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Title: An exploration of the role of alexithymia in the development of psychogenic voice disorders

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"Alexithymia” is a condition characterized by difficulties in identifying a person's own emotions, differentiating between emotional and physical arousal, and describing one’s emotions to other people (Bagby & Taylor, 1997). Alexithymia is inversely related to mentalization and is associated with emotional trauma, both of which are thought to influence the capacity to regulate affects induced by stressful events (Taylor, 2010).

Alexithymia has been implicated in a wide range of medical and psychiatric illness, (Vanheule, Desmet, Bogaerts, & Meganck, 2007, Taylor & Bagby, 2004), while a number of studies have linked the construct with psychogenic voice disorder (PVD).

This paper will provide an overview of both alexithymia and PVD, reviewing the available literature in order to help clarify how alexithymia might play a role in the development of PVDs. Various models of symptom formation will be discussed, and the therapeutic implications for the individual with PVD will be examined.
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CHAPTER 1: INTRODUCTION

Alexithymia

Alexithymia, meaning literally “no words for feelings,” was initially coined by Sifneos in (Sifneos, 1973) in an attempt to explain why patients with certain tendencies develop psychosomatic symptoms (Sifneos, 1973). Since its introduction, the term has rapidly gained recognition amongst practitioners and researchers alike (Weinryb, 1995). Current conceptualisations of alexithymia describe it cognitive deficit in the capacity to consciously experience emotional feelings in the context of autonomic activation indicative of emotional arousal (Lane et al, 2000). Knowing a patient’s level of alexithymia has significant implications for our understanding of health status, clinical presentation, behaviour, and responses to treatment (Lumley, Neely and Burger, 2007).

According to Taylor, Bagby and Parker (1999), alexithymia can cause a number of impairments in emotion processing, such as: difficulties identifying and describing feelings, problems distinguishing between feelings and physical arousal, constricted imaginary processes, and an externally oriented cognitive style. Alexithymia is thought to have a prevalence of 10% in the general population (ibid).

Alexithymia should not be seen as a dichotomous construct but rather a dimensional one, existing on a spectrum of severity, with people exhibiting different degrees of alexithymic traits (Ogrodniczuk, Piper & Joyce, 2011). Primary alexithymia is described as enduring, and non-changing, either as the as the result of neurological changes or entrenched defences that significantly impact on normal functioning (Taylor, 2010). Secondary alexithymia is a state-dependent form that develops in response to emotional trauma, acting as a mechanism whereby the individual represses emotional representations as a temporary defence against
further distress (Thompson, 2009; Freyberger, 1977). The high prevalence of Alexithymic features in traumatised individuals has been documented by a number of authors (Krystal, 2015, Yehuda et al, 1987, Berenbaum, 1996, Frewen, Dozois, Neufeld, & Lanius, 2008).

The connection between psychological trauma and alexithymia may explain why this disorder has been linked to Psychogenic Voice Disorders, which also linked to trauma and stress, in several studies, although the relationship between the two does not appear to be well explored in the literature.

**Alexithymia and Psychosomatic illness**

Stekel coined the term “somatization” in 1924 to describe the use of defence mechanisms for keeping intra-psychic conflicts, and their associated negative affects unconscious, while allowing for some expression through physical symptoms, and did not distinguish this from Breuer and Freud’s (2000) concept of conversion disorder, whereby motor and sensory symptoms developed as a result of hysteria (Marin and Carron, 2002).

Alexithymia has long been thought to contribute to the pathogenesis of psychosomatic illness (Nemiah, Freyberger and Sifneos, 1976, Nemiah, 1996, Berking and Wupperman, 2012), and there is a strong body of evidence that altered emotion processing and affect regulation are relevant factors in the development of ‘functional’ or psychosomatic symptoms (Taylor et al, 1999, Steffen, Fiess, Schmidt, & Rockstroh, 2015). The construct of Alexithymia has been linked to, and is currently being researched in relation to, a broad range of physical and mental illnesses, such as, eating disorders, somatization, mood disorders, psychosis, and various personality disorders (Dimaggio, Semerari, Carcione, Nicolo, & Procacci, 2007; Lumley, Neely, & Burger, 2007; Taylor, Bagby and Parker, 1999)

DeGucht and Heiser (2003) performed a meta-analytical review that indicated that
alexithymia was associated with a reduced ability to interpret somatic sensations as related to psychological distress, negative emotional states, and high levels of psychophysiological responses that lead to increased somatic discomfort and/or psychosomatic illness.

High levels of alexithymia are related to greater levels of functional symptoms, and a “difficulty identifying feelings” has been shown to have the single highest correlation with the number of identified symptoms (DeGucht and Heiser, 2003, Mattila, Kronholm, Jula, Salminen, Koivisto, Mielonen, & Joukamaa, 2008).

A 2014 study found that Alexithymia was present in 34.5% of patients with Functional Motor Symptoms (FMS), and 9.1% in those with organic movement disorders (Demartini, Petrochilos, Ricciardi, Price, Edwards, & Joyce, 2014). This represents a significantly higher prevalence in the FMS group, even after controlling for the severity of symptoms of depression. Other studies confirm that there is a clear independent relationship between medically unexplained symptoms (MUS) and alexithymia beyond the negative affect associated with the illness itself (Lumley, Neely and Burger, 2007, Mattila et al, 2008).

There is, therefore, an established link between alexithymia and psychosomatic illness. In relation to Psychogenic Voice Disorder (PVD) certain authors (Deary, Wilson, Carding & Mackenzie, 2003, Andersson & Schalen, 1998), have highlighted the presence of deficits in emotion processing. However, the overall significance of alexithymia in the formation of PVD is not well elucidated in the literature.

**Psychogenic Voice Disorder**

The human voice has been described as the “barometer” of emotion and is traditionally thought to give the listener an insight into the individual’s emotional state (Aronson, 1990, p.3, Seifert and Kolbrunner, 2005).
Voice disorders occur when the quality, pitch or loudness sounds different to from speakers of a similar gender, age, cultural context and geographic location (Stemple, Glaze, & Klaben, 2000, p. 85). Voice disorders are often associated with abnormal changes in the quality of a person’s voice, for example, hoarseness. Voice disorders can range from mild hoarseness to complete voice loss (Raming and Verdolini, 1998).

An important distinction between “organic” and “psychogenic” voice disorders is made in the literature. Organic voice disorders have a discernable physical cause, including but not limited to, structural abnormalities of the larynx, inflammation of the vocal cords, trauma, vocal cord paralysis, and benign or malignant lesions and tumours (Mathieson, 2013).

Where is there is a sudden or intermittent loss of volitional control over the initiation and maintenance of phonation in the absence of structural or neurological aetiology sufficient to account for the voice loss, the disorder is considered psychogenic in origin (Baker, 2013, Butcher, Elias and Cavalli, 2007, Aronson, 1990)

Such ‘non-organic’ dysphonias have been variously referred to as ‘conversion reactions’ (Breuer and Freud, 2000), ‘hysterical aphony or dysphonia’ (Neeleman and Mann, 1993), ‘psychogenic aphony or dysphonia’ (Baker, 1998, White, Deary, Wilson, 1997), ‘functional aphony or dysphonia’ (Wilson, Deary, and MacKenzie, 1995), ‘mutational falsetto’ or ‘puberphonia’ when observed in adult males (Froese and Sims, 1987, Woodson and Murry, 1994), and ‘muscle misuse or muscle tension disorders’ (Morrison and Rammage, 1994.). For the purposes of clarity, throughout this paper, the term psychogenic voice disorder (PVD) will be used, as it very clearly denotes that impairment is largely of psychological causation.

A differential diagnosis of psychogenic voice disorder should be established by experienced clinicians not only through exclusion of the presence of an organic aetiology (by direct or indirect visualisation of the vocal cords and their movements), but also by careful
It is thought that up to 80% of patients with PVD are female and this preponderance appears to be reflected in historical literature on the issue (Aronson, 1990 p. 144, Wilson, Deary & MacKenzie, 1995). Contemporary authors have sought to explain the high rate of PVD in women as due to variations in anatomy and vocal fold physiology, they are more likely to have vocally demanding occupations such as teaching, and are more vulnerable to a range of psychosocial difficulties that prompt them to seek help (Baker, Oates, Leeson and Bond, 2014)

**Alexithymia in Psychogenic Voice disorder**

There is a small but growing base of literature linking PVD to psychological factors within the patient such as depression, anxiety and specific traits, particularly neuroticism and alexithymia (Deary et al 1997, White at al 1997, Scott et al, 1997, Wilson et al, 2002). Studies also indicate that dysphonic patients have stronger histories of medically unexplained symptoms (or somatisation) than those in the general population (Deary et al 1997, Wessely et al 1999).

Deary, Wilson, Carding & Mackenzie (2003, p377) found a "striking” correlation between the severity of self-reports of dysphonia and degree of alexithymia, along with more emotional coping strategies, increased psychological distress, poorer quality of life, and a history of more unexplained medical symptoms. They describe voice disorder as a "symptom as much expressed as felt” (ibid, p377) and argue that the impact of the affective components of voice disorder have been recognised to some extent but are generally neglected in treatment plans.

Data published by Baker and Lane (2008), indicates that there is significantly more inhibition
or resistance to express emotion in women with PVD compared to those with organic voice disorder, that they tend to grow up in families with less emotional expression, and they display insecure attachment styles, supporting a link between alexithymia and the development of PVD.

Andersson and Schalen (1998) also support the case for the presence of alexithymia, stating clearly that the presence of PVD represents a deficit in “verbal emotional expression” (ibid, p.104) and have argued for the inclusion of communication skills training in management plans to help reduce the rate of relapse. Baker, Oates, Leeson, Woodford and Bond (2014) found that women with PVD had lower levels of emotional awareness, and those who reported lower physical and occupational demands on the voice scored higher on measures of alexithymia.

Deary and Miller (2011) agree that PVD is associated with multiple psychosocial factors, yet point out that these findings are shown to be true of other medically unexplained symptoms in which voice symptoms are absent, arguing that the notion that loss of voice is the symbolic representation of unexpressed emotion is appealing, but lacks scientific evidence.

**Chapter Summary**

A review of the available literature indicates that a link exists between alexithymia and both psychosomatic illness, and PVD. However, there is no clear consensus on the hypothesis that the development of vocal symptoms is related specifically to, or symbolic of, alexithymia. The next chapter will explore a number of theories on symptom formation in the hopes of adding further clarity.
CHAPTER 2: MODELS OF SYMPTOM FORMATION

Conversion disorders

The Freudian view of hysterical conversion disorders has arguably been more influential than any other in shaping our understanding of symptom development in psychogenic voice disorder (Butcher et al, 1993, Baker, 2002). Much of the earlier literature in relation to medically unexplained symptoms and conditions such as “psychogenic dysphonia” was understood against a background of Freud’s psychoanalytical model of the “hysterical” conversion reaction (Breuer & Freud, 2000). The psychoanalytical model of conversion reaction referred to a process whereby repressed or unconscious energy associated with instinctive sexual or aggressive instincts became converted into physical symptoms. The symptom was therefore a product of unconscious intra-psychic conflict that provided the primary gain of allowing the person to avoid the conflict that would have proven unbearable if brought into conscious awareness (Freud, 1905). The attention and illness role that might be adopted by the individual were thought to provide secondary gains, which served to reinforce the conversion reaction (Baker, 2003).

Freud published arguably his most important case study on a teenage girl, Dora, whose primary symptom was described as “hysterical aphonia”. Dora’s symptoms were viewed by Freud as a result of unconscious repression of a internal conflict regarding the advances of “Herr K”, and the role she played in the relationship between her father and “Frau K”, his mistress. Dora was not only repulsed by Herr K, but she was also excited sexually by him. Later, Freud questioned if Dora may have also had a sexual attraction to Frau K. Freud interpreted Dora’s voice loss as symbolic of her sense that when the person she loved was away, “speech had lost its value” for her (1905, p40) but also stressed that the interpretation
of the meaning of such symptoms would be different for each presenting case. Freud noted that although the primary mechanism for production of a hysterical symptom was psychological in nature, a certain degree of “somatic compliance”, or physical involvement, was also required (ibid, p40).

More recent authors have agreed that a symbolic relationship exists between the conversion symptom and the underlying conflict or threatening idea (Ziegler and Imboden, 1962, Viederman, 1996, Baker and Lane, 2008). Therefore the loss or disruption of voice might be symbolic of an alexithymic difficulty in verbalising emotional responses. Notably, the level of alexithymia in conversion disorder patients has been found to be higher than that of the healthy controls (Gulpek et al, 2014, Demartini et al, 2014).

**Modified conversion model**

Butcher and his colleagues (Butcher, Elias & Raven, 1993, Butcher 1995) have posited that in the majority of cases there is insufficient evidence for conversion reaction model of psychogenic voice disorder and suggested a reformulated psychoanalytic view of psychogenic voice disorders, which identifies two types (Butcher et al, 2007, p10). Type 1 cases are much rarer and demonstrate the features described in Freud’s hysterical conversion model, such as the use of unconscious repression of unacceptable instinctive desires and drives as a defence mechanism. Type 2 patients are distressed by their voice problem, and attempt to suppress emotion, which serves to create a conscious conflict regarding the expression or verbalisation of feelings (ibid). It could be argued that these two groups represent two different points of the same continuum, in that similar psychological processes are at play, the main difference between the two subtypes being their level of conscious
awareness of the conflict. Both groups convert the conflict into a vocal symptom, which appears be focused on the symbolic site of their psychological conflict.

**Trauma**

Severe and persistent psychogenic dysphonia may develop under innocuous circumstances, but at times can be traced back to traumatic stress experiences that occurred many months or years prior to the onset of the voice disorder (Baker, 2003). House and Andrews (1987) established that women with PVD very often experience difficulties in expressing negative affect specifically related to traumatic experiences. Janet’s (cited in Van der Kolk & Van der Hart, 1989, Van der Hart and Horst, 1989) model of conversion reaction emphasises that while recent stressful events appear to have precipitated symptom presentation, previously dissociated traumatic memories may also have been freshly triggered. In these cases, the original trauma must be explored and verbalised by the person for symptoms to resolve. Interesting, Freud (1919) supported Janet’s trauma theory relation to “war neuroses” in WWI soldiers with severe conversion reactions, many of whom presented with psychogenic aphonia, and were suffering from terrifying memories of war. Freud’s view in these cases, similar to current trauma theory, was that in the face of an inescapable threat, and an inability to use verbal expression as a coping mechanism, the individual’s normal psychic defences collapses (Freud, 1919).

Baker (2003) applies Janet’s model of dissociation and conversion to psychogenic voice disorders in order to explain how long forgotten traumatic experience can be triggered by more recent stressors. These traumas may be qualitatively embodied in the somatic symptoms, perhaps representing an experience of inability to cry out, fear of suffocation, or physical threat to the throat area (Baker, 2003). This approach ties in with current
perspectives in the field of trauma psychotherapy, that such patients react to reminders of the trauma with symptoms that are linked to the original threat (Van der Kolk, McFarlane and Weisarth, 1996). Taylor (2014) cites that alexithymia is common in trauma patients and that the area of the brain responsible for language, Broca’s area, is less active during reminders of trauma. Therefore, in some cases, the disordered voice could be the symbolic representation of an earlier trauma, combined with the inability to verbalise related emotions due to a trauma-related alexithymia. Recent research by Steffen et al (2015) supports the notion that adverse childhood experiences alter emotion processing, which in turn influences the severity of medically unexplained symptoms (MUS). They assert that such symptoms should be modelled as a trauma-induced ‘conversion’ of emotional stress responses into bodily symptoms (ibid, p140).

Levels of emotional awareness model

Lane and Schwartz (1987) developed a five-level model for the development of cognitive-emotional awareness, which has parallels to Piaget's model for intellectual development (Timoney and Holder, 2013). The five levels of emotional organization and awareness in the model are: awareness of physical sensations (level 1), action tendencies (level 2), single emotions (level 3), blends of emotions (level 4), and blends of blends of emotional experience (level 5, the capacity to appreciate complexity in the experiences of self and others) (Lane and Schwartz, 1987). The levels are hierarchically related, in that functioning at each level adds to and modifies function of the previous levels but does not eliminate them. Experience of emotion at level 1 is restricted to physiological reactions and sensations only. The fifth level of maturation ideally results in an individual who has fully developed the ability to identify, acknowledge, and communicate his or her own emotional states as well being able to recognise the emotional states of others with success (Lane and Schwartz, 1987,
In this context, alexithymia is viewed as occurring along a developmental spectrum subject to modification by the environment and personal experience, in which alexithymia “is not a distinct entity but represents one pole of a developmental continuum: the inability to put feelings into words may be a global trait, a circumscribed trait pertaining to emotions of a particular type, or a transient state” (Lane and Schwartz, 1987, p. 140)

In applying this model to PVD, Baker and Lane (2008, p150) argue that while such individuals are experiencing certain emotions at an undifferentiated, somatic level, placing them at the lower end of the developmental continuum, the individual with PVD could be operating at higher levels of emotional awareness at other times, or with regards to material unrelated to their disorder. This would support the case for a state-dependent alexithymia in the pathogenesis of PVD.

Neuropsychological Approaches

Recent progress in neuroimaging have allowed researchers (Kano and Fukudo, 2013, Moriguchi and Komaki, 2013, Laricchiuta et al, 2015) to use neuroimaging studies of people with alexithymia to support the “levels of emotional awareness” model (Lane and Schwartz, 1987). The presence of alexithymia correlates with weak responses in brain structures of cognitive-emotional processing (mediated by para-limbic structures and anterior cingulate cortex), potentially representing reduced function in more cognitively mature levels on the cognitive-developmental model (Kano and Fukudo, 2013). Alexithymia was also correlated with stronger responses in more primitive stages such as hypersensitivity to visceral pain, evidenced by greater brainstem activity.

Thayer and Lane’s neurovisceral model (2000) also supports the earlier cognitive-
developmental model in proposing that to become consciously aware of emotions requires a bottom up transfer of information from sub-cortical to cortical structures. Awareness of emotion at a cortical level mediates or inhibits sub-cortical and visceral activation, thus modulating emotional arousal and consequently leading to increased vagal tone (ibid). Taylor et al (1999) state that the mental representation of emotion assists in the moderation of duration and intensity of distress by allowing people to reflect and intentionally communicate emotional states in verbal form, which may help regulate responses. Therefore, if emotions are not adequately consciously processed, arousal is not modulated to the same extent, and as a consequence vagal tone may be reduced. As the larynx is supplied by vagal nerve innervation, this may have a special significance for PVD. Psychogenic voice disorder could be induced by alexithymia associated sympathetic arousal, attenuation of vagal nerve stimulation, or perhaps a combination of both responses (Baker and Lane, 2008, p154).

**Chapter Summary**

The theories described above appear significant in illustrating how a difficulty in the conscious experiencing of emotional arousal could lead to the development of a psychogenic voice disorder whether due to repression, suppression, trauma, dissociation, or cognitive-neuropsychological mechanisms. The alexithymic person's difficulty in identifying and describing feelings convert into chronic changes in physiological arousal due to lack of emotional expressiveness and modulation (Fiess, Rockstroh, Schmidt, & Steffen, 2015) which could lead to a deficit in vagal tone (Thayer and Lane, 2000). This might explain why people with PVD often experience constriction of the intrinsic and extrinsic laryngeal muscles, structures subject to vagal innervation. This constriction is known to lead to vocal quality changes or, in some cases complete voice loss (Aronson, 1990).
CHAPTER 3: DISCUSSION

Therapeutic Implications

Demartini et al (2014) state that considering alexithymia as a risk factor for the development of psychosomatic illness might help the clinician not only in the identifying the disorder and its underlying processes, but may also help the clinician communicate the diagnosis in a an appropriate way. This may be of particular relevance for people with PVD; in order to ensure that the role that emotion processing plays in the formation of their symptoms is explained in an accessible and sensitive manner.

Certain authors postulate that in all cases of PVD, unless treatment targets the reinstatement of the kinaesthetic model for normal phonation, dysphonia may persist, long after the precipitating conflict has passed (Rammage et al, 1987, Butcher et al, 2007, Aronson, 1990). Speech and Language Therapy intervention for psychogenic voice disorders receives generally favourable responses (Baker, 2002). Direct voice treatments have been successful in facilitating normal voice production, however, the reported rates of improvement using such approaches vary (Kent, 2004, p29). Although there is no consensus on the best techniques to use, a combination of direct and indirect approaches to voice therapy is supported by systematic review (Ruotsalainen, Sellman, Lic, Lehto, Verbeek, 2008). The long term effectiveness of voice therapy for this group has not been investigated and the overall rate of recurrence is not known. Clearly, if intervention has simply removed a symptom and not addressed the underlying emotional factors that led to the development of the issue, the chances of recurrence, development of new symptoms, or even breakdown in the face of removal of coping strategies, are increased (Tyron, 2014, Marmor, 2012, Andersson and Schalen, 1998). Clinical voice intervention may involve some psychological content, but in many cases specialised psychotherapeutic intervention is likely to be
necessary in achieving and maintaining improvements in emotional adjustment (Scott, Deary and MacKenzie, 1997, Butcher et al, 1993).

Alexithymia has negative implications for certain psychotherapeutic approaches, particularly those focusing on insight, emotional awareness and a close therapeutic alliance (Lumley, Neely and Burger, 2007, p240). The impact that alexithymia may have on the therapeutic interchange is arguably one of single most important factors responsible for the lack of success in psychotherapy regardless of therapist orientation (Krystal, 1982-1983, Roth and Fonagy, 2004). The severity of alexithymia is directly related to inferior treatment outcomes in both psychodynamic and supportive approaches (Grabe et al., 2008; Taylor et al., 1999).

Moriguchi et al (2006, p1480) found that alexithymia was related to impaired mentalizing, which in turn was linked to impairment in the ability to take a perspective different from the self, a skill that appears to highly important to the comprehension of the mental states of both self and others, an important part of the therapeutic interaction (Vanheule, Vandenberg, Verhaeghe and Desmet, 2010, p359). Body focused psychotherapy has been proposed as a route to help patients with alexithymia and medically unexplained symptoms, to mentalize and express what they experience (Calsius, De Bie, Hertogen, Meesen, 2016, p255).

Alexithymic patients often relate to others in a distant manner (Vanheule, et al 2007) and negative reactions and counter-transferences have been reported by therapists (Rasting, Brosig & Beutel, 2005, Vanheule, Verhaeghe, & Desmet, 2011) which may further impact on patient outcomes (Ogrodniczuk, Piper, & Joyce, 2011). Although it has been argued that due to the nature of alexithymia, these patients do not benefit from psychoanalysis (Sifneos, 1983; Nemiah, 1973, 1975; Nemiah, Freyberger, and Sifneos, 1976), Vanheule et al (2011, p91) propose that contemporary psychodynamic psychotherapy with alexithymic patients could be successful, if therapy addresses two underlying processes: problems in developing
accounts of one’s own experiences of arousal, due to which arousal remains present as bodily distress; and inability to manage distress using interpersonal relating and communication.

Baker (2003) suggests that in cases of psychogenic voice disorder where traditional approaches to facilitating normal voice are unsuccessful, and therapeutic focus on conflict over speaking out events does not lead to resolution, then the answer may lie in traumatic events experienced earlier in life. There is a growing body of research that shows the importance of personal history, attachment, and trauma where deficits in affect regulation and MUS are present (Van der Kolk, 1994, 2014, Taylor et al., 2000, Waller and Scheidt, 2006). Trauma focused psychotherapy, may provide a suitable model for some individuals with alexithymia and PVD, as non-verbal communication plays a significant role in establishing the therapeutic alliance, and the development of therapeutic narrative often starts at a somatic level (Taylor, 2014).

In contrast to other modalities, alexithymia may not represent a barrier to the success of more structured cognitive-behavioural treatments (Lumley, Neely and Burger, 2007, p240) and these approaches have also been used successfully in some cases of PVD (Butcher et al, 2007, p170).

According to Weiner and Craighead (2010), preliminary empirical and clinical reports indicate that modified group psychotherapies such as hypnosis, art therapy and meditation can help improve emotion processing in alexithymic patents. These, and other alternative routes for therapy such as therapeutic writing (Pennebaker, 1997, Pennebaker & Seagal, 1999, Baikie & Wilhelm, 2005) might yet prove beneficial if utilised in cases where the presence of alexithymia and PVD circumvents the use of more traditional verbal approaches.

Far from attempting to offer a homogeneous approach to treatment for people with PVD, and therefore neglect to integrate important individual or familial variations, this chapter
demonstrates that while there are therapeutic implications associated with alexithymia, and highlights an array of therapies exist that can be drawn upon, integrated and individualised as appropriate. Considering that there are degrees of both overlap and discord between the various theories of symptom development in PVD, the psychotherapist should be aware that both the presenting symptom and the significance of alexithymia may be different in each individual case.

**Conclusion**

A link between alexithymia and psychosomatic illness has been identified and researched in relation to many health problems, and although alexithymia appears significant to symptom development in psychogenic voice disorders, its exact mechanism can only be hypothesised at this time.

Several models for symptom formation in PVD have been presented. Although obvious differences exist, there appears to be a degree of overlap evident across all of these models, in that the symptom is commonly viewed as a consequence of failure to adequately express emotionally arousing material, through various mechanisms such as repression, suppression, lack of emotional awareness or dissociation. Freud’s model of conversion disorder remains influential, if suffering from lack of empirical validation (Baker, 1998). Butcher et al (2007, p10) have modified the traditional theory of conversion disorder to include two different but related subtypes of PVD: the classical conversion model, and a cognitive-conversion model that they argue reflects the profile of many contemporary patients with PVD. Baker (2003) argues that trauma may be a factor in some cases of PVD and that an updated model of conversion reaction might be strengthened if it better accommodated earlier traumatic events and dissociation. Modern neuroimaging techniques give weight and coherence to the cognitive developmental models of symptom formation, which helps account for the presence
of alexithymia in PVD as a part of a pervasive personality trait, a specific difficulty in processing particular emotional stimuli, or a transient state (Schwartz and Lane, 1987).

In conclusion, the hypothesis that alexithymia plays a role in the formation of symptoms in psychogenic voice disorder could be supported by a number of different theories. Although no single exact mechanism of symptom formation has been elucidated, it remains a pertinent issue for the management of such disorders. At this time it is unclear why one person with alexithymia develops a psychogenic voice disorder, and another person develops a different psychogenic symptom complex, such as chronic pain. It has long been advanced that the symptom is often symbolic of the underlying conflict not only in relation to unconscious conversions but also trauma. Therefore, alexithymia may have a symbolic relevance to PVD which is unique to the individual person concerned, yet this may only become meaningful in the context of a complex personal history of each client (Horwitz, 2002, p.46).

Understanding the role that alexithymia might play in PVD not only helps us to hypothesise potential routes for the genesis of symptoms, but also has implications for the selection of appropriate clinical pathways for each individual patient. The emotion processing deficits associated with alexithymia are reported to have implications for the therapeutic relationship and treatment approach, with some modalities potentially being contraindicated in this population (ref).

It is important that both direct voice therapy and psychotherapeutic interventions are both included as part of a holistic treatment plan in PVD. Not only does the underlying psychological distress need to be addressed, but it is crucial that the normal kinaesthetic model for voice production be facilitated and reinforced by experienced voice practitioners, particularly in cases of long standing PVD (Aronson, 1990, Butcher et al, 2007).

**Future Directions**
Research has not yet been able to offer an explanation of this phenomenon of gender bias in PVD (Baker, 2002), and this may be an area for further exploration in relation to sex specific styles of emotion processing. Future research may help shed light on the role of alexithymia in people with PVD through the use of neuroimaging techniques to explore if there are observable changes in neurobiological activity in response to treatment. Similarly, as vagal tone has been implicated in the disorder (Baker and Lane 2008, p154), this may be an area for further investigation. If measured, comparisons might be made to clarify if vagal tone changes in people with PVD after successful treatment.
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