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ANXIETY DISORDERS AND AUTONOMIC NERVOUS SYSTEM DYSFUNCTION: MECHANISMS AND CLINICAL IMPLICATIONS

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ABSTRACT

Anxiety disorders are prevalent and disabling psychiatric conditions, frequently associated with emotional, cognitive, behavioral, and somatic manifestations. Recent scientific evidence indicates that these disorders also involve relevant changes in the functioning of the autonomic nervous system, especially related to heart rate variability, vagal modulation, sympathetic activity, and cardiovascular regulation. The present study aimed to analyze the scientific evidence regarding the relationship between anxiety disorders and autonomic nervous system dysfunction, with emphasis on pathophysiological mechanisms and clinical implications. This is an integrative literature review, based on the analysis of 27 scientific studies published in peer-reviewed journals and indexed in recognized databases, with priority given to PubMed/ MEDLINE. Metanalyses, systematic reviews, observational, experimental, and clinical studies addressing anxiety disorders, heart rate variability, vagal modulation, sympathetic hyperreactivity, baroreflex function, and the physiological response to stress were included.The results demonstrated a consistent association between anxiety disorders and reduced heart rate variability, lower parasympathetic modulation, sympathetic hyperactivity, and changes in the physiological response to stress. These alterations were observed especially in generalized anxiety disorder, panic disorder, and social anxiety. It is concluded that anxiety disorders should be understood as complex psychophysiological conditions in which autonomic mechanisms participate in the expression, maintenance, and severity of symptoms. Assessing autonomic markers may contribute to a better clinical understanding, risk stratification, and the development of integrated therapeutic interventions.

Keywords: anxiety; autonomic nervous system; heart rate; psychophysiology.

INTRODUCTION

Anxiety disorders represent one of the most prevalent and disabling groups of psychiatric conditions in contemporary clinical practice, characterized by emotional, cognitive, behavioral, and somatic manifestations that go beyond normal adaptive responses to fear or threat. Although traditionally understood through psychopathological models centered on excessive worry, hypervigilance, avoidance, and the distorted interpretation of threatening stimuli, growing evidence shows that these disorders also involve measurable changes in physiological body-regulation systems, especially the autonomic nervous system (ANS). In this context, anxiety is no longer understood only as a subjective or psychological phenomenon; instead, it is analyzed as a condition associated with psychophysiological dysfunction, with repercussions for cardiovascular homeostasis, respiratory,
neuroendocrine, and inflammatory (1-4) .

The autonomic nervous system plays a central role in maintaining internal stability of the organism, regulating functions such as heart rate, blood pressure, vascular tone, respiration, sweating, gastrointestinal motility, and the physiological response to stress. Its action occurs predominantly through the dynamic interaction between the sympathetic and parasympathetic branches, which allow the body to alternate between states of rest, vigilance, mobilization, and recovery. Under conditions of real or perceived threat, sympathetic activation promotes defensive responses, while parasympathetic modulation, especially mediated by the vagus nerve, contributes to restoring physiological balance after the stressor. In anxiety disorders, this balance appears to be impaired, favoring patterns of hyperreactivity, low autonomic flexibility, and difficulty returning to the baseline state (1,4, 10, 11) .

Among the most investigated markers of autonomic function, heart rate variability (HRV) stands out, considered a noninvasive indicator of cardiac autonomic modulation. HRV reflects the temporal fluctuations between consecutive heartbeats and expresses the body's ability to adjust its cardiovascular activity in response to internal and environmental demands. In general, higher levels of HRV are associated with greater physio-

logical flexibility, better emotional regulation, and greater adaptive capacity, whereas reductions in HRV suggest reduced parasympathetic influence, autonomic rigidity, and vulnerability to stress. Meta-analyses have shown that individuals with anxiety disorders exhibit a significant reduction in HRV compared with healthy controls, reinforcing the hypothesis that autonomic dysfunction is a relevant component of the pathophysiology of these conditions (1,2) .

The association between anxiety and reduced HRV has been observed across different diagnostic categories, including generalized anxiety disorder, panic disorder, and social anxiety disorder. No

in generalized anxiety disorder, persistent worry appears to be related to reduced vagal tone and lower autonomic flexibility during tasks involving worry induction and aversive imagery, suggesting that anxious rumination may keep the body in a prolonged state of physiological vigilance (7,9) . In panic disorder, studies point to changes in cardiac modulation, blood pressure response, baroreflex function, and autonomic reactivity to physiological challenges, such as hyperventilation, perceived threat, and autonomic activation tests (13, 14, 16,20,21) . In social anxiety, in turn, alterations in HRV and in stress-related biomarkers have been described, including salivary alpha-amylase and cortisol, indicating the integrated participation of the autonomic and neuroendocrine systems in the response to threatening social stimuli (6,23) .

Beyond HRV, other physiological parameters have contributed to understanding the autonomic mechanisms involved in anxiety disorders. Respiratory sinus arrhythmia, often used as an indicator of cardiac vagal modulation, has been discussed as a transdiagnostic marker of emotional regulation and the ability to adapt to stress. Studies on respiratory sinus arrhythmia reactivity suggest that changes in vagal functioning may cut across different psychopathological dimensions, including anxiety, post-

traumatic symptoms, and states of persistent threat (25,26). Similarly, evidence on stress and HRV indicates that chronic exposures to states of emotional tension are associated with reduced cardiac variability, reinforcing the interface between

anxiety, physiological stress, and autonomic dysfunction (27).

From a neurobiological point of view, the relationship between anxiety and the ANS can be understood through the neurovisceral integration model. This model proposes that autonomic regulation depends on the interaction between cortical, subcortical, and autonomic structures, including the prefrontal cortex, amygdala, hypothalamus, brainstem, and vagal pathways. Cardiac vagal activity, expressed in part through HRV measures, would reflect the central nervous system's capacity to modulate emotional and physiological responses to threatening stimuli. Thus, reduced HRV may indicate lower efficiency of prefrontal inhibitory circuits over limbic and autonomic responses, favoring hypervigilance, an exacerbated fear response, and difficulty recovering after stress (10,11) .

The clinical implications of this association are broad. Symptoms often reported by anxious patients, such as palpitations, dyspnea, sweating, tremors, dizziness, a feeling of chest tightness, and gastrointestinal discomfort, may reflect, in part, disregulated autonomic activation. In many cases, these somatic manifestations intensify the perception of threat, feeding back into cycles of fear, catastrophic interpretation, and worsening of anxious symptoms. This dynamic is particularly evident in panic disorder, in which benign bodily sensations may be interpreted as signs of an imminent collapse, triggering new crises and reinforcing avoidance patterns (13,14,20,21).

Another relevant aspect concerns the possible cardiovascular impact of autonomic dysfunction associated with anxiety. Sustained reductions in HRV have been associated, in different populations, with worse cardiovascular prognosis, lower adaptive capacity, and greater physiological vulnerability to stress. Although the causal relationship between anxiety disorders and cardiovascular risk still requires in-depth investigation, available findings suggest that autonomic changes may represent a pathophysiological link between chronic psychological distress and systemic clinical outcomes (4,12,27). In addition, the use of psychotropics, especially antidepressants, may interfere with autonomic measures, which requires methodological caution when interpreting the

results and reinforces the need for studies that adequately control pharmacological variables, comorbidities, and patients' clinical characteristics (4,5, 15).

The understanding of autonomic dysfunction in anxiety disorders also has therapeutic relevance. Interventions pharmacological, psychotherapeutic, and behavioral interventions can indirectly modulate autonomic activity by reducing hyperarousal, persistent worry, and physiological threat responses. Studies suggest, for example, that pharmacological treatments may alter HRV parameters in patients with panic disorder, while non-pharmacological strategies, such as autogenic training, controlled breathing, biofeedback, physical activity, and emotional regulation techniques, can promote greater vagal balance and physiological recovery (15,24). In this sense, autonomic assessment can contribute not only to understanding the pathophysiology of anxiety, but also to monitoring treatment response and to developing integrated clinical approaches.

Despite advances in the evidence, the literature still presents methodological heterogeneity Significant differences in diagnostic instruments, in HRV measurement protocols, in recording time, in respiratory control, in the use of medications, in the presence of comorbidities, and in the types of experimental stimuli make direct comparison between studies difficult. In addition, part of the research focuses on specific disorders, such as panic disorder and social anxiety, while others address anxiety in a dimensional or transdiagnostic manner. This diversity reinforces the need for integrative syntheses capable of organizing the available findings, identifying consistent patterns, and highlighting relevant gaps for future investigations (1-4,25,26) .

In this scenario, the present integrative review aims to analyze the scientific evidence regarding the relationship between anxiety disorders and autonomic nervous system dysfunction, with an emphasis on pathophysiological mechanisms, markers autonomic and clinical implications. Specifically, we seek to understand how changes in HRV, in vagal tone, in sympathetic reactivity, in baroreflex function, and in the stress-response contribute to the manifestation, maintenance, and sev-

erity of anxiety disorders, as well as to discuss the potential of these markers to clinical assessment, risk stratification, and the development of integrated therapeutic interventions.

METHODOLOGY

This study is characterized as an integrative literature review, developed with the aim of bringing together, analyzing, and synthesizing scientific evidence on the relationship between anxiety disorders and dysfunction of the autonomic nervous system, with emphasis on pathophysiological mechanisms, autonomic markers, and the associated clinical implications. The choice of an integrative review is justified because it allows the inclusion of studies with different methodological designs, such as systematic reviews, meta-analyses, observational studies, experimental studies, psychophysiological studies, and clinical investigations, enabling a broad and critical understanding of the phenomenon under study.

The guiding question of the review was defined as follows: what scientific evidence demonstrates the association between anxiety disorders and dysfunction of the autonomic nervous system, and what are the main mechanisms and clinical implications described in the literature? The formulation of this question enabled the search to be directed toward studies that addressed, simultaneously, anxious manifestations, autonomic changes, and physiological or clinical outcomes related to cardiovascular regulation, the stress response, vagal tone, heart rate variability, and sympathetic hyperreactivity.

The bibliographic search was carried out in internationally recognized scientific databases, with priority given to PubMed/MEDLINE, in addition to complementary searches in indexed journals and editorial databases associated with the fields of psychiatry, neuroscience, psychophysiology, behavioral medicine, and cardiovascular sciences. Studies published in peer-reviewed scientific journals were considered, with availability of traceable bibliographic data, including title, authorship, journal, year of publication, volume, pages, and, when available, DOI. The selection prioritized studies with the greatest adherence to the central topic of the review and scientific relevance for understanding the autonomic

mechanisms involved in anxiety disorders.

Descriptors and combinations of terms in English were used because they are predominant in international biomedical databases, including: “anxiety disorders”, “autonomic nervous system dysfunction”, “heart rate variability”, “cardiac vagal tone”, “sympathetic nervous system”, “parasympathetic activity”, “baroreflex”, “panic disorder”, “generalized anxiety disorder”, “social anxiety disorder”, “respiratory sinus arrhythmia”, “cortisol”, “salivary alpha-amylase” and “stress response”. The combinations were organized with Boolean operators, such as AND and OR, in order to broaden the sensitivity of the search while preserving thematic specificity. Thus, combinations such as “anxiety disorders AND heart rate variability”, “panic disorder AND autonomic nervous system”, “generalized anxiety disorder AND cardiac vagal tone”, “social anxiety disorder AND cortisol AND salivary alpha-amylase” and “anxiety AND respiratory sinus arrhythmia” were prioritized.

Studies were included that addressed the relationship between clinically relevant anxiety disorders or anxious symptoms and functioning parameters autonomic, especially variability of heart rate, vagal tone, respiratory sinus arrhythmia, heart rate, blood pressure, baroreflex function, sympathetic activity, cortisol, salivary alpha-amylase, or physiological response to stress. Systematic reviews and meta-analyses were also included that synthesized evidence on autonomic dysfunction in psychiatric disorders, provided that they included specific data on anxiety or conditions directly related. Classic, highly cited studies were considered eligible when they made a relevant contribution to building the pathophysiological understanding of the topic, even if they were published in earlier years.

Studies were excluded if they did not show a direct relationship with anxiety disorders or with autonomic markers, publications without adequate bibliographic retrievability, studies exclusively focused on populations with cardiovascular diseases without symptom or anxiety-disorder assessment, opinion articles without an empirical basis, letters to the editor, conference abstracts without full text, or studies whose primary focus

was restricted to psychiatric disorders not related to the scope of the review. Work was also excluded in which autonomic changes were analyzed only as secondary findings without sufficient methodological description to allow critical interpretation.

The selection process was conducted in stages. Initially, studies were identified by reviewing titles and abstracts, considering adherence to the review topic. Next, potentially eligible studies were assessed for relevance scientific, clarity methodological rigor, relationship with the investigated autonomic mechanisms, and contribution to clinical discussion. After this screening, 27 studies were selected to form the final database of the review. This bibliographic sample was structured to include different levels of evidence, including meta-analyses, systematic reviews, observational studies, experimental studies, clinical studies, and theoretical integration models of neurovisceral mechanisms.

Data extraction was carried out from an analytically organized matrix in advance, including information such as author and year of publication, study type, investigated population, analyzed disorder or clinical condition, autonomic marker assessed, main findings, and contribution to understanding the topic. This matrix enabled comparison across the studies and the identification of recurring patterns in the literature, especially regarding reductions in heart rate variability, decreased vagal tone, sympathetic hyperreactivity, alterations in baroreflex function, and the psychophysiological stress response in individuals with anxiety disorders.

The analysis of the studies was conducted qualitatively and integratively, seeking to organize the findings into thematic axes compatible with the review's objectives. The main axes defined were: heart rate variability and vagal modulation; sympathetic hyperactivity and stress response; baroreflex function and cardiovascular regulation; differences among anxiety disorder subtypes; and clinical and therapeutic implications of autonomic dysfunction. This analytical strategy made it possible to integrate evidence from different methodologies and build a critical synthesis of the mechanisms by which anxiety disorders can interfere with autonomic regulation.

The references were organized according to the Vancouver style, with sequential numbering in accordance with the order of use and linkage in the development of the article. Citations in the text followed the numeric format, enabling a direct association between the findings discussed and the studies included in the bibliographic database. Since this is an integrative review based on secondary data published in scientific literature, there was no need to submit to an ethics committee for research, as no primary data were collected from human beings, nor were any identifiable individual information used.

The methodology adopted sought to ensure rigor, traceability, and scientific coherence in the selection and interpretation of evidence, recognizing, however, possible limitations inherent to integrative reviews, such as heterogeneity in methodological designs, differences in autonomic measurement protocols, variations in diagnostic criteria, the influence of psychotropic medications, and diversity of the instruments used to assess anxiety disorders. Despite these limitations, the selection of the 27 studies made it possible to bring together a consistent basis to discuss the relationship between anxiety and dysfunction autonomic in perspective pathophysiological, psychophysiological, and clinical.

RESULTS

This integrative review included 27 scientific studies that investigated the association between anxiety disorders and dysfunctions of the nervous autonomic system, including meta-analyses, systematic reviews, observational studies, experimental studies, psychophysiological studies, clinical studies, and theoretical models. The selected studies showed convergence regarding the existence of autonomic alterations in individuals with anxiety disorders, especially reduced heart rate variability, lower vagal modulation, changes in sympathetic response, cardiovascular regulation dysfunction, and increased physiological rigidity in response to threat or stress stimuli.

Among the included studies, meta-analyses and systematic reviews stood out, consistently demonstrating the association between anxiety disorders and reduced heart rate variability. Chalmers et al. found that individuals with anxiety

disorders show lower HRV compared with healthy controls, suggesting impairment of autonomic flexibility and reduced the body's adaptive capacity in response to emotional and environmental demands (1). Cheng et al. reinforced these findings by showing, in a systematic review and meta-analysis, that patients with anxiety disorders present significant alterations in autonomic parameters, especially those related to parasympathetic modulation (2). Wang et al., when comparing generalized anxiety disorder, panic disorder, and major depressive disorder, also observed relevant differences in HRV patterns, indicating that autonomic dysfunction may vary depending on the psychiatric diagnosis and the clinical expression of symptoms (3).

The analysis of the studies revealed that HRV was the most frequently used autonomic marker. This parameter appeared in studies involving generalized anxiety, panic disorder, social anxiety, transdiagnostic anxiety symptoms, and stress response. In general, reduced HRV was interpreted as a sign of lower parasympathetic influence, lower cardiac vagal tone, and greater physiological rigidity. This rigidity appears to limit the organism's ability to transition appropriately between states of rest, vigilance, activation, and recovery, which proved particularly relevant in conditions marked by persistent worry, hypervigilance, anticipatory fear, and intense somatic symptoms (1,2,7,9,24,27) .

In studies focused on generalized anxiety disorder, the findings indicated that chronic worry and the anticipation of negative events are associated with reduced autonomic flexibility. Thayer et al. observed specific autonomic characteristics in individuals with generalized anxiety and worry, suggesting a lower capacity for physiological regulation in the face of threatening cognitive stimuli (9). Levine et al. showed that patients with generalized anxiety disorder had changes in HRV during laboratory tasks of induced worry and aversive imagery, indicating that anxious cognitive processes can keep the body in a state of prolonged physiological activation (7). These results suggest that autonomic dysfunction in generalized anxiety disorder does not

occur only in response to external threats, but may also be triggered and sustained by internal processes, such as rumination, anticipation, and excessive worry.

In panic disorder, the included studies demonstrated a particularly relevant pattern of autonomic change. Reductions in HRV were identified, along with heart rate alterations, blood pressure changes, changes in baroreflex function, and exacerbated physiological responses to autonomic challenges. Classic studies by Yeragani et al., Klein et al., McCraty et al., and Slaap et al. showed changes in cardiac variability in patients with panic disorder, suggesting impairment of basal autonomic modulation and possibly sustained over time (16-19). Lambert et al. identified sympathetic changes and cardiac baroreflex function alterations, indicating that autonomic dysfunction in panic may involve both peripheral and central mechanisms of cardiovascular regulation (14). Martinez et al. and Wang et al. reinforced that patients with panic disorder show altered autonomic responses to physiological challenges or threat situations, which contributes to understanding the intense somatic symptoms observed in these patients (20,21).

Social anxiety also showed an association with relevant autonomic changes. Alvares et al. demonstrated reduced HRV in individuals with social anxiety disorder, with associations related to sex and symptom severity (6). Tamura et al. observed changes in salivary alpha-amylase and cortisol in patients with generalized social anxiety subjected to experimental stress, indicating an integrated involvement of the sympathoadrenergic and hypothalamic–pituitary–adrenal systems in the response to situations perceived as threatening (23). These findings suggest that, in social anxiety, exposure to or anticipation of negative social evaluation may mobilize autonomic and neuroendocrine relevant.

The table below presents a synthesis of the main result axes identified in the review, the predominant autonomic markers, the related disorders or clinical conditions, and the studies that support each finding.

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Table 1 - Synthesis of the main axes of results identified in the review

Outcome	Main physiological signals	Transdiagnostic	Principal identified	Study correspondent
Reduction in heart rate variability	HRV, HF-HRV, SDNN, RMSSD	General anxiety disorders, GAD, panic disorder, social anxiety	Consistent reduction in HRV in anxious individuals, suggesting lower autonomic flexibility and reduced parasympathetic modulation	1, 2, 3, 5, 6, 7, 16, 17, 18, 19
Lower vagal tone and autonomic rigidity	Cardiac vagal tone, respiratory sinus arrhythmia, HRV	GAD, trait/state anxiety, transdiagnostic symptoms	Lower capacity for physiological regulation and difficulty recovering after stress or persistent worry	9, 10, 11, 24, 25, 26
Sympathetic hyperreactivity	Heart rate, blood pressure, catecholamines, salivary alpha-amylase	Panic, social anxiety, stress response	Increased or dysregulated responses in the face of threat, hyperventilation, experimental stress, or autonomic challenges	13, 20, 21, 23, 27
Baroreflex and cardiovascular alteration	Cardiac baroreflex, blood pressure, heart rate	Panic disorder	Evidence of changes in baroreflex function and cardiovascular regulation during autonomic challenges	14, 20
Neurovisceral integration and regulation emotional	HRV, prefrontal circuits, amygdala, vagal regulation	Anxiety and transdiagnostic psychopathology	Relationship between lower HRV, lower prefrontal inhibitory control, and greater emotional/autonomic reactivity	10, 11, 25, 26
Influence of treatments and medications	HRV, vagal modulation, autonomic response	Panic, anxiety, psychiatric disorders	Psychotropic drugs and psychophysiological interventions can alter autonomic parameters, requiring methodological control	4, 5, 15, 24

Studies have also shown that autonomic dysfunction in anxiety disorders should not be understood as a single or uniform phenomenon. Although reduced HRV was the most recurring finding, the results indicated variations depending on the type of disorder, the severity of symptoms, the presence of comorbidities, the use of medications, and the assessment context. In generalized anxiety disorder, the findings were primarily related to persistent worry and reduced vagal flexibility. In panic disorder, greater emphasis was placed on somatic symptoms, cardiovascular responses, and baroreflex alterations. In social anxiety, a stronger relationship was observed with autonomic and neuroendocrine responses to social or experimental stressors.

Another relevant finding was the presence of evidence linking autonomic function to central circuits of emotional regulation. Friedman proposed the model of autonomic flexibility and neurovisceral integration, suggesting that cardiac vagal tone represents a functional marker of the central nervous system's ability to modulate emotional and autonomic responses (10). Thayer et al., through a meta-analysis involving HRV and neuroimaging, indicated that cardiac variability is related to brain regions implicated in emotional regulation, such as the prefrontal cortex, the amygdala, and structures involved in the stress response (11). These findings broaden the interpretation of HRV, not only as a peripheral marker, but as an expression of the integration between brain, emotion, and body.

The review also identified that some of the studies assessed methodological factors that could interfere with the interpretation of autonomic findings. Alvares et al. highlighted that psychiatric disorders present measurable autonomic dysfunction, but that the use of psychotropics can modify parameters such as HRV, heart rate, and cardiovascular responses (4). Licht et al., in the NESDA study, also indicated that the association between anxiety and HRV may be influenced by antidepressants and other clinical variables (5). These findings demonstrate the need to interpret results by considering potential confounders, especially medication, depressive comorbidities, age, sex, physical condition, smoking, substance use, and measurement protocols.

From a clinical standpoint, the results indicated that autonomic dysfunction

may contribute to understanding the physical symptoms often present in anxiety disorders. Palpitations, sweating, tremors, dyspnea, chest discomfort, dizziness, nausea, and interoceptive hypervigilance may be associated with patterns of sympathetic activation, reduced vagal regulation, and increased sensitivity to variations in internal physiological states. This relationship was particularly evident in studies of panic disorder, in which cardiovascular and autonomic changes appear associated with the intense fear response and the catastrophic interpretation of bodily signals (13, 14,20,21) .

Evidence was also identified regarding the therapeutic potential of autonomic modulation. Tucker et al. observed an increase in HRV after the use of paroxetine in patients with panic disorder, suggesting that pharmacological interventions can influence autonomic parameters (15). Miu et al. identified a reduction in HRV and vagal tone in anxiety, but they also observed effects of autogenic training, indicating that psychophysiological strategies may promote greater autonomic balance (24). These findings point to the possibility of using autonomic measures as complementary indicators of therapeutic response, although studies with greater methodological standardization are still needed.

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DISCUSSION

Overall, the results of this review demonstrated that anxiety disorders are associated with relevant autonomic changes, with emphasis on reduced HRV, lower vagal tone, sympathetic hyperreactivity, baroreflex dysfunction, and altered stress response. The convergence of the studies suggests that autonomic assessment can contribute to a more integrated understanding of anxiety disorders, bringing together emotional symptoms and somatic manifestations and mechanisms neurophysiological. These findings provide the basis for the discussion of anxiety as a psychophysiological condition complex, in which changes in the autonomic nervous system may participate both in the clinical expression and in the maintenance of symptoms.

The findings of this integrative review show that anxiety disorders are

associated with relevant changes in the functioning of the autonomic nervous system, especially with regard to reduced heart rate variability, lower vagal modulation, sympathetic hyperreactivity, baroreflex dysfunctions, and alterations in the physiological response to stress. The convergence among meta-analyses, experimental studies, clinical studies, and theoretical models suggests that anxiety should not be understood only as a psychological or behavioral phenomenon, but as a complex psychophysiological condition in which central and peripheral systems for emotional and bodily regulation participate in an integrated manner (1-4,10,11) .

Reducing heart rate variability was the most consistent finding among the analyzed studies. This marker expresses the body's ability to dynamically modulate cardiac activity in response to internal and external demands, being influenced by the interaction between the sympathetic and parasympathetic branches of the autonomic nervous system. The lower HRV observed in individuals with anxiety disorders suggests reduced physiological flexibility, impaired autonomic self-regulation, and a lower capacity to recover after situations of threat, worry, or stress (1,2,27) . These findings reinforce the hypothesis that pathological anxiety is associated with a state of autonomic rigidity, in which the body remains longer in patterns of vigilance and activation.

From a pathophysiological point of view, decreased HRV may reflect reduced influence of the cardiac vagal pathway and lower efficiency of the inhibitory mechanisms responsible for modulating intense emotional responses. The vagus nerve plays an essential role in parasympathetic regulation, contributing to cardiac deceleration, physiological recovery, and adaptation to environmental changes. When this modulation is reduced, the individual may show greater vulnerability to hyperarousal, difficulty relaxing, and prolonged maintenance of somatic symptoms, such as palpitations, muscle tension, sweating, tremors, and respiratory discomfort. This interpretation is consistent with studies linking anxiety, lower vagal tone, and altered respiratory sinus arrhythmia (9,24-26) .

The neurovisceral integration model provides a relevant theoretical basis for understanding these findings. According to this model, cardiac autonomic regula-

tion, in part, reflects the functional capacity of brain networks involved in emotional control, especially prefrontal, limbic, and brainstem regions. Lower HRV may indicate a reduced ability of the prefrontal cortex to exert inhibitory control over structures related to fear and threat, such as the amygdala. This imbalance favors hypervigilance, exaggerated interpretation of threatening stimuli, and intensified autonomic responses. Thus, HRV can be understood not only as a peripheral cardiac marker, but as a functional expression of communication between brain, emotion, and body (10, 11) .

No evidence of generalized anxiety disorder; the results suggest that persistent worry plays an important role in maintaining autonomic activation. Unlike brief physiological responses to real threats, chronic worry tends to prolong internal states of alert even in the absence of immediate danger. Studies included in this review showed that individuals with generalized anxiety exhibit autonomic changes during worry-induction tasks and aversive imagery, indicating that cognitive processes alone may be sufficient to sustain maladaptive physiological responses (7,9) . This reinforces the understanding of worry as a phenomenon that is not only psychological, but also bodily—able to keep the autonomic nervous system in a state of continuous mobilization.

In panic disorder, autonomic dysfunction takes on special clinical relevance, since this disorder is characterized by sudden and intense physical symptoms. Palpitations, shortness of breath, dizziness, tremors, sweating, and chest discomfort are often catastrophically interpreted by the patient, feeding the fear cycle and amplifying the autonomic response. The studies analyzed showed changes in HRV, heart rate, blood pressure, baroreflex function, and the response to autonomic challenges in patients with panic disorder (13, 14, 16-21). These findings suggest that panic may involve both greater interoceptive sensitivity and genuine alterations in the mechanisms regulating cardiovascular and autonomic function.

The alteration in baroreflex function observed in studies on panic disorder deserves emphasis. The baroreflex contributes to the rapid control of blood

pressure and heart rate, modulating cardiovascular responses in the face of hemodynamic changes. Dysfunctions in this system may favor autonomic instability, greater perception of bodily changes, and more intense cardiovascular responses in situations of threat or physical discomfort. The presence of sympathetic and baroreflex alterations in patients with panic suggests that the disorder may involve an important interface between emotional regulation, body perception, and cardiovascular control (14,20).

In social anxiety, the findings point to the participation of mechanisms autonomic and neuroendocrine in situations of assessment or social exposure. Reduced HRV in individuals with social anxiety indicates lower parasympathetic modulation, while changes in salivary alpha-amylase and cortisol suggest activation of the sympathoadrenérgic system and the hypothalamic-pituitary-adrenal axis in response to stressors. These results show that perceived social threat can trigger significant physiological responses, reinforcing the psychobiological nature of social anxiety (6,23). In this sense, social exposure is not only a cognitive experience of fear of judgment, but also an event capable of activating the body's defense systems.

Another relevant point is that the findings do not indicate a homogeneous autonomic dysfunction across all anxiety disorders. Although reduced HRV is a recurring pattern, the predominant mechanisms appear to vary according to the clinical subtype. In generalized anxiety disorder, the dysfunction seems more linked to persistent worry and vagal rigidity. In panic disorder, autonomic hyperreactivity stands out, the symptoms cardiorespiratory and baroreflex changes. In social anxiety, the findings suggest greater involvement of the physiological response to social stressors. This heterogeneity reinforces the need for clinical and investigative approaches that consider diagnostic and dimensional specificities (3,6-9,13,14,23).

The presence of autonomic changes also helps explain the high frequency of somatic symptoms in anxiety disorders. Many patients seek health services for physical manifestations before receiving a psychiatric diagnosis. Symptoms such

as tachycardia, pain or chest tightness, dyspnea, a sensation of fainting, sweating, tremors, and gastrointestinal discomfort may reflect dysregulated autonomic responses. When these symptoms are interpreted as signs of imminent danger, feedback occurs between bodily perception, fear, and physiological activation. This dynamic is particularly important in clinical practice because it can lead to repeated medical evaluations, excessive use of emergency services, and delays in the appropriate diagnosis of anxiety disorders.

The relationship between anxiety, autonomic dysfunction, and cardiovascular risk should also be considered. Sustained reductions in HRV have been associated, in different clinical contexts, with worse physiological adaptation and greater cardiovascular vulnerability. Although the studies included in this review do not allow us to establish a direct causal relationship between anxiety disorders and cardiovascular events, the findings suggest that autonomic dysfunction may represent an intermediate pathway between chronic psychological distress and systemic health changes (4, 12,27). This perspective broadens the clinical importance of anxiety disorders, indicating that their effects may extend beyond the emotional sphere and involve long-term physiological repercussions.

Autonomic assessment can therefore play a complementary role in clinical practice and research. Measures such as HRV, respiratory sinus arrhythmia, heart rate, blood pressure, baroreflex, cortisol, and salivary alpha-amylase may contribute to patients' psychophysiological characterization, severity stratification, monitoring therapeutic and understanding of clinical subgroups. However, these markers should not yet be interpreted in isolation as definitive diagnostic tools. Their value seems to be more associated with understanding the integrated functioning of the patient, especially when combined with clinical assessment, psychometric instruments, and analysis of contextual factors.

The therapeutic implications of the findings are relevant. If anxiety disorders involve low autonomic flexibility and reduced vagal modulation, interventions aimed at psychophysiological regulation may represent important complementary strategies. Techniques such as slow bre-

athing, HRV biofeedback, autogenic training, regular physical activity, mindfulness, cognitive behavioral psychotherapy, and interventions based on emotional regulation can help reduce hyperarousal and strengthen autonomic self-regulation. Studies included in this review indicate that both pharmacological interventions and psychophysiological strategies can modify autonomic parameters, although more robust and standardized clinical trials are still needed to confirm these effects (l5,24) .

The use of psychopharmaceuticals represents a central methodological issue. Some medications can directly alter heart rate, blood pressure, and HRV measures, making it difficult to interpret the relationship between anxiety and autonomic dysfunction. Studies such as those by Alvares et al. and Licht et al. highlighted the potential influence of psychotropic drugs on autonomic parameters, showing that part of the associations observed may be modulated by medication treatment (4,5). On the other hand, effective pharmacological interventions may also contribute to improved autonomic regulation, as suggested by the increase in HRV after paroxetine treatment in patients with panic disorder (l5). Thus, medications should be analyzed both as possible confounders and as therapeutic modulators of autonomic function.

The methodological heterogeneity of the included studies is an important limitation of the literature. Differences in the recording time of HRV, resting conditions, respiratory control, diagnostic instruments, the use of medications, comorbidities, sample size, and experimental protocols make direct comparison of results difficult. In addition, some studies used short HRV recordings, while others assessed prolonged periods, such as 24-hour measurements. This methodological variation may influence the findings and limit the standardization of conclusions. Future studies should adopt more uniform protocols, larger samples, and rigorous control of clinical and physiological variables.

Another limitation relates to the predominance of cross-sectional and observational studies, which makes causal inferences difficult. It is still not fully clear whether autonomic dysfunction precedes the development of anxiety diso-

rders, arises as a consequence of prolonged exposure to stress and worry, or whether both phenomena feed into each other over time. Longitudinal studies are needed to investigate whether autonomic measures can predict the onset, severity, recurrence, or response to treatment in different anxiety disorders.

It is also necessary to broaden understanding of transdiagnostic Transdiagnostic aspects. Anxiety frequently coexists with depression, post-traumatic stress disorder, obsessive-compulsive disorders, substance use, and chronic clinical conditions. This overlap makes it difficult to identify specific autonomic changes for each diagnosis. On the other hand, it may indicate that autonomic dysfunction is a shared marker of psychophysiological vulnerability,

present across different conditions marked by emotional distress, hypervigilance, and difficulty with regulation. Including studies on respiratory sinus arrhythmia and psychopathological dimensions reinforces this transdiagnostic perspective (25,26) .

Despite these limitations, this review shows that the literature provides consistent support for the association between anxiety disorders and autonomic dysfunction. Reduced HRV, lower vagal modulation, sympathetic hyperreactivity, and alterations in the stress response are recurrent findings, with pathophysiological and clinical relevance. These results strengthen the need for an integrated approach to anxiety disorders, considering not only subjective symptoms but also bodily and neurophysiological markers that participate in the expression and maintenance of the clinical condition.

Thus, anxiety disorders should be understood as conditions involving the interaction between cognitive, emotional, autonomic, and cardiovascular processes. This perspective broadens the scope of research and supports more comprehensive therapeutic approaches aimed at regulating the nervous system, reducing physiological hyperarousal, and strengthening adaptive capacity. Incorporating autonomic markers in future studies may help develop more accurate clinical models, personalized interventions, and follow-up strategies based on psychophysiological indicators.

CONCLUSION

This integrative review showed that anxiety disorders are associated with relevant changes in the functioning of the autonomic nervous system, especially due to reduced heart rate variability, lower vagal modulation, sympathetic hyperreactivity, baroreflex alterations, and an exacerbated physiological stress response. These findings demonstrate that pathological anxiety should not be understood only as a psychological or behavioral condition, but as a complex psychophysiological phenomenon resulting from the interaction between brain circuits for emotional regulation, peripheral autonomic mechanisms, and bodily defense responses.

Heart rate variability stood out as the main autonomic marker investigated in the studies analyzed, being consistently reduced in individuals with anxiety disorders. This reduction suggests less autonomic flexibility and less adaptive capacity in response to emotional, cognitive, and environmental stimuli. In addition, the decrease in vagal tone and alterations in respiratory sinus arrhythmia reinforce the hypothesis of impaired parasympathetic regulation, especially under conditions marked by persistent worry, hypervigilance, anticipatory fear, and intense somatic symptoms.

Findings also indicated that autonomic dysfunction may vary according to the subtype of anxiety disorder. In generalized anxiety disorder, chronic worry appears to contribute to the maintenance of prolonged patterns of physiological activation. In panic disorder, cardiovascular alterations, baroreflex changes, and intense autonomic responses to bodily cues and perceived threats stand out. In social anxiety, exposure to or anticipation of negative social evaluation may trigger relevant autonomic and neuroendocrine responses, involving both the sympathetic system and the hypothalamic–pituitary–adrenal axis.

From a clinical standpoint, understanding autonomic dysfunction in anxiety disorders broadens the interpretation of the physical symptoms frequently reported by patients, such as palpitations, sweating, tremors, shortness of breath, dizziness, and chest discomfort. These

symptoms may reflect real changes in physiological regulation and, when interpreted catastrophically, contribute to cycles of fear, avoidance, and worsening of the anxiety state. Thus, the assessment of autonomic markers may represent a complementary tool for characterization psychophysiological, monitoring therapeutic and clinical severity understanding.

Despite the consistency of the findings, the literature still presents important limitations, including heterogeneity in protocols for measuring heart rate variability, differences in diagnostic criteria, the influence of psychopharmaceuticals, the presence of comorbidities, and the predominance of cross-sectional studies. These limitations hinder causal inferences and reinforce the need for longitudinal studies, larger samples, greater methodological standardization, and rigorous control of clinical and physiological variables. and physiological.

It is concluded that anxiety disorders involve a significant interaction between emotional, cognitive, autonomic and cardiovascular. The incorporation of autonomic markers, especially heart rate variability and indices of vagal modulation, can contribute to more integrated and personalized clinical models. Future research should deepen the investigation into the role of these markers in risk prediction, in monitoring the response to therapy, and in developing interventions aimed at restoring autonomic flexibility and physiological self-regulation.

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