Aberration in hearing one’s own voice can cause not only stuttering but also depression

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SUMMARY

The etiology of depression has not yet been defined with certainty. The successful use of botulinum toxin recently to treat depression has brought to attention the theories which connect emotions with facial muscles. According to facial feedback hypothesis, facial muscles not only express emotions but also cause and/or alter them. It has been long suggested that a disturbance in the process of hearing one’s own voice can negatively affect the functioning of vocal muscles, and I suggest that the negative effect can extend to facial muscles leading at the end to depression. Using certain methods to restore the normal function of both vocal and facial muscles can hypothetically be a new treatment for depression.

Introduction

In community-dwelling US adults, major depression has a 12-month prevalence of 6.7% with rates for women about two times higher than those for men [1,2]. Psychosocial factors which can cause normal sadness can also trigger depression but their effect is not invariably equal on all people, and not in all cases of depression a preceding stressful life event can be defined, which led many scientists to believe that biological factors are also involved in the etiology of depression [3]. However, the clinical heterogeneity of depression disorders may preclude the possibility of finding a single biological defect, and it seems that a number of factors from different areas interact together to create the syndrome. Hence, my review below will include a discussion of some of the popular hypotheses related to the etiology of depression.

My particular interest is the relationship between vocal function, hearing, facial expression and mood in patients with depression. Facial expression is not only an indicator of emotional state but it can also, as I will show below, affect emotional state via sensory feedback. This has been shown in studies where facial expression is manipulated experimentally [7]. Thus a sad expression may induce feelings of sadness while a happy expression may induce feelings of happiness. Facial muscles are highly activated during speech, both for articulation and for non-verbal communication of emotional content of the message. The close coordination between voice, face and emotional state suggests that manipulating voice might be a way to influence facial expression and thus mental state in disorders such as depression.

Monoamine hypothesis

Monoamine impairment is known to be a manifestation or a correlate of depression [3], and this hypothesis says that depression is caused by an absolute or relative deficiency of monoamine transmitters in the brain. Many currently available pharmacotherapies that relieve depression or cause mania, or both, enhance monoamine activity. One of the foremost classes of drugs for depression, SSRIs, for example, boost the level of serotonin in the brain. The line of research in this area was initiated by the discovery many years ago that reserpine, a medication for hypertension, inadvertently caused depression. It did so by depleting the brain of both serotonin and the three principal catecholamines (dopamine, norepinephrine, and epinephrine). Such findings led to the “catecholamine hypothesis” and the “indoleamine (i.e., serotonin) hypothesis,” which in due course led to an integrated “monoamine hypothesis” [3]. My proposed hypothesis of auditory etiology for depression can actually be seen in consistency with the monoamine hypothesis, as one study has shown that serotonergic function is located predominantly in the primary auditory cortex [4], while another study showed that the primary auditory cortex had the highest concentration of cortical serotonin and the highest synthesis rate [4].

HPA axis and CRH

Stress is known to have a damaging effect on hypothalamic–pituitary–adrenocortical–axis (HPA axis), and depression can be the outcome of severe and prolonged stress [3]. Many aspects of the acute stress response are exaggerated, persistent, or dysregulated in depression [3]. Corticotropin releasing hormone (CRH) is the neuropeptide that is released by the hypothalamus to activate the pituitary in the acute stress response, and is found to be...
hypersecreted in depression [3]. CRH injections into the brain of laboratory animals produce the signs and symptoms found in depressed patients, including decreased appetite and weight loss, decreased sexual behavior and sleep, and other changes [3]. Furthermore, CRH is found in higher concentrations in the cerebrospinal fluid of depressed patients [3]. In autopsy studies of depressed patients, CRH gene expression is elevated, and there are greater numbers of hypothalamic neurons that express CRH [3]. A study found differences in dysregulations of the stress response between melancholic and atypical depression and suggested that studies of depression should examine each subtype separately [15]. In regard to the therapeutic implications of this hypothesis, a study on 20 patients with depression has shown that a CRH antagonist significantly reduced depression and anxiety scores, in addition to worsening of affective symptomatology after drug discontinuation [16]. However, a recent review notes that many issues remain to be addressed before agents which intervene with HPA axis function become available for clinical use as it is not yet clear whether these agents will have improved clinical profile in comparison to currently used drugs [17].

**Omega-3 fatty acids**

Lipids constitute 60% of the solid mass of the brain and are required for normal brain structure and function [5]. Compared with healthy control subjects, plasma and red blood cells from depressed patients show absolutely low levels of omega-3 fatty acids [5]. A strong inverse relationship exists between the consumption of omega-3 fatty acids in a population and the prevalence of both major depression and postpartum depression [5]. Treatment with ethyl-eicosapentaenoate, an omega-3 fatty acid, was effective in treating depression in patients who remained depressed despite adequate standard therapy [5]. However, a recent meta-analysis has concluded that despite the significant antidepressant efficacy of omega-3 fatty acids, it is still premature to validate this finding due to publication bias and heterogeneity; and noted the need for more large-scale, well controlled trials to find out the favorable target subjects, therapeutic dose, and the composition of omega-3 fatty acids in treating depression [18].

**The role of genetics**

Studies have established that depression run in families [3]. Numerous investigators have documented that susceptibility to a depressive disorder is twofold to fourfold greater among the first-degree relatives of patients with mood disorder than among other people [3]. In studies of monozygotic twins reared separately (“adopted away”), the results revealed an increased risk of depression compared with controls [3].

**Psychosocial factors**

Many episodes of depression are associated with some sort of acute or chronic stressful life event [3]. The compelling impact of past parental neglect, physical and sexual abuse, and other forms of maltreatment on both adult emotional well-being and brain function is now firmly established for depression [3]. However, it might be possible that in cases when negative feelings are out of proportion to the events attributed to them consciously or subconsciously, biological factors have had an impact on the size of the problem. One of the possible biological factors, as I will show in detailed discussion below, is a change in the tone and activity of certain facial muscles.

**Cognitive factors**

There is now ample evidence that the impact of a stressor is moderated by the personal meaning of the event or situation [3]. Heightened vulnerability to depression is linked to a constellation of cognitive patterns that predispose to distorted interpretations of a stressful event [3]. According to one model of cognitive vulnerability to depression, negative cognitions by themselves are not sufficient to engender depression. This model postulates, on the basis of previously gathered empirical evidence, that interactions between negative cognitions and mildly depressed mood are important in the etiology and recurrences of depression. Patterns or styles of thinking stem from prior negative experiences. When they are activated by adverse life events and a mildly depressed mood, a downward spiral ensues, leading to depression [3].

**The role of facial muscles**

A recent study [6] has shown that injecting the frown muscles with botulinum toxin was effective in treating depression. Botulinum toxin is thought to exert its effect peripherally through paralyzing muscles for a period of 2–4 months. Nine out of 10 patients included in the study, reported absence of depression after two months of receiving treatment, and the 10th patient reported improvement. The author concluded that as patients became no more able to frown, their ability to feel sadness was diminished. This study has a number of limitations including self-reporting depression scale, limited follow-up, lack of randomization, the absence of blind evaluation, the absence of control group, and especially the small number of individuals included.

However, the theoretical model which has been used to explain the effect of botulinum toxin on mood is not a new one, and has its roots back in the work of Piderit (1858), Gratiolet (1865), Darwin (1872) and James (1884), a concept which evolved about a century later into the facial feedback hypothesis (FFH) when Tomkins in 1962 gave a central role to facial muscles [7]. FFH suggests that facial muscles not only express emotions, but can also increase and/or initiate them through a feedback mechanism. Several studies have established the existence of a correlation between facial expressions and emotional experience [8]. Consistent with the above mentioned botulinum toxin study, increased activity in frown muscle corrugator was found to be associated with sadness, clinical depression and negative thinking [8]. Other studies have found modulating and initiating effects of facial muscles on mood [7]. Based on this understanding, the effect of botulinum toxin on mood has been explained to result from the toxin-induced lack of ability to frown, which, through interrupting feedback process, prevented patients from excessively feeling sad [6]. The assumption here is that the increased activity of corrugator muscle is a necessary component in the process of forming the excessive sad affect in patients with depression, so that when we paralyze this muscle we break the chain which produces the excessive sad feelings regardless of its etiology.

**Vocal-facial synchrony**

There is evidence that vocal and facial output is integrated. A study found that a rise in vocal frequency is associated with eyebrow movement during speaking – where the author also notes that speech production is always accompanied by facial and gestural activity [9]. Another study showed that facial motion which accompany speech process can be generated from speech acoustics, an evidence of correlation [10]. The fact that speech and facial gestures interact and cooperate to convey a desired message suggests that gestures and speech are controlled by the same...
internal control system [19]. Investigating the function of abnormal facial movements during stuttering, a study showed that they are likely due to an increase in physical tension of the speech related muscles [11]. The accessory activities of facial muscles that accompany speech disfluencies give the appearance of “speech-related struggle” and they are most visible during the blockages [20]. This synchrony between vocal and facial muscles during speech might mean that a disturbance of vocal function can cause a parallel disturbance of facial expressions. Theoretically, the resultant facial expression can take the form of sadness or, in other words, an increased activity of corrugator muscle. If this expression is persistent, it can virtually cause depression through facial feedback.

The role of auditory system

Voice is known to be controlled mainly through auditory feedback [12]. Stromsta [13] hypothesized that a disturbance in auditory feedback, namely the bone-conducted feedback, can be an etiological factor behind stuttering. A disturbance in the process of transmission of voice from larynx to inner ear can upset the function of vocal muscles, and as a consequence, I infer, the function of facial muscles because of the synchrony between vocal and facial muscles. Devices that produce delayed auditory feedback have been used for a long time as a successful treatment for stuttering [14]. Regardless of the etiology of stuttering, such a device has been shown to modify auditory feedback in a way that restructures the functioning of vocal muscles to restore fluency. I suggest that the effect of delayed auditory feedback can extend to facial muscles to restructure their functioning when it is applied, due to the aforementioned association between vocal and facial muscles. In parallel with its positive effect on speech process, its effect on the expression of emotion is expected to be a plausible one. This can in turn, through facial feedback, alter the emotional state in a positive direction.

The effect of auditory feedback alteration on facial muscles

Dewar et al. [21] has conducted a study which shed light on the effect of altering auditory feedback on facial muscles. He noticed while testing the effect of auditory feedback masking on 53 stutterers, that in many subjects abnormal facial movements appeared to be suppressed during auditory masking at a level which was effective in producing fluent speech. He followed with another study [21] designed to test this particular effect and found that in all eight stutterers abnormal movements of orbicularis oculi muscles concomitant with stuttering was abolished at the same time as the speech disfluency. He was also surprised by the finding that two ex-stutterers included in the study showed abnormal orbicularis oculi activity during apparently normal speech, which, however, ceased on the application of auditory masking. This finding might indicate that if fluency is achieved without modifying auditory feedback, the abnormal facial movements would continue to exist. Dewar concluded that speech and facial expressions are coordinated, and that factors which cause disharmony in speech are the same factors which cause abnormal facial movements [21].

Neurological correlates of auditory feedback distortion

The ability to recognize one’s own inner speech is essential for a sense of self. The verbal self-monitoring model proposes that this process entails a communication from neural regions involved in speech production to areas of speech perception. According to the model, if the expected verbal feedback matches the perceived feedback, then there would be no change in activation in the lateral temporal cortices [25]. In a meta-analysis on the neural correlates of picture naming [31] it has been suggested that regions were involved in the external loop of self-monitoring if they were reliably found to be activated in word listening tasks and were more strongly activated in experiments involving overt responses. This was the case for the bilateral superior temporal gyri (STG). More direct evidence on the areas involved in processing verbal feedback comes from studies, which manipulated auditory feedback. Here, authors assumed that the modulation of verbal feedback engages the self-monitoring process more strongly. For instance, in a positron emission tomography (PET) study [32], in which participants read aloud single words, increased activity was reported in the right STG, and a weaker similar left-sided activity when feedback was modulated by pitch distortion or by playing the voice of the

The role of facial muscles in emotional perception

A known feature of depression is the deficit in the area of emotional perception [22]. A popular hypothesis says that facial mimicry (also called imitation) is a necessary step in the process of emotional perception (also called emotional empathy) [23,24,30]. Facial mimicry has been extensively studied in psychophysiology, particularly with the aim to understand its links with empathy and emotional contagion or to determine an objective marker for empathy [27]. Facial mimicry is the spontaneous tendency of the subject to mimic the emotional expression seen in another face. This reaction occurs relatively quickly, within 500 ms after the stimulus onset, is unconscious, and independent of voluntary control, as it can still be observed when the subject is asked to inhibit any facial movement or even to react with incongruent facial movements [27]. A recent review on the neural correlates of imitation suggested that there is a core circuitry of imitation comprising the superior temporal sulcus and the ‘mirror neuron system’, which consists of the posterior inferior frontal gyrus and adjacent ventral premotor cortex, as well as the rostral inferior parietal lobe [28]. Furthermore, imitation as a form of social mirroring (in contrast to imitative learning) is supported by interaction of the core circuitry of imitation with the limbic system [28]. The limbic system is critical for emotional processing, and empathic individuals exhibit non-conscious mimicry of the postures, mannerisms, and facial expressions of others to a greater extent than non-empathic individuals [29]. In one study [24] aiming at examining how facial mimicry behavior in “face-to-face interaction situations” is related to individual differences in emotional empathy, seventy participants (36 men, 34 women) participated, with a median age of 22 years (range 19–35 years). Nine participants were excluded and 61 remained. Subjects were exposed to pictures of angry or happy faces, and electromyographic (EMG) activity was measured for Zygomaticus Major as an indicator of positive affect, and Corrugator Supercili as an indicator of negative affect. Subjects were then divided into low-empathy group and high-empathy group according to Questionnaire Measure of Emotional Empathy (QMEE). A significant difference in facial mimic reaction between high- and low-empathy participants emerged at short exposure times (56 ms), representing automatic, spontaneous reactions, with high-empathy participants showing a significant mimicking reaction. The low-empathy participants did not display mimicking at any exposure time. Based on this proposed connection between facial mimicry and emotional empathy, I suggest that the known overactivity in certain facial muscles in patients with depression might be the factor which disturbs the process of emotional perception in these patients. Without normal tone and responsiveness of facial muscles, they would not be able to properly imitate perceived expressions, and so the ability of the subject to empathize would be impaired.
experimentor rather than the participant’s own voice. An fMRI study [25] on 13 healthy subjects reported increased STG activation when auditory feedback was a distorted version of their voice, relative to the conditions of hearing their own voices undistorted.

On the other hand, a meta-analytic study of changes in brain activation in depression [26] reports that one of the most consistently identified regions is STG which shows decreased activation in patients with depression. This finding could be a result of a chronic overstimulation due to chronically hearing distorted own voice, ending up in a state of insensitivity (i.e. decreased activity) as an adaptive change.

A possible misconception

I expect that many misconceptions will arise around this hypothesis. One of the possible misconceptions is that I am saying that stuttering and depression share the same exact cause. This is simply not accurate. Actually, aberration of auditory feedback can take unlimited number of forms. Part of auditory feedback is performed with sound waves traveling from vocal folds to inner ear. In the acoustical terms, an aberration in this pathway might be called distortion of side tone. This is a distortion of sound waves which can take different shapes, some of which causes mere vocal problems, while others cause an effect which extends to facial muscles leading to emotional problems as explained above.

On the other hand, I am not trying to say that side tone distortion is the sole cause of stuttering or depression. Especially in the case of depression, central factors which affect brain or peripheral factors which affect facial muscles can hypothetically cause depression in the absence of side tone distortion.

Implications

This hypothesis suggests that a modulation of auditory feedback can treat depression in a subset of cases. The ideal target would be full restoration of normal auditory feedback. However, where this is not possible, patients might benefit from any modulation technique that restructures the function of vocal system in a positive direction, like delayed auditory feedback. A simple amplification of auditory feedback using a hearing aid can theoretically provide a competing input which is closer to the normal auditory feedback, and would hypothetically treat depression. It must however be noted that, when using auditory therapeutic methods, some chronic pathological changes in vocal and facial muscles might need a longer time to be reversed. At the muscular area it might be useful to use botulinum toxin or another treatment modality concomitantly with auditory modulation.

Another possible implication is providing a better understanding of why and how botulinum toxin works when injected to frozen muscles in patients with depression.

Conclusion

Depression is likely to result from multiple causative factors, I am trying to suggest one of them. The function of hearing one’s own voice is not expected to be perfect in all people. An aberration in this function can theoretically upset vocal system and result in a parallel disturbance in the functioning of facial muscles. If it ends up in a deviation of conveying emotions, the muscular feedback process upon which emotions rely would be disturbed, which can give rise to unwanted emotions including depression. Restoring normal auditory feedback and reversing the chronic pathological changes of facial muscles can thus be therapeutic for depression.

Conflicts of interest statement

None declared.

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