The difference between psychogenic and neurodegenerative dysphagia – demographics and treatment options

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Abstract

Psychogenic is a debilitating illness that can have multiple causes. Psychogenic dysphagia is a form of dysphagia that is caused by one or more psychological factors. 60.2% of psychogenic patients is female. Comorbidity is also frequently seen (31.6%). The precipitating cause is often a choking event (60.2%). Psychogenic dysphagia can occur at all ages, with a peak in early adolescence. Furthermore, weight loss can be up to one third of the initial weight. Psychogenic dysphagia can last for several years and sometimes for even more than a decade. Its duration until hospital admission is negatively correlated with weight loss rate. Relative weight loss is positively correlated with duration of the disease. The best medicinal treatment options are antidepressants and anxiolitics. The best behavioural treatment options seem to be EMDR and CBT.

On the other hand, we have neurogenic dysphagia. A vast body of literature shows that neurogenic dysphagia often occurs in Parkinson's disease. Tongue pumping and penetration or aspiration are the most serious problems that Parkinson patients seem to encounter during swallowing. A major underlying biological factor of these swallowing problems is the degeneration of cranial nerves that control swallowing mechanisms. EMST and supraglottic swallowing techniques seem to improve some of the swallowing issues in these patients.

Both psychogenic and neurogenic dysphagia seem to show facial similarities such as malnutrition and weight loss. Furthermore, brain studies demonstrate that both illnesses are accompanied by shifts in brain activities towards frontal and parietal lobes. These changes possibly represent changes in the salience network in both psychogenic and (early-stage) neurogenic dysphagia.

Table of contents

Introduction

Dysphagia in an individual is defined as having a (perceived) difficulty with swallowing. Dysphagia can have multiple underlying causes and pathologies. For example, it can be caused by a stroke neurodegenerative disease or psychological factors such as a choking experience. In this essay, the latter two are discussed in more detail. Dysphagia that is caused by a psychological factor is called psychogenic dysphagia. It can be caused by a choking or vomiting experience. Subjects often adapt their eating pattern to cope with the disease. Only around 100 to 150 cases have been described in the literature.

On the other hand, we have neurodegenerative dysphagia. This is dysphagia that is due to the loss of neurons and their connections. This is especially seen in Parkinson's disease. This is because Parkinson's disease is a neurodegenerative disease that causes movement disorders, including swallowing problems (Bloem, Okun, & Klein, 2021). It is estimated that around 6 million people are suffering from Parkinson worldwide (Ou et al., 2021). Around a quarter of These patients have serious swallowing difficulties (dysphagia) (Kalf, de Swart, Bloem, & Munneke, 2012). Hence, more than a million people with Parkinson have dysphagia, making it a serious worldwide problem in this group. In this essay, we try to unravel both psychogenic dysphagia and neurodegenerative or neurogenic dysphagia. In the end, we try to summarize their hallmarks and similarities and differences.

Psychogenic dysphagia – phagophobia and choking phobia

Causes and etiology of psychogenic dysphagia

Psychogenic dysphagia is a form of dysphagia that is caused by psychological factors. Usually, there is no organic cause present to explain the complaints. One of the first cases of psychogenic dysphagia was described in the late seventies of the 20th century by Kaplan and Evans (1978) in a woman. The subject experienced difficulty in swallowing, especially in public places. Additional symptoms that occurred were lengthy eating rituals and cutting food into small pieces. The precipitating event was a traumatic event, not related to choking. However, psychogenic dysphagia may be caused by more events, leading to the existence of more forms of psychogenic dysphagia. In this case, the form of dysphagia was only partially associated with a fear of eating (Kaplan & Evans, 1978). Other forms are directly related to a fear of eating.

Forms of psychogenic dysphagia that are a major focus of this article are phagophobia and choking phobia. Both forms are directly related to a fear of eating in general (Okada et al., 2007; Shapiro et al., 1997). Choking phobia is a subgroup of phagophobia. In addition to a fear of choking, phagophobia is also associated with a fear of vomiting (Okada et al., 2007). In choking phobia, there is a mere fear of choking that is present (McNally, 1994). Importantly, the fear of choking in choking phobia or vomiting in phagophobia is often precipitated by a choking or vomiting event, respectively (Banerjee, Bhandari, & Rosenberg, 2005; Batara et al., 2022; McNally, 1994; Okada et al., 2007).

The core symptom of psychogenic dysphagia is difficulty or fear of swallowing or choking, resulting in partial or entire food refusal and consequent weight loss (Franko, Shapiro, & Gagne, 1997). Secondary symptoms arise because of the concerning fear. Here, such symptoms include: a fear of eating alone or in public, a prolonged meal consumption time (Lopes, Melo, Curral, Coelho, & Roma-Torres, 2014), excessive chewing (Scemes, Wielenska, Savoia, & Bernik, 2009; Chorpita, Vitali and Barlow; 1997), spitting out food (seen in children) (Burklow and Lindscheid, 2004), having nightmares

about choking (Chatoor, Conley, & Dickson, 1988) and anxiety and distress. **Figure 1** shows a Venn diagram for psychogenic dysphagia, phagophobia and choking phobia.

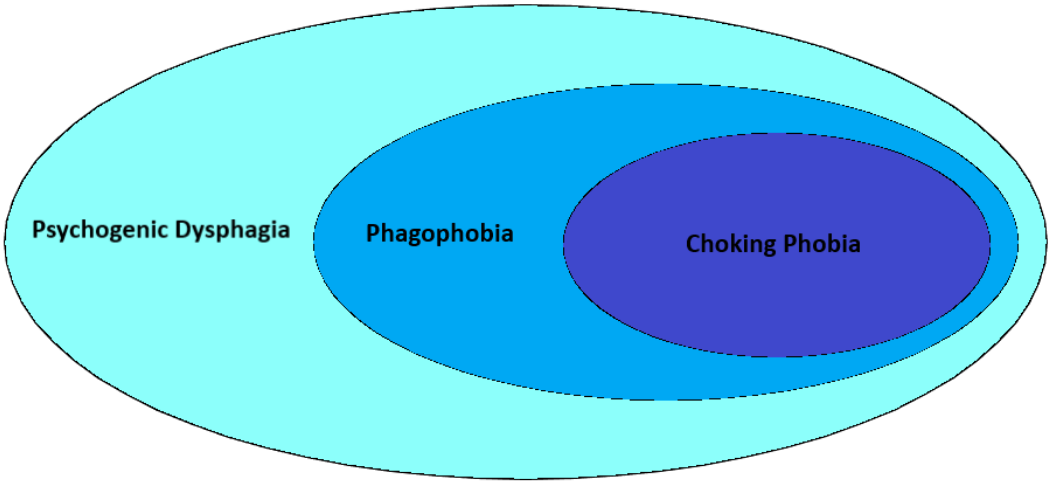

Figure 1. Venn diagram of psychogenic dysphagia, phagophobia and choking phobia.

Demographics and correlations on psychogenic dysphagia

Psychogenic dysphagia is a very rare phenomenon. Only up to some hundred cases have been reported in the literature. Data were extracted from 62 studies about psychogenic dysphagia (**Figure 2**). The data was analysed with help of R version 4.2.2. The packages *ggplot2, ggpubr,* and *stringi* were used.

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"Acikel and Ak (2018)"                    "Akbas and Akça (2018)"
"Atkins et al. (1994)"                    "Ball and Otto (1994)"
"Banerjee et al. (2005)"                  "Batara et al. (2022)"
"Begotka et al. (2021)"                   "Begotka et al. (2022)"
"Brown et al. (1986)"                     "Burklow and Linscheid (2004)"
"Carroll et al (2017)"                    "Celik et al. (2007)"
"Chatoor et al. (1988)"                   "Chorpita et al. (1997)"
"Çiyiltepe and Türkbay (2006)"            "Correia et al. (2010)"
"de Lucas-Taracena and Ibarra (2001)"     "De Roos and de Jongh (2008)"
"De Jongh and Ten Broeke (1998)"          "Dosanjh et al. (2017)"
"Epstein and Deyoub (1981)"               "Etoh et al. (2022)"
"Evans and Pechtel (2011)"                "Franko et al. (1997)"
"Greenberg et al. (1986)"                 "Greenberg et al. (1988)"
"Haberfellner (2008)"                     "Hambride et al. (2001)"
"Hosoglu and Akça (2018)"                 "Kaplan and Evans (1978)"
"Kardas et al. (2014)"                    "Khalifa and Job (2019)"
"Kim et al. (2018)"                       "Kim et al. (2022)"
"Kokanovic and Barron (2021)"             "Lane-Loney et al. (2022)"
"Liebowitz (1987)"                        "Lopes et al. (2014)"
"McNally (1986)"                          "Millikin and Braun-Janzen (2013)"
"Okada et al (2007)"                      "Öst (1992)"
"Penzer (1976)"                           "Premalatha et al. (2015)"
"Rachidi et al. (2022)"                   "Reid (2016)"
"Sahoo et al. (2016)"                     "Scemes et al. (2009)"
"Schurmans (2007)"                        "Seiverling et al. (2016)"
"Singer et al. (1992)"                    "Sivri et al. (2018)"
"Solyom and Sookman (1980)"               "Stoian and Rizeanu (2017)"
"Suraweera et al. (2014)"                 "Tanidir and Hergüner (2015)"
"Thomas et al. (2017)"                    "Thottam et al. (2015)"
"Vescovelli et al. (2017)"                "Williams et al. (2011)"
"Wulandari (2020)"                        "Zhu and Dow (2022)"
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Figure 2. The list of studies used in this data analysis.

Dichotomous data

Figure 3 shows dichotomous data on prevalence of psychogenic dysphagia among sexes, comorbidity rates and whether a choking event was the cause of the psychogenic dysphagia. Prevalence of psychogenic dysphagia seems to be higher among females. The ratio female: male is approximately 3:2 (**Figure 3A**). About one third of the subjects had another psychiatric diagnosis or history besides psychogenic dysphagia (e.g., depression, anxiety, obsessive-compulsive behaviour) (**Figure 3B**). In approximately 3 out of 5 subjects, a choking event related to themselves, or others triggered the psychogenic dysphagia (**Figure 3C**). Some other causes may include vomiting (Banerjee et al., 2005; Okada et al., 2007), a stressful event or period (Evans & Pechtel, 2011; Al-Haifi & Job, 2019) or a (viral) disease (Begotka, Silverman, & Goday, 2021).

A **B**

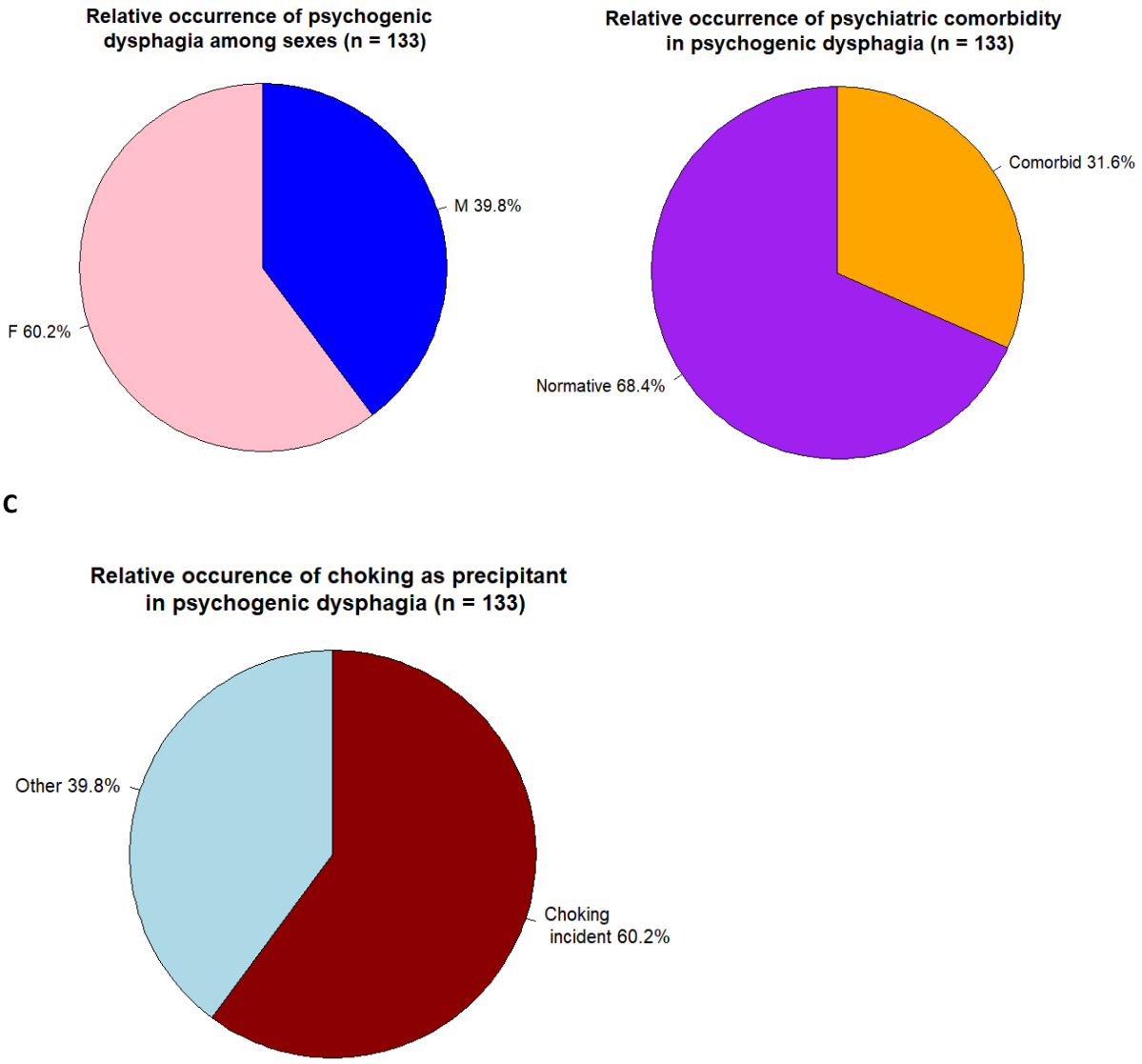

Figure 3. Pie charts showing demographics related to psychogenic dysphagia. **A**. The male-to-female ratio in psychogenic dysphagia. **B**. The relative occurrence of comorbidity among subjects with psychogenic dysphagia. **C**. The relative occurrence of a choking incident (self or other) as a cause of psychogenic dysphagia.

Univariate analyses

In the histograms, the blue, red, and yellow vertical line represent the median, mean and mode or, occasionally, the logarithm of the same, respectively. Of 101 participants, the age of onset could be determined. The mean age of onset was 20.7 $\pm$ 17.4 years (range 0.25 – 80). The median age of onset was 14.5 years (puberty). Furthermore, the onset seems to peak at an age of 10 years (pre-puberty) (**Figure 4A)**.

Whenever weight loss was present, the median weight loss was 6.75 kg. The mean absolute weight loss was 8.2 $\pm$ 5.6 kg and ranged from 1 to 22.7 kg (data not shown). A more robust measure of weight loss is the fraction of weight loss. This measure was calculated by the difference between postmorbid, and premorbid weight divided by the premorbid weight. Therefore, it corrects for limitations in weight loss due to lower absolute premorbid weight (seen in children). No literature on psychogenic dysphagia in obese individuals was found. The mean and median relative weight loss were 17.0% and 18.3%,

respectively and ranged from 3.9 to 31.3 percent (**Figure 4B**). Some Incidental studies reported absence of any weight loss in individuals, but these data were not included (Premalatha, Varghese, & Gundelli, 2015; Thottam, Silva, McLevy, Simons, & Mehta, 2015). Additionally, as a measure of gravity of the psychogenic dysphagia, the weight loss rate, was calculated (weight loss in grams divided by the period over which weight loss was reported in days). The median weight loss rate was 99 grams per day. The weight loss rate had a mean of 110 $\pm$ 94 grams per day and ranged from 2 to 375 grams per day (**Figure 4C**).

Participants presented to clinicians with a varying disease duration (time between onset and admission to patient care). The data for disease duration in months was severely right skewed. A log-transformation was performed to better visualize the data. The disease duration turned out to be log-normally distributed. The mean disease duration was approximately 23 (3.1) months, ranging from 0.1 to 360 months. The median disease duration was 5 (1.6) months. Most often, the disease duration was 3 (1.1) months (**Figure 4D**).

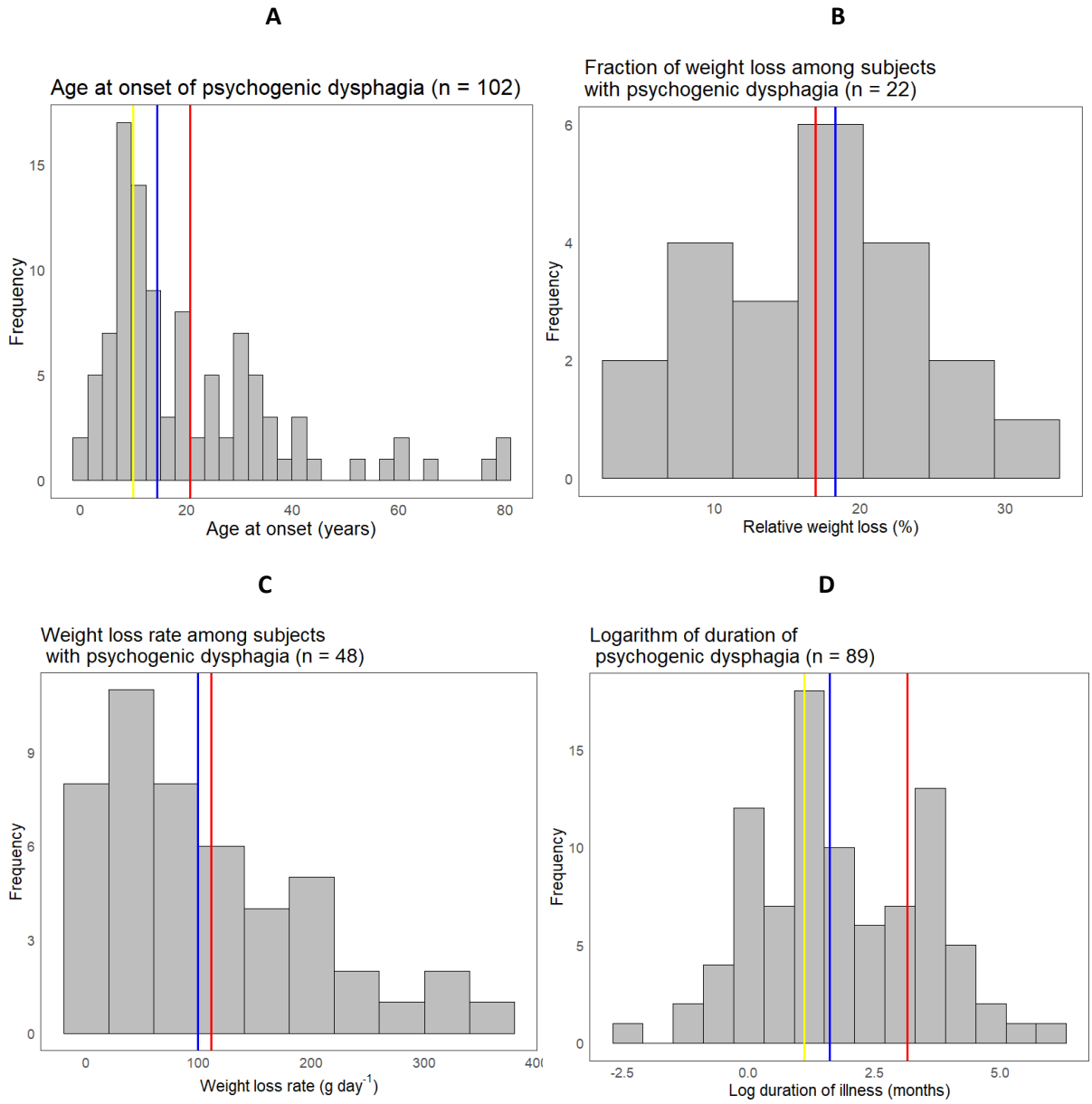

Figure 4. Univariate analysis of epidemiological disease variables on psychogenic dysphagia. **A**. Distribution of the age at onset of psychogenic dysphagia. **B**. Distribution of the relative weight loss in psychogenic dysphagia. **C**. Distribution of the rate of the weight loss in psychogenic dysphagia. **D**. Distribution of the logarithm of the duration of illness (in months) in psychogenic dysphagia. Whenever shown, blue lines: (logarithm of) median, red lines: (logarithm of) mean, yellow lines: (logarithm of) mode.

Association analyses

Association analyses were done to detect possible relationships between the assessed variables. Indeed, it was found that there was a moderately strong correlation between the age at onset and duration of psychogenic dysphagia. More specifically, there was a significant correlation between the logarithm of the two ($r = 0.55, p < 0.001$) (**Figure 5A**). Two non-missing datapoints from (Celik, Diler, Tahiroglu, & Avci, 2007) were deliberately removed, as they were considered outliers. With inclusion of the outliers the correlation coefficient was 0.36 (significant, data not shown). There was also a strong positive correlation between the relative weight loss and the duration of the disease ()$r = 0.68, p < 0.001$. This logically indicates that subjects lose more of their initial weight as the psychogenic dysphagia progresses (**Figure 5B**). No correlation was found between age at onset and weight loss rate (data not shown). A trend towards a positive correlation was found between age and weight loss rate (data not shown). Another finding was that the gravity (measured in weight loss per time unit) correlated negatively with the duration (time between onset and hospitalization) of psychogenic dysphagia ($r = -0.65, p < 0.001$). This indicated that subjects with a more serious illness sought help sooner (**Figure 5C**).

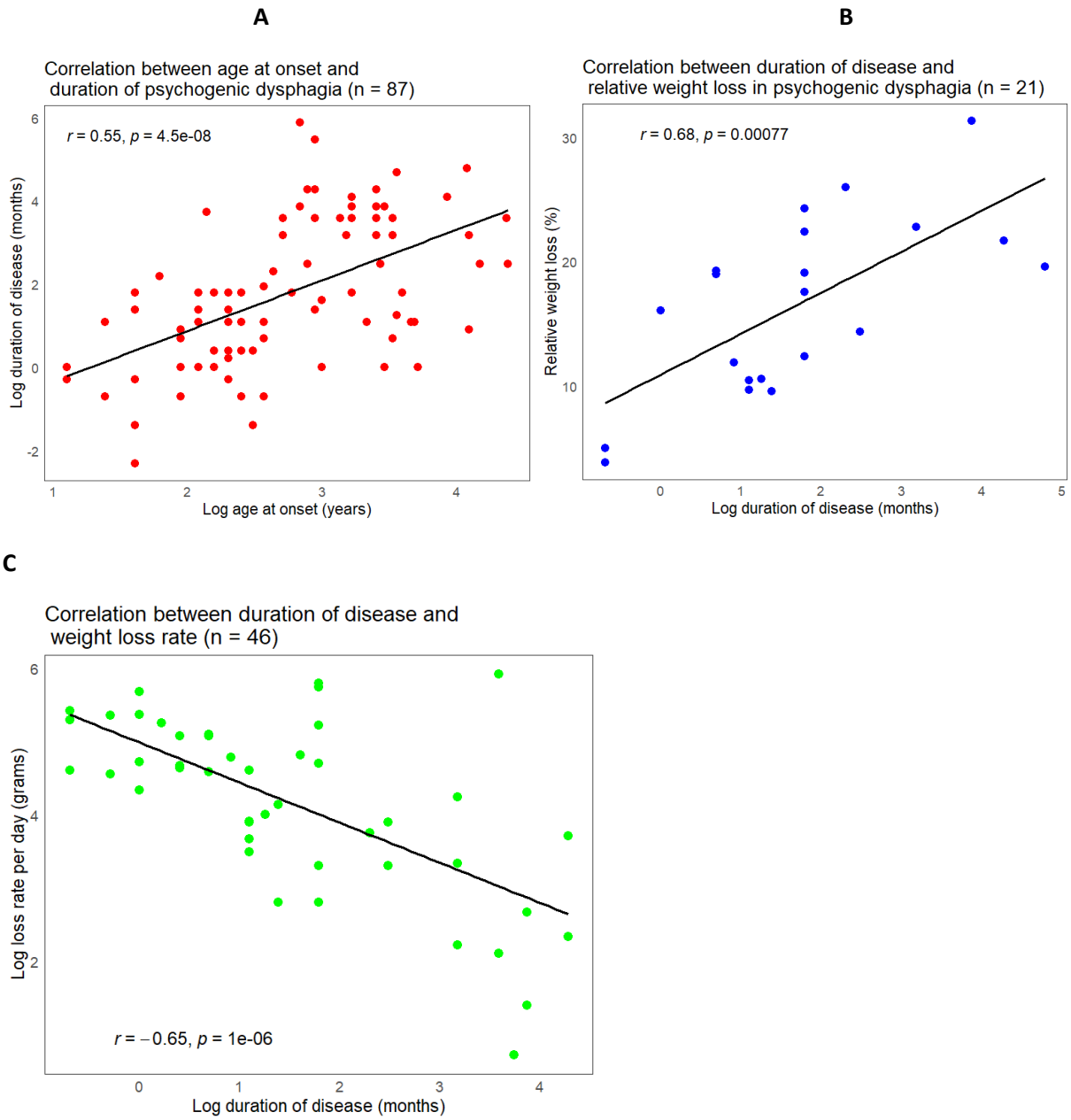

Figure 5. Correlational analyses between demographic variables. (**A**) The correlation between the age of onset and the duration of psychogenic dysphagia. B. The correlation between the duration and relative weight loss **(B)** and gravity of disease **(C)** in psychogenic dysphagia.

Diagnosis of psychogenic dysphagia

In the previous sections, we have seen that psychogenic dysphagia - in its severe form – is very rare. A possible cause of this is that it is often diagnosed very carefully. A thorough reevaluation of putative patients with psychogenic dysphagia illustrated that a wrong diagnosis could lead to attrition of the population of these patients. This is because they are then reclassified as *not* having psychogenic dysphagia (Ravich, Wilson, Jones, & Donner, 1989). The main criterion for a diagnosis of psychogenic dysphagia is the absence of any medical or organic causes of the dysphagia. In out of 73 of the 133 cases in this study a physiological test of some form was reported (See ***Appendix – Dataset psychogenic dysphagia***. These tests include cervico-cranial MRIs (Begotka et al., 2021; Begotka,

Silverman, & Goday, 2022; Okada et al., 2007), ear nose and throat examinations (Begotka et al., 2022), cranial nerve assessment (Premalatha et al., 2015), esophagoscopy (Singer, Ambuel, Wade and Jaffe, 1992), a fiber endoscopic study (Thottam et al., 2015) and, more frequently, a barium swallow study or video fluoroscopy (Burklow and Linscheid, 2004; Chatoor et al., 1988; Franko et al., 1997; Kim, Munshi, & Hussain, 2018).

Treatment options in psychogenic dysphagia

Therapies

After diagnosis, treatment options are considered to alleviate the psychogenic dysphagia. Therapies are often focused on treating the psychogenic dysphagia in such a way that subjects start to eat normally after a certain time period and gain weight. There are several ways in which the psychogenic dysphagia can be treated. In the light of the difference between psychogenic dysphagia and neurogenic dysphagia, it is important to mention that psychogenic dysphagia is often treated in a psychological way. Some treatment options include behavioural feeding therapy (BFT), eye movement desensitization, reprocessing (EMDR), and hypnosis.

Behavioural feeding therapy (BFT) is a form of therapy that can be maintained in and beyond a clinical setting. BFT is sometimes combined with a cognitive component. In the study of Ball and Otto (1994), the cognitive component consisted of psychoeducation and cognitive restructuring. With psychoeducation, subjects were educated to obtain knowledge and insights about the disease itself (Sarkhel & Arora, 2020). Cognitive restructuring is the act of challenging and replacing anxiogenic thoughts. In psychogenic dysphagia, this is done by evaluating the probability that one would choke. Desensitization through exposure can be comprised of interoceptive and in vivo exposure. An example of interoceptive exposure is that patients themselves create sensations of throat tightening by holding their swallow. In vivo exposure consists of practicing with eating feared foods. This is done by creating a food hierarchy ranging from least feared foods to most feared foods. In this way, subjects become desensitized in relation to the choking fear. Furthermore, subjects are supposed to reduce the number of chews per bite and decrease the use of avoidance behaviours. With this form of therapy, Ball and Otto managed to reduce the psychogenic dysphagia in all of their three patients in 11 to 13 sessions (Ball & Otto, 1994). In general, the treatment resulted in weight gain if significant weight was lost and significantly improved behaviour towards eating (Ball & Otto, 1994; Begotka et al., 2022; Çiyiltepe & Türkbay, 2006; Haberfellner, 2008; Millikin & Braun-Janzen, 2013; Okada et al., 2007; Suraweera, Hanwella, & de Silva, 2014). Sometimes, BFT may be combined with additional medication (Lopes et al., 2014). In a woman with fear of choking on fluids, a combination of the cognitive and behavioural elements resulted in almost complete remission (Öst, 1992). Combined with inpatient treatment, results from BFT may emerge even faster (Burklow & Linscheid, 2004). These studies clearly show that BFT is successful in treating psychogenic dysphagia.

Other forms of treatment are cognitive therapy alone, visual feedback and hypnosis. Firstly, from several studies it turned out that cognitive therapy alone could not improve the psychogenic dysphagia. This indicates the importance of a behavioural or exposure component in therapy (Atkins, Lundy and Pumariega, 1994; Banerjee et al., 2005). Secondly, with visual feedback or biofeedback the swallowing mechanism can be evaluated by the subjects themselves. In this way, subjects may feel reassured and more confident about the safety of swallowing, leading to a reduction in anxiety. However, studies show mixed evidence of efficacy of this form of therapy. Some report that it is clearly effective (Kim, Han, Shin, Yoon, & Kim, 2022). Others show that for some subjects biofeedback alone

is not sufficient and additional therapies are necessary (Thottam et al., 2015). Thirdly, hypnosis was also used sometimes. The idea of hypnosis in psychogenic dysphagia is that patients are given suggestions. These suggestions act in such a way that they should reduce the fears and symptoms related to psychogenic dysphagia. Evidence on the effectivity of hypnosis in psychogenic dysphagia seems scarce and is not always consistent. Some authors report improvements with hypnosis alone (Epstein & Deyoub, 1981) or in combination with other forms of therapy (Franko et al., 1997). Others imply that previous hypnosis was not sufficient to curb the psychogenic dysphagia (Millikin & Braun-Janzen, 2013).

Besides BFT and some less convincing treatments, a second and clearly successful therapy in the treatment of psychogenic dysphagia is EMDR. The basis of EMDR is that the processing of traumatic memories, such as a choking incident, is facilitated. As described in (de Jongh & Ten Broeke, 1998), this happens by 1) alleviating the distress that is related to one or more memories, and 2) decondition the effects of stimuli that trigger the anxiety response and 3) preparing the subject for future occasions in which the stimuli might occur. They managed to alleviate symptoms of choking phobia triggered by surgical interventions in a female subject in two EMDR sessions (de Jongh & Ten Broeke, 1998). In later studies, it could be observed that the eating pattern of children with a choking episode normalized. The children gained their weight as well. This happened after just one or two EMDR sessions (de Roos and de Jongh, 2008; Kokanovic & Barron, 2021). However, in a study of Schurmans (2007) 20 sessions were required to address the choking phobia. This was possibly because the subject had encountered more problems than only the choking phobia in earlier stages of life (Schurmans, 2007).

Medications

Therapies are regularly combined with medication, for example see (Acikel & Ak, 2018; Begotka et al., 2021; Lopes et al., 2014; Sivri, Gülsen, & Yilmaz, 2018). Several lines of medication have been prescribed to subjects with psychogenic dysphagia. The most frequently used groups are discussed hereafter and include anxiolytics, antipsychotics, monoamine-oxidase inhibitors (MAO-I), and antidepressants (SSRIs). Below, a table is drawn that shows the number of failures and successes for each medication group in treating psychogenic dysphagia (**Table 1**). This table can be reproduced with help of the data given in ***Appendix – Dataset psychogenic dysphagia***. Some entries had multiple entries that may indicate a combined use or consecutive failure of more than one medication type. Overall, anxiolytic agents and SSRIs performed best in reducing the symptoms related to psychogenic dysphagia. Data on MAO-I was scarce, so clear conclusions remain out. Antipsychotics performed worst in treating psychogenic dysphagia. Failures in the use of medication may be related to reported adverse effects (Greenberg, Stern, & Weilburg, 1986, 1988; Kim et al., 2018; Tanidir & Hergüner, 2015).

Table 1. Treatment outcome of medications in psychogenic dysphagia.

	Anxiolytic	**SSRI**	**Antipsychotic**	**MAO-I**
Success	10	16	2	2
Failure	2	3	4	1

Hence, we can already draw preliminary conclusions from behavioural and medicinal interventions on psychogenic dysphagia. However, the question remains what neuronal mechanisms are involved in this peculiar form of dysphagia. For example, we might wonder what the distinction between psychogenic dysphagia and neurodegenerative dysphagia at the level of the brain looks like. In the last section of this part, we summarize the literature on the neuronal mechanisms behind swallowing.

The cortical swallowing network

As the name implies, the cortical swallowing network (CSN) is a network in the brain that is involved in the process of swallowing. Here, we summarize some important findings and changes in the network in psychogenic dysphagia. An important brain area that is consistently involved in the CSN is the sensorimotor cortex (Babaei et al., 2013; Dziewas et al., 2003, 2005; Hamdy et al., 1999; Suntrup et al., 2014; Teismann et al., 2007). One way to achieve such a result is to perform a swallowing task in participants whilst brain imaging techniques are used. Importantly, in this task participants are given water via an oral injection or spray. In this way, any noise by other motor movements can be cancelled out (Suntrup et al., 2014; Teismann et al., 2007; Watanabe, Abe, Ishikawa, Yamada, & Yamane, 2004). In an experiment with the anesthetic lidocaine sprayed in the oral cavity, researchers could show a reduction of activity in the sensorimotor cortex during a swallowing experiment in healthy controls. This experiment solidifies that this area becomes specifically activated during swallowing (Teismann et al., 2007). Other brain areas that were repeatedly shown to be activated were the sensory and somatosensory cortex and supplementary motor area. This implies that these regions are also important in regulating swallowing movements (Dziewas et al., 2003; Hamdy et al., 1999; Teismann et al., 2007; Suntrup et al., 2014).

Above findings illustrate some of the brain areas that are important in normal swallowing. Studies assessing brain function in psychogenic dysphagia are very scant. In the one study found, Suntrup and others found a bilateral activation of the rostro-medial part of the primary and secondary sensorimotor cortex in healthy controls in a swallowing task mediated by infusion. However, in their diseased subjects suffering from functional (psychogenic) dysphagia they found activation of the right caudo-lateral part of the primary and secondary sensorimotor cortex and reduced activities in the sensorimotor area and supplementary motor area (SMA). In addition, the insula, dorsolateral prefrontal cortex and the inferolateral parietal lobe was also activated. The authors explain that the latter regions are part of the salience (rating) network that becomes active when psychogenic dysphagic patients are prompted to swallow (Peters, Dunlop, & Downar, 2016; Suntrup et al., 2014). According to a recent review, deficits in serotonergic signaling can disrupt the salience network by enhancing its activity (Conio et al., 2020). Therefore, hyperactivity in the salience network seen in the study of Suntrup et al. (2014) may have been caused by deficits in serotonergic neurotransmission. This could also explain why anxiolytics and especially antidepressants are effective in treating psychogenic dysphagia. In the next section, an organic equivalent of psychogenic dysphagia is discussed: neurodegenerative or neurogenic dysphagia. Later on, we will highlight similarities and differences between these two entities.

Neurodegenerative dysphagia – dysphagia in Parkinson's Disease

This section is about neurodegenerative dysphagia that is an organic form of dysphagia. Although this is not the only form of organic dysphagia, this essay only focuses on this form of organic dysphagia. Another form of dysphagia that was often described is dysphagia in strokes. Readers interested in this

topic are kindly referred to the book *Dysphagia Following Stroke* (3rd Ed.) by Daniels, Huckabee and Gozdzikowska (2019). Most of the remaining literature was about dysphagia in Parkinson's Disease. This is reviewed below.

Physiology of swallowing

The physiology of oropharyngeal (throat-and-mouth) dysphagia is rather complex. This is because sequential swallowing reflexes are coordinated by more than 25 muscles in six discrete swallowing phases. Notwithstanding the psychological complexity of psychogenic dysphagia, neurodegenerative dysphagia has proved more complex physiologically. Therefore, it is useful to first summarize the normal physiology of swallowing shortly before we delve into the squalor of neurodegenerative dysphagia often seen in Parkinson's disease.

Phases of swallowing

Several phases of swallowing are coordinated by a set of cranial nerves that are mediated by nuclei in the medulla and brain stem. First, we have the oral preparatory phase of swallowing. Essentially, it encompasses the formation of a bolus, or a swallowable solid piece of food within the oral cavity by mastication or chewing. The movements occurring in this phase by the tongue, mandible and lip and closure of the esophageal sphincter are regulated by the cerebellum. Next, in the oral transit phase, the tongue seals the food bolus to the palate and propels it towards the back of the oral cavity to initiate the pharyngeal phase. This is coordinated by several cranial nerves (Walton & Silva, 2018).

The pharyngeal phase is triggered by another pair of cranial nerves. During this phase, the nasopharynx and velopharyngeal junction is sealed off by the soft palate to prevent nasal outflow of food remainders. Next, the hyoid bone and larynx are elevated away from the cervical spine after which the bolus passes into the pharynx. Meanwhile, penetration of the larynx (cavity of the throat leading to the airways) through a food bolus is prevented in several ways. Firstly, the epiglottis moves posteriorly, thereby closing the airways and directing the food into the esophagus. Secondly, the airway is further protected from penetration as the focal cords close off. The pharyngeal phase ends as food passes through the upper esophageal sphincter (UES), which ushers in the esophageal phase (Walton & Silva, 2018).

The last and esophageal phase is triggered by opening of the UES after which food passes via the esophagus into the stomach. Although it is an essential phase of swallowing, it seems to be of lesser importance in Parkinson's disease. Where the oral and pharyngeal phase of swallowing are regularly accompanied by voluntary movements, the esophageal phase is mainly regulated by involuntary peristalsis (Costa, Brookes, & Hennig, 2000). In Parkinson's disease it is especially the voluntary movements that are compromised (Bloem et al., 2021). Also, most of the literature focuses on oropharyngeal dysphagia. Therefore, we will mainly focus on oropharyngeal dysphagia in Parkinson's disease.

Oropharyngeal dysphagia in Parkinson's disease

Oral dysphagia

Oral dysphagia is defined as dysphagia that solely relates to the oral phase of swallowing. There are several contributors to oral dysphagia. Firstly, mastication or chewing rates may be reduced due to

bradykinesia (slowed movements) of masticatory muscles (Baijens et al., 2012; Bushmann, Dobmeyer, Leeker, & Perlmutter, 1989). Piecemeal deglutition is defined as needing multiple swallows to successfully clear a bolus. In Parkinson patients, piecemeal deglutition happens more often than in healthy controls (Nagaya, Kachi, Yamada, & Igata, 1998; Robbins, Logemann, & Kirshner, 1986; Wang, Shieh, Weng, Hsu, & Wu, 2017). Besides, Parkinson patients show impairments in their tongue function. For example, bradykinesia and increased tongue pumping were found in some Parkinson patients in several studies (Bushmann et al., 1989; Troche, Sapienza, & Rosenbek, 2008, Umemoto, Tsuboi, Kitashima, Furuya, & Kikuta, 2011). It was seen that bradykinesia of the tongue caused impaired oral food transportation (Umemoto et al., 2011). Also, in one study, the number of tongue pumps was related to a delayed oral transit time; the longer the oral transit time, the more tongue pumps (Troche et al., 2008). However, another study shows that a shorter oral transit time is associated with more tongue pumps (Argolo, Sampaio, Pinho, Melo, & Nóbrega, 2015). The differences between studies may be explained by differences in bolus consistencies. Still, it seems certain that excessive tongue pumping changes oral transit time. Increased or accelerated tongue pumping does not only change oral transit times. It is also associated with unstable positioning and control of the food bolus. This leads to spillage of food in the sulci between lower teeth and lip and base of the mouth and retention in the pharyngo-esophageal transition zone (Argolo et al., 2015). Besides, reduced tongue and bolus control can lead to premature swallowing in which the bolus is not well prepared to be swallowed (Nagaya, Kachi, Yamada, & Sumi, 2004). Although these may be marginal and non-dangerous side effects of increased tongue pumping, there is one effect of increased tongue pumping that can be dangerous.

It turns out that increased tongue pumping is associated with higher chances of penetration or aspiration (P/A) (Nagaya et al., 1998; Argolo et al., 2015). PA seems to occur in approximately 32.8 percent (233/710) of Parkinson patients. Here, the counts are based on the studies (Bird, Woodward, Gibson, Phyland, & Fonda, 1994; Bushmann et al., 1989; Claus et al., 2020; Curtis, Molfenter, & Troche, 2020; Gaeckle, Domahs, Kartmann, Tomandl, & Frank, 2019; Labeit et al., 2020; Leopold & Kagel, 1997; Monteiro et al., 2014; Nagaya et al., 2004; Pflug et al., 2018; Pitts et al., 2010). In the same way, P/A occurred only in 6.7 percent (8/121) of the normal elderly population, using similar swallowing assessments (Allen et al., 2010; Monteiro et al., 2014) (see **Table 2**). Another study found a higher rate of P/A, occurring in 83% of the healthy elderly population. However, the number of swallows per participants was 32. This makes it more likely that a person has P/A in at least one of the swallows (Butler et al., 2010). Therefore, this study was not included in the analysis. Using Fisher's exact test, we find that penetration or aspiration is a significant problem in Parkinson's disease (OR: 6.89, CI: 3.30 – 16.62, $p < 0.001$). P/A is also a common cause of aspiration pneumonia. The latter was shown to be the most common cause of emergency admissions (Fujioka et al., 2016) and even death (Dilmaghani et al., 2021; Matsumoto et al., 2014).

To conclude, oral dysphagia seems to be especially driven by exaggerated tongue pumping. This then increases the chances of P/A. eventually, P/A is associated with increased risks of aspiration pneumonia and asphyxia, leading to increased chances of death. Hence, oral dysphagia plays a cardinal role in increasing the risk of adverse health outcomes in Parkinson patients. Next, we will look at the physiology of pharyngeal dysphagia and its possible risks in Parkinson's disease.

Pharyngeal dysphagia

The pharyngeal equivalent of oral dysphagia is pharyngeal dysphagia. This can be defined by dysphagia that is related to the pharyngeal phase of swallowing. Firstly, Parkinson patients seem to have a delayed velopharyngeal junction closure (Baijens et al., 2011), leading to lower velopharyngeal

pressure during swallowing (Jones & Ciucci, 2016). This also leads to a subjective difficulty of swallowing and an inherent reduced quality of life (Jones & Ciucci, 2016).

As swallowing continues, patients with Parkinson may have trouble letting the bolus pass their pharynx or posterior part of the throat.This can cause pharyngeal pooling leading to pharyngeal residue or the feeling of food that is stuck in the throat (Baijens et al., 2011; Labeit et al., 2020; Nagaya et al., 2004; Schröder et al., 2020). Also, researchers found a reduced pharyngeal peristalsis, which may explain why food sticks in the throat (Ali et al., 1996; Curtis et al., 2020; Robbins et al., 1986). In one study, a mechanical restriction was brought around the hyolaryngeal complex. This led to a reduction in the elevation of this complex and consequently to fatigue of pharyngeal musculature and reduced peristalsis (Shaker et al., 2016). Except for one study (Ellerston, Heller, Houtz, & Kendall, 2016) it has indeed been shown that there is a reduced elevation of the hyolaryngeal complex (Bushmann et al., 1989; Kim, Jeon et al., 2023; Leopold & Kagel, 1997). Hence, this reduced elevation can explain why reduced pharyngeal peristalsis and thus pharyngeal residue occurs in these patients.

As described previously, penetration of the airways by a food bolus is prevented in several ways. In healthy individuals, multiple components of the swallowing mechanism act harmoniously and fulfill this task. However, some of these components act abnormally in Parkinson's disease. For example, it has been seen that closure of the airways by the epiglottis is impaired relative to healthy control subjects. Presumably, this is caused by a restriction and slowness in the forward rotation of the epiglottis. In this way, the epiglottis does not close sufficiently and in time to protect the airways (Kim et al., 2015; Leopold & Kagel, 1997; Robbins et al., 1986). Thus, a malfunctioning epiglottis can alter the timing of airway closure in a significant way, being a serious risk for P/A (Curtis et al., 2020). In general, the pharyngeal swallowing reflex is the total picture of what happens during the pharyngeal phase. To connect with the oral phase, improper bolus control by the tongue can contribute to a delayed swallow reflex (Ertekin et al., 2002). Especially P/A happens if this reflex is not properly initiated, as the airways are open (Matsuo & Palmer, 2008). This can have consequences as described in the paragraph about oral dysphagia.

Consequences of oropharyngeal dysphagia

As already described in previous paragraphs, one of the consequences of oropharyngeal (organic) dysphagia is P/A. Because of this, patients with Parkinson may experience a fear of eating. Different from psychogenic dysphagia, only a small fraction of Parkinson patients with organic dysphagia seem to develop a genuine fear of eating (Leopold & Kagel, 1996). An explanation for this may be that many patients do not know that they have signs of organic dysphagia. Frequencies of subjective dysphagia are often much lower than that of objective dysphagia (Kalf, de Swart, Bloem, & Munneke, 2012). Still, dysphagia not only contributes to increased rates of P/A, but also to changes in attitudes towards eating. Furthermore, malnutrition and consequent weight loss may occur because of dysphagia (Bachmann & Trenkwalder, 2006; Nozaki, Saito, Matsumura, Miyai & Kang, 1999). To conclude, oropharyngeal dysphagia is a serious problem in Parkinson. In the next section, we see what neurobiological mechanisms underly the dysphagia in Parkinson.

Table 2. Prevalence of P/A in Parkinsonian subjects and healthy (age-matched) controls

Prevalence of P/A	Parkinson	Normal
Yes	233	8
No	477	113

Neuropathological mechanisms behind Parkinsonian dysphagia

The neuropathological mechanisms behind dysphagia in Parkinson are complicated and multifaceted. However, it is certain that Parkinson's disease is mainly caused by the accumulation of α-synuclein in the brain. This leads to the loss of nigrostriatal dopaminergic neurons and subsequently the disruption of both motor and non-motor pathways (Bloem et al., 2021). Below, an overview of most important pathological mechanisms in Parkinsonian dysphagia is given.

Neuropathological mechanisms in oral dysphagia

Previously, we discussed the oral phase of swallowing and mentioned that this phase was coordinated by a set of cranial nerves. In specific, these are the trigeminal nerve (cranial nerve, CN V) that controls chewing, the facial nerve (CN VII) that controls buccal and lip movement to assist in food positioning and the hypoglossal nerve (CN XII), which controls movement of the tongue (Walton & Silva, 2018). As we have seen previously, decreased mastication rates, tongue bradykinesia and pumping are cardinal to oral dysphagia. Especially in the late stages of the disease it could be seen that all of the above mentioned nerves were damaged as well as their inherent brainstem nuclei by accumulation of alpha-synuclein onto them (Seidel et al., 2015). Both brainstem nuclei and their efferent nerves are damaged likely because of anterograde neuronal transport of pathological alpha synuclein fibers from the brainstem nuclei to these nerves (Freundt et al., 2012). The damage done to these nerves by alpha-synuclein fibers thus possibly clarifies the slowed mastication, tongue bradykinesia and increased tongue pumping seen in Parkinson.

Next to pathological studies, there are neuroimaging studies. In one study, it was seen that Parkinson patients with dysphagia had lower radioactive binding in the ventral striatum (caudal), indicating a lower dopamine transporter (DAT) availability in that region (Booij & Kemp, 2008; Polychronis et al., 2019). Also, a weak positive correlation was observed between presynaptic dopamine levels in the ventral striatum (caudate) and subjective chewing and swallowing functions (Polychronis et al., 2019). A later study showed that premature swallowing was associated with decreased levels of DAT in the ventral putamen (striatum) (Kim, Jeon et al., 2023). An explanation for these findings is death of dopaminergic neurons in the substantia nigra that project to striatal brain areas. It is thought that especially this causes bradykinesia of the body, including the tongue (Bloem et al., 2021, Kim, Byung-Mo, et al., 2015; Kim, Youn et al., 2015).

Neurobiological mechanisms in pharyngeal dysphagia

The pharyngeal phase of swallowing is triggered by stimulation of the glossopharyngeal (CN IX) and vagal (CN X) nerves by the food bolus. This response is mediated by multiple nuclei located in the medulla. These include the nucleus of the solitary tract and the dorsal motor nucleus of the vagal nerves (Saito, Ezure, & Tanaka, 2002; Walton & Silva, 2018). Also in these nerves, accumulation of α-synuclein was found in most Parkinson patients (Mu et al., 2013a, 2013b). This can explain why patients show a delayed swallowing reflex seen in several studies (Bushmann et al., 1989; Robbins et al., 1986; Claus et al., 2020; Wintzen et al., 1994). Also, the internal superior laryngeal nerve was damaged in

one study in all patients (Mu et al., 2013b). This nerve controls the coughing reflex and damage to it can impair this reflex (Kiray, Naderi, Ergur, & Korman, 2006). This may explain why patients suffer from silent aspiration and aspiration pneumonia (Bushmann et al., 1989; Bird et al., 1994; Nóbrega, Rodrigues, & Melo, 2008).

As with oral dysphagia, the same neuroimaging studies focused on pharyngeal dysphagia as well. For example, an impaired triggering of pharyngeal swallow, laryngeal elevation, delayed pharyngeal transit time and aspiration were all associated with decreased DAT availability in several subareas of the striatum, including the caudate nucleus and putamen (Kim, Jeon et al., 2023). Like oral dysphagia, also pharyngeal dysphagia seems to be multifaceted.

Neuropathological findings in generic Parkinsonian dysphagia

This paragraph is about studies that did not assign findings to a specific swallowing phase. Still, useful information about neuropathological mechanisms in dysphagia in Parkinson's disease was provided. For example, patients showed higher activation of the right insula, lateral premotor and motor cortex, dorsolateral prefrontal cortex and inferolateral parietal cortex (Suntrup et al., 2013). Also, Gao and others (2019) found that Parkinson patients with dysphagia showed an enhanced functional connectivity in cerebellar regions, premotor and supplementary motor cortex. Other areas include the temporal, frontal and orbitofrontal gyrus (Gao et al., 2019). Some researchers found just a decreased activity of the supplemental motor area (Kikuchi et al., 2013; Suntrup et al., 2013). A possible explanation for this is that disease progression in dysphagic patients was lower in the group of Gao et al. (2019). This was indicated by a lower Hoehn and Yahr disease scale (Gao et al., 2019; Hoehn & Yahr, 1967; Kikuchi et al., 2013; Suntrup et al., 2013). Furthermore, Gao et al. think that some of these brain regions, such as the cerebellum is overactive as a cause of compensation for swallowing mechanisms that do not work properly anymore (Gao et al., 2019). Others observed a shift in brain activation in non-dysphagic patients towards the lateral motor, premotor and parietal cortices, suggesting a prodromal stage of dysphagia. This was not seen in dysphagic patients, possibly reflecting the decay of compensatory mechanisms by neuronal degeneration (Suntrup et al., 2013). All in all, the dysphagia seen in Parkinson seems to be bifactorial, with degeneration of cranial nerves on one side and dopaminergic alterations on the other side.

Treatment options

Treatment of dysphagia in Parkinson can be multimodal. In the earlier decades, it has been tried to treat patients with the dopaminergic agent levodopa. However, this has not been very effective. An improvement in dysphagia was seen in less than half of a group of twenty patients (Bushmann et al., 1989). A later study also found little consistent improvement in a group of patients treated with apomorphine and levodopa (Hunter et al., 1997). Others even suggest that levodopa may worsen the function of the brainstem swallowing reflex (Michou et al., 2014).

There are also physiological treatments that seem to improve swallowing. These include the supraglottic swallow and expiratory muscle strength training (EMST). The supraglottic swallow offers the patient a way to voluntarily protect their airways (Bushmann et al., 1989). With this technique, subjects are instructed to hold their breath and tilt their chin to the chest, swallow and cough and then swallow another time. In this way, a bolus is cleared from the airways that are hence protected (Bülow, Olsson, & Ekberg, 1999; Bushmann et al., 1989). This method seems to have mixed effectiveness. In one study this method was effective in clearing aspiration in 2 out of 3 patients (Bushmann et al, 1989). However, another study reached this effect in only 1 out of 6 patients (Nagaya et al., 2004). A possible explanation for this is the difference in mean Hoehn and Yahr disease state between the two studies.

Patients in a later state would not or less be able to perform this technique (Nagaya et al., 2004). On the other hand, we have EMST. Results show that Parkinson patients benefit from this treatment. For example, it was found that patients showed reduced severity of P/A, as shown by a reduction on the Penetration-Aspiration scale (Rosenbek, Robbins, Roeker, Coyle, & Wood, 1996; Troche et al., 2010). Others found an improvement in residue scores, indicating an improved swallowing function (Claus et al., 2021). As explained, the rationale behind this treatment is that it improves airway clearance. This is because muscles that are used for coughing are strengthened. In this way, P/A is improved (Claus et al., 2021; Troche et al., 2010).

Discussion

In this essay, we have tried to give an overview of psychogenic and neurogenic dysphagia. We have found that psychogenic dysphagia leads to significant weight loss and that it is associated with mental comorbidities. Choking mostly precipitated the psychogenic dysphagia. With a fluoroscopic barium swallow study and other physiological test an organic cause can mostly be precluded. Albeit cross-sectionally, patients tend to lose more weight as the disease progresses. Patients with a more severe disease progression were also sooner admitted to medical care. Cognitive behavioural therapy and EMDR were consistently effective against psychogenic dysphagia. Antidepressants and anxiolytics are also most consistently effective as pharmacological agents. Brain areas that are altered in their activity include the sensorimotor cortex, SMA, insula, the frontal and parietal lobes. Some of these brain areas are part of the salience network. This salience network can be modulated by medication and therapies (Schienle, Schäfer, Stark, & Vaitl, 2009).

On the other hand, we have neurogenic or neurodegenerative dysphagia. Oral dysphagia manifested as delayed mastication, piecemeal deglutition, and tongue pumping. Important disruptions in the pharyngeal phase presented as slowed pharyngeal peristalsis, pharyngeal residue, and impaired pharyngeal reflexes. In Parkinson, we have seen that dysphagia can be caused by the degeneration of cranial nerves essential in the coordination of swallowing. It is very likely that this is caused by the accumulation of α-synuclein in brainstem nuclei and their descendant cranial nerves (Seidel et al., 2015). Another possibility is that decreased dopaminergic neurotransmission in the striatum contribute to the bradykinesia seen in Parkinson and thus oral and pharyngeal dysphagia. Some possible treatments include the supraglottic swallow technique and EMST. Of both it is known that they are effective in the early stages of the disease (Bushmann et al., 1989; Troche et al., 2010; Claus et al., 2021).

In conclusion, psychogenic and neurogenic dysphagia clearly have different underlying pathologies that need different treatment approaches. Psychogenic dysphagia patients rather show functional brain abnormalities. However, Parkinson patients show additional structural impairments, such as degeneration of cranial nerves and basal ganglia. This explains why dysphagia in this group worsens over time. Besides these differences, there are also similarities. Especially on the level of brain imaging, psychogenic and neurogenic dysphagia show facial similarities. For example, in both (prodromal) neurogenic and psychogenic dysphagia, we see a reduced activity in the supplementary motor area during swallowing tasks. This activity tends to drift away to lateral motor cortices, parietal and frontal areas in both diseases. This possibly means that compensatory mechanisms become active as normal swallowing gets interrupted (**Figure 6**).

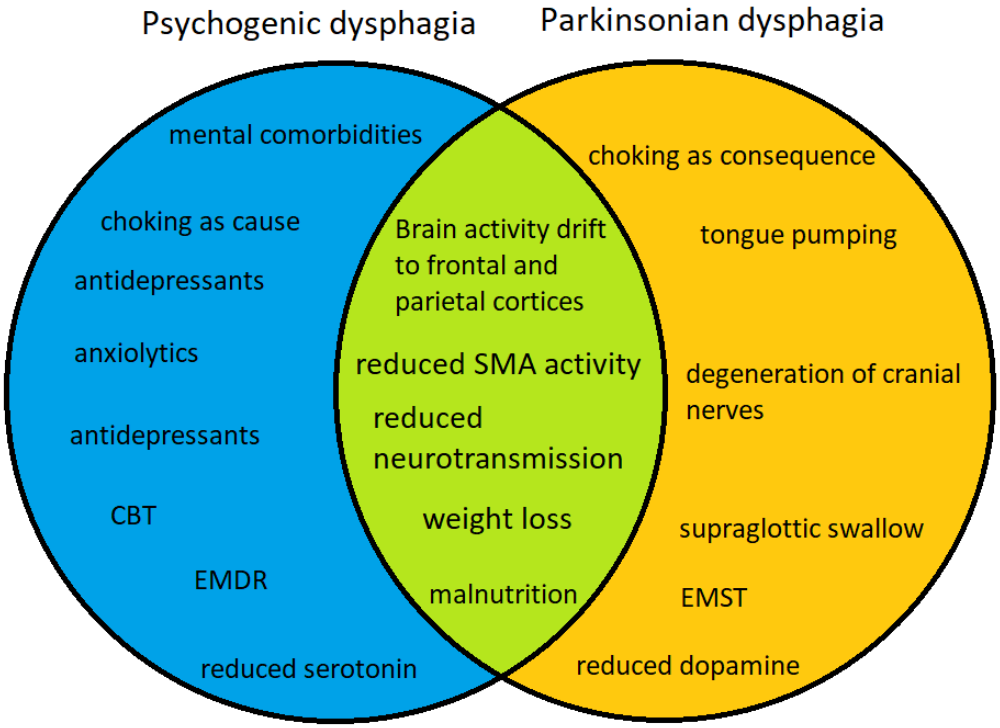

Figure 6. Differences and similarities between psychogenic and Parkinsonian (neurodegenerative) dysphagia.

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Appendix – Dataset psychogenic dysphagia

This appendix contains the dataset for psychogenic dysphagia. The reader is adviced to copy the table below from the digital version of this thesis with the name *mBIO_2023_deGrootJ* and paste it in an empty excel or csv-file to make it readable.

Study	Precipitant of false dyspha gia	Age at onset (years)	Sex	Psychiatric Comorbidity	Duration of illness (months)	Diet composition	Weight loss (kg)	Loss rate (kg per day)	Fraction weight loss	Resolvent medicaments	Resolvent therapies	Physiological tests	Indications for true dyspha gia	Previous unsuccessful therapies	Previous unsuccessful medicaments	Duration of treatments (months)	Number of sessions	Time to last follow-up (months)	Weight gain (kg)	Fraction weight gain
Akikel and Ak (2018)	food stuck in throat	15	F		36	soft foods			SSRI	CBT	upper GI, endoscopy	negative								
Akbas and Akça (2018)	stressful event	5	M	autism	0.1	restricted NOS				anxiolytic, antipsychotic										
Atkins et al. (1994)	choked	6	M		9	liquids				anxiolytic		videofluoroscopy	negative	CT		2.333.333.333	3			
Ballan	choked	30	F	pho	36	noso	9	0.008			BFT				anxioly	2.75	11	3	9	

dOtto (1994)				bia		lids		219							tic					
BalandOtto (1994)	choked	20	F	phobia	1	nosolids	9	0.29589	0.1611		BFT					3.25	13		2.25	0.048077
BlandOtto (1994)	choked	25	F		36	mashedfoods				BFT					2.75	11	2	4.5		
Banerjeeetal. (2005)	7	M	anxiety		nosolids				SSRI		streptococcalscreen, chestx-ray, videofluorosc	negative	CT				2		

											opy								
Banerjee et al. (2005)	10	F	OCB, phobia	3	no solids				SSRI		esophagram, gastroscopy, cervical x-ray	negative							
Banerjee et al. (2005)	choked	8.5	F	OCB, phobia	42	no solids	2.25	0.0020555		SSRI		esophagogram	negative	SOS		9		8		
Batra et al. (2022)	7	M		2		6	0.09863	0.193				negative								
Begotka et al. (2021)	choked		F								BFT	EGD, x-ray, braain	negative			2	15			

												MRI								
Begotka et al. (2021)	choked		F			NG-tube					BFT	EGD	negative			1	3			
Begotka et al. (2021)	choked		M	tic disorder					PPI, fluticasone		BFT	EGD	positive			5	31			
Begotka et al. (2021)	choked		F			NG-tube					BFT	esophagogram	negative			1	5			
Begotka et al. (2021)	viral illness	F	OCB						SSRI		BFT	EGD	negative			1	4			
Begotka et al. (2021)	viral illness	M	anxiety						SSRI		BFT	EGD, esophagogram	negative			2	8			

Begotka et al. (2021)	viral illness	M							SSRI	BFT	EGD, barium study	negative			1	6			
Begotka et al. (2021)	viral illness + choked	M			NG-tube				PPI	BFT	cervical x-ray, videofluoroscopy, naso-pharyngoscopy	negative			2	6			
Begotka et al. (2022)	choked		M	tic disorder							EGD| upper GI radiography								
Begotka et	choked		M	anxiety, depr							EGD | cervi								

al. (2022)				session							calx-ray							
Begotka et al. (2022)	choked		M								EGD |bronchoscopy							
Begotka et al. (2022)	choked		M								EGD							
Begotka et al. (2022)	choked		M								EGD							
Begotka et al. (2022)	choked		M								EGD							
Begotka et al. (2	choked		M								EGD							

022)																				
Begotka et al. (2022)	choked		M									EGD								
Begotka et al. (2022)	choked		M									EGD								
Begotka et al. (2022)	choked		M									EGD								
Begotka et al. (2022)	choked		M									EGD								
Begotka et al. (20	choked		M									EGD								

22)																				
Begotka et al. (2022)	choked		F									EGD								
Begotka et al. (2022)	choked		F									EGD								
Begotka et al. (2022)	choked		F									esophagogram								
Begotka et al. (2022)	choked		F									esophagogram								
Begotka et al. (2022)	choked		F									esophagogram								

Begotka et al. (2022)	choked		F									videofluoroscopy							
Begotka et al. (2022)	choked		F									videofluoroscopy							
Begotka et al. (2022)	illness		F									videofluoroscopy							
Begotka et al. (2022)		F										Ear Nose and Throat evaluation							
Brown et al. (1986)	throat tightness	65	F	depression	12					SSRI								12	

Brown et al. (1986)	40	F	depression, phobia	no solids	11.7		0.26	MAO-I			negative			1		12	12.6	0.378378
Brown et al. (1986)	78	F				13.5	0.222 1918		MAO-I		videofluoroscopy	negative		neuroleptic, anxiolytic	1		4		
Burklow and Linscheid (2004)	choked	8	F		3	soft foods					BFT	videofluoroscopy	outpatient treatment	0.1	7	6		
Burklow and Linscheid (2004)	choked	5	M		0.25	liquids					BFT			outpatient treatment	0.1	7	12		
Burklow an	choking of	7	F		1	soft foods	3.15	0.112329			BFT	endoscopy	negative	outpatient	0.133333	9	0.5		

d Linscheid (2004)	acquaintance														treatment				
Burklow and Linscheid (2004)	other	12	F		1.5	soft foods	2.93	0.10411			BFT	video fluoroscopy, esophageal manometry	0.066667	5					
Burklow and Linscheid (2004)	choked	9	F		1	soft foods				BFT	upper GI, endoscopy	negative	outpatient treatment	0.06667	5				
Burklow and Linscheid	choked	10	F		4	liquids	7.65	0.063014			BFT					0.1	7			

(2004)																				
Carroll et al (2017)	choked	14	M																	
Celik et al. (2007)	stressful event	0.25	F		21	NG-tube				SSRI				desensitization	antipsychotic	6		36		
Celik et al. (2007)	stressful event	0.25	F		21	NG-tube				SSRI				desensitization	antipsychotic	6		36		
Chatoor et al. (1988)	choked	8	F		3	mashed foods	3.6	0.039452			BFT					0.75				
Chatoor et al. (1	choked	9	F	oppositional	1.5	no solids	6.75	0.160274			BFT					1				

988)				disorder																
Chatoor et al. (1988)	choked	10	M		1.5	liquids	4.5	0.1068499			BFT	x-ray, bronchoscopy, laryngoscopy, videofluoroscopy	negative			1				
Chatoor et al. (1988)	choked	10	M	non-specific emotional and cog	1.25		6.75	0.1923299			BFT					1.5				

				nitiveinstabilities																	
Chatoor et al. (1988)	choked	11	F		1.5	liquids	6.75	0.1602 74			BFT		negative	outpatient treatment	0.75						
Chorpita et al. (1997)	choked	5	F	panic disorder, phobia	6	soft foods				BFT					4	14	9				
Çiyiltepe and Türkbay (2006)	choked, choking of acquaintance	13	M	ADHD	3	soft foods	4	0.0328 77			BFT	video fluoroscopy, Ear Nose and T	negative			0.5			5		

												hroatevaluation								
Correia et al. (2010)	choked	34	M		36															
de Lucas-Taracena and Ibarra (2001)	illness	13	F		7	soft foods				BFT					0.75				
De Roos and de Jongh (20	choked	3	M	anxiety	0.75	liquids					EMDR						2			

08)																				
De Roos and de Jongh (2008)	choking of acquaintance	7	F	anxiety	1						EMDR						2			
De Roos and de Jongh (2008)	choking of acquaintance	9	F		6						EMDR						2			
De Roos and de Jong	choked	15	F		24	liquids					EMDR						1	3		

h (2008)																				
De Jong and Ten Broeke (1998)	choked	25	F	depression, panic disorder	60						EMDR						2	15		
Dosanjh et al. (2017)	other	8	M		3	restrictedNOS	4.5	0.05	0.0970887		BFT						5	0.75		
Epstein and Deyoub (19	other	35	M		108						hypnosis					15	56	36		

81)																				
Etoh et al. (2022)	choking of acquaintance	7	F	tic disorder	0.5	liquids	1	0.2	0.039		CBT	head and neck MRI, laryngoscopy	negative			0.75				
Evans and Pechtel (2011)	stressful event	28	M								CBT	laryngoscopy, videofluoroscopy	negative	physical exercises			3			
Franko et al. (1997)	other	41	F		1	not restricted NOS					videofluoroscopy	negative								
Franko et al. (1	stressful ev	34	F		24	soft foods					videofluorosc	negative								

997)	ent										opy								
Frankoetal.(1997)	choked	18	F		12	softfoods	4.5	0.049315			hypnosis	videofluoroscopy	negative						
Frankoetal.(1997)	24	F		24	liquids	20.5	0.028082				videofluoroscopy	negative							
Frankoetal.(1997)	choking of acquaintance	19	F		36	nodairyproducts	11.4	0.3747795				videofluoroscopy	negative						
Frankoetal.(1997)	19	F		72	not restricted NOS						videofluoroscopy	negative							
Frankoetal.(1	choked	32	F		48	liquids						videofluoroscopy	negative						

997)												py								
Frankoetal.(1997)	other	36.5	M		6	liquids	22.7	0.186575				videofluoroscopy	negative							
Frankoetal.(1997)	foodstuckinthroat	32	M		1	softfoods	2.3	0.076667				videofluoroscopy	negative							
Frankoetal.(1997)	foodstuckinthroat	30	M		48	nomeat	5.9	0.004038				videofluoroscopy	negative							
Greenbergetal.(1986)	choked	18	F		72	nosolids	11.25	0.010274	0.217	anxiolytic					MAO-I	4.5			9.45	0.233333

Greenberg et al. (1986)	60	F		24	liquids	12.6	0.07	0.228	anxiolytic		videofluoroscopy	negative		SSRI	8			13.5	0.3157 89
Greenberg et al. (1988)	choked	28	F		3	restrictedNOS	4.5	0.049315	0.105	anxiolytic		videofluoroscopy, upperGI	negative			3		3		
Greenberg et al. (1988)	choked	43	M			mashedfoods			anxiolytic						1				
Greenberg et al. (1988)	choked	30	F		24		6.75	0.009247		anxiolytic					SSRI				
Hab	cho	35	F	pani	3.5	noso	5	0.05	0.1		BFT		negat				6		4	0.09

erfellner(2008)	ked			cattacks		lids		4795	06				ive							5238
Hambrideetal.(2001)	other		F								CBT							2		
HosoglucandAkça(2018)	choked	3	M		1	nosolids	1.5	0.2142866		SSRI	BT	x-ray,laryngoscopy,upperGI	negative			0.5	3			
KaplanandEvans(1978)	other	25	F		48	noconsumptioninpublic				CBT		negative			7.5	15	6			
Kard	cho	12	M		0.25					anxio	dese	fiber	negat			0.1				

as et al. (2014)	ked									lytic	nsitization	endoscopic evaluation, bronchoscopy, upper GI	ive						
Khalifa and Job (2019)	stress ful event	29	F	depression	soft foods				BT	strobos copy, fiber endosc opic eval uation of swallow	negative			1	4				

Kim et al. (2018)	other	79	F	anxiety	36	NG-tube					ECT	video fluoroscopy	negative		antipsychotic, SSRI	2	7			
Kim et al. (2022)	other	25	M		6		19	0.312329	0.224		visual feedback	video fluoroscopy, fiberendoscopic evaluation	negative			3			15	0.227273
Kokanovic and Barron (2021)	choked	4	F		0.5	soft foods					EMDR						2	3		

Lane-Loneyetal.(2022)	choked	11	F	anxiety	3	softfoods	9	0.1			CBT					2.25				
Liebowitz(1987)	choked	19	F		240					anxiolytic										
Liebowitz(1987)	19	F	panicattacks	4						anxiolytic										
Lopesetal.(2014)	choked	31	M		12	nomeat	10	0.027397	0.144	SSRI	CBT					9	9			
McNally(1986)	chokedd	16	M		6	softfoods	20.25	0.11059	0.243		BFT			psychotherapy	2.25	6	6			
Milliken an	stressfulev	59	M	OCB	120	liquids	14.85		0.196		BFT	videofluoro	negative	hypnosis,bi	19	49	48	5.4	0.08484848	

d Braun-Janzen (2013)	ent											scopy		of eedback, craniosacral therapy, couples consseling					
Okada et al (2007)	other	5	F		4		2	0.016438	0.096		SFT					6				
Okada et al (2007)	other	15	F								SFT					4				
Okada et al (20	other	8	F	OCB	6		3	0.016438	0.124		SFT	cervico-cranial	negative			48			7	0.3684211

07)												MRI, Ear Nose and Throat, upper GI								
Okada et al (2007)	other	10	F	OCB							SFT					18				
Okada et al (2007)	other	11	M	ADHD-like	6		5	0.027397	0.191		SFT	cranial MRI and upper GI	negative			5			3.8	0.1792245
Okadaet al (2	other	10	F	ADHD-like							SFT					3				

007)																				
Öst (1992)	stressful event	39	F		3	no fluids					CT					1.5	7	12		
Penzer (1976)	choking on food	51	F		60								positive							
Premalatha et al. (2015)	stressful event	40	M		3							oral peripheral mechanism examination, cranial nerve ase	negative				3			

												ssment, swallowing tests								
Rachidi et al. (2022)	stressful event	6	F	phobia							BFT		negative			5				
Reid (2016)	choked + other	13	F	phobia	2	no solids	9	0.160274	0.19							7	16	36	7.65	0.2
Shooo et al. (2016)	choked	20	F	depressive	5	liquids	7.5	0.1232888			BT	video fluoroscopy	negative			3	25	12		
Scemes et al. (20	choked	30	F		72		15	0.0410096		SSRI	BFT						12		6	

09)																				
Schurmans (2007)	choked	30	F		48	completely restricted NOS	21.15	0.014486	0.313333		EMDR			eating disorder treatment, psychodynamic therapy, CBT	20					
Seiverling et al. (2016)	choked	4	F		3	liquids					BFT					0.5		1		
Singer et al. (1992)	choked	8	M		1	liquids						videofluoroscopy, eso	negative				45	24	2.7	0.123853

												phagoscopy								
Sivri et al. (2018)	choking of acquaintance	14	F		10	no solids	13	0.042 74	0.26	SSRI, antipsychotic	CBT					0.5			10	0.27027
Solyomand Sokman (1980)	17	M	OCB	48										sensory feedback						
Solyomand Sokman (1980)	M	OCB, phobia		9	0.1			aversion therapy												
Solyomand	17	M		360						behavioural th					12					

Sokman (1980)											erapy							
Solyo mand Sokman (1980)	60	M	phobia	2.5		9	0.12	0.11 9048			behavioural therapy				0.75	9	12		
Stoian and Rizeanu (2017)	other	23	M	anxiety	36						multiple						24		
Suraweera et al. (2014)	choked	23	F			soft foods					relaxation, in vivo exposure, cogniti	negative		antipsychotic	3	20			

											therapy									
Tanidir and Hergüner (2015)	choked	4	F	OCB		nosolids				SSRI			negative	desensitization	SSRI	6		6	8	0.3076922
Thomas et al. (2017)	choked	11	F		0.5	liquids	1.4	0.1	0.0501799		CBT		negative							
Thottam et al. (2015)	choked	13	M		0.5	nosolids	4	0.2279455			visual feedback	fiberendoscopic evaluation	negative					2		

Thottam et al. (2015)	choked	5	M		0.75	no solids	4.5	0.2136399				fiber endoscopic evaluation	negative	visual feedback			6	
Thottam et al. (2015)	choked	10	M		0.75	no solids	2	0.0950688			visual feedback	fiber endoscopic evaluation	negative				8	
Thottam et al. (2015)	choked	8	M		4	no solids						fiber endoscopic evaluation	negative	visual feedback				
Thottam et al. (2015)	7	F		2.5	restricted NOS				visual feedback	fiber endoscopic eval ua	negative							

											tio n								
Vescovelli et al. (2017)	other	10	M	anxiety, depression	6	no meat	9.9	0.33	0.176		CBT, PNT		negative			3	12	12	
Williams et al. (2011)	other	8	F		4	NG-tube					exposure					0.25	33	3	
Wulandari (2020)	stressful event	34	F	panic disorder	2		10	0.164384		SSRI	BFT	laryngoscopy, videofluoroscopy	negative				12	6	
Zhuand Do	choked	80	F	phobia	12	no solids					exposure	endoscopy, es	negative			4			

w (2022)												ophagustest								