Eur J Pediatr (2012) 171:409–414
DOI 10.1007/s00431-011-1570-y

REVIEW

Clinical practice

Swallowing problems in cerebral palsy

Corrie E. Erasmus · Karen van Hulst · Jan J. Rotteveel · Michel A. A. P. Willemsen · Peter H. Jongerius

Received: 18 May 2011 / Accepted: 5 September 2011 / Published online: 20 September 2011

Abstract Cerebral palsy (CP) is the most common physical disability in early childhood. The worldwide prevalence of CP is approximately 2–2.5 per 1,000 live births. It has been clinically defined as a group of motor, cognitive, and perceptive impairments secondary to a non-progressive defect or lesion of the developing brain. Children with CP can have swallowing problems with severe drooling as one of the consequences. Malnutrition and recurrent aspiration pneumonia can increase the risk of morbidity and mortality. Early attention should be given to dysphagia and excessive drooling and their substantial contribution to the burden of a child with CP and his/her family. This review displays the important functional and anatomical issues related to swallowing problems in children with CP based on relevant literature and expert opinion. Furthermore, based on our experience, we describe a plan for approach of investigation and treatment of swallowing problems in cerebral palsy.

Keywords Cerebral palsy · Children · Swallowing problems · Neuroanatomical control · Practical approach · Multidisciplinary swallowing/drooling team

C. E. Erasmus (✉) · J. J. Rotteveel · M. A. A. P. Willemsen
Donders Institute for Brain, Cognition and Behaviour,
Department of Paediatric Neurology,
Radboud University Nijmegen Medical Centre,
Nijmegen, The Netherlands
e-mail: c.erasmus@neuro.umcn.nl

K. van Hulst
Department of Rehabilitation,
Radboud University Nijmegen Medical Centre,
Nijmegen, The Netherlands

P. H. Jongerius
Department of Rehabilitation, Sint Maartenskliniek,
Nijmegen, The Netherlands

Introduction

Cerebral palsy (CP) is the most common physical disability in early childhood. The worldwide prevalence of CP is approximately 2–2.5 per 1,000 live births [27]. It has been clinically defined as a group of motor, cognitive, and perceptive impairments secondary to a non-progressive defect or lesion of the developing brain [4]. Epilepsy is a common problem in patients with CP [27]. Up to 80% of CP cases arise from antenatal factors; birth asphyxia contributes approximately 10% of CP cases [16]. Acquired cases in the postnatal period are usually related to central nervous system infection, trauma, strokes, and severe hypoxic events such as near-drowning. Genetic disorders and acquired insults follow a pattern of selective vulnerability during early brain development. For example, the neonatal neuropathological correlates of hypoxic–ischemic encephalopathy include specific and well-known patterns of brain injury [12, 51] (see Table 1) that interfere with the frontal/insular–basal ganglia–brainstem swallowing pathway [6, 10, 13, 15, 19–21, 23, 24, 26, 39, 52]. We propose that an understanding of paediatric dysphagia might be facilitated by a heightened awareness of the topography pertaining to the neuronal damage. This article focuses on the pathophysiology, clinical features, assessment, and management of swallowing problems in children with CP.

Members of the Multidisciplinary Outpatient Swallowing/Drooling Clinic at the Radboud University Nijmegen Medical Centre in the Netherlands continually review literature on dysphagia and drooling in neurologically affected patients. References for this review were obtained from personal reprint files, supplemented by PubMed and Scopus searches with varying search periods. The search terms "drooling," "sialorrhoea," "swallowing," "dysphagia," "cerebral palsy," "children," "brain regions," "fMRI," "MEG," and "EMG" were used. Only English-language articles, published from

Table 1 Regions with a predilection for hypoxic–ischemic neuronal injury to swallowing

Site of lesion [12, 51]	Swallowing elements[a]		
	Oral	Pharyngeal	GOR
Periventricular leucomalacia (preterm babies)	+	+/−	+/−
Cortical and subcortical injury in a watershed parasagittal distribution (term babies and prolonged partial hypoxic events)	+	+/−	+/−
Relatively selective injury to the putamen, thalamus, and peri-rolandic cerebral cortex, and often including injury to the brainstem (term babies and acute anoxic events)	+	++	+

GOR gastro-oesophageal reflux, +/− probably present, + very likely present, ++ evident

[a] Expert opinion

1970 up to 2011, were included. The final reference list was generated based on originality and relevance to the topics covered in the review.

Neural control of swallowing

Normal swallowing is a goal-directed sequential behaviour that requires the coordinated action and inhibition of the muscles located around the oropharynx and oesophagus. The swallowing process is controlled in a complex manner involving the brainstem as well as cortical and subcortical central pathways. In addition, it requires a higher level of fine-tuning between the central circuits and the enteric nervous systems (ENS) (see Fig. 1).

Efficient swallowing relies on sensory input from the oropharynx that triggers bilateral afferents in specific regions of the trigeminal sensory nuclei. Subsequently, the inputs reach the brainstem regions responsible for the patterned motor actions of swallowing. Sequential and rhythmic patterns of swallowing are formed and organized by a central pattern generator (CPG) located in the medulla oblongata. The CPG consists of two hemi-CPGs which, under physiological conditions, are tightly synchronized. The swallowing motor sequence is primarily generated in the ipsilateral hemi-CPG which transfers premotor neuron signals to the contralateral CPG [17]. The CPG itself is organized into two groups of neurons: the dorsal swallowing group (DSG) in and around the nucleus of tractus solitarius (NTS) and the ventral swallowing group (VSG) just cranial to the nucleus ambiguus. The DSG contains the generator neurons involved in the triggering, shaping, and timing of the sequential or rhythmic swallowing pattern. The DSG activates all VSG premotor neurons, which in turn distribute the swallowing drive to the various motor neuron pools involved in swallowing. The multifunctional pattern-generating circuits of the brainstem allow rapid

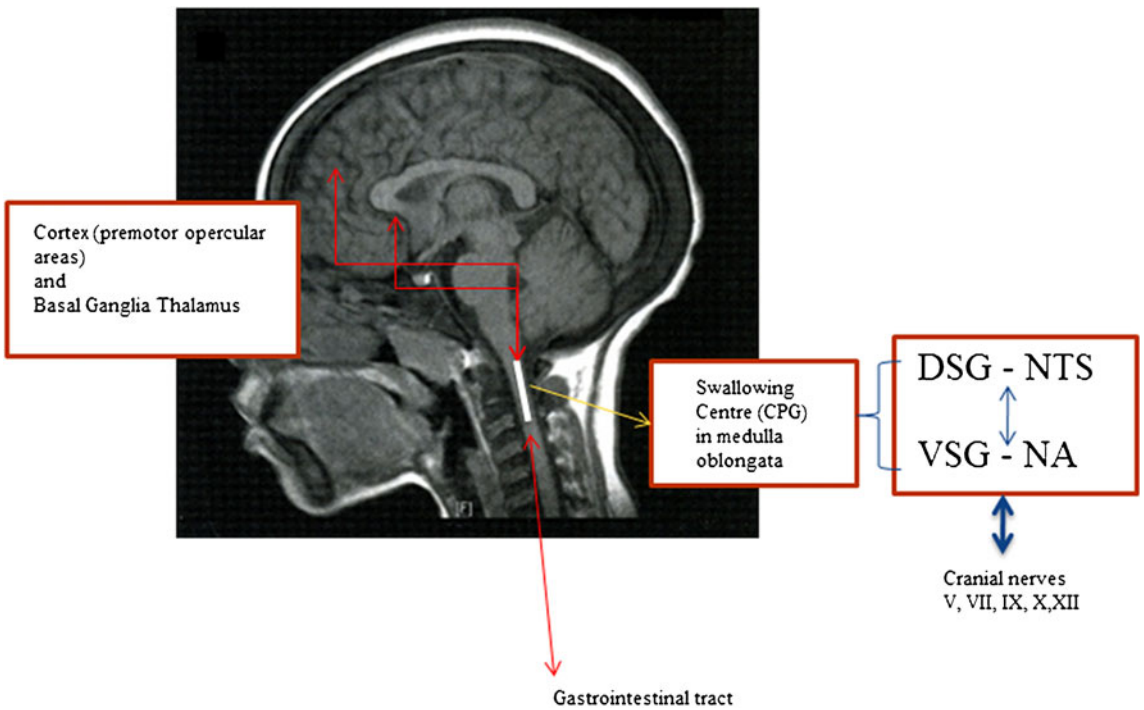

Fig. 1 Overview of the swallowing pathway. *DSG* dorsal swallowing group, *NTS* nucleus of tractus solitarius, *VSG* ventral swallowing group, *NA* nucleus ambiguus

modulation of orofacial behaviours (swallowing, respiration, chewing, coughing, and vomiting) [5].

Although our knowledge of the cortical regions involved in swallowing has grown substantially through functional magnetic resonance imaging studies, the exact central control mechanism for swallowing is still not fully understood. The involvement of many functionally and spatially different cortical sites suggests multilevel control for the swallowing pathways. It has been proposed that the control system consists of parallel loops which are able to coordinate and integrate the complex, sequentially based activation for swallowing [26]. The primary motor area and cingulate and insular cortices might all have essential roles in the coordination of the entire swallowing process [21, 24, 52]. Some investigators assume a functional dominance in swallowing [8] or a time-dependent shift of cortical activation from the left to the right sensorimotor cortex during voluntary swallowing [45].

In summary, voluntary and reflexive swallowing are controlled by widely distributed bilateral and multifocal cortical networks which apparently involve overlapping cortical regions. The primary sensory, motor, and cingulate cortices have a major role in these networks. The execution of the sensorimotor aspects related to swallowing relies on functionally connected pathways between (extra) pyramidal cortical motor planning regions, centres controlling the brainstem and cranial nerves, as well as lower motor neurons.

The brain–gut axis

Normal gastrointestinal tract (GI) function results from a balanced interaction between the enteric nervous system (ENS) and the central nervous system (CNS) which is called “the brain–gut axis”. Both neural and hormonal ENS communications have important integrative functions. A detailed discussion of the hormonal pathways is beyond the scope of this article. The ENS neural communications consist of the intrinsic afferent and motor neurons distributed along the gut wall (located in the mesenteric Auerbach and submucosal Meissner plexuses).

Afferent (vagal) sensory fibres terminate in the NTS of the hindbrain. The preganglionic motor innervations to the plexus arise from the dorsal motor nucleus of the vagus in the brainstem. The NTS and the vagal dorsal motor nucleus together comprise the dorsal vagal complex, important in the coordination of the muscular gut activity (by the vago-vagal reflex) [1].

The oesophagus consists of a proximal striated muscle portion (upper oesophageal sphincter, UOS) and a distal smooth muscle portion (lower oesophageal sphincter, LOS). At rest both sphincters are tonically contracted. Relaxation of the UOS (glossopharyngeal and vagal nerves) is initiated in the swallowing centre located in the medulla. Relaxation and contraction of the LOS (vagal and splanchic nerves) are initiated through local peristaltic activity of the oesophagus or distension of the gastric wall.

Thus far, the exact coupling of distinct interneurons (also called local circuit neurons or connector neurons) in the NTS is not known. Also, it is not totally clear which cortical regions are mainly involved in processing information to the GI tract. It has been suggested that the anterior insular cortex (called “visceral cortex”), the prefrontal and sensory/motor regions, the cingulate gyrus, as well as the limbic regions, all participate in the integration of neuronal information to the GI tract [18].

Swallowing problems in cerebral palsy

A recent epidemiological study among 1,357 children recorded by the Northern Ireland Cerebral Palsy Register between 1992 and 2009 showed a dysphagia prevalence of 43% in children with CP in any degree [29]. Results from speech pathology testing and video fluoroscopic swallowing studies in CP children demonstrate the relationship between typically affected brain regions and the associated characteristic patterns of dysfunctional swallowing (see Table 1). Usually, clinical features such as delayed initiation and segmented swallowing during attempted volitional movement might be determined by cortical neuronal networks, while dysfunctional pharyngeal components of swallowing (i.e. automatic components of deglutition, such as throat clearing, laryngeal closure tasks) suggest subcortical brain injury and/or basal ganglia necrosis [23]. In CP, dysphagia is often characterized by problems in both the volitional oral movements and the more reflexive pharyngeal phase of swallowing. Moreover, impaired ability to plan and coordinate swallowing with ventilation (e.g. greater propensity to swallow at abnormal times within the respiratory cycle, such as early inspiration after a thin liquid swallow and variable duration of the deglutition apnoea) are consistent with brainstem involvement [6]. A clinico-pathological correlation between differences in the breath–swallow pattern and the risk for aspiration is likely. Clinically, aspiration or episodic aspiration manifests as frequent coughing and occasional pneumonia. The overall incidence of pulmonary aspiration in CP due to oral motor dysfunctions is not known precisely. Admission to the hospital for presumed aspiration pneumonia in children with CP is common. An earlier study among 238 children with recurrent pneumonia showed that 48% had oropharyngeal incoordination with an aspiration syndrome whereas 50% of these children were diagnosed with CP [28]. Video fluoroscopic study of swallowing (VFSS) has

demonstrated pulmonary aspiration in 38% [32] to over 70% of the cases [25], and frequently, the aspiration occurred without coughing, referred to as "silent" [32, 38]. Repeated pulmonary aspiration leads to chronic coughing, sleep-disordered breathing, impaired clearance of airway secretions, colonization of the respiratory tract by pathogenic bacteria, and a high risk of progressive lung parenchymal damage. This process may be lethal [14, 22].

Besides dysphagia, chronic pulmonary aspiration may also occur as a result of the gastro-oesophageal reflux (GOR) [53]. The incidence of GOR has been estimated at approximately 50% [40] and might be explained by lesions in the neuronal-anatomic swallowing centre located in the medulla oblongata leading to dysfunction of the vago-vagal reflex. In addition, GOR in children with CP may also result from direct lesions in cortical areas that modulate brainstem activity [1, 3].

Constipation, a common dysmotility disorder of the gut in children with CP, is often overlooked. More than half of the children with severe generalized CP are constipated [50]. The high incidence of the dysmotility disorders emphasizes the defective integration and modulation of information in the brain–gut axis in CP [30, 34, 35, 41, 43, 49], for which some investigators had proposed the term "Dysphagia–GOR complex" with a central role for the vagal nerve [33, 34]. It is reasonable to assume that vagal disruption is responsible for defective feedback to the distinct cortical regions and to the brainstem, those features being associated with swallowing disorders, defective ventilation, as well as dysmotility problems. At this time, more studies are needed to investigate the clinical relevance of integrated breathing, GI and swallowing function on the health and nutritional outcomes of children with CP.

Drooling is caused by the swallowing disorder and occurs in 10–58% of children with CP [11, 44, 46]. From a clinical point of view, it makes sense to distinguish between "anterior" and "posterior" drooling. Anterior drooling is the unintentional loss of saliva from the mouth; it can impose a significant disability on children with CP, leading to

Table 2 Recommendations for evaluation of dysfunctional swallowing (expert opinion)

The paediatric neurologist, paediatrician, rehabilitation specialist, speech pathologist, ENT specialist, pedagogue, dentist, nurse practitioner, occupational therapist, physiotherapist, plastic surgeon may be involved in the multidisciplinary swallowing/drooling teams.
Assessments:
Medical and social–emotional history of the patient. Does the child suffer from intractable seizures?
Medication, benzodiazepines or neuroleptic-induced drooling?
Respiratory status (cough, wheezing, recurrent pneumonia) → Consider examination by the paediatric pulmonologist.
Comment: although common practice, the prophylactic use of antibiotics with suspected or proven aspiration is not recommended.
Presence of gastro-oesophageal reflux, which, if severe, can be associated with hyperstimulation of the salivary glands or indirect aspiration → Consider GOR treatment.
Nutrition and hydration. Safe feeding programme? Does the feeding result in normal growth? → Consider nasotube feeding, laxative.
Comment: see www.LifeExpectancy.org/articles/GrowthCharts.shtml and [9]
Neurological examination (consciousness, cranial nerves, general motor skills/posture, and tone)
Orofacial examination (nasal breathing, upper airways obstruction) → Consider examination by the ENT specialist.
Oral hygiene, occlusion, and dental examination
Assessment by a speech pathologist → objective: modify food bolus such as consistency, size and texture, positioning of the patient, and examining compensatory swallow manoeuvres:
Posture and head control; mouth closure, lip seal
Oral sensorimotor examination (tongue lateralisation, sensation, tone, strength, (pathological) reactions)
Oropharyngeal stage of swallowing during eating and drinking (swallow on demand, oral control, frequency/efficiency/safety)
Speech (dysarthria/dyspraxia) and communication skills
Management of secretions → Consider drooling treatment [31].
VFSS confirms silent aspiration and defines the pathophysiology of oropharyngeal swallow with various types of bolus
Comments: VFSS is the study of choice for complete evaluation of the feeding and swallowing process; aspiration is suspected in case of recurrent pneumonia and in children who are prone to gagging and coughing; silent aspirators do not exhibit overt symptoms of aspiration; aspiration risk is increased in non-ambulant children with CP (Gross Motor Functioning Classification System III or higher). See also [2]

Table 3 Drooling treatment (expert opinion)

Severe anterior drooling
• <3 years: oral motor therapy for training motor skills
• >4 years: botulinum toxin therapy (submandibular glands) [36]
If no response or developmental progress → Consider
1. Injection of the submandibular and parotid glands concurrently
2. Intense behavioural treatment [47]
3. Surgery: submandibular duct relocation [37]
Comment: Behavioural therapy is not given nor indicated in adults because no research is done in this field: no evidence exists that it is effective.
Posterior drooling
• <3 years: oral motor therapy, feeding advices for safer swallowing
• >4 years: botulinum toxin therapy (submandibular and/or parotid glands)
If no response → Consider surgery (duct ligation or gland removal, no submandibular duct relocation)
Comment: Consider anticholinergic medication for drooling control in case of contraindications to botulinum toxin therapy or surgery. Glycopyrrolate (glycopyrronium bromide) appears to be a more acceptable anticholinergic drug in the management of drooling in children. Randomized controlled trials with this drug in children with CP are warranted. Dosage: oral suspension 40–100 μg/kg per day with a maximum of 175 μg/kg per day, dosage given once daily [42]

psycho-social, physical, and educational consequences. The most severely affected children may be rejected by their peers and even by their caregivers. Excessive anterior drooling damages books, computer, and keyboards and as such threatens essential tools for education and communication in neurologically disabled patients. In addition to cosmetic effects, drooling can produce peri-oral infections and can impair dentition. In contrast to anterior drooling, the so-called "posterior drooling" refers to the spill of saliva over the tongue through the faucial isthmus [18]. In particular, the children with most severe pharyngeal dysphagia are at medical risk due to saliva aspiration to the lungs. As mentioned above, aspiration in a child with CP often occurs without obvious coughing or choking (i.e. silent), and therefore, chronic aspiration of saliva might not be diagnosed prior to development of significant lung injury.

In case of chemical irritation such as that caused by GOR, salivary secretion is increased to protect the oral, pharyngeal, and oesophageal mucosa mediated by the vago-vagal complex in the brainstem. Unfortunately, in children with oral motor dysfunction, this protective increased saliva volume may accumulate in the pharynx and/or oesophagus, leading to an increased risk for aspiration. It is still a matter of debate whether GOR can cause severe drooling and whether or not treatment of pathological GOR diminishes drooling in children with CP.

Assessment and management of swallowing problems in CP

The investigation and treatment of swallowing problems in children with CP are challenging. Individualized care plans should be formulated accounting for the degree of oral motor impairment, feeding ability, aspiration, epilepsy, and ambulation. Generally, swallowing is more problematic in non-ambulatory children with CP. Furthermore, marked disturbed consciousness such as drug overdose and seizures interfere with the voluntary swallowing act and is a common cause of aspiration. Some children develop aspiration in association with GOR. Some anti-epileptics, such as clobazam and clonazepam, and neuroleptic drugs will induce the drooling risk.

In short, there is a growing awareness among clinicians that at early stage, particular note should be given to the importance of dysphagia [7] and excessive drooling contributing substantially to the burden of a child with CP and his or her family [48]. Ideally, the management of patients with swallowing problems requires the coordinated expertise of a number of health care professionals. Regular reassessment is necessary to gauge the response to oral motor training, nutrition, and drooling interventions. Tables 2 and 3 summarize our recommendations for evaluation and treatment of dysfunctional swallowing and drooling in children with CP.

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Altaf MA, Sood MR (2008) The nervous system and gastrointestinal function. Dev Disabil Res Rev 14:87–95
2. Arvedson JC (2008) Assessment of pediatric dysphagia and feeding disorders: clinical and instrumental approaches. Dev Disabil Res Rev 14:118–127
3. Aziz Q, Andersson JL, Valind S, Sundin A, Hamdy S, Jones AK, Foster ER, Langstrom B, Thompson DG (1997) Identification of human brain loci processing esophageal sensation using positron emission tomography. Gastroenterology 113:50–59
4. Bax M, Goldstein M, Rosenbaum P, Leviton A, Paneth N, Dan B, Jacobsson B, Damiano D (2005) Proposed definition and classification of cerebral palsy, April 2005. Dev Med Child Neurol 47:571–576
5. Bianchi AL, Gestreau C (2009) The brainstem respiratory network: an overview of a half century of research. Respir Physiol Neurobiol 168:4–12
6. Casas MJ, Kenny DJ, McPherson KA (1994) Swallowing/ventilation interactions during oral swallow in normal children and children with cerebral palsy. Dysphagia 9:40–46
7. Cooper-Brown L, Copeland S, Dailey S, Downey D, Petersen MC, Stimson C, Van Dyke DC (2008) Feeding and swallowing dysfunction in genetic syndromes. Dev Disabil Res Rev 14:147–157
8. Daniels SK, Corey DM, Fraychinaud A, DePolo A, Foundas AL (2006) Swallowing lateralization: the effects of modified dual-task interference. Dysphagia 21:21–27
9. Day SM, Strauss DJ, Vachon PJ, Rosenbloom L, Shavelle RM, Wu YW (2007) Growth patterns in a population of children and adolescents with cerebral palsy. Dev Med Child Neurol 49:167–171
10. Dziewas R, Soros P, Ishii R, Chau W, Henningsen H, Ringelstein EB, Knecht S, Pantev C (2003) Neuroimaging evidence for cortical involvement in the preparation and in the act of swallowing. Neuroimage 20:135–144
11. Ekedahl C, Mansson I, Sandberg N (1974) Swallowing dysfunction in the brain-damaged with drooling. Acta Otolaryngol 78:141–149
12. Fatemi A, Wilson MA, Johnston MV (2009) Hypoxic-ischemic encephalopathy in the term infant. Clin Perinatol 36:835–858, vii
13. Hamdy S, Mikulis DJ, Crawley A, Xue S, Lau H, Henry S, Diamant NE (1999) Cortical activation during human volitional swallowing: an event-related fMRI study. Am J Physiol 277:G219–G225
14. Hemming K, Hutton JL, Pharoah PO (2006) Long-term survival for a cohort of adults with cerebral palsy. Dev Med Child Neurol 48:90–95
15. Humbert IA, Robbins J (2007) Normal swallowing and functional magnetic resonance imaging: a systematic review. Dysphagia 22:266–275
16. Jacobsson B, Hagberg G (2004) Antenatal risk factors for cerebral palsy. Best Pract Res Clin Obstet Gynaecol 18:425–436
17. Jean A (2001) Brain stem control of swallowing: neuronal network and cellular mechanisms. Physiol Rev 81:929–969

18. Jongerius PH, Joosten F, Hoogen FJ, Gabreels FJ, Rotteveel JJ (2003) The treatment of drooling by ultrasound-guided intraglandular injections of botulinum toxin type A into the salivary glands. Laryngoscope 113:107–111
19. Kern M, Birn R, Jaradeh S, Jesmanowicz A, Cox R, Hyde J, Shaker R (2001) Swallow-related cerebral cortical activity maps are not specific to deglutition. Am J Physiol Gastrointest Liver Physiol 280:G531–G538
20. Kern M, Chai K, Lawal A, Shaker R (2009) Effect of esophageal acid exposure on the cortical swallowing network in healthy human subjects. Am J Physiol Gastrointest Liver Physiol 297: G152–G158
21. Kern MK, Jaradeh S, Arndorfer RC, Shaker R (2001) Cerebral cortical representation of reflexive and volitional swallowing in humans. Am J Physiol Gastrointest Liver Physiol 280:G354–G360
22. Lefton-Greif MA, Carroll JL, Loughlin GM (2006) Long-term follow-up of oropharyngeal dysphagia in children without apparent risk factors. Pediatr Pulmonol 41:1040–1048
23. Malandraki GA, Sutton BP, Perlman AL, Karampinos DC, Conway C (2009) Neural activation of swallowing and swallowing-related tasks in healthy young adults: an attempt to separate the components of deglutition. Hum Brain Mapp 30:3209–3226
24. Martin RE, Goodyear BG, Gati JS, Menon RS (2001) Cerebral cortical representation of automatic and volitional swallowing in humans. J Neurophysiol 85:938–950
25. Mirrett PL, Riski JE, Glascott J, Johnson V (1994) Videofluoroscopic assessment of dysphagia in children with severe spastic cerebral palsy. Dysphagia 9:174–179
26. Mosier K, Bereznaya I (2001) Parallel cortical networks for volitional control of swallowing in humans. Exp Brain Res 140:280–289
27. Odding E, Roebroeck ME, Stam HJ (2006) The epidemiology of cerebral palsy: incidence, impairments and risk factors. Disabil Rehabil 28:183–191
28. Owayed AF, Campbell DM, Wang EE (2000) Underlying causes of recurrent pneumonia in children. Arch Pediatr Adolesc Med 154:190–194
29. Parkes J, Hill N, Platt MJ, Donnelly C (2010) Oromotor dysfunction and communication impairments in children with cerebral palsy: a register study. Dev Med Child Neurol 52:1113–1119
30. Ravelli AM, Milla PJ (1998) Vomiting and gastroesophageal motor activity in children with disorders of the central nervous system. J Pediatr Gastroenterol Nutr 26:56–63
31. Reddihough D, Erasmus CE, Johnson H, McKellar GM, Jongerius PH (2010) Botulinum toxin assessment, intervention and aftercare for paediatric and adult drooling: international consensus statement. Eur J Neurol 17(Suppl 2):109–121
32. Rogers B, Arvedson J, Buck G, Smart P, Msall M (1994) Characteristics of dysphagia in children with cerebral palsy. Dysphagia 9:69–73
33. Saito Y (2009) Reflections on the brainstem dysfunction in neurologically disabled children. Brain Dev 31:529–536
34. Saito Y, Kawashima Y, Kondo A, Chikumaru Y, Matsui A, Nagata I, Ohno K (2006) Dysphagia-gastroesophageal reflux complex: complications due to dysfunction of solitary tract nucleus-mediated vago-vagal reflex. Neuropediatrics 37:115–120
35. Sanger GJ, Lee K (2008) Hormones of the gut-brain axis as targets for the treatment of upper gastrointestinal disorders. Nat Rev Drug Discov 7:241–254
36. Scheffer AR, Erasmus C, Van Hulst K, Van Limbeek J, Jongerius PH, van den Hoogen FJ (2010) Efficacy and duration of botulinum toxin treatment for drooling in 131 children. Arch Otolaryngol Head Neck Surg 136:873–877
37. Scheffer AR, Erasmus C, Van Hulst K, Van Limbeek J, Rotteveel JJ, Jongerius PH, van den Hoogen FJ (2010) Botulinum toxin versus submandibular duct relocation for severe drooling. Dev Med Child Neurol 52:1038–1042
38. Smith HC (2008) Cough and aspiration of food and liquids due to oral pharyngeal dysphagia. Lung 186(Suppl 1):S35–S40
39. Soros P, Inamoto Y, Martin RE (2009) Functional brain imaging of swallowing: an activation likelihood estimation meta-analysis. Hum Brain Mapp 30:2426–2439
40. Spiroglou K, Xinias I, Karatzas N, Karatza E, Arsos G, Panteliadis C (2004) Gastric emptying in children with cerebral palsy and gastroesophageal reflux. Pediatr Neurol 31:177–182
41. Staiano A, Cucchiara S, Del GE, Andreotti MR, Minella R (1991) Disorders of oesophageal motility in children with psychomotor retardation and gastro-oesophageal reflux. Eur J Pediatr 150:638–641
42. Stern LM (1997) Preliminary study of glycoyrrolate in the management of drooling. J Paediatr Child Health 33:52–54
43. Sullivan PB (2008) Gastrointestinal disorders in children with neurodevelopmental disabilities. Dev Disabil Res Rev 14:128–136
44. Tahmassebi JF, Curzon ME (2003) The cause of drooling in children with cerebral palsy—hypersalivation or swallowing defect? Int J Paediatr Dent 13:106–111
45. Teismann IK, Dziewas R, Steinstraeter O, Pantev C (2009) Time-dependent hemispheric shift of the cortical control of volitional swallowing. Hum Brain Mapp 30:92–100
46. Van de Heyning PH, Marquet JF, Creten W (1980) Drooling in children with cerebral palsy. Acta Otorhinolaryngol Belg 34:691–705
47. van der Burg JJ, Didden R, Engbers N, Jongerius PH, Rotteveel JJ (2009) Self-management treatment of drooling: a case series. J Behav Ther Exp Psychiatry 40:106–119
48. van der Burg JJ, Jongerius P, van Limbeek J, van Hulst K, Rotteveel J (2006) Drooling in children with cerebral palsy: a qualitative method to evaluate parental perceptions of its impact on daily life, social interaction, and self-esteem. Int J Rehabil Res 29:179–182
49. Vane DW, Shiffler M, Grosfeld JL, Hall P, Angelides A, Weber TR, Fitzgerald JF (1982) Reduced lower esophageal sphincter (LES) pressure after acute and chronic brain injury. J Pediatr Surg 17:960–964
50. Veugelers R, Benninga MA, Calis EA, Willemsen SP, Evenhuis H, Tibboel D, Penning C (2010) Prevalence and clinical presentation of constipation in children with severe generalized cerebral palsy. Dev Med Child Neurol 52:e216–e221
51. Volpe JJ (2006) Brain injury in premature infants: a complex amalgam of destructive and developmental disturbances. Lancet Neurol 8:110–124
52. Watanabe Y, Abe S, Ishikawa T, Yamada Y, Yamane GY (2004) Cortical regulation during the early stage of initiation of voluntary swallowing in humans. Dysphagia 19:100–108
53. Weir K, McMahon S, Barry L, Ware R, Masters IB, Chang AB (2007) Oropharyngeal aspiration and pneumonia in children. Pediatr Pulmonol 42:1024–1031