Chapter #27

ANXIETY DISORDERS

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ABSTRACT
In modern society characterized by conflicts and crises, almost every person experience anxiety which is most often characterized by a diffuse, unpleasant feeling of fear, accompanied by symptoms such as headaches, sweating, digestive discomfort, palpitations. Anxiety disorders are widely spread among the population, with a predilection for women in a ratio of 2:1 In most cases, anxiety disorders begin in childhood, becoming exacerbated in adulthood if not properly treated. Anxiety disorders differ from normal, everyday anxiety in that they involve anxiety that is much more intense (e.g., panic attacks), lasts longer (anxiety that persists for months or more, instead of fading after the stressful situation passes), or leads to phobias that affect your life. Being among the most common mental disorders that are associated with significant individual and social costs, this chapter aims at the theoretical and scientific approach to anxiety disorders.

Keywords: anxiety, fear, distress, panic attack, anxiety disorders, phobias.

1. INTRODUCTION

In the ICD-10 International Classification of Mental and Behavioral Disorders, it is specified that "for anxiety disorders, the dominant symptoms are variable, but include common complaints of constant nervousness, tremors, muscle tension, sweating, dizziness, palpitations and epigastric discomfort. The fear that the subject or a close relative of the subject will have an accident or will become ill is often expressed, along with a variety of other fears and bad premonitions". We live in an uncertain and effervescent world, in the true sense of the word, where the changes and certainty of tomorrow affect the population of the entire globe. Society is in a continuous and dizzying change, carrying the individual through a multitude of mental states, some of which cause emotional distress and even serious imbalances in his functionality. Thus, anxiety is an inevitable part of life in contemporary society and there are many situations that occur in our routine to which it is normal to react with anxiety. Anxiety is a signal that alerts us to a possible danger long before pain. Moderate anxiety accompanies the escalation of desire, while excess diminishes performance.

Anxiety disorders are associated with increased morbidity and are often chronic and resistant to treatment. They can be seen as a family of pathologies, having common aspects, but also important differences and include agoraphobia, panic disorder, social anxiety

disorder or phobia, specific phobia, and generalized anxiety disorder. (Sadock, Sadock, & Ruiz, 2014)

Thus, if for a long time, until the end of the 80s, anxiety was a subject of psychological and psychiatric research and a theme for scientific meetings, today it has become a problem that almost any individual in society can testify to. Moreover, it has become a really successful media topic. Media coverage mobilized many individuals to a more careful introspection on this issue.

Thus, certain questions inevitably arise: "What is the boundary between normal anxiety and pathological anxiety?", "What causes anxiety?", "How do anxiety disorders manifest themselves?" "What are the consequences of pathological anxiety?", "Is there more than one kind of anxiety?", "Do we have to treat all forms of anxiety?", "How?", "What are pathological anxiety disorders?"

Out of the desire to contribute to society's evolutionary approach by providing answers to these questions, the following chapter will address the theoretical and scientific aspects of anxiety disorders.

Anxiety disorders are differentiated from each other by the types of objects or situations that induce fear, anxiety, or avoidance behavior as well as by the associated ideation. Thus, although anxiety disorders tend to be frequently comorbid with one another, they can be differentiated by analyzing the types of situations that are feared or avoided and the content of associated ideas or beliefs (American Psychiatric Association, 2013). Anxiety disorders differ from normal fear and anxiety, specific to developmental stages, in that they are excessive or persist after the completion of the appropriate developmental stages. They also differ from transient fear or anxiety, often induced by stress, by their prolonged nature (for example, a duration of more than 6 months). The duration criterion represents a general recommendation, allowing a certain degree of flexibility, the duration being sometimes shorter for children (American Psychiatric Association, 2013).

Normal anxiety is characterized by a vague, diffuse, unpleasant feeling of fear. Often it is accompanied by symptoms such as stomach discomfort, tightness in the chest, palpitations, tremors, and dizziness.

It is necessary to differentiate anxiety from fear. Anxiety is a warning signal. It announces an imminent danger and empowers the person to take measures against that danger. Fear is also a warning signal, but it must be differentiated from anxiety. Fear is a response to an external, defined danger, while anxiety is a response to an undefined, vague, internal danger (Sadock et al., 2014).

2. BACKGROUND

Regular anxiety can be felt by every individual, but it is different from anxiety disorders that require specialized intervention and even medication treatment. Anxiety disorders are classified as follows: separation anxiety disorder; selective mutism; specific phobia; social anxiety disorder (social phobia); panic disorder; panic attack; agoraphobia; generalized anxiety disorder; substance/medication-induced anxiety disorder; anxiety disorder due to another medical condition; other specified anxiety disorder; unspecified anxiety disorder (American Psychiatric Association, 2013).

Anxiety was defined by as fear without an object, manifested by psychomotor restlessness, vegetative changes, and behavioral dysfunctions, having a character of potentiality, deforming the present experience in relation to the future perceived as hostile and predetermined as such (Tudose, Tudose, & Dobranici, 2011, p. 226).

Anxiety is common and contributes to the activation of the body's alert mechanisms. Together with fear and the instinct to run away, they constitute defense mechanisms in the face of danger and adaptation to new situations. Anxiety is the way of reaction of any person who feels threatened. When there is a potentially dangerous situation or such a situation is anticipated, the feeling of fear appears first. Fear triggers a biological mechanism in the body that leads to the secretion of adrenaline. Adrenaline is a hormone that alerts the whole body and prepares it for flight or fight, normal reactions in case of danger. Thus, the man becomes more alert, his heart beats faster and pumps blood with greater pressure, breathing becomes more alert, and muscles tense. Fear is the expression of a concrete situation, of an immediate threat.

There is a connection between anxiety and stress. An event is perceived as stressful or not depending on a person's psychological resources and coping mechanisms. All this involves the ego. The ego is an abstraction according to which a person perceives, thinks, and behaves being stimulated by internal and external stimuli. A person with a functional ego is in an adaptive balance with the external and internal worlds, but if the ego does not function correctly, the person will suffer from chronic anxiety. If the imbalance is internal, between the person's impulses and consciousness, or external, between the pressure of the external world and the person's ego, the lack of stability produces a conflict. There is also the possibility of a combination of these two imbalances, for example, employees who have very critical bosses are subject to impulses that must be controlled in order not to lose their jobs (Sadock et al., 2014).

Anxiety has 2 major components: the awareness of being scared or nervous and the awareness of the psychological sensations. The feeling of shame can increase anxiety, and the individual not wanting others to notice these reactions to him. Many people are amazed when they find out that those around them have not noticed that they have anxiety, or if they have, they have not appreciated its intensity. In addition to visceral and motor effects, anxiety affects perception, thinking, and learning. It often produces confusion, and distorted perceptions, not only regarding time and space but also regarding people and the significance of events.

These distortions can decrease the ability to concentrate, reduce memory, and decrease the ability to make associations.

An important aspect of emotions is their effect on the selectivity of attention. Anxious people tend to select certain aspects of the environment and ignore other aspects, these things making them feel entitled to consider the situation as dangerous (Sadock et al., 2014).

Pathological anxiety differs from usual restlessness or fear when it becomes permanent and can cause major disruptions in the individual's life, not being linked to a certain object or a certain situation, but being possible by only imagining a potential danger that does not exist in the immediate vicinity and is even unlikely to happen.

Anxiety affects the entire being of the individual. It is a physiological, psychological, and behavioral reaction at the same time. At a physiological level, anxiety can cause an acceleration of the heart rate, muscle tension, nausea, feeling of dry mouth, sweating, and feeling of suffocation. At a behavioral level, anxiety can paralyze the ability to act, express or experience everyday situations. The psychological impact translates into a subjective state of fear and embarrassment.

The etymology of the word anxiety comes from the Latin "anxietas" which means experience marked by agitation, insecurity, fear, and fright. Although it is attested in the dictionary since 1771, it is considered that the notion of anxiety was introduced for the first time by S. Kierkegaard, who presents anxiety as a thrill, a fear of something undefined and indeterminable, as opposed to the feeling of fear, in which the object is concrete and individual (Kierkegaard, 1998).

3. EXPLANATORY THEORIES OF ANXIETY

Anxiety is a complex psychological phenomenon and there are various explanatory theories of this concept. These theories are psychological (psychoanalytic, behavioral, existential theories) and biological theories: genetics, neurotransmitters (noradrenaline, serotonin, GABA).

In the psychoanalytic view, Freud connects the term "anxiety" with libido and explains anxiety through the frustrations of the libido and the prohibitions dictated by the "Superego". Anxiety is the danger signal addressed to the "Ego", that is the conscious personality; it is an emotional state with two distinct aspects: a specific note of discomfort and a motor determinant, both felt, and experienced by the subject (Freud, 2004). Anxiety is a preparation reaction for an effective confrontation with dangers and threats, a reaction whose place is at the level of the "Ego". Freud believes that anxiety, at its origin, is adaptive, requiring an intensive consumption of psychic energy, thus, when anxiety becomes chronic, when it manifests itself over long periods of time, it makes it necessary for individuals to use some means of managing it (Freud, 2004).

Nowadays, many neurobiologists continue to emphasize many of Freud's ideas and theories, such as the role of the amygdala. The amygdala serves the fear response without any reference to conscious memory and this underlines Freud's concept of an unconscious memory system for anxious responses. A shortcoming of considering anxiety as a disease and not a signal is that the real reason that led to anxiety can be ignored. From a psychodynamic perspective, the goal of therapy is not necessarily to eliminate anxiety, but rather to increase tolerance to it. This would allow the client to experience the anxiety and use it as a signal to investigate the inner conflict (Sadock et al., 2014).

Adler believes in his theory that there is a connection between anxiety and the inferiority complex. The individual who lives in this complex feels bad and unfit. Anxiety appears in connection with the need to restore the lost social emotion when the social environment puts certain tasks before the individual. Even when the task is very easy, one perceives it as a verification of his integrity, which leads to an emotional reaction and a strong tension during its solving (Adler, 1996). If the anxiety comes from the inferiority complex, then the person who relives it has an additional motivation that is not related to the current situation. Also, Adler (1996) believes that anxiety is determined by tasks that endanger the individual's self-esteem.

According to psychoanalytic theories, anxiety is a signal for the danger of penetration into the unconscious of unacceptable, repressed infantile desires of a sexual nature. If the defense mechanisms fail to neutralize the danger, anxiety arises. Psychoanalysts state that by perceiving anxiety as a disorder, the meaning of the signal is lost sight of and there is a danger of ignoring the underlying causes (Freud, 2004).

Neo-Freudian representatives give anxiety new meanings and dimensions. K. Horney believes that anxiety is the dynamic center of all neuroses, the main source of anxiety being, not sexual impulses (as Freud claimed), but hostile impulses. Horney uses the term anxiety as synonymous with fear, thereby indicating a relationship between anxiety and fear. She believes that both are actually emotional reactions to danger and both can be accompanied by physical sensations such as: shaking, sweating, and violent palpitations, which can be so strong that an instant and intense fright can lead to death. Anxiety is a proportional reaction in relation to hidden and subjective danger (Horney, 1998).

Although cognitive psychology and humanistic psychology have different conceptual frameworks, the approach to anxiety in these two currents is practically similar. From the perspective of cognitive and humanistic psychology, anxiety occurs in the event of a

collision with a new experience that does not correspond to human knowledge or representations and presents a threat. Cognitive and humanistic theories are of great importance because they go beyond psychoanalytic and learning theory to explain anxiety by introducing the cognitive pattern model. According to this view, the subject who manifests anxiety tends to overestimate the degree of danger of a certain situation and, at the same time, underestimate his ability and capacity to face that physical or mental threat perceived by him.

3.1. Biological Approach to Anxiety Disorder

Research into the biological etiology of anxiety disorders aims to identify a direct relationship between symptoms, mental syndromes, and brain activity, thus establishing the pathophysiological bases of anxiety. Anxiety disorders have in common some disorders of serotoninergic and noradrenergic neurotransmission of the hypothalamic-pituitary-adrenal and hypothalamic-pituitary-thyroid axes functioning as well as the response to lactate, CO2 and other anxiogenous substances (Udristoiu, Marinescu, Podea, & Dehelean, 2011).

Stimulation of the autonomic nervous system causes certain symptoms of anxiety, such as headache, diarrhea, tachycardia, and tachypnea. The autonomic nervous system of certain patients with anxiety disorder, especially those with panic disorder, shows excessive sympathetic manifestations and adapts slowly to repeated stimuli.

Regarding the neurotransmitters, animal studies have revealed 3 main neurotransmitters to be involved in anxiety disorders: norepinephrine (NE), gamma-aminobutyric acid (GABA), and serotonin.

Some of the symptoms experienced by patients with anxiety disorders, such as panic attacks, insomnia, and intramuscular tension, are characteristic of excessive noradrenergic function. The general theory regarding the involvement of norepinephrine in these pathologies is that the affected patients have a deficient regulation of the noradrenergic system with bursts of activity occurring at certain times. Noradrenergic neurons are located mainly in the locus ceruleus, and their axons project into the cerebral cortex, limbic system, spinal cord, and brainstem. Experiments on animals have shown that stimulation of the locus ceruleus leads to a fear response, while ablation of this area eliminates the fear response in animals.

Human studies have shown that in people with panic disorder, the use of beta-agonist drugs increases the severity and frequency of panic attacks.

The role of GABA in anxiety is best highlighted by the spectacular effect that benzodiazepines have in these pathologies, they increase GABA activity as they act on GABA type A receptors. Benzodiazepines with low potency are useful for the treatment of generalized anxiety disorder, while benzodiazepines with high potency are useful for the treatment of the panic disorder. These observations, together with several studies, have led to the hypothesis that certain patients with anxiety disorder have abnormal functioning of the GABA A receptor.

Numerous types of acute stress led to an increase in serotonin in the prefrontal cortex, amygdala, nucleus accumbens, and lateral hypothalamus. Interest in this direction was also stimulated by the fact that it was observed that certain serotonergic antidepressants have an effect on anxiety disorders, such as obsessive-compulsive disorder. The effectiveness of buspirone, a serotonin receptor agonist in the treatment of anxiety disorders also suggests the possibility of a link between serotonin and anxiety. Serotonergic neurons are located in the raphe nuclei in the rostral brainstem and project to the cerebral cortex, hypothalamus, and limbic system.

There is multiple evidence in the literature regarding the fact that psychological stress increases the synthesis and release of cortisol. Cortisol has the role of mobilizing and restoring energy reserves and contributes to increasing vigilance, attention, and memory. Excessive and sustained secretion of cortisol can have serious adverse effects, such as hypertension, osteoporosis, immunosuppression, insulin resistance, dyslipidemia, coagulation disorders, atherosclerosis, and cardiovascular disease. Alterations of the Hypothalamic-Pituitary-Adrenal Axis (HPA) have been demonstrated in PTSD. In patients with panic disorder, blunted adrenocortical hormone response to corticotropin-releasing factor has been observed in certain studies (Sadock et al., 2014).

Serotoninergic pathways (originating in the dorsal nucleus of the raphe), innervate the amygdala and the frontal cortex, facilitating avoidance behavior as well as escape behavior. The noradrenergic system (originating in the locus coeruleus) and the dopaminergic system sensitize autonomic activation and vigilance in response to a threat (Rang, Dale, Ritter, & Moore, 2003).

Structural studies, such as those of MRI or CT, have shown in certain patients the increase in the size of the cerebral ventricles (Engel, Bandelow, Gruber, & Wedekind 2009). In one study, the increase was correlated with the duration of benzodiazepine administration. Another MRI study highlighted a specific defect in the right temporal lobe in patients with panic disorder (Fontaine, Breton, Déry, Fontaine, & Elie 1990). Other imaging studies have highlighted abnormal changes in the right hemisphere, but none in the left hemisphere. These changes raise the issue that certain asymmetries could be important in the development of anxiety in certain people (Martin, Ressler, Binder, & Nemeroff 2009).

Functional brain imaging (fMRI) has highlighted in certain studies abnormalities in the frontal cortex, temporal and occipital areas of patients with anxiety disorders. One interpretation of these results is that certain patients with anxiety disorders may have structural and functional brain abnormalities (Sadock et al., 2014).

Genetic studies have produced strong evidence that certain genetic elements contribute to anxiety disorders. Heredity is a predisposing factor for this category of disorders. Almost half of the patients with anxiety disorders have one or more affected members. It is obvious that there is a connection between genetics and anxiety disorders, but these disorders are not only due to genetics. One study attributed anxiety disorders to an abnormality in the genes encoding the serotonin transporter. People with this variant produce less transporter and have increased levels of anxiety (Sadock et al., 2014).

The raphe nuclei and the locus coeruleus project especially in the limbic system and the cerebral cortex in combination with the results of imaging studies, have become the focus of most hypotheses regarding anxiety disorders (Morris, McCall, Charney, & Murrough, 2020).

In addition to being innervated by the serotonergic and noradrenergic systems, the limbic system also contains a high concentration of GABA A receptors. Two areas of the limbic system have been highlighted in many studies over time, namely the increase in activity in the septohippocampal pathway that can the cingulate gyrus, which is mainly involved in OCD, also leads to anxiety (McGovern & Sheth, 2017).

The frontal cerebral cortex is connected with the cingulate gyrus and the hypothalamus. and may be involved in anxiety disorders. The temporal cortex has also been implicated in anxiety disorders (Sadock et al., 2014).

Anxiety disorders are among the most widespread psychiatric pathologies. The National Comorbidity Study reported that one in four people meet the diagnostic criteria for at least one anxiety disorder. From an epidemiological point of view, the prevalence of

anxiety disorders is 30.5% in women and 19.2% in men. According to the statistics carried out by WHO globally, they indicate significant effects of the COVID-19 pandemic on the population, increasing anxiety disorders by 25.6% compared to 2019 (76.2 million new cases), with women and young people being the most affected categories.

Anxiety is accompanied by a multitude of changes at the biological, affective, cognitive, and behavioral levels: at the biological level - the changes induced by the imbalance of the vegetative nervous system dominate, with the predominance of the sympathetic; at the affective level – the person describes feelings of fear, immediate catastrophe, helplessness, and horror; at the cognitive level – maladaptive processing and informational contents lead to preferential processing of anxiogenic environmental stimuli, ignoring neutral or positive stimuli from an affective point of view - the existence of a discrepancy between what the person wants or must do and what he thinks he can do; at the behavioral level - the behavior of avoiding anxiety-provoking situations appears.

Among the specific manifestations of anxiety, the following categories can be mentioned:

• physiological manifestations: tremors, agitation, muscle tension, sweating, dizziness, palpitations, weakness, cold and wet hands, dry mouth, short and rapid breathing, hot flushes, or cold shivers, feeling sick, nausea, feeling of emptiness in the stomach, generalized fatigue, hyperventilation, insomnia;

• affective manifestations: mental tension, fear, nervousness, restlessness, irritability, and a permanent state of worry;

• cognitive manifestations: decreased ability to concentrate, exhaustion and mental tension, intellectual confusion, and mental discomfort;

• behavioral manifestations: avoidance, psychomotor agitation, disorganized activity, and the tendency to overcome the state of discomfort through defensive mechanisms.

It is not necessary that anxiety-specific changes to occur simultaneously at all four levels in a way that the person is aware of.

4. GENERAL INFO REGARDING THE CLASSIFICATION

4.1. Panic Attack

Panic attack is defined as a distinct state in which there is a sudden onset of feelings of fear, terror, and a feeling of impending disaster. These are associated with somatic symptoms (palpitations, chest pain, feeling of suffocation) and the fear of going crazy or losing control; A panic attack can occur in the context of any anxiety disorder, as well as in the case of other mental disorders (for example in depression, PTSD, substance use disorders, etc.) but also in case of medical conditions (cardiac, respiratory, etc.) the diagnosis of a panic attack can be established, according to the following symptoms:

1. Palpitations, pounding heart or accelerated heart rate.
2. Sweating
3. Trembling or shaking
4. Sensation of difficult breathing (dyspnea) or suffocation
5. Feeling of choking
6. Precordial pain or discomfort
7. Nausea or abdominal distress
8. Feeling dizzy, unsteady, light-headed, faint
9. Chills or heat sensations
10. Paresthesias (numbness or tingling sensations)

11. Derealization (feeling of no reality) or depersonalization (being detached from oneself)

12. Fear of losing control or "going crazy"

13. Fear of dying (p. 208)

The average age of onset of panic attacks in the US is about 22-23 years in adults. In preadolescent children, panic attacks are uncommon, and unpredictable panic attacks are rare. Risk factors are temperament factors (negative affectivity, anxious sensitivity) and environmental factors (smoking, interpersonal stressors, stressors related to physical well-being, and negative experiences) (American Psychiatric Association, 2013).

4.2. Separation Anxiety Disorder

It is a mental condition that is part of the anxiety disorders category and that manifests itself, according to the DSM-5 by an excessive fear and anxiety inappropriate for the developmental stage regarding separation from attachment persons, evidenced by at least three of the following:

1. Recurrent excessive distress when anticipating or experiencing separation from home or major attachment figures.

2. Persistent and excessive worry about losing attachment persons or about possible harm to them, such as illness, injury, disasters, or death.

3. Persistent and excessive worry about experiencing an event (e.g., getting lost, being kidnapped, having an accident, becoming ill) that could cause separation from the major attachment figure.

4. Persistent reluctance or refusal to go out, away from home, to school, to work, or elsewhere because of fear of separation.

5. Persistent and excessive fear or reluctance about being alone or without a major attachment figure at home or in other settings.

6. Persistent reluctance or refusal to sleep away from home or go to sleep without being near a major attachment figure.

7. Repeated nightmares involving the theme of separation.

8. Repeated complaints of physical symptoms (e.g., headache, stomachaches, nausea, vomiting) when separated from major attachment figures occurs or is anticipated (American Psychiatric Association, 2013, p. 190-191).

The prevalence of separation anxiety disorder among the population is 4.8% and the onset is considered to be around 18 years (Schiele & Domschke, 2021).

Periods characterized by intense separation anxiety from close people are normal in the early stages of development and may indicate the development of secure attachment relationships (Sadock et al., 2014). The onset can occur in the preschool age, the earliest, anytime during childhood, and less often in adolescence (American Psychiatric Association, 2013). The disorder evolves with exacerbations and remissions if it is not treated in time or case of non-compliance with treatment.

The manifestations of separation anxiety disorder vary with age. Young children are more reluctant to go to school or avoid school altogether. They do not show concern or specific fears regarding certain dangers to which the parents, the house, or themselves could be subjected, and the anxiety is manifested only when the separation takes place. As children grow, worry appears, there are often concerns about certain types of danger or mild worry related to the lack of attachment figures. In adults, a separation anxiety disorder can limit the ability to adapt to changes in circumstances. In some situations, the symptoms persist into adulthood. In most cases, however, it is remitted until the patient becomes an adult.

The risk factors include a stressful event in life, especially a loss (death, illness, major changes, divorce) combined with the overprotection and intrusiveness of the parents. More than that it seems that this disorder is genetically passed through generations.

The functional consequences of separation anxiety disorder are multiple, and affected persons usually limit their independent activities outside their environment or in the absence of major attachment figures (e.g., children avoid school, do not go to camps, have trouble sleeping alone, adolescents do not want to go to high school) (American Psychiatric Association, 2013).

4.3. Selective Mutism

It is an anxiety disorder manifested by an individual's constant inability to speak in social situations where it is necessary to speak (e.g., school), although the individual can do so in other contexts, according to the definition given by the American Psychiatric Association (2013). This disturbance affects the person's social and professional relationships and academic performance, in the case of children. The dysfunction is not better explained by a communication disorder (e.g, childhood-onset fluency disorder) and does not occur exclusively in the setting of an autism spectrum disorder, schizophrenia, or other psychotic disorder. It is a relatively rare disorder and occurs more frequently in younger children compared to adolescents and adults, usually starting before the age of 5. The disorder usually does not come to the doctor's attention until the start of school, when social interactions and performance demands increase, such as reading aloud.

As far as selective mutism is concerned, it must be kept in mind by the cultural environment because children of families who have emigrated to a country where another language is spoken may refuse to speak the new language due to the lack of knowledge of it. If the understanding of the new language is adequate but the refusal to speak it persists, the diagnosis of selective mutism is justified.

Functional consequences of selective mutism led to impairments in social functioning because children may be too anxious to engage in social interactions with other children. As they grow up, these children will face increasing social isolation. At school, they may show academic deficits because they do not communicate their academic or personal needs to the teachers. There is frequently a severe deficit in school and social functioning, to which the teasing of colleagues also contributes.

When we are looking at risk factors and prognosis, we see that maternal and paternal psychopathology and high paternal age have a high risk of selective mutism for their children (Koskela et al., 2020), neurotic character or behavioral inhibition can play a role, as can parental history of shyness, social isolation, and social anxiety. Parents' social inhibition can serve as a model for social reticence and selective mutism in children. Also, parents of children with selective mutism were described as hyper-protective or exerting more intense control than parents of children with other anxiety disorders or without disorders.

The treatment considered several approaches: long term cognitive behavioral therapy as recommended therapy (Oerbeck, Overgaard, Stein, Pripp, & Kristensen, 2018) and some other solutions like psychomotor 45 minutes sessions - the development of how children process information through movement during play sessions (Esposito et al., 2017).

4.4. Specific Phobia

It is characterized by the presence of clinical-level anxiety, due to the confrontation with a situation or object that causes fear; frequently leading to behavioral avoidance of the anxiogenic stimulus. Scientific literature highlights the following main characteristics of a

phobia: a stimulus-bound fear reaction that is distressing to the point of causing emotional, social, or occupational disruptions; recognized as excessive or unreasonable and leads to avoidance or intense anxiety upon exposure to the feared stimulus (Andrews et al., 2013, p. 261).

The ICD-10 Classification of Mental and Behavioral Disorders proposes meeting all the conditions listed below for a definite diagnosis: symptoms, psychological or vegetative, must be primary manifestations of anxiety, and not secondary to other symptoms such as delusions or obsessive thoughts: anxiety must be limited to the presence of particular situations or objects; phobic situations are avoided whenever possible (World Health Organization, 1993, p. 230-231).

Phobias are characterized by agitation or severe anxiety when a person is exposed to specific objects or situations or when the respective person anticipates exposure to certain situations or objects. Exposure almost invariably results in panic attacks in a susceptible person. People with phobias try to avoid phobic stimuli, some even becoming very complicated in this regard. For example, a person with a phobia of airplanes will travel very long and tiring distances by bus or car, just to avoid meeting the phobic stimulus, i.e. the airplane. Many patients associate substance use disorders with their attempt to reduce the stress of the phobic stimulus. In addition to these, approximately one-third of those with social phobia suffer from a major depressive disorder (Sadock et al., 2014).

Phobias are common in the general population, with 60% of adults reporting some form of phobic fear, but for most of them, the levels of distress and impairments presented are not severe enough for a specific phobia diagnosis (Leahy, Holland, & McGinn, 2012). However, the National Comorbidity Study found that 12.5% of respondents had at some point in their lifetime significant enough deficits to meet diagnostic criteria for specific phobia; this places specific phobia as the third most common psychiatric diagnosis in the general population (Kessler et al., 2005).

It is the most common anxiety disorder, with a prevalence in the general population of 10-11.3%. It manifests itself more often in women, with a ratio of men to women – 1:2 (American Psychiatric Association, 2013). Approximately 75-90% of people with animal phobias, natural phenomena, or situational forms of phobias are women.

Depending on the individual's phobia, one will show decreased functioning in the domain adjacent to the phobia (e.g. individuals with blood phobia avoid seeking medical help even when they suspect the presence of a medical condition, or the fear of vomiting or suffocation can substantially reduce food intake) (American Psychiatric Association, 2013).

The production of a specific phobia usually results from coupling an object or situation with fear or panic. When a specific event (for example driving a car) is coupled with an emotional experience (for example a car accident), the person is susceptible to the permanent association between the two (for example driving a car with anxiety). The emotional experience can be a response to an external incident, such as a car accident, or an internal incident, most commonly a panic attack. Other mechanisms of association between the phobic object and phobic emotions include modeling, in which one person observes the reaction of another (e.g., a parent), and the person is taught or warned about the dangers of specific objects (e.g., snake venom).

Specific phobias sometimes occur following an event that has traumatically affected the individual (e.g., after the individual is attacked by an animal or gets stuck in an elevator), after seeing other people go through such an event (e.g., witnessing someone's drowning), after the transmission of information (e.g., very detailed media coverage of a plane crash or other traumatic events). However, most individuals with a specific phobia cannot remember the particular cause of the onset of the phobia.

Specific phobia tends to be present in several family members. The blood-injection-injury type has an increased prevalence among family members. Studies have reported that two-thirds of those affected have at least one first-degree relative with the same type of phobia (Sadock et al., 2014).

The specific phobia shows two peaks from the point of view of the age of onset, a peak being in childhood for animal phobia, blood-injection-injury phobia, and natural environment phobia and a peak in adulthood for other phobias, such as situational phobias. Since patients with isolated specific phobias rarely present themselves for treatment, there are not many studies on the evolution of the disease. The limited information available in the literature up to this point suggests that most specific phobias that begin in childhood and persist in adulthood will persist for many years (Sadock et al., 2014).

4.5. Social Anxiety Disorder (Social Phobia)

Social anxiety disorder (under the older name of social phobia) is the exaggerated fear of one or more social situations. Among the situations that generally trigger social anxiety symptoms, we include public speaking and other types of public performances, social gatherings, meeting new people, eating in public, using public restrooms, contradictory discussions, and discussions with higher-status persons. In such situations, people with social anxiety disorder fear that they will be criticized or judged in negative terms, either because they did not perform well or because they are simply seen as being anxious (Leahy et al., 2012).

In the case of social phobia, avoidance is the clearest behavioral symptom. Many patients will avoid feared situations as much as possible, but will experience intense anxiety when the negative consequences of avoidance are perceived to be greater than those of participation (Andrews et al., 2013).

The specific symptoms of social phobia are flushing, sweating, shaking, palpitations and nausea. This disorder can lead to alcoholism as many individuals consume alcohol to relieve these symptoms, unaware that they are socially phobic (Bourne, 2015).

The lifetime prevalence for social anxiety disorder is between 3% and 13% (Ponniah & Hollon, 2008). The US National Comorbidity Survey Replication (Kessler et al., 2005) shows that 12.1% of the US population meets diagnostic criteria for social anxiety disorder at some point in their lifetime, this fact placing this disorder in fourth place on the list of the most common psychiatric disorders encountered. Lower values of the prevalence estimated at 12 months are found in several areas of the globe, clustering around the value of 0.5 - 2.0%; the average prevalence in Europe is 2.3%. The prevalence rate at 12 months for children and adolescents is comparable to that for adults. The prevalence rate decreases with age (American Psychiatric Association, 2013).

Several studies have reported that certain children have a pattern characterized by behavioral inhibition (Lahat et al., 2014; Henderson, Pine, & Fox, 2015). This characteristic is common in children whose parents suffer from panic disorder and can develop into severe shyness as the children grow. At least some of the people with social anxiety presented behavioral inhibition during childhood. Probably together with this characteristic, which is believed to be biologically determined, there are psychological data that indicate that the parents of people with social anxiety are usually less caring, more rejecting, more overprotective than other parents.

The success of drug therapy on social anxiety led to the emergence of two neurochemical hypotheses regarding two types of social anxiety. Specifically, the success of beta-adrenergic antagonists in performance phobia (e.g public speaking) led to the development of the adrenergic theory. Patients with performance phobia might release

more adrenaline and noradrenaline both peripherally and centrally compared to people without phobia, or they might release a normal amount, but show an excessive sensitivity to these neurotransmitters. The observations that MAOIs are more effective than tricyclic antidepressants in the treatment of this pathology led to the hypothesis that there is a dopamine disorder in this pathology (Kim & Amsterdam, 2019). A study found in patients with social anxiety significantly lower levels of homovanillic acid than in those without this pathology (Wingerson, Cowley, Kramer, Petty, & Roy-Byrne, 1996).

First-degree relatives of people with social anxiety are more likely to be affected by social anxiety than first-degree relatives of those without mental disorders. Just like in other anxiety disorders, there is a genetic component, but also an environmental one in the cause of the emergence of social anxiety disorder. If one identical twin has this issue, there is a 30-50% chance that the other will also have this problem. Heritability in first-degree relatives is five to six times higher than in other relatives. At the same time, the social anxiety of adoptive parents is significantly correlated with the social anxiety of adopted children (Kendler, Karkowski, & Prescott, 1999).

The onset age of social anxiety disorder is between 11 and 16 years. However, many patients report the onset in early childhood. Social anxiety disorder can rarely appear after the age of 20, except in situations where there are certain precipitating factors such as physical suffering or other major changes in the patient's life (Leahy et al., 2012).

A significant percentage of individuals who suffer from social anxiety disorder are also diagnosed with depression, or other anxiety disorders (panic disorder, generalized anxiety disorder, or substance abuse). Among patients diagnosed with social anxiety disorder, up to 50% of them experience spontaneous remissions within two or three years; the other 50% continue to exhibit symptoms of the disorder for much longer, without receiving treatment (Bourne, 2015).

Social anxiety disorder has temperamental risk factors (basic personality traits that predispose individuals to social anxiety disorder through inhibited behavior and fear of negative judgment;) environmental factors (maltreatment and difficulties in childhood); genetic and physiological factors (there is a genetic component, first-degree relatives of patients with social anxiety disorder are 2-4 times more likely to suffer from the same disorder) (American Psychiatric Association, 2013).

Social anxiety disorder can lead to serious impairments in academic performance, occupational and social functioning. Individuals suffering from social phobia have below average academic performance and economic status; also, a significant number of them have poor interpersonal relationships, not having a partner and a couple life (Belzer, McKee, & Liebowitz, 2005). Despite the discomfort and social impairments, in Western societies only about half of these individuals seek treatment, and this happens after 15-20 years of experiencing symptoms.

4.6. Panic Disorder

A panic attack is a sudden and distinct episode of intense discomfort and/or fear, accompanied by various physical symptoms (dizziness, palpitations, tremors, feeling of suffocation or lump in the throat, sweating, chest pain, nausea, dizziness, numbness, tingling, chills or hot flashes, feeling faint) and cognitive symptoms (fear of losing control, fear of dying and feelings of detaching from reality and derealization). Panic attacks come on suddenly and are brief, rarely lasting more than 30 minutes, with the maximum level of anxiety being reached within the first 10 minutes (Leahy et al., 2012).

In the process of diagnosing panic disorder, all causes of possible medical conditions (hypoglycemia, hyperthyroidism, reaction to excess caffeine, or abstinence from alcohol, tranquilizers, or sedatives) must be ruled out. The causes of panic disorders are a combination of heredity, chemical imbalances in brain and recent personal stress (Bourne, 2015, p. 24). Panic attacks can also occur in other anxiety disorders, but to be diagnosed with panic disorder, the patient must have repeated and unexpected panic attacks. At the same time, the patient experiences a permanent fear regarding the suffering of other panic attacks, or of the repercussions of these panic attacks (Leahy et al., 2012).

Panic attacks without an anxiety disorder occur in 22% of the general population (Kessler et al., 2005), and the estimated 12-month prevalence of panic disorder in the US and some European countries is approximately 2–3% in adults and adolescents, women being more affected than men, the ratio being approximately 2:1. The lower prevalence of panic disorder is in older adults and may be attributed to age-related attenuation of the autonomic SN response (American Psychiatric Association, 2013).

People tend to develop panic disorder in their late teens or early 20s. About half of those who have a panic disorder developed it before the age of 24. In a third of these cases, panic is complicated by the appearance of agoraphobia, because individuals suffering from panic disorder will avoid going out in open spaces for fear of having panic attacks and the situation could get out of control.

The first panic attack is often spontaneous. Sometimes, panic attacks occur in situations of excessive effort, excitement, sexual activity, and emotional trauma. Therapists must do a brief analysis of the situations that happened before the panic attacks. Such activities may include excessive consumption of caffeine, alcohol, nicotine, or other substances. Unusual eating or sleeping patterns can contribute to panic attacks. The attack often begins with a 10-minute period of increasing symptoms.

The main symptoms are extreme fear and a feeling of imminent death. People usually cannot name the source of the fear, often having trouble concentrating and being confused. Physical signs often include tachycardia, palpitations, dyspnea, and sweating. Often, patients leave their situation and seek help. The attack usually lasts between 20 and 30 minutes, rarely more than an hour. Patients may feel depressed or depersonalized during the attack. Symptoms can disappear quickly or gradually.

Between attacks, patients may have anticipatory anxiety about other attacks. Worries about death by cardiac or respiratory arrest are the main focus of patients during panic attacks. Up to 20% of patients have episodes of syncope during a panic attack. Such patients are found in emergency rooms and although the doctors say they are healthy, they insist on the fact that they are about to die from a heart attack. Hyperventilation can cause respiratory alkalosis, breathing in a bag is useful in this regard (Sadock et al., 2014).

Depressive symptoms are often found in panic disorder and some patients, a depressive disorder coexists with panic disorder. Studies show that the risk of suicide in those with panic disorder is higher than in those without mental disorders (Sadock et al., 2014).

Panic disorder, according to DSM-V presents temperamental risk factors (individuals presenting negative affectivity, neurotic character, tendency to experience negative emotions, anxious sensitivity); environmental risk factors (childhood sexual abuse, maladaptive environment, smoking, illicit substances, alcohol consumption); genetic and physiological factors (children of parents with anxiety, depressive and bipolar disorder have an increased risk for being diagnosed with panic disorder; medical conditions – bronchial asthma). Panic attacks and a diagnosis of panic disorder are associated with a higher

frequency of suicidal ideation and suicide attempts, even when other suicidal risk factors are considered (American Psychiatric Association, 2013).

In case of panic disorder, the costs imposed by this diagnosis are high both for the affected individual and for society. Individuals diagnosed with panic disorder experience significant occupational, interpersonal, and physical impairments. Because panic disorder accounts for the highest number of consultations of all anxiety disorders (American Psychiatric Association, 2013), individuals diagnosed with this disorder are among the highest users of health care services, including emergency department and hospitalizations (Barlow, 2002). Also, for this reason, they miss work twice as much as patients with other psychiatric conditions, thus creating greater deficits than those caused by the chronically ill, thus generating direct costs (hospitalization) as well as indirect costs (labor productivity) for the health system (Barlow, 2002).

Panic disorder severely impairs the interpersonal functioning of affected individuals. The fear of suffering panic attacks in public places severely limits their ability to socialize with other people (Leahy et al., 2012).

4.7. Agoraphobia

The word agoraphobia denotes the fear of open spaces, but the essence of agoraphobia is the fear of having panic attacks (Bourne, 2015). Individuals suffering from agoraphobia are afraid of being put in situations from which escape may be difficult, or of being put in embarrassing situations, should they suffer a panic attack. Fear and shame play a key role. The most common feature of agoraphobia is the anxiety caused by the thought of the agoraphobic individual being away from the person to whom there is a primary attachment (Bourne, 2015).

According to the data presented in the Manual of Diagnosis and Statistical Classification of Mental Disorders, "each year approximately 1.7% of adolescents and adults are diagnosed with agoraphobia. Women are twice as likely as men to experience agoraphobia. The 12-month prevalence in individuals older than 65 is 0.4%" (World Health Organization, 1993, p. 219).

Agoraphobia can begin in childhood, but the peak incidence is towards the end of adolescence and young adulthood. Chronic remission is rare unless the agoraphobia is treated. The development of agoraphobia can be complicated by various conditions, especially other anxiety disorders, depressive disorders, substance use disorders, and personality disorders (American Psychiatric Association, 2013). In many cases, agoraphobia appears to arise from panic disorder, as the affected individual practically avoids facing the symptoms of a panic attack in public, reaching in severe cases the restriction of all activities (Bourne, 2015).

4.8. Generalized Anxiety Disorder

Generalized anxiety disorder is characterized by chronic anxiety that persists for at least six months but is not accompanied by panic attacks, phobias, or obsessions (Bourne, 2015). Patients with generalized anxiety disorder express extreme and/or chronic apprehensive worry and related physical symptoms, failing to control their worries and reporting various problems such as fatigue, irritability, restlessness, muscle tension, and insomnia. (Leahy, et al., 2012).

As with all mental disorders, before establishing a correct diagnosis of generalized anxiety disorder, the assessment will be made according to the criteria established by the Diagnostic and Statistical Manual of Mental Disorders, DSM-5. As with all other disorders, the clinician must rule out the possibility that the patient being evaluated and suspected of

having a generalized anxiety disorder is caused by physical illness or alcohol or drug abuse/withdrawal (Leahy et al., 2012).

Epidemiological studies indicate that the lifetime prevalence of generalized anxiety disorder is estimated to be between 5.8 and 9%, with the risk being higher in women (2.5:1 ratio), young adults, and people of color (Blazer, George, & Winfield, 1991). Individuals of European descent tend to experience this disorder more frequently than individuals of other origins. Peak prevalence is in midlife and declines in later years of life (American Psychiatric Association, 2013).

Generalized anxiety disorder is probably the pathology that is most frequently associated with another mental disorder, usually social phobia, specific phobia, panic disorder, or depressive disorder. In this way we can see the disorder as part of a spectrum of mood and related disorders. Probably between 50 and 90 percent of those with a generalized anxiety disorder also have another mental disorder (Tyrer & Baldwin, 2006).

The therapeutic effect of benzodiazepines and buspirone has focused scientific research on GABA and serotonin regarding this pathology (Garakani et al., 2020). Although there is no convincing data to attest to the fact that there are benzodiazepine receptor abnormalities in generalized anxiety disorder, we suspect dysfunctions of the occipital lobes, and secondarily of the basal ganglia, the limbic system, and the frontal cortex. Other neurotransmitter systems that have been researched in generalized anxiety disorder are those that regulate norepinephrine, cholecystokinin, and glutamate.

Neuroimaging studies of patients with generalized anxiety disorder have revealed important changes. A PET study reported a decreased metabolic rate in the basal ganglia and white matter in patients with generalized anxiety disorder compared to people without mental illness (Wu et al., 1991).

The two schools that studied this pathology in depth are the cognitive-behavioral school and the psychoanalytic school. According to the cognitive-behavioral school, patients with generalized anxiety disorder respond to dangers that are incorrectly perceived. The inaccuracy starts from the fact that they pay selective attention to the negative details in the environment, thus distorting the information processing.

The psychoanalytic school believes that anxiety comes from unresolved, unconscious conflicts. Sigmund Freud first presented this theory in 1909 with the description of Little Hans. Before that, Freud conceptualized anxiety as having a physiological basis.

The onset of generalized anxiety disorder is rare in adolescence, with most individuals reporting that they have felt anxious and nervous throughout their lives, with the average age of onset for this disorder being 30 years (American Psychiatric Association, 2013). Because of its chronicity and poor compliance with treatment, some clinicians have considered generalized anxiety disorder to be a lifelong disorder (like diabetes), and others consider it a personality disorder (Leahy et al., 2012).

Generalized anxiety disorder can be exacerbated by any stressful situation that evokes fears of failure, illness, rejection, abandonment, demanding performance, marital conflict, or any situation that increases the impression of danger and threat (Bourne, 2015).

4.9. Anxiety Disorder due to Another Medical Condition

This diagnostic category is reserved for situations where considerable anxiety (which may take the form of panic attacks or generalized anxiety) is a direct effect of an illness. Individuals may be affected by different types of illnesses that can cause anxiety, including endocrine conditions, cardiovascular or respiratory conditions (asthma), metabolic conditions (vitamin deficiencies, porphyria), gastrointestinal illness (irritable bowel

syndrome, gastroesophageal reflux disease) or neurological conditions (fibromyalgia, epilepsy, cerebral palsy) (Bourne, 2015; Meuret, Tunnell, & Roque, 2020).

To correctly establish this diagnosis, clinicians can review the diagnostic criteria established by DSM-5: panic attacks or anxiety predominate the clinical picture; there is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition; the disturbance is not better explained by another mental disorder; the disturbance does not occur exclusively during delirium; the disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (American Psychiatric Association, 2013, p. 230).

4.10. Substance or Medication-Induced Anxiety Disorder

The diagnosis of substance and medication-induced anxiety disorder will be made when the patient being evaluated has generalized anxiety or panic attacks that are caused by the physiological effect of a substance, whether it is a drug of abuse, a medication, or exposure to toxins. Anxiety can be caused by exposure to a substance or withdrawal from a substance (Bourne, 2015). Substance-induced anxiety is common, both as a result of ingesting recreational drugs and also as a result of ingesting certain medications. Sometimes is difficult to distinguish this disorder and delirium, that is included in the differential diagnosis.

Several medications can cause anxiety. Although sympathomimetic substances, such as cocaine, amphetamines, and caffeine have been most frequently associated with the production of anxiety, many serotonergic substances can cause chronic anxiety in users. The clinical characteristics of substance-induced anxiety vary depending on the substance involved. Even the rare use of psychostimulants can lead to anxiety disorder in certain people. These cognitive deficits are usually reversible when the substance is stopped.

The most frequently reported symptoms when using steroids, antiepileptic drugs, antimalarial drugs, and antiretroviral drugs are corelated with psychosis with persecutory delusions and auditory hallucinations. Mood changes and anxiety may precede psychosis after steroids and antimalarials (Niebrzydowska & Grabowski, 2022).

5. CONCLUSION/DISCUSSION

The complexity and high prevalence of anxiety disorder have led many specialists to conceive various materials about this issue, in different forms, bringing their beneficial contribution to the information of those interested. The present work has addressed all anxiety disorders in a somewhat conventional form as brief reviews intended for clinicians and beyond. Anxiety and related disorders are generally defined by the characteristics of excessive anxiety, fear, worry, and avoidance. While anxiety may be a normal part of everyday life, anxiety disorders are associated with functional impairment and cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. Anxiety disorders constitute a major clinical and public health problem, being associated with a significantly increased risk of mortality, and the co-occurrence of these disorders led to an increased risk of death. Due to the high prevalence of anxiety disorders, the associated excess mortality has a huge impact on public health. A recent meta-analysis indicated that approximately five million deaths worldwide can be attributed to mood and anxiety disorders each year (Olariu et al., 2015). It is also important for clinicians to realize that anxiety disorders and depression are associated with increased mortality from both natural and unnatural causes and that physical health assessment in

these patients is always important. Low rates of recognition of anxiety disorders have been described at the level of primary care (Hofmann & Smits, 2008).

The current conceptualization of the etiology of anxiety disorders includes an interaction of psychosocial factors, for example, the person's adversity, stress, or trauma, and a genetic vulnerability, which manifests itself in neurobiological and neuropsychological dysfunctions. Evidence for potential biomarkers for anxiety disorders in the fields of neuroimaging, genetics, neurochemistry, neurophysiology, and neurocognition has been summarized in two recent consensus papers (Bandelow et al., 2017). Despite extensive and high-quality neurobiological research in the field of anxiety disorders, reviews indicate that specific biomarkers for anxiety disorders have not yet been identified. Thus, it is difficult to make recommendations for specific biomarkers (e.g., genetic polymorphisms) that might help identify individuals at risk for anxiety disorder. Developing an appropriate approach to patients with anxiety disorders and comorbid general medical conditions requires the involvement of a multidisciplinary team, with an emphasis on early recognition of risk factors and appropriate treatment. Personal, social, and disease factors can each intervene to delay or complicate treatment. All patients with anxiety disorders require supportive discussion and attention to the emotional problems associated with the anxiety disorder. Psychoeducation includes information about the physiology of bodily symptoms, anxiety reactions, and the justification of available treatment possibilities. Many patients may require formal psychological treatment interventions, which are mostly done in outpatient or private psychological practices. It is a common belief that patients with anxiety disorders treated with medication relapse immediately after stopping the medication, whereas gains from psychological therapies are maintained months or years after treatment discontinuation. This would give psychological therapy a considerable advantage over medication treatment of anxiety.

The impact of the increasing prevalence of anxiety disorder on society is considerable, and it is imperative that specialists are well-documented and trained to contribute favorably to public health.

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