American Journal of
Biomedical Science & Research

www.biomedgrid.com
ISSN: 2642-1747

Mini Review

Factors Influencing the Development of Anticipatory Nausea and Vomiting in Cancer Patients

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To Cite This Article: *Batoul Barari Tajani*, Factors Influencing the Development of Anticipatory Nausea and Vomiting in Cancer Patients. Am J Biomed Sci & Res. 2025 29(2) AJBSR.MS.ID.003779,* **DOI:** 10.34297/AJBSR.2025.29.003779

Received: November 11, 2025; **Published:** November 24, 2025

Abstract

Anticipatory Nausea and Vomiting (ANV), a distinct subtype of chemotherapy-induced nausea and vomiting, is primarily associated with psychological processes. The key factors contributing to ANV include classical conditioning, demographic and treatment-related variables, as well as anxiety or negative expectations. Empirical evidence from laboratory models has validated these underlying mechanisms of ANV. The management of ANV may involve medical or pharmacological interventions, such as benzodiazepines and other psychotropic medications. However, behavioural treatments, particularly systematic desensitization, are considered the primary therapeutic approach for ANV. Additionally, certain complementary treatment modalities have demonstrated potential in alleviating ANV symptoms, warranting further investigation. This review will examine the foundational models of ANV and discuss the various treatment options available.

Keywords: ANV, Nausea and vomiting, Cancer patients, SACTs, CINV

Introduction

Anticipatory Nausea and Vomiting (ANV) is a prevalent issue among cancer patients, often arising as a consequence of chemotherapy-induced nausea or vomiting. Chemotherapy-Induced Nausea (CINV), which may occur acutely or with a delay following chemotherapy infusion, represents a common adverse effect of treatment. Notably, up to 20% of patients report experiencing nausea prior to any chemotherapy cycle, and up to 30% report this anticipatory, learned, or psychological nausea by the fourth chemotherapy cycle. The likelihood of developing ANV increases with repeated exposure to chemotherapy. Consequently, the development of ANV appears to conform to a classical conditioning model, wherein repeated pairings of unconditioned stimuli (i.e., chemotherapy) and conditioned stimuli (e.g., the clinic, the nurse) elicit nausea and vomiting even before the administration of emetogenic agents. This model, along with other psychological factors contributing to the development of ANV and laboratory validation of these models, is discussed below [1-2]. ANV frequently exhibit resistance to standard antiemetic pharmacological interventions. Given its psychological and

behavioural underpinnings, psychotropic medications may offer a more effective pharmaceutical approach. However, behavioural therapy remains the most effective strategy for managing ANV. This discussion examines these therapeutic modalities and considers emerging complementary and alternative therapies.

Psychological Mechanism in ANV

Psychological mechanisms and demographic factors play a significant role in influencing the onset, frequency, severity, and duration of ANV. Three distinct yet interrelated factors contribute to ANV: 1) classical conditioning, which may result in anticipatory nausea; 2) demographic, clinical, and treatment-related factors, which can predict the risk of anticipatory nausea; and 3) anxiety or negative expectancies, which may trigger and heighten sensitivity to anticipatory nausea. The National Cancer Institute has identified Pavlovian classical conditioning as the most suitable theoretical framework for elucidating the development of anticipatory nausea and vomiting [3]. Within the classical conditioning paradigm of ANV, an unconditioned stimulus, such as chemotherapy, which inherently provokes an unconditioned response, namely nausea, is associated with a conditioned stimulus. In the context of ANV, potential conditioned stimuli may encompass the visual and olfactory cues of the clinic, the presence of nurses, and the treatment room environment. Following repeated pairings of the unconditioned stimulus with the conditioned stimulus, the conditioned stimulus alone becomes adequate to trigger the conditioned response, which is nausea [4-6]. The nausea experienced by cancer patients is anticipatory, as it may commence even before the administration of the chemotherapeutic agent. The conditioned nature of ANV is corroborated by evidence indicating that the incidence and severity of ANV tend to escalate following successive chemotherapy cycles; this is attributable to the repeated pairings of the unconditioned and conditioned stimuli.

Cancer Patients Status and ANV

Various personal characteristics and events related to cancer treatment appear to increase the likelihood of a patient experiencing ANV. The National Cancer Institute identifies several variables associated with ANV. In addition to the emetogenic potential of the chemotherapeutic agent, being under the age of 50 and female are the most prevalent indicators of CINV and ANV. Other common factors associated with ANV include susceptibility to motion sickness, the emetogenic potential of chemotherapy agents, weakness, dizziness, or light headedness following chemotherapy, morning sickness during pregnancy, and nausea and vomiting after the most recent chemotherapy session [1,3,7].

New Insights into the Psychological Mechanisms Underlying Anticipatory Nausea and Vomiting: The Influence of Negative Expectancies

In addition to demographic and clinical variables that contribute to ANV, psychological, cognitive, and social factors also significantly influence the conditioned response associated with ANV. For instance, anxiety, self-absorption, and response expectancies have been identified as substantial contributors to the development of ANV [8-10]. Anxiety may affect the onset of ANV, at least partially, through negative expectancies [11,12]. as expectancies are known to influence the development of other learning-based conditioning effects [13].

Recent research into the behavioural determinants of ANV underscores the importance of patients' beliefs concerning symptom manifestation. Within the framework of the patient belief model of ANV development, healthcare professionals assume a crucial role by disseminating information about treatment side effects and addressing patients' concerns and expectations. Effective communication between patients and healthcare providers is vital in shaping patients' expectations for treatment outcomes, referred to as "response expectancies," which can significantly influence physical, emotional, and mental outcomes throughout the treatment process [14]. In a seminal contribution to this field, Kirsch (1985) argues that the anticipation or response expectancy of experiencing a physiological sensation, such as nausea, can evoke corresponding subjective experiences and directly affect both physiological and psychological outcomes [15]. The influence of response expectancies is substantial, as these expectancies explain variance in ANV beyond the emetogenicity of the chemotherapeutic agent and other established predictive factors for nausea, such as gender and age [14,16,17]. For instance, Roscoe et al. conducted a study involving 194 breast cancer patients who were about to commence chemotherapy with a doxorubicin-based regimen. The study revealed that patients who anticipated they were "very likely" to experience severe nausea were five times more likely to actually experience severe nausea compared to those who believed they were "very unlikely" to do so [18].

Expectations regarding nausea may reflect an individual's awareness of their susceptibility to this condition, informed by previous experiences such as nausea during pregnancy or a predisposition to motion sickness. However, these expectations are also influenced by socio-cultural factors and the information patients receive from clinicians, hospital staff, other patients, family, friends, and broader societal sources. Consequently, nausea expectations can be attributed to both internal and external origins, with the latter offering potential avenues for mitigating chemotherapy-induced nausea. Unlike other risk factors for nausea, nausea expectations are adaptable, presenting an opportunity for targeted intervention.

The British National Coordinating Centre for Health Technology Assessment conducted an extensive review of the expectancy literature. The authors evaluated 47,600 references for relevance and quality, ultimately selecting the 93 highest-quality studies for their analysis. Their findings suggest that expectancies significantly influence the development of symptoms and side effects,

concluding that response expectancies serve as a fundamental mechanism underlying the placebo effect [19]. Similarly, the nocebo hypothesis, as discussed by Hahn and Barsky et al., [20,21], posits that the anticipation of side effects can lead to the manifestation of symptoms. Furthermore, studies linking anxiety to the onset of nausea may, to some extent, reflect the construct of negative expectancies [22]. In conclusion, the assessment of patients for demographic, clinical, and psychological variables, alongside other cognitive and emotional vulnerabilities previously discussed, may assist healthcare providers in identifying individuals at risk of developing ANV. Currently, there is a need to establish a concise and comprehensive screening process for ANV-related risk factors and to integrate this process into the evaluation protocols of cancer treatment centres.

Aetiology of ANV

ANV is primarily conceptualized as a result of classical Pavlovian conditioning [23,24] which is theorized to occur within the central nervous system [25]. Nonetheless, the emergence of ANV may also be partially attributed to insufficient initial management of chemotherapy-induced nausea and vomiting [26]. Given the association between ANV and CINV, it is crucial to examine the aetiology of CINV.

The Pathophysiology of CINV Encompasses a Complex Interplay of Neuroanatomical and Peripheral Centres Associated with ANV

The mechanism underlying Chemotherapy-Induced Nausea and Vomiting (CINV) is not restricted to a single anatomical region; rather, it involves a complex interaction among neuroanatomical and peripheral centres, as well as various neurotransmitters and receptors. The central and peripheral regions implicated in this process include: a) the emetic or Vomiting Centre (VC), a cluster of neurons located in the medulla oblongata, which serves as the primary structure for coordinating nausea and vomiting; b) the Chemoreceptor Trigger Zone (CTZ) situated in the area postrema at the floor of the fourth ventricle of the brain; c) the vagal nerve afferents, which project from the Gastrointestinal (GI) tract to the nucleus of the solitary tract (NTS) and the dorsal motor nucleus of the vagus; and d) the Enterochromaffin Cells (EC) lining the GI tract [27-30]. Chemotherapeutic agents can induce emesis through afferent input at various sites, involving distinct mechanisms. These agents are toxic to ECs lining the GI mucosa, prompting them to release neurotransmitters such as dopamine, serotonin (5-HT), substance P (SP), acetylcholine, histamine, and gamma-aminobutyric acid (GABA) [31-34]. These neurotransmitters bind to the corresponding receptors on the abdominal vagal afferents [35-37], thereby activating them, which subsequently transmit the stimuli to the dorsal vagal complex, comprising the emetic/VC, the area postrema (CTZ), and the NTS. These sensory inputs are integrated, culminating in the activation of the emetic response [31]. Another potential source of afferent input inducing emesis involves the CTZ [38,39]. which is sensitive to chemical stimuli from drugs [40]. The blood-brain barrier in the CTZ is permeable to circulating mediators, allowing them to directly interact with the VC [40], thereby resulting in emesis.

Research has established that no single neurotransmitter is solely accountable for all occurrences of CINV [41], a phenomenon particularly evident in ANV. Furthermore, while the inhibition of specific pathways can mitigate vomiting, it does not necessarily alleviate nausea. This observation implies that the mechanisms underlying nausea and vomiting may involve distinct pathways and mediators. Additionally, post-treatment CINV is initiated by stimuli affecting the Chemoreceptor Trigger Zone (CTZ) and the Vomiting Centre (VC), whereas ANV arises when the VC is activated by perceptual stimuli, which are generated by personal thoughts, emotions, or sensory stimuli associated with chemotherapy [42]. Although ANV is less prevalent than post-treatment CINV, it poses a significant challenge as it causes greater discomfort in cancer patients undergoing chemotherapy and is generally more difficult to manage than acute CINV or Delayed Nausea and Vomiting (DNV) [43]. A more comprehensive understanding of the mechanisms underlying different types of CINV may facilitate the development of more effective antiemetic medications.

There exists a notable deficiency in animal models for CINV, and currently, no laboratory model adequately represents Anticipatory Nausea and Vomiting (ANV). Some investigations into ANV have utilized models such as body rotation to induce nausea and related symptoms in healthy individuals [44]. Body rotation elicits a range of symptoms collectively identified as motion sickness, with the most prominent in humans being nausea, vomiting, pallor, and cold sweating [45]. In this model, the afferent signals from the vestibular system serve as the unconditioned stimulus [44]. Research employing the body rotation model indicates that techniques such as the overshadowing procedure and latent inhibition may be effective in preventing and mitigating [46,6,47].

Pharmacological and Psychological Intervention

Evidence supports the efficacy of benzodiazepines administered on the day of or immediately prior to chemotherapy in mitigating ANV, Razavi et al conducted a double-blind, placebo-controlled study to evaluate the impact of incorporating low-dose alprazolam (0.5-2 mg per day) into a psychological support program that included progressive relaxation training. The study involved fifty-seven women undergoing adjuvant chemotherapy for early-stage breast cancer. The findings revealed a higher incidence of anticipatory nausea in the placebo group compared to the alprazolam group (18% vs. 0%, p=0.038). Additionally, there was a significantly greater use of sleep aids (hypnotics) in the placebo group (19% vs. 0%, $p < 0.05$). This small-scale study demonstrated that the adjunctive use of benzodiazepines may effectively reduce anticipatory symptoms in patients undergoing chemotherapy [48].

The implementation of recommended pharmacotherapy for

the prevention and treatment of chemotherapy-induced acute and delayed emesis is essential in mitigating the conditioned development of anticipatory symptoms. Advances in 5-HT3 antagonists and the development of NK-1 antagonists have significantly enhanced the management of acute and delayed CINV, thereby potentially reducing the incidence of ANV. Current guidelines [49] advocate for a three-drug regimen comprising a serotonin (5-HT3) receptor antagonist (palonosetron), dexamethasone, and an NK-1 receptor antagonist (aprepitant or fosaprepitant) for highly emetogenic chemotherapy agents, to be administered prior to the initiation of chemotherapy to prevent acute CINV. For acute CINV associated with moderately emetogenic agents, the combination of palonosetron and dexamethasone is recommended. In the context of low-risk chemotherapy regimens, the use of dexamethasone, a 5-HT3 receptor antagonist, or a dopamine receptor antagonist is advised. For delayed CINV, treatment options include dexamethasone and aprepitant, aprepitant alone, or dexamethasone alone. To prevent ANV, the Multinational Association of Supportive Care in Cancer (MASCC) committee appropriately recommends optimal control of acute and delayed CINV in conjunction with behavioural therapies [49].

Behavioural and psychological interventions remain the most efficacious strategies for managing ANV. The investigation of behavioural treatments for cancer-related nausea has been extensively documented in the medical literature for over three decades. Initial research on these interventions primarily focused on alleviating ANV rather than CINV, as ANV is a conditioned response that can be effectively managed through behavioural approaches grounded in learning principles. Studies on the behavioural treatment of conditioned adverse effects of chemotherapy have predominantly concentrated on two principal methods: Systematic Desensitization (SD) and hypnosis [50]. Additional behavioural techniques that have been explored include biofeedback [51], imagery, and various relaxation methods [52]. These interventions have consistently demonstrated efficacy in reducing ANV and decreasing levels of cancer-related anxiety and distress [53]. Notably, trials examining the behavioural treatment of ANV have shown these approaches to be effective even when patients' anxiety levels are not controlled [54], indicating that the impact of these interventions extends beyond mere anxiolytic effects. Regarding timing, interventions based on learning principles are most effective when implemented prior to the full development of the undesired conditioned response, such as nausea and vomiting [52].

Acknowledgement

The authors express their deepest gratitude to the Faculty of Pharmacy, M.S. Ramaiah University of Applied Science, all the nursing staff, and resident doctors of M.S. Ramaiah Medical College Hospital for their incredible cooperation.

Competing Interest

The authors declare no competing interests.

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