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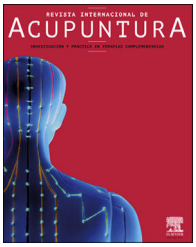

CONTINUING EDUCATION

A hypothesis about acupuncture's anxiolytic mechanism of action and its effect on the formation of self-efficacy beliefs and academic performance

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Abstract Pre-exam anxiety is highly prevalent among students of all educational levels, leading to decreased performance. This anxiety causes psychological and physiological changes related to stress, which can affect the formation of self-efficacy beliefs. Studies have shown a correlation between pre-exam anxiety, self-efficacy and academic performance. Acupuncture (AC) has an anxiolytic effect recognized in the treatment of generalized anxiety disorder and anxiety in the pre-operative and pre-exam period. Stress-related variables such as cortisol level, blood pressure and heart rate can be modulated by AC. Brain regions related to the control of the autonomic nervous system, emotional, memory and cognitive processing are also modulated by AC. Therefore, the aim of this study is present a hypothesis about how the AC reduces anxiety and stress through neuroendocrine modulation, optimizing the formation of self-efficacy beliefs, which promote behaviors responsible for improved performance.

PALABRAS CLAVE
Acupuntura;
ansiedad;
autoeficacia;
rendimiento;
exámenes académicos

Una hipótesis sobre el mecanismo de acción ansiolítico de la acupuntura y su efecto en la formación de creencias de autoeficiencia y rendimiento académico

Resumen La ansiedad previa a los exámenes es muy frecuente entre los estudiantes de todos los niveles educativos, lo que lleva a una disminución del rendimiento. Esta ansiedad provoca cambios psicológicos y fisiológicos relacionados con el estrés, que pueden afectar la formación de creencias de autoeficacia. Los estudios han demostrado una correlación entre la ansiedad previa al examen, la autoeficacia y el rendimiento académico. La acupuntura (AC) tiene un efecto ansiolítico reconocido en el tratamiento del trastorno de ansiedad generalizada y la ansiedad en el período preoperatorio y preexamen. Las variables relacionadas con el estrés, como el nivel de cortisol, la presión arterial y la frecuencia cardíaca, pueden ser moduladas por

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AC. Las regiones cerebrales relacionadas con el control del sistema nervioso autónomo, emocional, memoria y procesamiento cognitivo también son moduladas por AC. Por lo tanto, el objetivo de este estudio es presentar una hipótesis acerca de cómo la AC reduce la ansiedad y el estrés a través de la modulación neuroendocrina, optimizando la formación de creencias de autoeficacia, lo que promueve comportamientos responsables para mejorar el rendimiento.

Introduction

The anxiety related to academic tests, which is a type of situational anxiety, is considered a common problem among students of all levels.[1] This pre-exam anxiety often leads to physiological effects and mental symptoms that can negatively influence academic performance.[2] Studies estimate that between 15 and 22% of students have high levels of pre-exam anxiety,[3] reaching 65% in medical students.[4] Some research demonstrates a relationship between pre-exam anxiety and a number of negative variables, including increased risk of subsequent anxiety and depression,[5] difficulties in engaging with the content taught, and poor performance in the exams.[6] Studies have shown that anxiety decreases the formation of self-efficacy beliefs leading to decreased academic performance.[7,8]

From a therapeutic point of view, pharmacotherapy and psychotherapy are the conventional treatments for anxiety with the former being considered the standard treatment and with the latter being insufficient when used alone in most cases. Regarding pharmacotherapy, anxiolytics, antidepressants or monoamine oxidase inhibitors are used, with benzodiazepines being the most used pharmacological resource as anxiolytics.[9] However, these drugs can cause habituation, serious adverse effects and drug interactions.[10]

In this context, we hypothesized that the anxiolytic effect of AC[11] can increase the formation of self-efficacy beliefs and, consequently, increase the academic performance. One great advantage of AC on conventional treatments is that AC does not cause side effects in the vast majority of patients.[12]

Therefore, the aim of this study is to present the empirical data, which support our hypothesis, and discuss the implications for students submitted to AC treatment, if this hypothesis is confirmed in future studies.

Hypothesis

From a mechanistic point of view, the hypothesis is that the anxiolytic and anti-stress effect of AC on pre-exam anxiety may occur due to the decrease in cortisol production, via modulation of the hypothalamic-hypophyseal-adrenal (HHA) axis and by the regulation of brain regions involved in emotional, cognitive and memory processing, via modulation of salience network (SN), default mode network (DMN), limbic-paralimbic-neocortical network (LPNN) and memory network (MN). Thus, the decrease in the level of anxiety and stress facilitates the formation of self-efficacy beliefs, which in turn, modulates the behaviors responsible for the increase in academic performance (Fig. 1).

Foundation of the hypothesis

Pre-exam anxiety, self-efficacy and academic performance

In recent decades, some theoretical models have been formulated to explain pre-exam anxiety affecting academic performance. Among these, the interference model postulates that academic performance is reduced by anxiety, due to the disturbance caused by anxiety in the process of retrieving and using information.[13] In the attentional control model, anxiety affects performance through an adverse effect on attentional control.[14] From the point of view of neuronal networks, these changes postulated in the models mentioned above, occur in brain regions related to memory (hippocampus), emotional processing (amygdala) and control of executive functions (prefrontal cortex), which are regions related to anxiety psychopathology (described in the next topic).[15] In addition, it has been shown that anxiety activates sympathetic nervous system responses, such as, blood pressure, heart rate and cortisol level.[2] Cortisol, in turn, affects the brain regions responsible for the psychopathology of anxiety, increasing its level.[16]

These neuroendocrinal and psychological changes related to anxiety may decrease academic performance by means of decreasing of the self-efficacy beliefs, defined as people's judgment on their abilities to organize and execute courses of action necessary to achieve certain types of performance.[8,17] Studies has demonstrated correlation between anxiety, self-efficacy beliefs and performance.[7,8] In other words, these studies indicate that increased anxiety decreases self-efficacy beliefs that lead to poorer performance. This correlation is explained by the role of anxiety in the formation of such beliefs. According to literature, somatic and emotional states, such as anxiety, stress, excitement and mood states, provide information to create self-efficacy beliefs. That is, people's degree of confidence is affected by their physiological and psychological state. Thus, when self-efficacy decreases, initiation and perseverance in behaviors related to improve performance is compromised.[8,17]

Neural circuits of anxiety

In the previous topic we analyzed how anxiety affects the formation of self-efficacy beliefs and, consequently, academic performance. Now, we will analyze which neural circuits are responsible for anxiety symptoms.

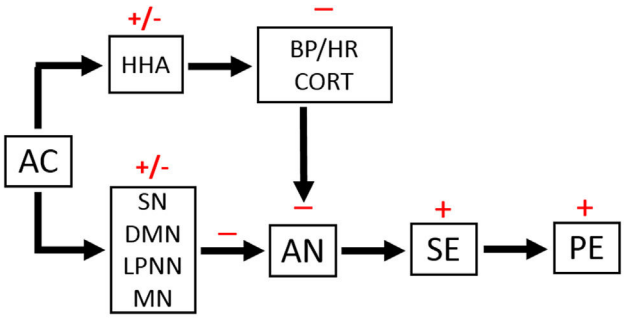

Fig. 1 Hypothesis about the mechanism of action of AC increasing academic performance.

According to studies of unpredictable shock threat and high-resolution functional magnetic resonance imaging (MRI), several brain areas are involved in the psychopathology of anxiety. The dorsolateral prefrontal cortex (dlPFC) regulates emotion and attention control and is related to anxiety.[18] When the individual is engaged in cognitive tasks that activate the dlPFC, this activation inhibits anxiety caused by the unpredictable threat of shock.[19] Patients with psychiatric disorders show hypoactivation of dlPFC during tasks that cause anxiety. Anxious patients also have a cognitive deficit due to low dlPFC activation, which impairs working memory.[20] These studies demonstrate that normal dlPFC activity is crucial in controlling pathological anxiety.

The dorsomedial prefrontal cortex (dmPFC) is involved in the pathogenesis of anxiety by means of regulating the conscious threat assessment and worry.[21] In addiction, the dmPFC is also involved, together with the anterior hippocampus, in consolidation and retrieval of memories.[22] It has been demonstrated a positive correlation between activation of the anterior hippocampus, measured with magnetoencephalography, and increased level of anxiety in humans.[23] Thus, dlPFC, dmPCF and the anterior portion of the hippocampus form a memory network (MN) that, when altered, contributes to the pathogenesis of anxiety. In addition to MN, the other brain regions mentioned above make up other functional brain networks, such as the salience network (SN) and the default mode network (DMN). Changes in these networks are also related to anxiety disorders, as mentioned below.

The cortical nodes of the SN are the dlPFC, ACC (anterior cingulate cortex), PCC (posterior cingulate cortex) and anterior insula. These nodes are activated by sensory stimuli that attract attention because they are unexpected, new or behaviorally relevant. Thus, the hyperactivation of these circuits are relevant to anxiety disorders due to their integral role in emotional, cognitive and behavioral self-regulation.[24]

The DMN is a resting state network responsible for an introspective self-referential state. This network is composed of the dmPFC, PCC, precuneus and medial and lateral parietal cortex.[25] A study with electroencephalography showed that individuals with high trait anxiety fail to synchronize DMN during the resting state, reflecting a possible deficit in top-down cognitive control. In these individuals, there was a decrease in connectivity between dmPFC and ACC/PCC/retrosplenial cortex. Furthermore, the strength of functional DMN connectivity was negatively related to the total score of Stait and Trate Anxiety Index (STAI-T).[25] In the next topic, we will see that AC modulate the brain areas cited, responsible for the pathogenesis of anxiety and stress.

Acupuncture in the treatment of anxiety

The AC has been used to treat various diseases for at least 2500 years in China. According to oriental medicine theory, AC is defined as the insertion of needles into the skin and underlying tissues, at specific locations known as acupoints, for curative or preventive purposes.[26] Systematic reviews have shown that AC has been used to control generalized anxiety in pre-operative and pre-exam conditions.[12,26] In these studies, in addition to the significant results in decreasing anxiety, there were no relevant side effects. Among the 360 points of AC, located in the main energy channels (meridians), the acupoints PC 6 *Neiguan*, HT 7 *Shenmen*, L 3 *Taichong* and EX-HN 3 *Yintang* are the points that the scientific literature indicates as the most effective in controlling anxiety.[12,26]

From a mechanistic point of view, the anxiolytic effect of AC can occur through the reduction of cortisol production,[27] via modulation of the HHA axis, and regulation of brains regions involved in cognition, memory, and emotion.[28] Numerous studies have shown that regions of the brain modulated by AC largely overlap with regions related to psychopathology of anxiety.[29] These studies, brought together in a meta-analysis of brain activities associated with AC stimulation, reveal activation in the sensorimotor cortical network (insula, thalamus, ACC and primary / secondary somatosensory cortex) and deactivation of the limbic-paralimbic-neocortical network (LPNN) composed of mPCF, caudate, amygdala, PCC and parahippocampus. This "LPNN" terminology was given by the authors for the network of brain regions involved in the AC response. The LPNN is composed of the main nodes of the DMN, amygdala, hypothalamus and nodes of SN (PCC) and MN (dmPCF). The activation and deactivation patterns cited in these studies suggest that hemodynamic responses in the brain simultaneously reflect sensory, cognitive and affective modulation after AC stimulation.

Future perspectives and conclusion

According to the literature reviewed, the pre-exam anxiety decreases the formation of self-efficacy beliefs and the

academic performance. AC acts on brain regions responsible for the pathogenesis of stress and anxiety, which is the possible anxiolytic mechanism of acupuncture. This emirical data support our hypothesis that the anxiolitic effect of AC has the potential to increases the formation of self-efficacy beliefs and academic performance. To confirm this hypothesis, future studies can verify the effect of AC on stress-related, psychological and performance variables. In these studies, the application of the correlation coefficient can establish positive correlations (eg, between self-efficacy and performance) and negative correlations (eg, between stress-related variables and performance-related variables). The neuroendocrine effect of AC can be verified by neuroimaging studies on the modulation of brain areas involved in the psychopathology of anxiety and by cortisol dosage as well. If this hypothesis is confirmed, AC can be used as part of a protocol that aims to improve physical and psychological well-being of students, and consequently, a better performance.

AC promotes the modulation (+/−) of the hypothalamic-hypophyseal-adrenal axis (HHA) and the decrease (−) of stress (BP/HR/CORT) and anxiety (AN). AC also promotes modulation (+/−) on the brain areas responsible for cognitive, emotional and memory processing (SN, DMN, LPNN, MN), leading to a decrease in AN. With the decrease in AN, there is an improvement in the somatic and emotional state, facilitating the development (+) of self-efficacy beliefs (SE). The increase in SE leads to behavioral changes that promote a better (+) performance (PE). BP: blood pressure; HR: heart rate; CORT: cortisol; SN: salience network; DMN: default mode network; LPNN: limbic-paralimbic-neocortical network; MN: memory network.

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Conflict of interest

"The Authors declares that there is no conflict of interest".

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