

## REVIEW ARTICLE

# Drooling

Crispian Scully<sup>1</sup>, Jacobo Limeres<sup>2</sup>, Michael Gleeson<sup>3</sup>, Inmaculada Tomás<sup>2</sup>, Pedro Diz<sup>2</sup>

<sup>1</sup>Eastman Dental Institute, University College London, London, UK; <sup>2</sup>Special Needs Unit, School of Medicine and Dentistry, Santiago de Compostela University, Spain; <sup>3</sup>Institute of Neurology, University College London, London, UK

**INTRODUCTION:** Drooling is the overflowing of saliva from the mouth. It is mainly due to neurological disturbance and less frequently to hypersalivation. Drooling can lead to functional and clinical consequences for patients, families, and caregivers. The aim of this review is to emphasize the clinical aspects of the assessing and management of drooling.

**METHODS:** All papers and clinical reviews of drooling in the electronic data bases (Medline, PubMed, Embase and the Cochrane Library) for the past 40 years in any languages have been evaluated.

**RESULTS:** The severity of drooling and the effects on the quality of life of the patient and family, help to establish a prognosis and to decide the therapeutic regimen. Treatment options range from conservative therapy to medication, radiation, or surgery, and often a combination is needed.

**CONCLUSIONS:** Chronic drooling remains a problem that can be difficult to manage. Despite the acceptable results obtained with most of the treatments, none is free of undesirable effects.

J Oral Pathol Med (2009) 38: 321–327

**Keywords:** drooling; oral health; saliva; sialorrhea

## Introduction

Drooling or salivary incontinence is an indication of an upset in the coordinated control mechanism of orofacial musculature and palato-lingual musculature leading to excessive pooling of saliva in the anterior mouth and resulting in unintentional loss of saliva from the mouth (1). Drooling is normal in healthy infants; but usually drooling stops by about 18 months of age and is considered abnormal if it persists beyond the age of 4 years (2) (Fig. 1). Drooling can lead to functional, social, psychological, and clinical consequences for

patients, families, and caregivers. Saliva soils clothing and patients may have perioral skin breakdown and infections, disturbed speech and eating, and can occasionally develop aspiration-related and pulmonary complications.

Sialorrhea, hypersialia, hypersalivation and ptialism, are terms used to describe increased salivary flow (3). Drooling it is not usually associated with an increased production of saliva; in fact, in most cases, the volume of saliva produced is normal (4).

The aim of this review is to emphasize the clinical aspects of the assessment and management of drooling.

## Method

All papers and clinical reviews of drooling in the electronic data bases (Medline, PubMed, Embase and the Cochrane Library) for the past 40 years in any of the languages have been evaluated. The main search terms used were 'saliva', 'drooling', 'sialorrhea', 'hypersalivation' and 'ptialism'. We also perused relevant reports and abstracts from the past five symposiums of the International Association for Disability and Oral Health and the International Association for Dental Research.



Figure 1 Drooling in a cerebral palsy patient.

Correspondence: Jacobo Limeres, Special Needs Unit, School of Medicine and Dentistry, Santiago de Compostela University, C/Enterríos sn, 15782 Santiago de Compostela, Spain. Tel: 00 34 981 56 31 00, Fax: 00 34 981 56 22 26, E-mail: jacobo.limeres@usc.es  
Accepted for publication September 25, 2008

## Salivary secretion and physiology of swallowing

Salivary secretion is regulated by a reflex arch. The afferent part is mainly activated by stimulation of chemoreceptors (located in the taste buds) and mechanoreceptors (located in the periodontal ligament). Olfaction and stretch of the stomach are other surprisingly poor stimuli (5). Other impulses affecting secretion depend on, for example, the emotional state (5). The afferent cranial nerves V, VII, IX and X carry impulses to the salivary nuclei (salivation center) in the medulla oblongata (6). The efferent part of the reflex is mainly parasympathetic. The cranial nerve VII provides control of the submandibular, sublingual, and minor glands, whereas the cranial nerve IX controls the parotid glands (6). The flow of saliva is enhanced by sympathetic innervation, which promotes contraction of muscle fibers around the salivary ducts (7).

Saliva may be secreted in the absence of exogenous stimuli referred to as the resting or unstimulated salivary flow. In the resting state, 70% of saliva is secreted by the submandibular and sublingual glands. When stimulated, salivary flow increases up to five fold, with the parotids providing most of the saliva. On average, in healthy non-medicated adults, the unstimulated and chewing-stimulated salivary flow rate are about 0.3 and 1.5 ml/min respectively (3), but the range is wide and the limits of normalcy in all age groups and both genders are considerable. The pathophysiology of sialorrhea remains unclear.

Spontaneous swallowing is a pre-requisite for normal development of feeding and drool control. Swallowing is a complex neuromuscular activity involving rapid coordination of structures in the oral cavity, pharynx, larynx, and esophagus. Swallowing involves three stages, oral, pharyngeal and esophageal, with the oral phase under voluntary neuromuscular control and the latter two phases under involuntary neuromuscular control (8).

The oral phase of swallowing can be further subdivided into the oral preparatory and the oral transport stages. In the oral preparatory stage, the main neuromuscular actions include: lip closure, tension in the labial and buccal musculature, rotary motion of the jaw, lateral rolling motion of the tongue, and bulging forward of the soft palate (to prevent nasal aspiration). The oral transport stage lasts 1 s and occurs as the musculature of the lips and cheeks contract followed by tongue contraction against the hard palate, and velum musculature contraction against the nasopharyngeal mucosa. Normal tongue motion is essential for carrying out the tasks of the oral phase of swallowing. Pharyngeal phase also lasts <1 s and the neuromuscular actions involved are: velopharyngeal closure, tongue-base retraction (starts pharyngeal peristalsis), elevation and closure of larynx (airway protection to prevent aspiration), and opening of the cricopharyngeal region. The esophageal transport from the upper esophageal sphincter to the stomach lasts 8–20 s, and the main neuromuscular action is the generation of the 'primary

wave' (and eventually 'secondary wave' when there is increased pressure in mid-esophagus).

Swallowing is initiated by sensory impulses transmitted as a result of stimulation of touch receptors on the fauces, tonsils, soft palate, base of the tongue, and posterior pharyngeal wall. Sensory impulses reach the brainstem primarily through the cranial nerves IX and X, while the efferent function is mediated through the cranial nerves V, VII, IX, X and XII (9). Cricopharyngeal sphincter opening is reflexive. The respiratory center of the medulla is directly inhibited by the swallowing center for the very brief time that it takes to swallow (deglutition apnea). Swallowing is a centrally coordinated process where supratentorial and infratentorial regions of control are involved. The supratentorial area of control is located in the frontal cortex (10). The infratentorial or brainstem areas are located in the dorsal region within the nucleus of the tractus solitarius as well as in the nucleus ambiguus (11). Both, the cortical and sub-cortical control regions are important pathways in the voluntary initiation of swallowing (oral phase). The brainstem is responsible for the involuntary (pharyngeal and esophageal) phases of swallowing.

In healthy individuals the daily production and swallowing of saliva ranges from 0.5–1.5 l. In a 24-h cycle, about 600 swallowing actions occur (12). Under normal circumstances, individuals are able to compensate for increased salivation by swallowing. Hypersalivation does not necessarily lead to drooling. Poor swallowing function leads to excessive pooling of saliva in the anterior portion of the oral cavity and drooling.

## Etiology and prevalence of drooling

Drooling may be caused by an oral motor dysfunction, inadequate swallowing capacity, a deficit of the oral sphincter and, less frequently, sialorrhea (4). Drooling usually occurs as result of some form of neurological disturbance, either central (e.g. cerebral palsy) or peripheral (e.g. facial palsy) (13) (Table 1). Oral preparative and oral transportation phases of swallowing are usually altered. Muscle incoordination then hampers the initiation of the swallowing reflex, and is characterized by incoordination of the smooth transition of saliva from the mouth to the oropharynx (14). Other factors that may influence the severity of drooling include inadequacy of lip seal, muscle hypotonia, macroglossia, dental malocclusion, abnormal posture, emotional state, impairment of concentration, decreased oral sensory awareness and impaired nasal airway patency (15).

In children, cerebral palsy is the most common cause. It has been estimated that drooling abnormally persists in 10–38% of individuals with cerebral palsy (16), although in some series this percentage exceeds 50% (4). It has been suggested that in about 10% of children with cerebral palsy drooling interferes with daily social activities and functions (17). Drooling is also a common problem in Down syndrome (18) and learning disability (19, 20).

**Table 1** Most common causes of drooling

<i>Decreased swallowing</i>		
<i>Neuro-muscular diseases</i>	<i>Anatomic abnormalities</i>	<i>Excessive saliva production (sialorrhea)</i>
-Cerebral palsy	-Macroglossia	-Irritating oral disease (i.e. ulceration, infection, trauma)
-Amyotrophic lateral sclerosis	-Orthodontic problems (i.e. anterior open bite, lip seal incompetence)	-Medication side-effects (i.e. clozapine, risperidone, nitrazepam, bethanecol)
-Parkinson's disease	-Surgical defects following major head and neck resection	-Gastroesophageal reflux (water brash)
-Heavy metal neurotoxins (e.g. mercury, thallium, copper, arsenic, antimony)	-Ankylosis of the temporo-mandibular joint	-Other less frequent causes (i.e. otolaryngologic diseases such as laryngitis, pharyngitis, tonsillitis and epiglottitis, secretory phase of the menstrual cycle, pregnancy)
-Intellectual disability		
-Wilson disease		
-Angelman syndrome		
-Moebius syndrome		
-Pseudobulbar palsy		
-Bulbar palsy		
-Stroke		
-Facial palsy		

In adults, Parkinson's disease is the most common cause. About 45% of parkinsonian patients complain about drooling, which in 15% of cases is detected in the early phases of disease (21). Drooling is also commonly related to other neurological conditions such as stroke, pseudobulbar palsy, or bulbar palsy, where it is detected in almost 30% of the patients (22).

People with pharyngeal or esophageal obstruction may also be affected, and oropharyngeal and esophageal cancers are important causes to exclude (23). Surgical defects following major head and neck resection may also impede the ability to manage increased secretions leading to drooling (24).

**Clinical features of drooling**

Saliva soils clothing, furniture, carpets, teaching materials, communicative devices and toys, etc. such that some patients need their clothes and bibs changing up to 15 times a day and almost always wear a towel around the neck (4, 25). Patients may experience repeated perioral skin breakdown and infections. Aspiration-related respiratory and pulmonary complications are more frequent in those with a diminished sensation of salivary flow and hypopharyngeal retention. Drooling may not only be unaesthetic but can also affect speech and eating (26), and thus stigmatism is common.

Drooling can lead to functional, social, psychological, and clinical consequences for patients, families, and caregivers (19).

**Assessment of drooling**

A thorough history helps assess the severity of drooling, the effects on the quality of life for the patient and family, and to establish a prognosis and to decide the therapeutic regimen. Volumetric measurement for absolute quantification of saliva spill or intraoral pooling (e.g. external collection devices, intraoral suction hook, etc.) can help guide treatment and assess outcomes. Counting the number of bibs or items of clothing soiled each day provides a subjective estimate. Physical findings such as skin maceration on the neck, chest, and hands because of dampness and constant wiping confirm impressions of severity and indicate the need for treatment. The most popular scales categorize drooling into dry, mild, moderate or frequent, and evaluates the results of drooling interventions into excellent, good, fair and poor (27) (Table 2). The drooling quotient is a validated, semi-quantitative, direct observational method; the presence or absence of drooling is assessed every 15 s during two 10-min periods separated by a 60-min break (28, 29). A thorough head and neck and oral examination is indicated and the main topics have been included in Table 3.

**Table 2** Qualitative evaluation of drooling

<i>Quantification of drooling</i>		<i>Wilkie and Brody classification of efficacy therapeutic procedures for drooling (27)</i>	
Dry	Never drools	Excellent	Normal salivary control
Mild	Only lips wet	Good	Slight loss of saliva with or without dried froth on the lips
Occasional drooling	Not every day	Fair	Improved, but with significant residual saliva loss or with thickened, offensive, brown, gummy froth
Moderate	Lips and chin wet		
Frequent	Drooling every day	Poor	Failure to control or too dry
Constant	Constant drooling		
Severe	Clothing soiled		
Profuse	Hands, and tray moist and wet		

**Table 3** Assessment of drooling

---

Measurement of salivary flow
Head position and control
Competence of labial sphincter
Coordination of the swallowing neuromuscular mechanisms
Perioral skin condition
Dentition and occlusion
Mandible and palatal position
Presence of mouth breathing
Gag reflex and intraoral tactile sensitivity
Nasal obstruction and appearance of tissues on anterior rhinoscopy
Flexible nasopharyngoscopy or lateral neck film to detect adenoid hypertrophy
Swallowing efficiency: determined by observation, barium swallow, or fiber optic endoscopy
Salivary gland size
Neurologic examination (cranial nerve examination)
Radiosialography for evaluating salivary secretory function

---

**Table 4** Treatments of drooling

---

Oral motor training
Speech therapy
Behavior therapy
Pharmacotherapeutics (e.g. benztropine, glycopyrrolate, scopolamine)
Botulinum toxin A
Surgery
Radiotherapy
Others (photocoagulation of salivary ducts, tongue acupuncture)

---

**Management of drooling**

When drooling impacts on the quality of life, treatment is indicated, but to reduce drooling and not to completely eliminate salivation – as that risks complications from xerostomia. A team including at least an otolaryngologist, neurologist, dentist, speech, occupational and physical therapists is needed (30). Options range from conservative therapy (e.g. oral motor training) to medication, radiation, or surgery, and often a combination is needed (Table 4).

The first objective of treatment is to correct or minimize anything that facilitates drooling, such as a head-down posture, inadequate lip seal, nasal obstruction, etc. (7). Regular oral health care is recommended (31, 32).

*Speech/physical therapy*

Oral motor training can be used to try to normalize muscle tone, stabilize the positions of body, head,

and jaw, decrease tongue thrust, increase lip closure, and promote swallowing but is time-consuming, and requires motivation. Although few data are available to confirm the effectiveness of these therapies, a 6-month trial is worth considering. Choices include customized dental appliances to aid lip closure (33, 34), stimulate tongue movement to deflect saliva toward the pharynx (26, 29, 35) or to stimulate the musculature (17, 33). The speech therapist plays a crucial role in improving drooling for example by using vibrations applied to the masseter, the digastrics and the upper lip (36). Biofeedback and automatic cueing techniques can decrease drooling (37, 38). Specific programs have been developed for cerebral palsy, including attendance at ‘anti-drooling’ classes and use of saliva-collecting cups (39). Success depends on the patient’s cognitive level but also, patients tend to become habituated to the stimulus, and thus the effectiveness of the devices wanes (40, 41).

*Pharmacologic treatment*

Pharmacologic treatment of drooling attempts to decrease salivation by reducing cholinergic activity, either systemically (e.g. atropinics) or more locally (e.g. sublingual ipratropium spray); or increasing adrenergic activity (e.g. clonidine patches) (Table 5) (7). However, these drugs may have adverse effects and are contraindicated in patients with asthma, or glaucoma. A systematic review including seven comparable studies, found evidence that benztropine, glycopyrrolate, and benzhexol hydrochloride are effective but no one drug was preferable (42).

Oral or sublingual administered atropine sulfate can reduce salivation (43), although some authors have suggested that there is a lack of scientific evidence supporting it (44). Transdermal scopolamine, applied as a patch behind the ear, has been well tolerated in short-term studies (45, 46). No data confirm its efficacy over long periods. Nebulized scopolamine has been also used (47). Benztropine in one double-blind trial produced a 65% decrease in drooling in adults but this did not apply to pediatric population (48, 49). Glycopyrrolate has, in prospective randomized trials, significantly reduced drooling in patients with cerebral palsy and other developmental disorders, but 20 percent of patients had adverse effects including behavior changes (50, 51). Clonidine patches can control hypersalivation induced by antipsychotics such as clozapine (52).

**Table 5** Medications most commonly used to reduce sialorrhea/drooling

---

<i>Agent</i>	<i>How supplied</i>	<i>Adult dosage</i>	<i>Potential adverse effects apart from xerostomia</i>
Glycopyrrolate	Tablets, 1 or 2 mg	Start 0.5 mg orally, one to three times daily; titrate to effectiveness and tolerability	Constipation, urinary retention, blurred vision, hyperactivity, irritability
Scopolamine	Patch, 1.5 mg	One patch every 3 days	Pruritus at patch site, urinary retention, irritability, blurred four vision, dizziness, glaucoma
Botulinum toxin A	Vial, 100 U per vial	Single injections of 10–40 units under ultrasound guidance into all major glands	Pain at injection site Local swelling Difficulty in swallowing

---

Adapted from Hockstein et al. (7).



Several studies on botulinum toxin serotype A in the treatment of drooling have been published, confirming the outcome with very rare reports of serious complications (28, 53, 54). However, in a recently published review it has been stated that the effective therapeutic dose and ideal form of application remain to be established (55). The parotid gland is usually injected in two or three sites and the submandibular gland in one to three. The efficacy of treatment varies from 6 weeks to 6 months. The reported side-effects include weakness of the adjacent muscles producing difficulty in swallowing because of toxin diffusion to masseter or pharyngeal muscles, risk of aspiration, facial weakness, and local infection. Other potential effects are hematoma, salivary duct calculi, local injuries of the carotid arteries or branches of the facial nerve, and recurrent jaw dislocation. Percutaneous injection under ultrasound guidance improves efficacy and safety (56).

Irradiation of the salivary glands has been used to decrease saliva secretion, but has variable success, a significant risk of xerostomia and loss of taste (57), and has the potential risk of malignancy in the irradiated field (58). However, it has been successful in treating drooling in patients with amyotrophic lateral sclerosis (59, 60) and in patients with parkinsonism (57).

*Surgical therapy*

Surgery is indicated when drooling persists after at least 6 months of conservative therapy or is moderate to profuse in a patient whose cognitive function precludes use of conservative approach (2). It is best deferred until the patient is at least 6-year-old as by this time, there should be full maturation of oral motor function and coordination.

Surgical procedures used to control drooling are aimed at decreasing salivary flow, or redirecting the salivary duct flow to a location more advantageous to promote swallowing (7). Although a range of techniques

to control drooling have been proposed, controversy exists regarding indications and results (Table 6).

A number of procedures has been proposed in the past and they have been found to be wanting. These have ranged from excision of the major glands and denervation procedures to relocation or ligation of the salivary ducts. Resection of the major glands is a significant undertaking and leaves the patient with xerostomia and a visible scar, at the least, with a facial nerve weakness at worst (61). The results of denervation by section of the chorda tympani nerve or tympanic neurectomy are frankly disappointing. Only 50–80% of patients are reported to have achieved a satisfactory outcome and some of them regress over time because of neural re-growth. Furthermore, bilateral tympanotomy is inevitably attended by a period of hearing loss until middle ear exudates have dissipated and section of the chorda tympani results in a permanent taste deficit (62). It is perhaps not surprising that these operations have become obsolete.

Nowadays, the gold standard surgical procedure is submandibular duct relocation as the submandibular salivary glands are the major contributors to resting salivary flow (61). In this way, the salivary flow is re-directed to the back of the mouth, a site more conducive to swallowing than drooling. There are significant advantages to both the patient and surgeon from duct relocation. The most obvious is the absence of an external scar and a potential facial nerve weakness that might have been sustained during resection of the gland and compromised the oral competence of a cerebral palsied child. Two complications are regularly encountered – ranula formation and submandibular duct obstruction. Crysdale and White (19) reported an 8% incidence of post-operative ranula formation in their series of 194 patients. The frequency of this complication prompted them to modify their technique and resect all sublingual tissue at the time of relocation. It was also

**Table 6** Surgical treatment of drooling/sialorrhea

<i>Surgical therapy</i>	<i>Advantages</i>	<i>Disadvantages</i>
Submandibular gland excision	Good control of sialorrhea	General anesthesia usually needed External scar Potential for dental caries
Submandibular duct re-routing	No external scar	Risk of facial nerve damage General anaesthesia usually needed Potential for anterior dental caries Potential for aspiration Ranula and duct obstruction Additional surgery for persistent drooling Lateral neck cyst
Parotid duct re-routing	Redirects flow in the stimulated state	Risk of lingual nerve damage General anesthesia usually needed Risk of sialocele
Parotid duct ligation	Simple procedure Decreases flow in the stimulated state	Potential for aspiration Risk of sialocele
Transtympanic neurectomy	Does not require general anesthesia Technically simple, fast procedure	Predictable return of salivary function Taste deficit Transient hearing loss Requires repeating

Adapted from Hockstein et al. (7).

thought that this might reduce salivary flow further. Unfortunately, the addition of sublingual gland resection was and is still found to be ineffective and increases morbidity in terms of hemorrhage and pain (63). Late obstruction of relocated submandibular ducts has been attributed to excessive tonsil size and recurrent tonsillitis. Surgical removal of these obstructed and re-routed glands has been found to be difficult because of adhesions. Patients with a longstanding history of tonsillar infection should have a tonsillectomy some weeks before relocation.

The results of treatment by submandibular duct relocation are difficult to interpret. Reduction in the severity of drooling should be achieved in at least 80% of patients, but that is not synonymous with the cessation of sialorrhea. Saliva is still present on the chins of up to 70% of those cases deemed by the surgeon to be successful. Success is therefore very difficult to assess and greatly depends on the expectations of the patient or parents (64).

In summary, despite the acceptable results obtained with most of the treatments however, none is free of undesirable effects. Therefore, chronic drooling remains a problem that can be difficult to manage and a multidisciplinary approach is required.

## References

- Blasco PA, Allaire JH, and participants of the Consortium on Drooling. Drooling in the developmentally disabled: management practices and recommendations. *Dev Med Child Neurol* 1992; **34**: 849–62.
- Crysdale WS. Management options for the drooling patient. *Ear Nose Throat J* 1989; **68**: 820, 825–6, 829–30.
- Sreebny LM. Saliva in health and disease: an appraisal and update. *Int Dent J* 2000; **50**: 140–61.
- Tahmassebi JF, Curzon ME. The cause of drooling in children with cerebral palsy – hypersalivation or swallowing defect. *Int J Paediatr Dent* 2003; **13**: 106–11.
- Pedersen AM, Bardow A, Jensen SB, Nauntofte B. Saliva and gastrointestinal functions of taste, mastication, swallowing and digestion. *Oral Dis* 2002; **8**: 117–29.
- Garrett JR, Proctor GB. Control of salivation. In: Linden RWA ed. *The Scientific Basis of Eating*. *Ront Oral Biol*. Basel: Karger, 1998; 135–55.
- Hockstein NG, Samadi DS, Gendron K, Handler SD. Sialorrhea: a management challenge. *Am Fam Physician* 2004; **69**: 2628–34.
- Dodds WJ. Physiology of swallowing. *Dysphagia* 1989; **3**: 171–8.
- Hamdy S, Aziz Q, Rothwell JC, Hobson A, Barlow J, Thompson DG. Cranial nerve modulation of human cortical swallowing motor pathways. *Am J Physiol* 1997; **1272**: 802–8.
- Hamdy S, Aziz Q, Rothwell JC, et al. The cortical topography of human swallowing musculature in health and disease. *Nature Med* 1996; **2**: 1217–24.
- Zoungrana OR, Amri M, Car A, Roman C. Intracellular activity of motoneurons of the rostral nucleus ambiguus during swallowing in sheep. *J Neurophysiol* 1997; **77**: 909–22.
- Jenkins GN. Saliva. In: Jenkins GN ed. *The Physiology and Biochemistry of the Mouth*, 4th edn. Oxford: Blackwell Scientific Publications, 1978; 284–359.
- Nunn JH. Drooling: Review of the literature and proposals for management. *J Oral Rehabil* 2000; **27**: 735–43.
- Myer C. Sialorrhea. *Pediatr Clin North Am* 1989; **36**: 1495–500.
- Lespargot A, Langevin MF, Muller S, Guillemont S. Swallowing disturbances associated with drooling in cerebral-palsied children. *Dev Med Child Neurol* 1993; **35**: 298–304.
- Johnson H, Scott A. *A Practical Approach to Saliva Control*. Tucson, AZ: Communication Skill Builders Inc., 1993.
- Harris SR, Purdy AH. Drooling and its management in cerebral palsy. *Dev Med Child Neurol* 1987; **29**: 807–11.
- Limbrock GJ, Hoyer H, Scheyling H. Regulation therapy by Castillo-Morales in children with Down's syndrome: primary and secondary orofacial pathology. *ASDC J Dent Child* 1990; **57**: 437–41.
- Crysdale WS, White A. Submandibular duct relocation for drooling: A 10-year experience with 194 patients. *Otolaryngol Head Neck Surg* 1989; **101**: 87–92.
- Limbrock GJ, Hoyer H, Scheyling H. Drooling, chewing and swallowing dysfunctions in children with cerebral palsy: treatment according to Castillo-Morales. *ASDC J Dent Child* 1990; **57**: 445–51.
- Volonte MA, Porta M, Comi G. Clinical assessment of dysphagia in early phases of Parkinson's disease. *Neurol Sci* 2002; **23**(Suppl. 2): S121–2.
- Sullivan PB, Lambert B, Rose M, Ford-Adams M, Johnson A, Griffiths P. Prevalence and severity of feeding and nutritional problems in children with neurological impairment: Oxford Feeding Study. *Dev Med Child Neurol* 2000; **42**: 674–80.
- Boyce HW, Bakheet MR. Sialorrhea: a review of a vexing, often unrecognized sign of oropharyngeal and esophageal disease. *J Clin Gastroenterol* 2005; **39**: 89–97.
- Donald PJ. Surgical rehabilitation following anterior resection for oral cavity carcinoma. *Laryngoscope* 1981; **91**: 1941–56.
- Reddihough D, Johnson H, Ferguson E. The role of a saliva control clinic in the management of drooling. *J Paediatr Child Health* 1992; **28**: 395–7.
- Fischer-Brandies H, Avalle C, Limbrock GJ. Therapy of orofacial dysfunctions in cerebral palsy according to Castillo-Morales: first results of a new treatment concept. *Eur J Orthod* 1987; **9**: 139–43.
- Wilkie TF, Brody GS. The surgical treatment of drooling. A ten-year review. *Plast Reconstr Surg* 1977; **59**: 791–8.
- Jongerius PH, Rotteveel JJ, Van Limbeek J, Gabreels FJ, Van Hulst K, Van Den Hoogen FJ. Botulinum toxin effect on salivary flow rate in children with cerebral palsy. *Neurology* 2004; **63**: 1371–5.
- Limbrock GJ, Fischer-Brandies H, Avalle C. Castillo-Morales' orofacial therapy: treatment of 67 children with Down syndrome. *Dev Med Child Neurol* 1991; **33**: 296–303.
- Blasco PA. Management of drooling: 10 years after the Consortium on Drooling, 1990. *Dev Med Child Neurol* 2002; **44**: 778–81.
- Crysdale WS, Mccann C, Roske L, Joseph M, Semenuk D, Chait P. Saliva control issues in the neurologically challenged. A 30 year experience in team management. *Int J Pediatr Otorhinolaryngol* 2006; **70**: 519–27.
- Meningaud JP, Pitak-Arnop P, Chikhani Lbertrand JC. Drooling of saliva: a review of the etiology and management options. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2006; **101**: 48–57.

33. Johnson HM, Reid SM, Hazard CJ, Lucas JO, Desai M, Reddihough DS. Effectiveness of the Innsbruck Sensorimotor Activator and Regulator in improving saliva control in children with cerebral palsy. *Dev Med Child Neurol* 2004; **46**: 39–45.
34. Haberfellner H. ISMAR: an autotherapeutic device assisting patients from drooling to articulated speech. *Pediatr Rehabil* 2005; **8**: 248–62.
35. Inga CJ, Reddy AK, Richardson SA, Sanders B. Appliance for chronic drooling in cerebral palsy patients. *Pediatr Dent* 2001; **23**: 241–2.
36. Mcracken A. Drool control and tongue thrust therapy for the mentally retarded. *Am J Occup Ther* 1978; **32**: 79–85.
37. Domaracki LS, Sisson LA. Decreasing drooling with oral motor stimulation in children with multiple disabilities. *Am J Occup Ther* 1990; **44**: 680–4.
38. Koheil R, Sochaniwskyj AE, Bablich K, Kenny DJ, Milner M. Biofeedback techniques and behaviour modification in the conservative remediation of drooling by children with cerebral palsy. *Dev Med Child Neurol* 1987; **29**: 19–26.
39. Harris MM, Dignam PF. A non-surgical method of reducing drooling in cerebral palsied children. *Dev Med Child Neurol* 1980; **22**: 293–9.
40. Lancioni GE, Brouwer JA, Coninx F. Automatic cueing to reduce drooling: a long-term follow-up with two mentally handicapped persons. *J Behav Ther Exp Psychiatry* 1994; **25**: 149–52.
41. Van Der Burg JJ, Didden R, Jongerius PH, Rotteveel JJ. A descriptive analysis on behavioural treatment of drooling (1970–2005). *Dev Med Child Neurol* 2007; **49**: 390–4.
42. Jongerius PH, Van Tiel P, Van Limbeek J, Gabreels FJ, Rotteveel JJ. A systematic review for evidence of efficacy of anticholinergic drugs to treat drooling. *Arch Dis Child* 2003; **88**: 911–4.
43. Dworkin J, Nadal J. Nonsurgical treatment of drooling in a patient with closed head injury and severe dysarthria. *Dysphagia* 1991; **6**: 40–9.
44. De Simone GG, Eisenclas JH, Junin M, Pereyra F, Brizuela R. Atropine drops for drooling: a randomized controlled trial. *Palliat Med* 2006; **20**: 665–71.
45. Talmi YP, Finkelstein Y, Zohar Y. Reduction of salivary flow with transdermal scopolamine: a four-year experience. *Otolaryngol Head Neck Surg* 1990; **103**: 615–8.
46. Lewis DW, Fontana C, Mehallick LK, Everett Y. Transdermal scopolamine for reduction of drooling in developmentally delayed children. *Dev Med Child Neurol* 1994; **36**: 484–6.
47. Zepetella G. Nebulized scopolamine in the management of oral dribbling: three case reports. *J Pain Symptom Manage* 1999; **17**: 293–5.
48. Owen SE, Stern ML. Management of drooling in cerebral palsy: three single case studies. *Int J Rehabil Res* 1992; **15**: 166–9.
49. Camp-Bruno JA, Winsberg BG, Green-Parsons AR, Abrams JP. Efficacy of benztropine therapy for drooling. *Dev Med Child Neurol* 1989; **31**: 309–19.
50. Mier RJ, Bachrach SJ, Lakin RC, Barker T, Childs J, Moran M. Treatment of sialorrhea with glycopyrrolate: a double-blind, dose-ranging study. *Arch Pediatr Adolesc Med* 2000; **154**: 1214–8.
51. Blasco PA, Stansbury JC. Glycopyrrolate treatment of chronic drooling. *Arch Pediatr Adolesc Med* 1996; **150**: 932–5.
52. Grabowski J. Clonidine treatment of clozapine-induced hypersalivation. *J Clin Psychopharmacol* 1992; **12**: 69–70.
53. Benson J, Daugherty KK. Botulinum toxin A in the treatment of sialorrhea. *Ann Pharmacother* 2007; **41**: 79–85.
54. Nobrega AC, Rodrigues B, Torre AC, Enzo A, Melo A. Does botulinum toxin decrease frequency and severity of sialorrhea in Parkinson's disease? *J Neurol Sci* 2007; **253**: 85–7.
55. Fuster Torres MA, Berini Aytés L, Gay Escoda C. Salivary gland application of botulinum toxin for the treatment of sialorrhea. *Med Oral Patol Oral Cir Bucal* 2007; **12**: 511–7.
56. Mancini F, Zangaglia R, Cristina S, et al. Double-blind, placebo-controlled study to evaluate the efficacy and safety of botulinum toxin type A in the treatment of drooling in parkinsonism. *Mov Disord* 2003; **18**: 685–8.
57. Postma AG, Heesters M, Van Laar T. Radiotherapy to the salivary glands as treatment of sialorrhea in patients with parkinsonism. *Mov Disord* 2007; **22**: 2430–5.
58. Stalpers LJ, Moser EC. Results of radiotherapy for drooling in amyotrophic lateral sclerosis. *Neurology* 2002; **58**: 1308.
59. Andersen PM, Gronberg H, Franzen L, Funegard U. External radiation of the parotid glands significantly reduces drooling in patients with motor neurone disease with bulbar paresis. *J Neurol Sci* 2001; **191**: 111–4.
60. Neppelberg E, Haugen DF, Thorsen L, Tysnes OB. Radiotherapy reduces sialorrhea in amyotrophic lateral sclerosis. *Eur J Neurol* 2007; **14**: 1373–7.
61. Burton MJ. The surgical management of drooling. *Dev Med Child Neurol* 1991; **33**: 1110–6.
62. Chilla R, Nicklatsch J, Arglebe C. Late sequelae of iatrogenic damage to chorda tympani nerve. *Acta Otolaryngol* 1982; **94**: 461–5.
63. Glynn F, Dwyer TP. Does the addition of sublingual gland excision to submandibular duct relocation give better overall results in drooling control? *Clin Otolaryngol* 2007; **32**: 103–7.
64. Martin TJ, Conley SF. Long-term efficacy of intra-oral surgery for sialorrhea. *Otolaryngol Head Neck Surg* 2007; **137**: 54–8.