Neurogenic Dysphagia

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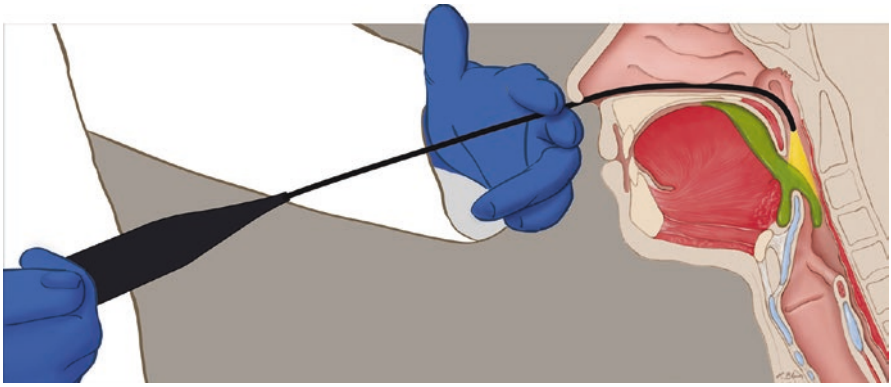
"*Over the next 20 years, the face of dysphagia evaluation and treatment may change drastically. […] It is an exciting and stimulating area in which to practice. Our patients benefit from our efforts, and we derive pleasure from witnessing their progress. The rewards are great; after all, to many people, fewer pleasures are more satisfying than a glass of wine and a good meal.*"

(Langmore 2001, p. 249)

"*The best answer to the question of who should perform FEES is whoever understands oropharyngeal dysphagia best. […] FEES now has a secure place in the armamentarium of tools used to evaluate and manage this disorder. As the scoring system becomes better validated and technology allows more quantification of findings, its use will become ever more valuable.*"

(Langmore 2017, pp. 34–35)

Preface



Neurological disorders, such as stroke, dementia, Parkinson's disease, or neuromuscular diseases, are the most common causes of swallowing disorders. Neurogenic dysphagia has significant consequences for those affected as it impacts on the quality of life, and is associated with malnutrition, aspiration pneumonia, and even death. According to recent data, up to 50% of all neurological patients suffer from a swallowing disorder. Today, neurogenic dysphagia plays an important role in the daily practice of acute-care hospitals, rehabilitation clinics, nursing homes, and outpatient care. With the aging of the population and the greater emergence of neurodegenerative diseases, neurogenic dysphagia will take on an even more important role in the future. In the next few years, the demographic changes in our population will lead not only to a steady increase in neurological diseases but thereby also to a corresponding increase in the frequency of neurogenic dysphagia.

Against this background, a rapid development of different diagnostic modalities and new treatment options for neurogenic dysphagia has taken place. Over the years, the use of flexible endoscopic evaluation of swallowing (FEES) has grown incrementally. When Susan Langmore first published on this procedure in 1988, there were only a handful of clinicians using this tool to assess swallowing. Today, FEES is a well-established procedure done throughout the world. It has been embraced by clinical specialists who evaluate dysphagia, including speech and language pathologists (SLPs), gastroenterologists, otolaryngologists, phoniatrists,

geriatricians, rehabilitation physicians, physiatrists, and neurologists. There have been very few textbooks written about FEES in general (Langmore 2001; Aviv and Murray 2005). The content of the present book is focused on neurological disorders but will be relevant to practitioners who see other patients as well. The main approach presented here is to customize the exam in order to uncover the nature of the disorder and to address the specific purpose of the exam. This means not to adhere to the "one size fits all" principle at all and this may be a refreshing change from some overly rigid protocols in use.

The use of FEES to assess neurogenic dysphagia has become firmly established in neurology and has significantly enhanced the clinical understanding of the complex patterns of neurogenic dysphagia by enabling a direct visualization of the act of swallowing. Today, specific endoscopic protocols allow standardized examinations of special subtypes of neurogenic dysphagia. In the future, swallowing endoscopy is likely to become just as common as the instrumental procedures routinely used by neurologists, such as EEG, EMG, and ultrasound. In order to take account of the increased knowledge in the field and to enable a broad spectrum of training in swallowing endoscopy, a FEES training curriculum for neurogenic dysphagia was first developed by the German Neurological Society, the German Stroke Society and the German Society for Geriatrics. A similar FEES training program was recently also established by the European Society for Swallowing Disorders (ESSD). This book covers all content requirements of these FEES training curriculums.

With the aid of FEES and other modern instrumental methods of investigation, it is becoming increasingly possible to classify the various forms of dysphagia both phenomenologically and pathophysiologically. These findings are facilitating the development of completely novel therapeutic approaches in the future. Due to the specific and complex combination of different symptoms that result from underlying neurological disorders, it is becoming clear that neurogenic dysphagia is not a simple symptom but rather a multi-etiological syndrome. Analogous to aphasia syndromes, it is now possible to speak of neurogenic dysphagia syndromes, although etiology is considerably more heterogeneous than in the field of aphasias. In this book, modern neurological systematics are consistently applied to neurogenic dysphagia syndromes, whose clinical relevance is emphasized.

The modern diagnosis of and therapy for neurogenic dysphagia are thus a highly differentiated, expansive, and extremely exciting field of medicine in which different specialist groups work together in an interdisciplinary manner. The aim of this book is to impart practical knowledge on the current state of clinical research. Wherever possible, practical guidance is provided for diagnostics and therapy in the daily care of patients with neurogenic dysphagia. The supplemental electronic material makes use of video samples to demonstrate various patterns of neurogenic dysphagia that have direct practical relevance. In addition to already available diagnostic and therapeutic procedures, the various chapters of the book also reveal the areas in which there is a particularly urgent need for additional research. The book is also intended to provide easy access and fast information to colleagues who have no special knowledge in this field. We would also be very pleased to see the work used as a reference source that can prove helpful to its readers.

In medical textbooks, drawings provided by illustrators with special medical expertise are very rare. We are therefore proud that Heike Blum and Esther Gollan have created numerous informative illustrations for this book that considerably enhance readers' understanding of the highly complex process of swallowing and its coordination. These images are not mere schematic drawings but rather extremely precise and detailed representations that are intended to aid in the understanding of even more complex relationships by visually depicting the described details.

Despite our careful attention to detail, this book will naturally contain room for improvement. It is therefore our great wish that readers inform us of any mistakes, send suggestions and comments, and contribute constructively to the further optimization of a future edition.

We are most grateful to Andrea Ridolfi from Springer for his exceptional collaboration and patient support during the completion of this book. We would like to thank Ryan DeLaney for expert help with the English translation. We also wish to thank our families for their continued support and understanding of the requirements of our work.

Finally, we are impressed by the enthusiasm and motivation with which neurogenic dysphagia is today being examined and treated by multidisciplinary teams consisting of SLPs and physicians of various disciplines. These dysphagia teams have recently been described as ideal "think tanks" because they can examine issues from a variety of perspectives and thereby generate creative ideas and solutions together. In this sense, we hope that the readers of this book will profit from its lessons.

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Contents

Abbreviations

AD	Alzheimer's disease
AFM	Airflow method
ALS	Amyotrophic lateral sclerosis
AWMF	Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften e.V. [Association of Scientific Medical Societies in Germany]
ÄZQ	Ärztliche Zentralstelle für Qualitätssicherung [German Agency for Quality in Medicine]
BMD	Becker muscular dystrophy
BMI	Body mass index
BODS	Bogenhausener Dysphagia Score
BRACS	Boston Residue and Clearance Scale
CBD	Corticobasal degeneration
CBS	Corticobasal syndrome
CEA	Carotid endarterectomy
CIM	Critical illness myopathy
CIP	Critical illness polyneuropathy
CNS	Central nervous system
COPD	Chronic obstructive pulmonary disease
CPEO	Chronic progressive external ophthalmoplegia
CPG	Central pattern generator
CRT	Cough Reflex Test
CSS	Collet–Sicard syndrome
DES	Diffuse esophageal spasm
DGEM	Deutsche Gesellschaft für Ernährungsmedizin [German Society for Nutritional Medicine]
DGN	Deutsche Gesellschaft für Neurologie [German Neurological Society]
DISH	Diffuse idiopathic skeletal hyperostosis
DM	Dermatomyositis
DMD	Duchenne muscular dystrophy
DSG	Dorsal swallowing group
DSI	Digital spot imaging
DTI	Diffusion tensor imaging

EBM	Evidence-based medicine
EDSS	Expanded disability status scale
EEG	Electroencephalography
EMG	Electromyography
EMGBF	EMG biofeedback
EMST	Expiratory muscle strength training
EPI	Echo-planar imaging
ESPEN	European Society for Clinical Nutrition and Metabolism
F.O.T.T.®	Facial-Oral Tract Therapy
FDT	Functional dysphagia therapy
FEDSS	Flexible endoscopic dysphagia severity scale for acute stroke patients;
FEES	Flexible endoscopic evaluation of swallowing
FEESST	Flexible endoscopic evaluation of swallowing with sensory testing
fMRI	Functional magnetic resonance imaging
FSHD	Facioscapulohumeral muscular dystrophy
FST	Fatigable swallow test
FTD	Frontotemporal dementia
FTLD	Frontotemporal lobar degeneration
GBS	Guillain–Barré syndrome
GCS	Glasgow Coma Scale
GUSS	Gugging Swallowing Screen
HBE	Harris–Benedict equation
HRM	High-resolution manometry
HSP	Hereditary spastic paraplegia
IBM	Inclusion body myositis
ICU	Intensive care unit
IDDSI	International Dysphagia Diet Standardisation Initiative
KD	Kennedy's disease
KSS	Kearns–Sayre syndrome
LBD	Lewy body dementia
LEMS	Lambert–Eaton myasthenic syndrome
LGMD	Limb-girdle muscular dystrophy
LSVT®	Lee Silverman Voice Treatment
LTD	Long-term depression
LTP	Long-term potentiation
LTV	Long-term ventilation
MBS	Modified barium swallow
MC	Myasthenic crisis
MDS	Münster Dysphagia Score
MDT	Munich Dysphagia Test
MEBDT	Modified Evan's blue dye test
MEG	Magnetoencephalography
MEP	Motor-evoked potential
MFS	Miller Fisher syndrome

MG	Myasthenia gravis
MJD	Machado–Joseph disease
MM	Mendelsohn maneuver
MNA	Mini nutritional assessment
MNDs	Motor neuron diseases
MS	Multiple sclerosis
MSA	Multiple system atrophy
mSv	MilliSievert
MUST	Malnutrition Universal Screening Tool
NA	Nucleus ambiguus
NAS	Neuroacanthocytosis syndromes
NBIA	Neurodegeneration with brain iron accumulation
NMES	Neuromuscular electrical stimulation
NMO	Neuromyelitis optica
NMT	Neuromyotonia
NPC	Niemann–Pick disease, type C
NPD	Niemann–Pick disease
NRS	Nutritional risk screening
OFC	Orbitofrontal cortex
OM	Overlap myositis
OPDM	Oculopharyngodistal myopathy
OPMD	Oculopharyngeal muscular dystrophy
OPP	Oral preparatory phase
ORL	Otorhinolaryngology
OSAS	Obstructive sleep apnea syndrome
OTT	Oral transit time
PAS	Penetration–Aspiration Scale
PD	Parkinson's disease
PEG	Percutaneous endoscopic gastrostomy
PES	Pharyngeal Electrical Stimulation
PET	Positron-emission tomography
PKAN	Pantothenate kinase-associated neurodegeneration
PM	Polymyositis
PNS	Peripheral nervous system
PPA	Primary progressive aphasia
PSP	Progressive supranuclear palsy
PTT	Pharyngeal transit time
RASSS	Rapid Aspiration Screening for Suspected Stroke
RLN	Recurrent laryngeal nerve
rTMS	Repetitive transcranial magnetic stimulation
SBMA	Spinal and bulbar muscular atrophy
SCA	Spinocerebellar ataxia
SCLC	Small-cell lung cancer
SD	Semantic dementia
SGSM	Supraglottic swallow maneuver

SjS	Sjögren syndrome
SLE	Systemic lupus erythematosus
SMA	Supplementary motor areas
SN	Solitary nucleus
SPT	Swallowing Provocation Test
SSA	Standardized Swallowing Assessment
SSGSM	Super supraglottic swallow maneuver
SU	Stroke unit
TBI	Traumatic brain injury
tDCS	Transcranial direct-current stimulation
TMS	Transcranial magnetic stimulation
TOR-BSST©	Toronto Bedside Swallowing Screening Test
UES	Upper esophageal sphincter
UPDRS	Unified Parkinson's Disease Rating Scale
VAST	Video-assisted swallowing therapy
VD	Vascular dementia
VFS	Videofluoroscopy
VFSS	Videofluoroscopic swallowing study
VLM	Ventrolateral medulla oblongata
VPC	Velopharyngeal closure
VSG	Ventral swallowing group
VVST	Volume-Viscosity Swallow Test
WD	Wilson's disease
YPRSRS	Yale Pharyngeal Residue Severity Rating Scale

List of Videos

Electronic Supplementary Material is available in the online version of the related chapters on https://doi.org/10.1007/978-3-030-42140-3_3; https://doi.org/10.1007/978-3-030-42140-3_4; https://doi.org/10.1007/978-3-030-42140-3_6

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Neuroanatomy and Physiology of Deglutition

1

Contents

1.1 The Unimpaired Swallow

Swallowing is a vital human motor activity. The act of swallowing transports saliva and food from the oral cavity into the stomach while simultaneously protecting the respiratory tract. Although swallowing can be initiated voluntarily, most swallows occur without conscious input. While awake, a healthy person swallows about once a minute between meals depending on saliva production, whereas during deep sleep, salivation and swallowing are almost completely suspended. The rate of swallowing increases to up to three times per minute when a person sucks on a piece of candy, and a small meal requires about 30 swallows on average (Martin et al. 1994). During a single day, a healthy adult swallows approximately 1000 times (Dodds 1989).

Swallowing can be divided into four phases: the oral preparatory phase, the oral phase, the pharyngeal phase, and the esophageal phase. These phases are not strictly separated; rather, they smoothly merge into each other. Hiimae and Palmer have described the oral phase of swallowing in detailed reports (see Heiimae and Palmer 1999).

1. In the *oral preparatory phase*, food is chewed and mixed with saliva. The soft palate (velum palatinum) is lowered and raised in synchrony with jaw movement

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during mastication. During this process, the airway remains open and the pharynx and larynx remain in the resting position. As a food bolus is processed by the tongue and mixed with saliva, the part that has been titrated is moved to the back of the mouth while new sections are processed in the more anterior oral cavity (stage 1 and 2 transport). Over time, the titrated portion of the bolus falls into the oropharynx, gradually filling the valleculae. Toward the end of this phase, as the oral processing and transport stages end, the oral propulsive stage is seen where the tongue propels the entire bolus, both the part in the oropharynx and the part still in the oral cavity into the pharynx to be swallowed. Synchronously with tongue propulsion, the pharyngeal phase of the swallow begins. Therefore, spillage of food into the hypopharynx prior to swallow initiation is normal, not pathologic.

Liquids have a very different pattern of oral preparation. The tongue contains the liquid by cupping it within a depression in the anterior one- or two-thirds of the tongue (Dodds 1989). There is no spillage of portions of the liquid bolus into the oropharynx; instead, the entire liquid bolus is swallowed together. However, the head of the bolus may fall as low as the piriforms before the pharyngeal phase begins. The duration of this spillage is usually less than 2 s and sometimes shorter.

2. In the oral phase, whether for food or liquid, the tongue elevates and rolls posteriorly in a peristaltic motion, making sequential contact with the hard and soft palate and thereby propelling the bolus into the pharynx (Dodds 1989). The lips remain closed and the buccinator muscles remain contracted, thereby enabling slight negative pressure in the oral cavity to facilitate the bolus transport. The oral propulsive phase is executed voluntarily, (Fig. 1.1) and lasts less than 1 s.
3. Once the pharyngeal phase begins, it is invariant in sequence (Kendall et al. 2003). At the beginning of the reflexive movement pattern, the velum rises to close the nasopharynx (velopharyngeal closure) and prevent nasal regurgitation of bolus material. The swallow is usually initiated during the expiratory phase of respiration. During the swallow, breathing is interrupted briefly. A rapid, piston-like backward movement of the base of the tongue presses the bolus into the hypopharynx. At the same time, the airway begins its closure, first at the level of the arytenoids, followed immediately by the hyoid and larynx rising and the epiglottis retroflexing. The vocal folds close last, about 0.6 s after the arytenoids begin their medial and anterior movement to contact the petiole of the epiglottis. The pharyngeal constrictors are the last muscle groups to contract. They squeeze medially in a sequential fashion to close the airspace. At the same time that the pharyngeal constrictors begin contracting, the upper esophageal sphincter relaxes and opens. The rising of the larynx and the expansion of the opened esophageal entrance result in negative pressure, which pulls the bolus downward. The bolus slides over the epiglottis and the piriform sinus and is transported downstream by sequential contractions of the pharyngeal constrictors into the upper esophageal sphincter and the esophagus (Fig. 1.1). The pharyngeal phase lasts about 0.7 s (Kendall et al. 2003; VanDaele et al. 2005).

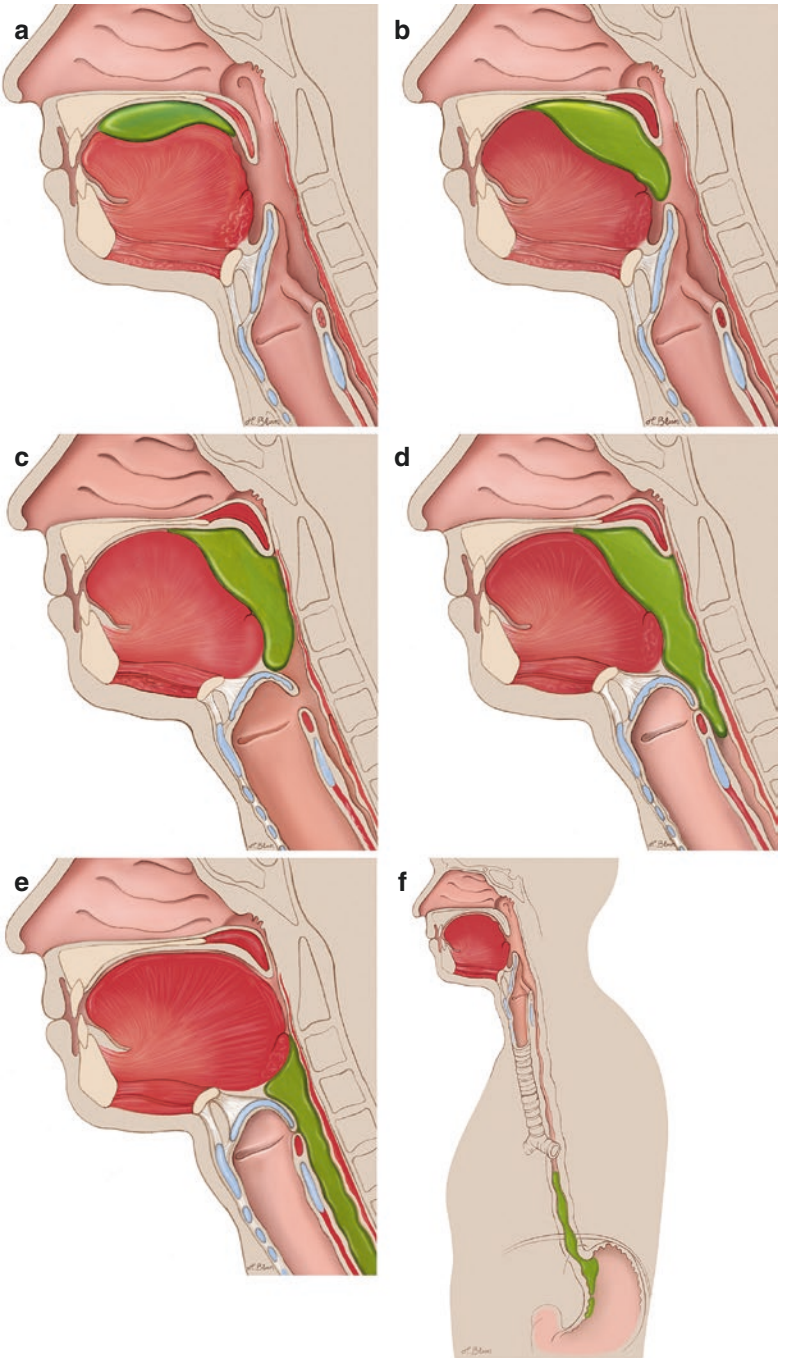

Fig. 1.1 Phases of swallowing (© 2012–2017 Heike Blum, University Hospital Muenster). (**a**) Oral preparatory phase; (**b**) oral phase; (**c**) triggering of swallow reflex at beginning of pharyngeal phase; (**d**) pharyngeal phase, (**e**) end of pharyngeal phase shortly before closure of upper esophageal sphincter; (**f**) esophageal phase. Reproduced with permission

4. The passage of the pharyngeal peristalsis contraction wave through the upper esophageal sphincter terminates its relaxation and marks the transition to the esophageal phase of swallowing. During this phase, the hyoid, the larynx, and the epiglottis return to their resting positions. The velum relaxes, thereby reopening the nasopharynx and allowing breathing to continue. A primary peristaltic wave of the esophageal musculature propagates the bolus into the stomach. Secondary peristaltic cleaning waves are induced by local extensor stimuli. The esophageal phase can take up to 10 s, depending on bolus consistency and size (Fig. 1.1; Dodds et al. 1990; Bartolome and Schröter-Morasch 2013).

Overall, the seemingly straightforward yet highly complex process of swallowing requires bilateral, coordinated activation and inhibition of more than 25 pairs of muscles in the oral cavity, pharynx, larynx, and esophagus (Fig. 1.2).

The act of swallowing involves five cranial nerves as well as the ansa cervicalis (C1–C3). The coordination of the masticatory muscles is mediated by the 3rd branch (V_3) of the trigeminal nerve (V). The orofacial musculature—which is important for oral closure—is innervated by the facial nerve (VII). The hypoglossal nerve (XII) supplies the intrinsic tongue muscles, whereas accompanying spinal nerves C1–C3 innervate the extrinsic muscles of the tongue. The muscles of the soft palate and the pharyngeal isthmus as well as the constrictor and levator muscles of the pharynx are activated by the glossopharyngeal (IX) and vagal (X) nerves (Fig. 1.3). The vagus nerve (X) innervates the intrinsic laryngeal muscles and the esophagus. The trigeminal nerve (V_3), the facial nerve (VII), and the ansa cervicalis innervate the supra- and infrahyoid muscles, which coordinate the movement of the hyoid and larynx (Donner 1985; Dodds et al. 1990).

A timely opening and closing of the upper esophageal sphincter (UES) is particularly important to the unimpaired swallow. This central element of the pharyngeal phase is executed by the fine-tuned contraction and relaxation of the opening and closing muscle groups of the UES. The *UES opening muscles* are differentiated into *anterior and posterior muscle groups* (Lang 2012). The *anterior muscle group* includes the *upper hyoid muscles* (geniohyoid muscle, mylohyoid muscle, stylohyoid muscle, hypoglossal muscle, anterior digastric muscle). Each of these muscles originates above the hyoid and attaches to the top of it. When they contract, they pull the hyolaryngeal complex forward and upward. The *lower hyoid muscles* include the thyrohyoid muscle, the sternohyoid muscle, the sternothyroid muscle, and the omohyoid muscle. These muscles attach to the underside of the hyoid and pull the hyolaryngeal complex forward and downward. The *posterior muscle group* is formed by the stylopharyngeus muscle, the palatopharyngeus muscle, and the pterygopharyngeus muscle. These muscles attach to the back of the pharynx and pull it backward and upward. The *UES closing muscles* consist of the cricopharyngeal muscle, caudal portions of the inferior pharyngeal muscle, and the cervical esophageal musculature. The cricopharyngeal muscle attaches to the cricoid cartilage and forms a muscular band at the junction of the pharynx and the

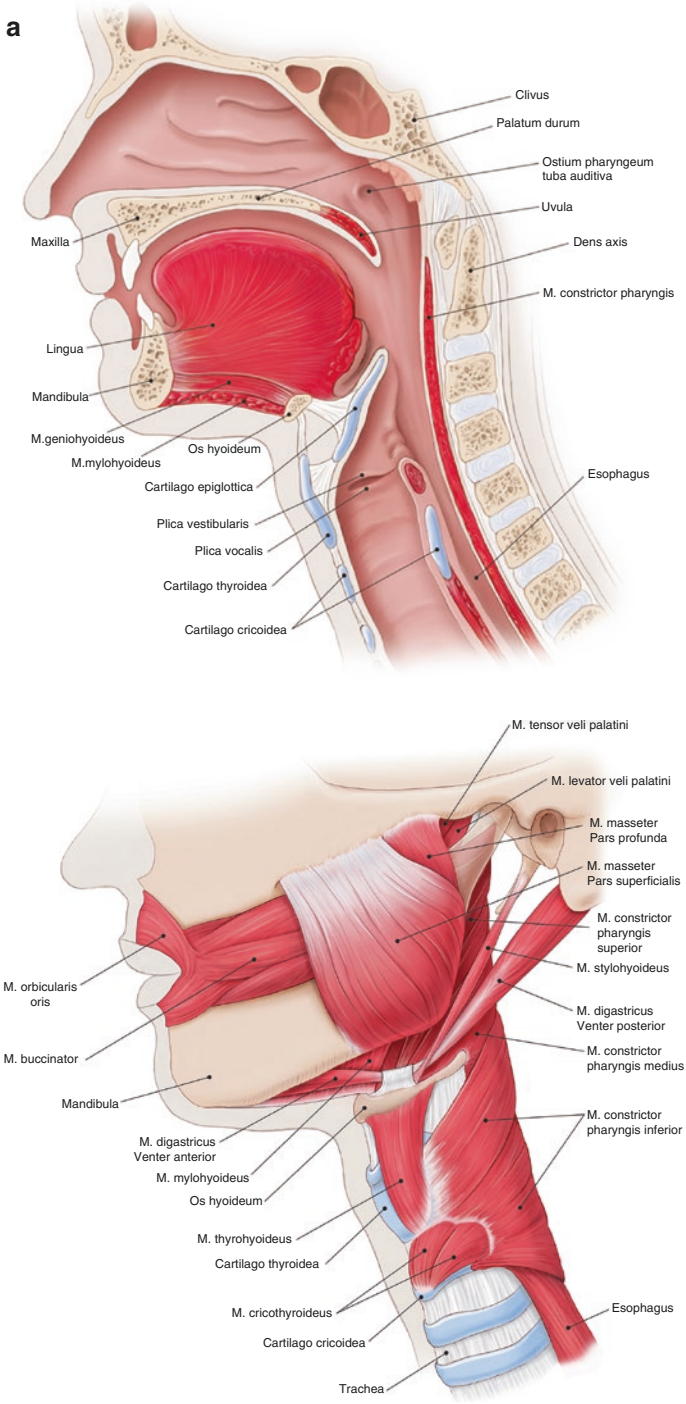

Fig. 1.2 (**a**, **b**) Anatomy of swallowing muscles (© 2017 Esther Gollan). Reproduced with permission

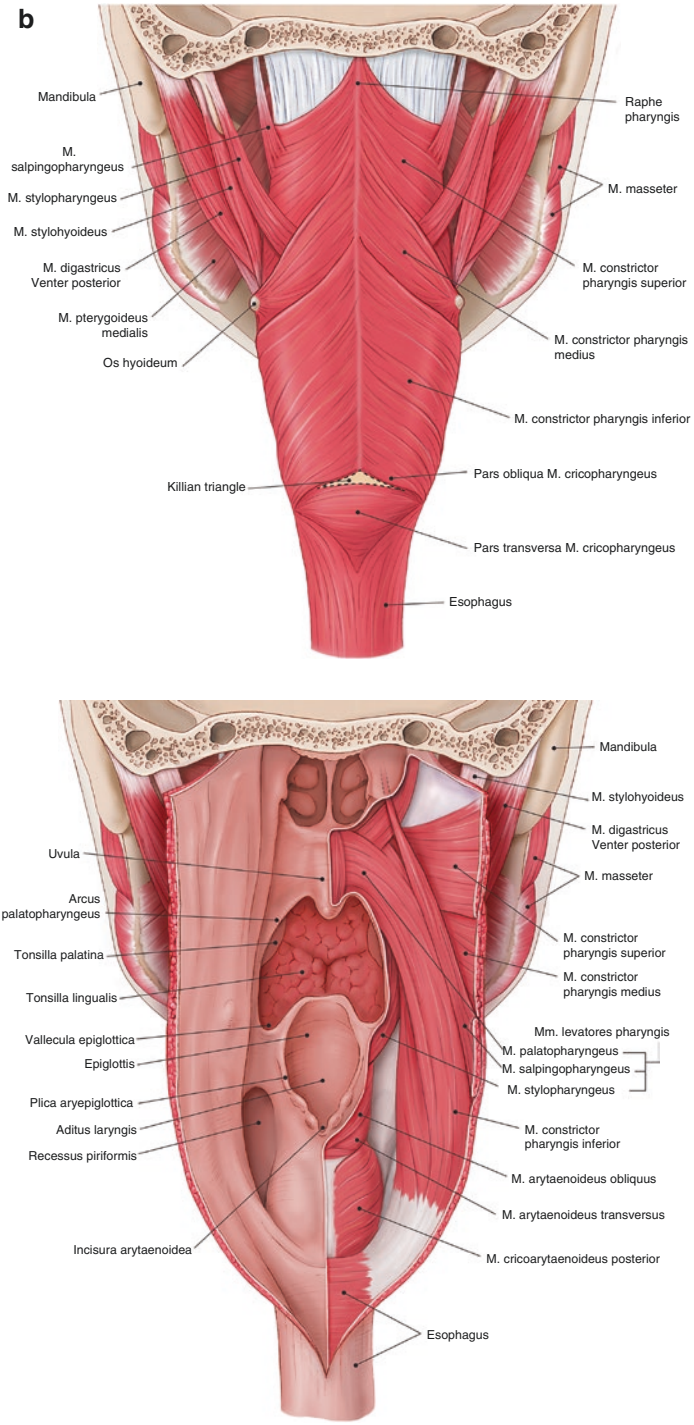

Fig. 1.2 (continued)

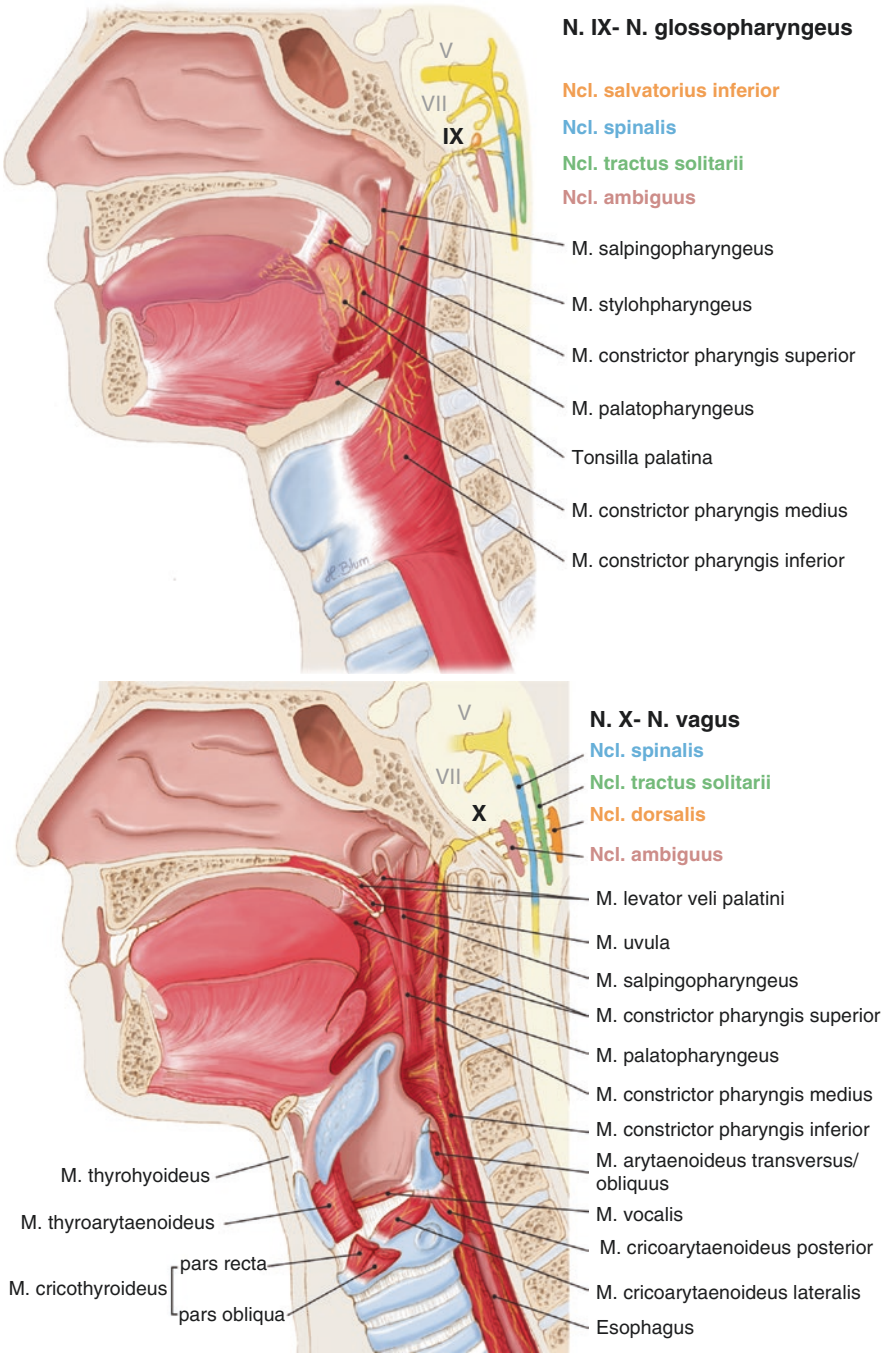

Fig. 1.3 Pathway of glossopharyngeal and vagal nerves (© 2017 Heike Blum, University Hospital Muenster). Reproduced with permission

esophagus. Its pars obliqua ends in a median raphe of the inferior pharyngeal constrictor muscle, while its pars transversa does not form a median raphe. The Kilian's triangle is formed by these two parts of the cricopharyngeal muscle and constitutes a weak spot and predilection site of the Zenker's diverticulum. The striated musculature of the cervical esophagus is attached below the pars transversa (Lang 2012).

The act of swallowing is subdivided into the oral preparatory, oral, pharyngeal, and esophageal phases. Cranial nerves V, VII, IX, X, and XII as well as more than 25 pairs of muscles are involved in the control and execution of swallowing. The upper esophageal sphincter—which consists of opening and closing muscles—has special clinical significance for the unimpaired swallow.

1.2 The Impaired Swallow

The impaired swallow is referred to medically as dysphagia. The term "dysphagia" is derived from the ancient Greek prefix "dys," meaning "disturbed," and the verb "phagein," meaning "to eat." The literal meaning of the term is thus "eating disturbance." Although the epidemiology of dysphagia in the general population has not been sufficiently studied, it has been estimated that more than 5% of the general population suffer from a swallowing disorder. Oropharyngeal dysphagia—which is more common than esophageal dysphagia—occurs more frequently among the general population and is about as common as the most widespread metabolic disorder, diabetes mellitus. Oropharyngeal dysphagia affects 13% of the total population over the age of 65. The highest prevalence of oropharyngeal dysphagia is among old patients with neurological diseases. The prevalence of oropharyngeal dysphagia increases with increasing age. It remains at 16% among individuals between 70 and 79 who live independently and increases to 33% for individuals 80 and older. As a result, the prevalence of oropharyngeal dysphagia has been increasing worldwide in so-called aging societies (Kuhlemeier 1994; Clavé and Shaker 2015; Wirth et al. 2016; Dziewas et al. 2017).

Neurological disorders are the most common cause of dysphagia. Swallowing impairments caused by disorders affecting the central swallowing network or downstream peripheral nerves and muscles are referred to as neurogenic dysphagia. The term "neurogenic" in this context thus includes both all types of neurological diseases and dysphagia caused by myopathies. No differentiation is made between "neurogenic" and "myogenic," a differentiation that is otherwise fundamental in neurology. The term "myogenic dysphagia" is therefore not common in German- or English-speaking countries. It is necessary to differentiate swallowing disorders caused by diseases in the field of otorhinolaryngology (e.g., tumors or inflammation of the pharynx or larynx—usually called structural dysphagia), internal medicine and gastroenterology (e.g., Zenker's diverticulum or reflux disease), and psychiatry (e.g., globus

pharyngis) from neurogenic dysphagia. It is also important to note a special element of this classification: If an otorhinolaryngological disease causes a swallowing impairment due to lesions of cranial nerves that are relevant to the act of swallowing, the dysphagia could also be formally classified as "neurogenic" because the affection of the (peripheral) nervous system would then constitute the underlying pathophysiology. Usually, however, swallowing disorders caused in this manner are allocated to the field of otorhinolaryngology since their diagnosis and therapy essentially fall into this field of specialization.

It is estimated that about 50% of all neurological patients suffer from neurogenic dysphagia (Clavé and Shaker 2015). As early as 2001, Doggett et al. (2001) calculated that approximately 300,000–600,000 people develop neurogenic dysphagia every year in the USA. The most common form of neurogenic dysphagia is stroke-related dysphagia. Neurogenic dysphagia can lead to disorders in one, several, or all phases of swallowing described in Sect. 1.1, which leads to a variety of symptoms, the most important of which are briefly described below:

- Premature spillage: The bolus slides forward and out of the mouth uncontrolledly (anterior spillage) or backward into the throat (posterior spillage).
- Delayed triggering of the swallow reflex: The swallowing reflex is triggered too late. The result is often a pooling (accumulation) of bolus parts in the hypopharynx before the swallowing reflex is triggered.
- Penetration: The bolus enters the laryngeal vestibule but remains above the true vocal folds.
- Aspiration: The bolus enters the laryngeal vestibule and descends below the level of the true vocal folds and into the upper trachea.
- Silent penetration/aspiration: Bolus material enters the laryngeal vestibule or the subglottic region without triggering a cough or swallow reflex. Silent penetration/aspiration is particularly dangerous because it is neither perceived by the patient nor detected in the clinical examination.
- Residue or retention: Bolus material remains in the swallowing tract after the swallow and is not transported further (in the oral cavity: oral residue; in the throat: pharyngeal residue; in the esophagus: esophageal residue). Retention actually refers to the process that leads to residue, but both terms are usually applied interchangeably in international usage.
- Reflux: Material from lower parts of the swallowing tract flows back into higher parts. Gastro-pharyngeal or esophago-pharyngeal reflux are the most common types of reflux.
- Odynophagia: Swallowing is painful. Painful swallowing is rarely seen as an isolated symptom of neurogenic dysphagia.
- Hypersalivation: There is an increase in salivation, which—in neurogenic dysphagia—is mostly due to the reduced ability to swallow saliva and not due to an increased salivary production. The terms pseudo-hypersalivation and sialorrhea are also used.

Figure 1.4 illustrates major symptoms of neurogenic dysphagia. A detailed description can be found in Sect. 3.1.

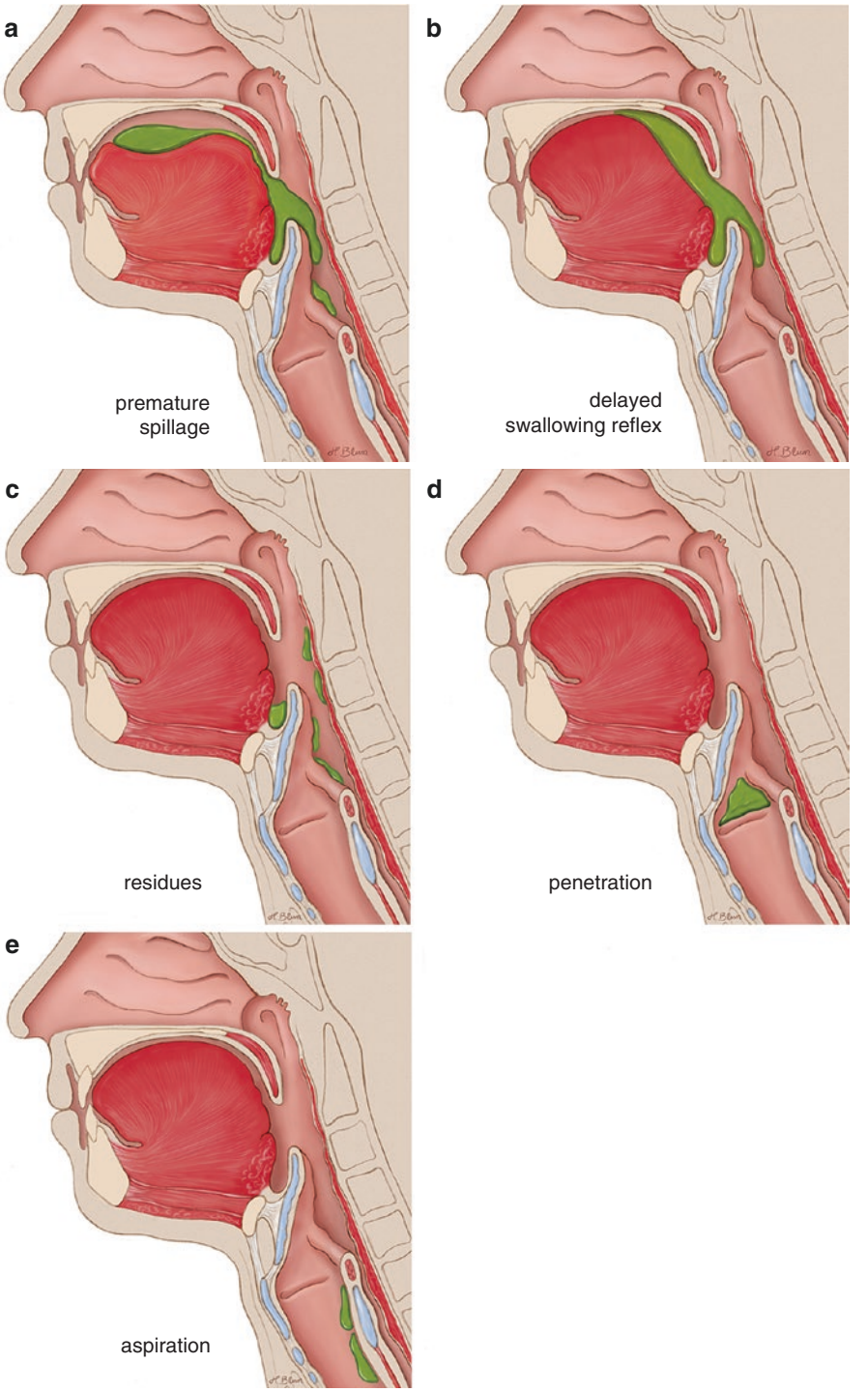

Fig. 1.4 Clinically relevant symptoms of neurogenic dysphagia (© 2017 Heike Blum). (**a**) Spillage (a disorder of the oral preparatory phase and/or of the oral phase); (**b**) delayed swallowing reflex (a disorder of the pharyngeal phase); (**c**) residue; (**d**) penetration; (**e**) aspiration. Reproduced with permission