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An auditory-neuroscience perspective on the development of selective mutism

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ABSTRACT

Selective mutism (SM) is a relatively rare psychiatric disorder of childhood characterized by consistent inability to speak in specific social situations despite the ability to speak normally in others. SM typically involves severe impairments in social and academic functioning. Common complications include school failure, social difficulties in the peer group, and aggravated intra-familial relationships. Although SM has been described in the medical and psychological literatures for many years, the potential underlying neural basis of the disorder has only recently been explored. Here we explore the potential role of specific auditory neural mechanisms in the psychopathology of SM and discuss possible implications for treatment.

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Selective mutism (SM) is characterized by consistent failure to speak/vocalize in specific social situations (e.g., at school) despite the ability to speak normally in other situations (American Psychiatric Association, 2013). SM typically involves impairments in social and academic functioning. Reported complications include school failure, social difficulties in the peer group, and aggravated intra-familial relationships (e.g., Bergman et al., 2002; Cunningham et al., 2004; Steinhausen and Juzi, 1996).

In the early 90s Black and Uhde (1992, 1995) reported an overwhelming incidence of avoidant disorder or social phobia in children with SM. These authors argued that SM should thus be treated as an extreme manifestation of

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social phobia. Although this position has been challenged by data indicating that parents, teachers, and clinicians do not necessarily report greater social anxiety in children with SM compared with children with social anxiety (Manassis et al., 2003), and that children with SM do not report greater social anxiety than children with social phobia alone (Yeganeh et al., 2003), the high comorbidity between SM and social anxiety has still shaped clinical practice to a great extent (Viana et al., 2009).

Notably, although many children with SM display shy temperament and social anxiety, only a very small portion of socially anxious children meet DSM diagnostic criteria for SM. This suggests that SM may involve a unique component that is absent in typical manifestations of social anxiety disorder. Thus, other factors, perhaps more directly associated with the core symptom of SM and their potential effect on speaking behavior should be considered. In the current opinion-report we describe evidence for the involvement of sub-optimal function of the auditory efferent system in the psychopathology of SM. First, we briefly describe findings delineating associations between auditory efferent activity and vocalization. We then describe a series of studies from our laboratory providing evidence for efferent aberrations in SM. Finally, based on the auditory aberrations discovered we discuss potential implications for the development of alternative treatments for children with SM. Importantly, the current report is not intended to provide a comprehensive review of the SM literature but rather to provide a neuroscience perspective on one of its potential neural generators.

1. The role of auditory efferent activity in vocalization

Because a specific inability to produce speech in certain circumstances is the hallmark of SM, it makes sense to consider possible anomalies in the neural mechanism supporting this specific behavior. Self-monitoring of one's own voice has a vital role in the development and enduring maintenance of vocalizations in both humans and animals (Oller and Eilers, 1988; Doupe and Kuhl, 1999). Continuous transaction between speech and hearing mechanisms (Curio et al., 2000; Borg et al., 2009; Ventura et al., 2009) enables constant monitoring of the quality of voice and speech, perception of external sounds while vocalizing, and prevention of desensitization due to possible overstimulation by self-vocalization (Hoy, 2002). Aberrations in auditory feedback induced via experimental manipulations such as the presentation of background noise (e.g., Lombard effect) or delayed auditory feedback result in significant alterations in vocalization in humans (e.g. Lee, 1950; Lamprecht, 1988) and animals (Osmanski and Dooling, 2009).

In humans, two distinct efferent mechanisms are known to be involved in monitoring and regulating vocalization: the middle-ear acoustic reflex (MEAR) and the medial olivocochlear bundle (MOCB) reflex. The neural circuit of the MEAR controls the contraction of the stapedius and tensor-tympani middle-ear muscles upon presentation of loud low-frequency sounds. This results in stiffening of the ossicular chain and subsequent attenuation of sound (Borg and Counter, 1989). When the MEAR is activated by self-vocalization, it is assumed to produce an anti-masking effect by attenuating potential overloading of the cochlea and thereby maintaining a fairly constant level of sensitivity that prevents interference by the speaker's own voice (Curio et al., 2000). Furthermore, activation of the middle-ear muscles during vocalization has been allocated an important role in reducing distortion, nonlinearities, and upward spread of masking (Borg and Zakrisson, 1975).

The sound-evoked MOCB reflex originates in the medial portion of the superior olivary complex on both sides of the brainstem and is activated via myelinated fibers that project directly onto the outer hair cells in the cochlea (Guinan, 2006). The functioning of the MOCB can be tested non-invasively in humans by means of contralateral suppression of otoacoustic emissions (Collet et al., 1990). Contralateral acoustic stimulation can attenuate, through fibers of the MOCB, the acoustic energy generated by outer hair cells activity and can be measured in the ear-canal (Guinan, 2010). The functional significance of the MOCB reflex is still debated. Most of the research regarding MOCB function during vocalization has been conducted in animals. Data from the singing cricket (Poulet and Hedwig, 2002) and mustached bat (Goldberg and Henson, 1998) suggest that during self-vocalization inhibitory activation of the MOCB takes place. In the singing cricket, for example, intercellular recordings indicated that presynaptic inhibition of auditory afferents and postsynaptic inhibition of an interneuron occur in phase with the song pattern. The authors postulate that inhibitory action decreases the auditory interneuron's response to self-generated sounds, and thus reduces self-induced desensitization (Poulet and Hedwig, 2002).

In humans, the functional role of the MOCB during vocalization is not fully understood (Robertson, 2009). Recent evidence suggest that MOCB feedback protects the ear from noise-induced cochlear damage caused by exposure to moderate sound intensities similar to those created by vocalizations in various natural environments (Maison et al., 2013). These authors propose that chronic self-stimulation by vocalization may present a significant damage risk to the ear without protection from efferent feedback, a hypothesis supported by the notion that the MOCB reflex is activated in anticipation of vocalization (Suga and Jen, 1975; Xie and Henson, 1998). The MOCB reflex has also been shown to play an anti-masking role in normal hearing subjects during signal detection/perception in background noise. For example, activation of the MOCB reflex improved threshold detection and intensity discrimination of tones in noise (Micheyl and Collet, 1996; Micheyl et al., 1997), and enhanced perception of speech in background noise (Messing et al., 2009; Brown et al., 2010; Kumar and Vanaja, 2004; Giraud et al., 1997). Altered pre-neural amplification via outer hair cell activity that leads to an increase in signal-to-noise ratio for certain frequency bands has been suggested as a potential underlying mechanism for such improvements (Cooper and Guinan, 2006).

Valuable information regarding the functional role of MEAR and MOCB during vocalization may be gained by studying clinical populations that exhibit aberrations in