600–1200 mg every 12 hours	GI upset
Albuterol	0.083%/3 cc	3 cc via nebulizer up to 4 times per day	tremor, nervousness insomnia, tachycardia
Acetylcysteine (Mucomyst)	10% or 20% per 2 cc	Start with 10%/2cc via nebulizer mixed with albuterol BID	Bronchospasm, GI upset

[a]As a class, anticholinergics can produce the side effects of constipation, dry mouth, urinary retention, blurry vision, altered mental status, tachycardia and reduced sweating. Anticholinergics can also precipitate narrow angle glaucoma.

[b]TCAs have anticholinergic properties and therefore share all of the side effects of the anticholinergic group as listed above.

IV, intravenous; IM, intramuscular; SL, sublingual, PO, orally; GI, gastrointestinal; BID, twice daily; BP, blood pressure.

Percussion and vibration. Manual chest clapping can be used to mobilize bronchial secretions and stimulate a cough. Mechanical vibration of the chest using vests that provide high frequency chest wall oscillations is believed to aid in the mobilization of tracheobronchial secretions by producing shearing forces that thin mucus. The vest can be self-applied and may provide an alternative to conventional postural drainage with percussion.[51,53–55]

Incentive spirometry. An incentive spriometer is a device that provides visual and possibly auditory feedback for preset inspiratory flows or volumes. While a single deep breath, multiple deep breaths, and a deep breath held for 5 seconds has been demonstrated to reduce atelectasis there is no evidence to support incentive_spirometry in an airway clearance regimen.[51,56]

Glossopharyngeal breathing or frog breathing. Glossopharyngeal breathing is a form of voluntary positive pressure breathing accomplished in a series of air gulping actions generated by the lips, tongue, pharynx, and palate. The larynx acts as a valve maintaining air in the lungs between gulps. Patients with ineffective cough related to low vital capacity, such as those with neuromuscular disorders and spinal cord injury, can use glossopharyngeal breathing to achieve greater chest inflation prior to a cough. This allows more effective mobilization of secretions.[51,57–59]

Active cycle of breathing. Active cycle of breathing is comprised of a series of techniques of breath control, deep breathing, and forced expiration called huffing at both low and high lung volumes. It has been demonstrated to mobilize secretions more effectively than manual percussion.[51,60,61]

Forced expiratory maneuvers. The Flutter VRP1® (Scandipharm Inc., Birmingham, AL) is a pipe-shaped device with a high-density stainless-steel ball enclosed in a cone within the bowl. Upon expiration the ball rises and falls creating oscillatory positive pressure and vibration within the airways.[51,62] The RC-Cornet® (Pari Respiratory Equipment, Midlothian, VA) is a curved plastic tube containing a flexible hose and valve. During expiration a positive and oscillating airway pressure is created.[51,63] The positive expiratory pressure (PEP) device consists of a facemask or mouthpiece to which various resistors can be attached in order to achieve a desired level of more continuous positive airway pressure.[51,64–66] All of these techniques aid in the mobilization of secretions that can then be cleared by huffing or coughing. All are superior to conventional postural drainage and percussion and are preferred by patients. There are no convincing data to recommend one technique over another.

Cough assist

Weakness of inspiratory and expiratory respiratory muscles and weakness of the vocal cords may contribute to a poor cough and contribute to the development of mucus plugging, atelectasis and pneumonia. An insufflator–exsufflator or cough-assist device may benefit such patients. With the nose occluded, the patient places a tube between the teeth and seals the lips. With the push of a button the device delivers a preset volume of air via the sealed oral cavity to inflate the lungs. Pushing a second button causes the device to generate negative pressure. Timing a voluntary cough with the expiratory phase of the insufflator-exsufflator produces a greater velocity of air movement than can be achieved by weakened expiratory muscles alone, leading to more effective secretion clearance. The device can be used as needed or on a set daily schedule to maintain airway clearance. With appropriate adaptation the device can be used in patients with a tracheostomy. Administration of nebulized medications prior to use of the insufflator-exsufflator may facilitate airway clearance.[67–77]

RADIATION

Radiation of the salivary glands is an option in patients who fail or cannot tolerate medical therapy and for whom surgical treatments are not appropriate. The dose of radiation should be gradually titrated to reach the desired effect so that severe xerostomia may be avoided. The submandibular and parotid glands can be targeted individually or together. Radiation can be complicated by the induction of head and neck malignancies but this has an expected onset after a decade or more. Thus radiation therapy may be appropriate for management of sialorrhea and excessive pharyngeal secretions in patients for whom life expectancy is reduced to this level.[32,78]

SURGERY FOR SIALORRHEA

Surgery should be considered in the management of severe sialorrhea when all medical therapies have failed. Options include surgery upon the salivary glands and or the salivary ducts and surgery to denervate the salivary glands.[6,32,79]

Bilateral redirection of the submandibular ducts can transfer salivary flow from the front to the back of the mouth. The ducts are dissected free and then relocated posterior to the tonsillar pillars. This may require a tonsillectomy. The procedure may be complicated by the development of a ranula secondary to extravasation of saliva into the soft tissues of the floor of the mouth and may increase the chance for anterior dental caries. The parotid ducts (Stetson's duct) can be similarly redirected posterior to the tonsillar pillars. This type of surgery is not an option for patients with pharyngeal weakness because it will increase posterior pharyngeal secretions and may thereby increase the chance of aspiration.[32,80,81] The most aggressive surgical option entails bilateral resection of the submandibular glands and ligation of the parotid ducts and avoids the potential complication of increased pharyngeal secretions. Parotid duct ligation may be complicated by the production of a sialocele or salivary retention cyst.[32,80,81]

The simplest surgical option for management of sialorrhea is transtympanic neurectomy. The procedure can be accomplished without general anesthesia and is performed by sectioning the tympanic plexus and at times the chorda tympani in the middle ear, thereby interrupting parasympathetic innervation to the major salivary glands. It can be complicated by loss of taste on the anterior two thirds of the tongue. The procedure may have to be repeated since salivary function predictably returns after 12–18 months, when nerve regeneration has occurred.[32]

SURGERY FOR EXCESSIVE PHARYNGEAL OR TRACHEOBRONCHIAL SECRETIONS

Surgery for the management of excessive pharyngeal or tracheobronchial secretions is usually restricted to patients with intractable neurogenic aspiration who have failed medical management and have incurred multiple aspiration pneumonias. Multiple options are available and the appropriate procedure should be dictated by the neurophysiologic cause of dysphagia and the overall status of the patient.

A common option is tracheostomy, which provides access to the trachea for mechanical cough assist and or manual suctioning, along with placement of an enteral feeding tube to limit or eliminate the passage of food and drink. This does little to prevent the aspiration of oropharyngeal secretions. Impaired relaxation of the cricopharyngeus (upper esophageal sphincter) during swallowing is a cause of neurogenic dysphagia. Incoordination or increased tone in this muscle may lead to functional obstruction of the upper esophagus and pharyngeal dysphagia. Cricopharyngeal myotomy combined with arytenoid adduction or epiglottic plication has improved aspiration in patients with this form of dysphagia. In patients with bilateral vocal cord paralysis the vocal cords can be surgically medialized, which will decrease but not eliminate aspiration. Recurrent neurogenic aspiration may also be treated with endotracheal stenting. In this procedure a stent is endoscopically placed into the larynx. Aspiration is minimized by tightly fitting the stent such that the lateral walls of the stent directly abut the laryngeal wall. The superior aspect of the device is closed; therefore a tracheostomy is required. Laryngeal diversion with laryngotracheal separation is another albeit more invasive option. There are two forms of this procedure. Both require sectioning the upper trachea just below the larynx and suturing the lower trachea to the skin of the anterior neck. In one version of this procedure the superior larynx is sutured closed. In another it is anastamosed to the esophagus. The most drastic option for the treatment of intractable aspiration is total laryngectomy with tracheostomy.[3,6,7,32,79]

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Address reprint requests to:
Leo McCluskey, M.D., M.B.E.
Penn Neurological Institute
330 South 9th Street
Philadelphia, PA 19107

E-mail: lfmcclusky@pahosp.com