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The Development of Anticipatory Nausea and Vomiting in Cancer Chemotherapy Patients: A Prospective Study

by

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled, "The Development of Anticipatory Nausea and Vomiting in Cancer Chemotherapy Patients: A Prospective Study", submitted by Gary B. Challis in partial fulfillment of the requirements for the degree of Master of Science.

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ABSTRACT

The occurrence of nausea and/or vomiting in anticipation of a chemotherapy treatment session for cancer (ANV) is typically described in terms of a conditioned response. The respondent learning model predicts that ANV develops when an unconditioned response of postchemotherapy nausea and/or vomiting (PCNV) becomes associated with a treatment session which acts as the unconditioned stimulus. After a number of associations the unconditioned stimulus and other relevant stimuli act as conditioned stimuli capable of eliciting ANV. ANV has been reported in 25% to 35% of those chemotherapy patients who experience PCNV. Although levels of anxiety and the severity of PCNV may mediate the development of ANV, they are not reliable predictors of its occurrence. This research attempted to establish that two person-variables, absorption (a proclivity to become involved in imaginative pursuits) and autonomic perception (an awareness of one's physiological reactions), accurately predict which cancer patients will develop ANV.

Seventy cancer patients receiving chemotherapy were interviewed at home prior to their second treatment session in order to obtain baseline measures of absorption, autonomic perception, depression, state-trait anxiety and basic demographic information. Patients were then interviewed prior to each of their next six treatment

sessions at which time measures of depression, state anxiety, the severity and duration of PCNV, and their experience of ANV were obtained.

Previous findings suggesting that motion sickness, anxiety, depression, gender and age are predictors of the development of ANV were not replicated. Patients with ANV did score significantly higher on measures of absorption and autonomic perception than patients who did not develop this response. The results also provided support for a respondent learning model of ANV development. Those variables hypothesized to mediate conditioning (i.e., toxicity of treatment drugs, severity of PCNV and levels of state anxiety) accurately predicted which patients developed ANV (85.71% of cases correctly classified). When absorption and autonomic perception were added to the learning variables a significantly greater proportion of cases were correctly classified (95.71%).

These findings suggest that a respondent learning model is a necessary but not sufficient model for describing the development of ANV. The influence of cognitive processes needs to be considered in attempting to explain the occurrence of this response.

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To my best friend and wife Gail, I owe my love and thanks. Her contributions lie not only in the body of this thesis, but also in its spirit. The sacrifices which she has made can never be fully appreciated and it is to her that I dedicate this work.

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"Just as you cannot specify the contribution of each brain cell to an image, so you cannot specify the contribution of an individual to the collective representation". Emile Durkheim

INTRODUCTION

Individuals diagnosed with cancer must adjust physically and psychologically to the presence of a potentially life-threatening illness (Silberfarb, 1982). Although some of the stresses involved in this adjustment may abate once the disease and the effectiveness of various treatments are explained, for some patients the treatment and its side effects may prove more stressful than the actual diagnosis (Altmaier, Ross & Moore, 1982; Harrell, 1972; Ingle, Burish & Wallston, 1984; Worden & Sobel, 1977).

Chemotherapy is frequently employed in an attempt to control the abnormal cellular activity associated with some cancers (Cotanch, 1983; Holland, 1977; Redd, Rosenberger & Hendler, 1982). The drugs used in cancer treatment function to disrupt the DNA synthesis of malignant cells thereby rendering them benign (Burish & Lyles, 1981; Jerry & Challis, 1984). Unfortunately, the actions of these drugs are not strictly limited to the neoplastic cells but may also have a toxic effect upon all cells which normally undergo rapid differentiation (e.g., gastrointestinal tract, hair follicles, bone marrow; Burish & Lyles, 1981; Frytak & Moertel, 1981; Jerry & Challis, 1984). Interference with the normal functioning of these cells may lead to the occurrence of such physiological difficulties

as nausea, vomiting, alopecia, anorexia and diarrhea (Holland, 1979; Jerry & Challis, 1984; Morrow, 1982). A number of studies have indicated that these side effects may become so severe that they interfere with the effectiveness of a patient's specified treatment program (e.g., missed or delayed treatments, withdrawal from therapy; see Dolgin, Katz, McGinty & Seigel, 1985; Holland, 1979). Penta, Poster and Bruno (1983) reported that two-thirds of physicians surveyed stated that 1-5% of cancer chemotherapy patients refused treatment due to the severity of their postchemotherapy nausea and/or vomiting (PCNV). An additional 15.3% of physicians reported that 5-10% of their patients refused treatment for the same reason. A study by Maule and Perry (1983) reported that as many as 10% of cancer chemotherapy patients refuse further treatment due to PCNV.

Of the side effects which may develop as either a direct or secondary response to treatment, one of the most prevalent is nausea and/or vomiting (Ahles, Cohen, Little, Balducci, Dubbert & Keane, 1984; Frytak & Moertel, 1981; Golden, 1975; Zeltzer, Kellerman, Ellenberg & Dash, 1983). Nausea is defined as "an awareness of the urge to vomit and is accompanied by loss of gastric tone and peristalsis with contraction of the duodenum and reflux of intestinal contents into the stomach" (Seigel & Longo, 1981, p.352). Vomiting is the "forceful expulsion of gastrointestinal

contents through the mouth and is associated with powerful sustained contractions of the abdominal muscles and opening of the cardia" (Seigel & Longo, 1981, p.352). Chemotherapeutic agents interact with an area near the postrema of the fourth ventricle which has been named the 'chemoreceptor trigger zone' (Frytak & Moertel, 1981; Harris, 1978; Maule & Perry, 1983; Scott, Donahue, Mastrovito & Hakes, 1986). This interaction induces activation of another area located in the lateral reticular formation identified as the 'vomiting centre' (Lumsden & Holden, 1969). When the stimulation from these centres reaches the threshold for elicitation, vomiting occurs (Harris, 1978).

Post-Chemotherapy Nausea and Vomiting: PCNV occurs in direct response to the presence of toxic chemotherapeutic agents in one's system (e.g., Redd et al., 1982). In addition to the side effects which the drug treatments may themselves produce (see above), the persistence of PCNV can lead to a variety of additional, debilitating conditions (e.g., anemia, anorexia, severe fatigue, stomatitis; Cotanch, 1983; Frytak & Moertel, 1981; Nicholas, 1982; Penta, Poster, Bruno & MacDonald, 1981; Scogna & Smalley, 1979). As mentioned, it is possible that the severity of PCNV, and its related side effects, may actually force some patients to discontinue their treatment program (Nicholas, 1982; Penta et al., 1981; Zeltzer et al., 1983).

Anticipatory Nausea and Vomiting: Although previous research has primarily been concerned with the frequency and severity of PCNV (e.g., Weddington, Miller & Sweet, 1984), in recent years there has developed a growing body of literature concerned with those responses which may develop prior to a chemotherapy session (e.g., Andrykowski, Redd & Hatfield, 1982; Burish & Lyles, 1980; Chang, 1981; Morrow, 1982; Morrow, 1984b; Redd et al., 1982). Many of the problems associated with the occurrence of PCNV can be further compounded by the development of nausea and/or vomiting in anticipation of treatment (Cotanch, 1983).

The frequency and severity of anticipatory nausea and/or vomiting (ANV) is substantial enough to interfere with the treatments of a significant number of chemotherapy patients (Redd & Hendler, 1982; Weddington et al., 1984). The frequency of this response has been reported to range from 10% to 65% of those patients who are also experiencing PCNV (Altmaier et al., 1982; Coons, Leventhal, Nerenz, Love & Larson, 1987; Scogna & Smalley, 1979). The most commonly reported finding is that 25% to 35% of patients with PCNV will also develop ANV (Andrykowski et al., 1982; Morrow, 1982; Nerenz, Leventhal & Love, 1982; Redd et al., 1982).

Difficulties in Establishing the Incidence of ANV

The wide range of incidence rates for ANV reflects the difficulties that are encountered by investigators when they try to define the nature of the development of this response. These difficulties arise primarily in four specific areas.

First, there are concerns about the effect that the site or stage of cancer may have upon establishing accurate measures of the incidence of ANV. To this point the majority of studies have paid little attention to these variables due primarily to the fact that it is the treatment process and its effects, rather than the type of cancer, that are of importance (e.g., Morrow, 1982; Weddington et al., 1984). Weddington et al. (1984) found no significant differences between those who did or did not develop ANV on the basis of their diagnosis. Although the type and stage of cancer necessarily determine the drugs involved in treatment, and thus the potential for ANV, it remains the treatment and not the cancer which mediates response development. This is not to say that the type of cancer should not be considered. In fact, a number of cancers which directly involve the gastrointestinal tract may predispose an individual to the occurrence of nausea and/or vomiting as either a direct or indirect result of treatment (Berkow, 1977; Morrow, 1986). Obviously such a predisposition would interfere with an accurate

determination of the incidence of ANV. If, however, as a direct result of the nature of a patient's cancer their susceptibility to experiencing nausea or vomiting does not increase, then the site and stage of cancer need not be considered a determining factor in the development of ANV (e.g., Morrow, 1982).

Second, a number of difficulties arise concerning the method by which the severity, frequency and duration of both PCNV and ANV are measured (Burish & Carey, 1986). Previous retrospective studies have required patients to recall, not only the occurrence of PCNV or ANV episodes, but also their severity and duration (Morrow, 1984d). The difficulties which are related to the accuracy of retrospective reporting can be minimized by obtaining self-reported measures of these responses within a short interval after each occurrence. Additionally, the use of a standardized measure for recording various components of these responses would result in a more accurate, replicable reporting of these events.

Third, there has been considerable variation in the actual definition of what constitutes an occurrence of conditioned nausea and/or vomiting (Burish & Carey, 1986). Nausea, retching and gagging have all been treated as similar responses with no consideration given to the differences in their incidence rates or developmental processes. Additionally, some studies have classified

nausea and vomiting together as one response (e.g., Nicholas, 1982; Redd & Andrykowski, 1982). There are significant differences which exist with respect to the severity and incidence of these two responses making it essential that they be treated as two separate entitites.

Last, there is no accepted procedure that defines when a patient should be questioned about their experience of ANV during a course of treatment (Burish & Carey, 1986). Given that the incidence of ANV varies as a function of the number of treatment sessions a patient has received (see section below; Respondent Learning Model) then the reported incidence of this response obviously depends upon whether patients were asked after their second, third, fourth or fifth treatment. It is not surprising therefore, that reports of the incidence of ANV vary by as much as 50%. In order to obtain an accurate estimation of this rate it is essential that patients be followed for a minimum of four treatments to ensure that the response has had a sufficient time to develop. Prospective longitudinal studies would also alleviate this problem since they are not restricted to measuring prevalence rates after a certain minimal number of treatments, but rather, can follow patients across many treatment sessions.

Respondent Learning Model

The development of ANV is typically described in terms of a respondent learning model whereby the events and surroundings related to the treatment sessions become associated with the adverse effects of the chemical agents (e.g., Katz, 1982; Redd & Andrykowski, 1982; Redd et al., 1982). The injection of the toxic drugs used in therapy (unconditioned stimulus; UCS) function to elicit PCNV (unconditioned response; UCR). An association of the environmental factors related to treatment (conditioned stimulus; CS) with the occurrence of the UCS "may ultimately endow these stimuli with the capacity of eliciting anticipatory nausea" (conditioned response; CR) (Andrykowski et al., 1985, p.447). Once this response has been 'learned' it can be present during treatment, immediately prior to the injection of the drug, or up to twenty-four hours before treatment (Redd et al., 1982; Weddington et al., 1984).

If it is assumed that the development of ANV follows a respondent learning model, then patients exhibiting this response should be exposed to factors that facilitate its acquisition (e.g., more severe PCNV, greater number of UCS-UCR pairings; Andrykowski, 1985). Studies conducted by Weddington, Miller and Sweet (1982) and Wilcox et al. (1982) found that of those patients who experienced severe PCNV, 53% and 56%, respectively, also experienced ANV.

This rate is much higher than the standard 25% which is commonly reported and indicates that the stronger the salience of the UCR, the stronger the "conditioning" (e.g., Dolgin et al., 1985; Duigon, 1986; Love, Nerenz & Leventhal, 1983; Morrow, 1982; Morrow, 1984b; Nesse, Carli, Curtis & Kleinman, 1980; Nicholas, 1982; Scogna & Smalley, 1979; vanKomen & Redd, 1985). It follows, therefore, that the severity of a patient's ANV should be a function of the emetic potential of the drugs used in treatment, with higher emetic potential resulting in a greater severity of ANV (e.g., Morrow, 1982; Morrow, 1984b; Nesse et al., 1980; Nerenz, Leventhal, Easterling & Love, 1986).

Additionally, a respondent learning model of development would require that at least one UCS-UCR pairing be present before the CR can occur. At present there are no reported cases of ANV developing without the patient first experiencing PCNV. In fact, two to four treatment sessions (UCS-UCR pairings) are usually required before ANV will develop (Morrow, 1984b; Redd et al., 1982). Nicholas (1982) reported that, on average, patients developed ANV after 2.6 treatment sessions. Olafsdottir, Sjoden & Westling (1986) found that ANV was present, on average, by the fourth treatment ($\underline{M}$ = 3.3 treatment sessions).

In accordance with this model, the probability of the CR occurring should increase as the number of UCS-UCR pairings increase. Increased pairings, in turn, result in

stronger contiguous and contingent associations between the UCS and UCR, thereby facilitating the conditioning process. A variety of studies to date (e.g., Burish & Lyles, 1982; Morrow, 1982; Nesse et al., 1980; Redd et al., 1982; vanKomen & Redd, 1985; Yasko, 1985) have shown that as the number of treatment sessions increases there is a corresponding rise in the incidence of ANV.

The occurrence of ANV outside of the treatment setting indicates that even those elements which indirectly initiate some cognition concerning the upcoming treatment session (e.g., preparing to come to the clinic, the drive to the centre) may be capable of eliciting the response (Ahles, et al., 1984; Dobkin, Zeichner & Dickson-Parnell, 1985; Nicholas, 1982; Yasko, 1985). Duigon (1986) reported that patients experiencing ANV did so, on average, 17 hours prior to their treatment session. This behavior pattern of stimulus generalization (Marks, 1969; Nesse et al., 1980) also fits within the constraints of a respondent learning model.

Models of ANV Development

Bolles's (1972) expectancy theory of learning may explain the process by which ANV develops. If an individual has been exposed to a stimulus in conjunction with some other set of stimuli or cues, then these cues have a particular meaning for the individual. Due to the

development of an association between the two stimuli, the presence of one stimulus becomes a signal for the presentation of the second one (Tarby, 1982). Bolles states that given a particular contingency or relationship between two stimuli there develops a corresponding stimulus-stimulus expectancy. After a number of associations it becomes expected that when certain stimuli and/or behaviors related to a particular outcome are present, a second stimulus will be presented.

It is possible that ANV can be elicited due to the predictive qualities that the environmental and situational cues associated with treatment possess. Any reinforcement that maintains the accuracy of this stimulus-stimulus contingency (i.e., the expected PCNV occurs once treatment has been given) will improve the predictive quality of these cues. After a number of treatment session-PCNV associations the environmental stimuli associated with treatment act as accurate and predictive cues capable of eliciting "some response from the organism that is conditioned to (these) external stimuli" (Bolles, 1973, p.296).

Although the respondent learning paradigm is widely accepted as the most accurate model for describing the course of development of ANV, it is unable to explain why only a small percentage of cancer patients ever develop the response (Burish & Carey, 1986). In light of the

inadequacy of this model as it is currently stated, numerous attempts have been made to establish better models to identify prospectively those individuals at risk for the development of ANV. These will be discussed below along with the relevant research studies that have led to their postulation.

Taste Aversion: One theory proposes that the development of ANV is a result of learned taste aversions. Taste aversion is similar in development to poison bait shyness whereby animals quickly learn avoidance of particular tastes or smells that have previously resulted in illness (Bernstein & Webster, 1980; Nesse et al., 1986). In this paradigm, learning of the avoidance or shyness response can occur in one trial (Bernstein & Webster, 1980) even when there is little temporal contiguity between the UCS and UCR (Nerenz et al., 1986). One trial learning can account for the rapid development of ANV (after only one UCS-UCR pairing) that is problematic for the assumptions of a respondent learning model (Morrow, 1982). Additionally, the taste aversion model accounts for the persistence of conditioning despite the presence of a large temporal gap between the UCS and the UCR (Bernstein & Webster, 1980; Nerenz et al., 1986). The respondent learning model requires a close temporal association between the UCS and UCR to ensure the conditionability of the response.

Studies conducted by Nerenz, Leventhal and Love, (1982) and Fetting, Wilcox, Iwata, Criswell, Bosmajian and Sheilder (1983) provided further support for this theory by showing that individuals who reported changes in their sense of taste were more likely to develop the response of ANV. It follows that if learning taste aversions is the process through which ANV develops, then blocking tastes associated with treatment should significantly decrease the potential for ANV. A study conducted by Nerenz et al. (1986) found that the blocking of tastes does result in a lowered incidence of ANV. Additionally, the authors went on to recommend that in order to avoid negative conditioning towards some foods patients should eat a varied assortment of meals within the 24-hour period prior to their treatment.

Despite the findings in support of this theory, there is considerable evidence against a taste aversion model of ANV development (Andrykowski, 1987). Nerenz et al. (1986) subsequently discovered that once the effects of PCNV were accounted for statistically, the effects of taste were no longer significant. Whereas Nerenz et al. concede that these results prevent taste from being described as a direct cause of ANV, they point out that taste may serve as a predictor of the conditions which are necessary for ANV development, and further, that its presence may actually facilitate conditioning.

Additionally, Bernstein & Webster (1980) reported that it is possible for learned taste aversions to develop in patients receiving chemotherapy even if they do not experience PCNV. However, current research indicates that the occurrence of PCNV must occur before ANV can develop (Nesse et al., 1980; Andrykowski, 1985).

This is not to say that learned taste aversions are not occurring in cancer patients, nor that they do not function in some capacity within the respondent learning model; however, the implication that this is a complete model for the development of ANV is not supported by the research literature.

Locus of Control: Another proposed causal model concerns an individual's perception of locus of control over their disease and/or its treatment (Kellerman, Zeltzer, Ellenberg & Dash, 1983; Morrow, 1982; Zeltzer et al., 1983). It has been suggested that a patient's active participation in their own therapy may be an essential component to recovery (Altmaier et al., 1982; Hoffman, 1982; Zeltzer et al., 1983) and that the loss of control concerning the disease, treatment and side effects may have a severe effect upon their psychological adjustment to cancer (Morrow, 1982; Zeltzer et al., 1983). It is further argued by Zook and Yasko (1983) that such severe psychological effects may in turn lead to an increased level of nausea and/or vomiting in the individual.

This theory can account for the proven effectiveness of behavioral interventions due to the active role that the patients assume in their own therapy. Once patients have obtained control over some component of their disease or treatment, it is hypothesized that their susceptibility to ANV should be diminished.

Whereas a link between locus of control and the severity of nausea and/or vomiting may be present, studies to date have failed to find any significant associations between these factors and the development of ANV (Morrow, 1982; Zook & Yasko, 1983). Although feelings of loss of control are likely to be common amongst cancer patients undergoing chemotherapy, it does not appear that this factor predisposes an individual to ANV.

Conditioned Anxiety: It has also been proposed that the development of ANV is a result of conditioned anxiety. This model states that "nausea and vomiting are not conditioned directly, but are the result of an emotional state of anxiety or aversion that is conditioned to external cues" (Nerenz et al., 1986, p.225; see also, Rhodes, Watson & Johnson, 1986). In support of this model, behavioral interventions effective in eliminating the conditioned response of ANV all function to lower anxiety (Redd et al., 1982). There is also reported to be a significant positive correlation between those individuals who experience high levels of both state and trait anxiety

and their susceptibility to ANV (Burish & Lyles, 1981; Redd & Andrykowski, 1985; Redd et al., 1982). It is difficult to determine whether Burish and Lyles are correct in their assumption that high levels of state anxiety are precursors to the development of ANV. It may be that state anxiety is a result of ANV rather than a cause.

Although an association between anxiety and anticipatory nausea would be consistent with a conditioned anxiety model, it does not establish this as a causative mechanism. Nesse et al. (1980) failed to find any consistent pattern of results which would indicate that the level of anxiety which one experiences is capable of controlling the prevalence of ANV. The authors reject a theory of ANV development based solely upon levels of anxiety. They do, however, acknowledge that ANV develops as a conditioned response to external stimuli and that the removal of certain response cues, such as odours, may help to alleviate ANV. Morrow and Morrell (1982), although acknowledging the negative influences that anxiety can have upon the psychologic functioning of the individual, did not view anxiety reduction as the primary reason for the effectiveness of their procedure (systematic desensitization) in reducing ANV. They found that patients, both with ANV and without, exhibited the same levels of anxiety reduction as a result of the behavioral intervention.

Nesse et al. (1980) point out that there are no data to indicate that patients who experience ANV have previously experienced nausea or vomiting as a result of elevated levels of anxiety (see also, Morrow, 1982). It could be argued, however, that previous anxiety-producing situations were not comparable to the treatment situation. Nevertheless, Nesse et al. also point out that despite high anxiety levels, not all chemotherapy patients develop ANV. In fact further research by Nerenz et al. (1986) shows that, as with taste aversions, when the effects of PCNV are statistically controlled, the effects of anxiety are no longer significant.

Andrykowski et al. (1982) have argued that Spence's (1964) anxiety-conditionability theory is probably correct in suggesting that anxiety may facilitate, but not determine, the acquisition process of a variety of conditioned responses such as ANV (e.g., Carey & Burish, 1985).

Pharmacological Interventions for ANV

If it were possible to relieve the compounding effects of PCNV and ANV, perhaps fewer patients would be forced to prematurely end their treatment programs. The acceptance and tolerance of a specified chemotherapy program therefore requires the practitioner to ensure that both the direct effects of PCNV and its potential side effects, including

ANV, be kept at an absolute minimum (Scogna & Smalley, 1979).

A number of studies (e.g., Frytak & Moertel, 1981; Redd et al., 1982; Scogna & Smalley, 1979) have been conducted in an attempt to determine the effectiveness of antiemetic drugs in reducing the frequency and severity of both PCNV and ANV. Unfortunately, present pharmacological interventions have not proven to be entirely effective in controlling these responses (Burish & Carey, 1986; Chang, 1981; Morrow, 1986; Scogna & Smalley, 1979). Although resulting in a reduction of the severity of PCNV, the overall frequency of PCNV has proven relatively resistant to antiemetic intervention (Burish & Carey, 1986; Frytak & Moertel, 1981; Morrow, 1986; Nesse et al., 1980; Redd et al., 1982; Scogna & Smalley, 1979). Furthermore, the use of antiemetics has had little effect upon either the severity or frequency of ANV (Frytak & Moertel, 1981; Harris, 1981; Morrow, 1986; Redd et al., 1982; Zeltzer et al., 1983). In addition to the effects of the drugs used in treatment, the antiemetics may themselves produce unacceptable or detrimental side effects such as drowsiness and diarrhea (e.g., Burish & Carey, 1986).

A continued inability to alleviate the severity of both PCNV and ANV may lead to a severe deterioration of the patient's physiological and psychological condition (Cotanch, 1983; Hoagland, Morrow, Bennett & Carnike). If

patients are unable to endure the side-effects of the chemotherapy they receive, practitioners may occasionally reject other potentially useful treatments (Frytak & Moertel, 1981). As a result of continued PCNV/ANV the depressed nutritional status of the patient can also lead to a weakness in natural immunities, and thus, to an increased susceptibility to other illness and infection (Cotanch, 1983). Ultimately, this depressed state may further decrease the ability of the patient to withstand their current treatment procedures (Harris, 1978; Morrow, 1982). Wilcox, Fetting, Nettesheim and Abeloff (1982) established that of the 19% of patients in their study who stopped chemotherapy due to the occurrence of severe PCNV, over 70% were also experiencing ANV. This finding raises the question would patient noncompliance decrease if ANV were prevented or controlled?

Behavioral Interventions for ANV

Due to the ineffectiveness of present pharmacological interventions to control the response of ANV, a number of behavioral procedures have been applied in an attempt to ameliorate this response. It follows that if the respondent learning model illustrates the true nature of the acquisition process of this response, then counter-conditioning strategies should prove effective in suppressing ANV (Hoffman, 1982; Morrow & Morrel, 1982; Redd

& Andrykowski, 1982; Redd et al., 1982). Morrow (1982) states that the argument that "ANV is a conditioning phenomenon because conditioning-derived treatments have been shown effective is clearly circular" (p.396). However, he goes on to point out that due to the variety of behavioral interventions that have been proven effective in controlling ANV, there is "a degree of support for viewing ANV as a conditioned response" (p.396).

Systematic Desensitization: Systematic desensitization has been suggested as a method of behavioral intervention that may be capable of controlling the occurrence of ANV (Hoffman, 1982; Morrow, 1982; Morrow, 1984a; Zeltzer et al., 1983). While this form of intervention has been shown to be successful in reducing the severity and duration of anticipatory nausea, its effectiveness with anticipatory vomiting has been questioned (Morrow, 1986).

Hoffman (1982) successfully combined systematic desensitization with hypnosis in an attempt to control these responses. In his conclusion he argued that systematic desensitization was the essential component for his successful procedure, with hypnosis being the most effective 'tool' through which desensitization can be introduced. Morrow (1986) established that systematic desensitization was more effective than muscle relaxation alone in reducing the severity and duration of anticipatory nausea. Morrow noted that the increased effectivness of

this procedure may have been a result of the effect that it had upon the patients' cognitively evaluated anxiety. The conditioned response of ANV may be disrupted if patients "are not able to focus on (or remember) potential conditioned stimuli connected with the clinic" (p.442).

Hypnosis: LaBaw, Holton, Tewell and Eccles (1975) taught self-hypnosis procedures in group sessions to pediatric oncology patients. Patients who experienced hypnosis prior to their treatment session reported a reduction in their subjective levels of anxiety, depression and the occurrence of anticipatory nausea (see also, Dempster et al., 1976). Redd et al. (1982) also reported that patients who were given hypnosis before a treatment were able to reduce their anticipatory nausea and completely eliminate their anticipatory vomiting. Six subjects who had previously experienced ANV were involved in this study. Each was taught a hypnotic technique that induced deep muscle relaxation. After successfully controlling their ANV, three patients chose not to utilize their hypnotic techniques during one of their treatments. It is interesting to note that the anticipatory symptoms returned at this time and then were once again controlled when hypnosis was reintroduced. The authors point out that the hypnotic procedures are quite similar to those utilized with deep muscle relaxation. They state, "one might well wonder if that was not what was operating" (p.18).

Hypnosis consists of a package of techniques and interventions of a psychological nature and is not a single "technique" (Spanos, 1986). As with the use of hypnosis to alleviate chronic pain, it is not clear how each of these may be relevant to the reduction of ANV (Stam, McGrath & Brooke, 1984).

Relaxation: Another technique reported to be effective for reducing ANV is muscle relaxation therapy (Burish & Lyles, 1979, 1981; Cotanch, 1983; Morrow, 1986; Redd et al., 1982). A number of studies have shown that muscle relaxation, with or without guided imagery or hypnosis, is effective in reducing both the severity and frequency of ANV (e.g., Burish & Lyles, 1979, 1981; Cotanch, 1983; Dash, 1980; Redd et al., 1982).

Mechanisms of Action of Behavioral Interventions

Distraction: The effectiveness of these procedures in reducing the severity of ANV and PCNV may be due, in part, to the presence of a "large psychogenic element involved in both (the) etiology and treatment of nausea and vomiting" (Frytak & Moertel, 1981, p.394). Thus, distraction has been proposed as the mechanism responsible for the success of these intervention procedures (Burish & Lyles, 1981; Cotanch, 1983). Behavioral interventions necessarily focus the patient's attention on the procedure itself. The literature consistently identifies patients at risk for ANV

as reporting cognitions or ruminations concerning their upcoming treatment (Burish & Lyles, 1981; Cotanch, 1983; Morrow, 1986). Distracting activities may interrupt a patient's ruminations concerning the treatment and its side effects, thereby distracting the individual from those cues which have become conditioned to the chemotherapy session. Recent research (Redd, Jacobsen, Die-Trill, Dermatis, McEvoy & Holland, 1987) has shown that the cognitive distraction achieved by involving pediatric patients in video games has resulted in significant decreases in the occurrence of ANV.

Locus of Control: Active participation in one's own therapy has also been suggested as a possible reason for the effectiveness of these behavioral interventions. It has been hypothesized by some authors (e.g., Burish & Lyles, 1981; Cotanch, 1983) that the perception of control may actually help to reduce a patient's level of anxiety. It is further stated that this increased calm may function to interrupt the physiological arousal that accompanies stressful situations (see above; Burish & Lyles, 1981). Despite these theoretical processes, locus of control has not been shown to be related to reductions in ANV (Morrow, 1982; Zook & Yasko, 1983).

Relaxation: It is possible that procedures such as hypnosis, muscle relaxation and systematic desensitization are effective simply by reducing "anxiety and physiological

arousal, thereby reducing side effects such as gastrointestinal upset" (Lyles, Burish, Krozely & Oldham, 1982, p.522). The feelings of relaxation and lowered levels of arousal are incompatible with feelings commonly associated with nausea and vomiting (Paul, 1969). Muscle relaxation and the subsequent lowered levels of physiological arousal function so as to interrupt and/or inhibit the muscle activity which is necessary for nausea and/or vomiting to occur (Lumsden & Holden, 1969).

Social Support: It is also possible that the increased attention which is garnered by a patient's involvement in a program of psychological treatment may be important in the success of the intervention. The involvement of the therapist may function to increase the supportive network of the patient (Cotanch, 1983). This, however, implies that patients who require intervention (i.e., those with ANV) are somehow deficient with respect to their social support network. Previous studies indicate that it is unlikely that social support is sufficient in and of itself to account for the noted effectiveness of behavioral procedures (Burish & Lyles, 1981). Burish and Lyles also point out that patients commonly develop a close relationship with their primary care nurses and thus the added support that would come from a therapist is unlikely to account for the effectiveness of these procedures. Additional prospective research concerning baseline levels

of patients' social networks are required to establish the relevance of this factor in ANV development.

An unfortunate drawback of these behavioral interventions is that they are not employed until such time as ANV has been noted to be reliably occurring (commonly after two to four treatment sessions). It is not practical to enroll every individual patient in a form of behavioral therapy for ANV when only a minority of patients are at risk for development of the response. On the other hand, the time lag between identification of a patient who develops ANV and the successful implementation of a behavioral intervention exposes that patient to a number of sessions of both PCNV and ANV. It is therefore essential that prospective studies be conducted so that behavioral interventions can begin prior to the development of these controllable side effects to treatment.

Individual Differences as Mediators of Conditioning

The respondent learning paradigm remains the most parsimonious model of ANV. Unfortunately, as was noted earlier, this model fails to address the problem of why only 25% of patients develop ANV (Burish, 1986; Dolgin, 1985). It has been suggested that the lower rate of conditioning is due to the presence of individual differences that function to mediate the conditionability of the patient (vanKomen & Redd, 1985).

VanKomen and Redd (1985, p.191) argued "it is possible that patients who experience ANV can be characterized by a personality profile distinct from patients who do not develop this response" (see also, Andrykowski, 1985; Carey & Burish, 1985). Most studies to date have been conducted with patients who have, already, reliably exhibited the conditioned response (after 2-4 treatments). The inability to identify those patients at risk for this response makes it difficult to retrospectively differentiate those factors that are causes of the response from those that are reactions to it (Burish & Carey, 1986). Prospective identification would permit pre-treatment interventions capable of preventing this response (Carey & Burish, 1985). In addition to the learning variables already mentioned, the identification of treatment, environmental or psychological factors that may be mediating this response should allow for a more accurate explanation of the process by which ANV develops.

Whereas environmental (salience of cues) and treatment (toxicity of drugs, number of treatments) factors may determine whether or not ANV can develop in a given setting, they are unable to account for the fact that only some patients develop ANV in the same setting. Given the constraints of the respondent learning model, a number of personality variables have been proposed that may be capable of mediating the conditioning process.

Locus of Control: Although locus of control was insufficient as a model of ANV development, within the respondent learning model it has been argued that it may provide important information concerning conditionability (Morrow & Morrell, 1982). Kellerman et al. (1983), Morrow and Morrell (1982) and Zeltzer et al. (1983) have each failed to find any significant differences in measures of locus of control between cancer patients who developed ANV and those who did not. There is evidence which also suggests that those patients who sense a lack of control may actually be able to "minimize the severity of any pain or discomfort which they experience" (Nehemkis et al., 1982, p.226). Therefore, despite numerous studies relating to the importance of this variable, the available literature does not suppport locus of control as either a mediating factor in the conditioning process or as a theory of response development.

Taste Aversion: Whereas taste aversion was also an inadequate model for explaining the developmental process of ANV, it may be possible that within the respondent learning paradigm it functions to mediate conditioning. Individuals who are capable of "tasting" the drugs used in treatment may be those same individuals who are at risk for ANV development (Nerenz et al., 1986). However, as with the taste aversion model, studies to date have failed to indicate any significant differences between those patients

who develop ANV and those who do not (Andrykowski, 1987). Furthermore, Andrykowski (1987) found that only a small percentage (5.8%) of patients develop taste changes, thereby preventing this variable from being considered an accurate predictor of ANV (see also, Fetting et al., 1984).

Depression: A number of authors have argued that a patient's level of depression may function to increase or decrease their susceptibility to ANV (e.g., Altmaier et al., 1982; Nerenz et al., 1982; Yasko, 1985). Studies have shown that those patients who exhibit ANV are characterized by both a significantly higher incidence (Altmaier et al., 1982) and level of depression (Nerenz et al., 1982). However, as previously stated, the lack of information from prospective studies has made it impossible to determine whether this depression is characteristic of at risk individuals or simply a response to the occurrence of ANV.

Demographic Variables: Demographic variables such as age, sex, race and marital status have also been proposed as factors within the respondent learning model that may mediate ANV conditioning (e.g., Morrow, 1982; Morrow, 1984b; Duigon, 1986). As evidence of the inconsistency in results that currently exists concerning the importance of these factors, a study by Weddington et al. (1982) failed to find significant differences between those patients with or without ANV on any of these demographic variables.

There are reports that suggest those individuals at risk for ANV are younger and more likely to be female (Morrow, 1984b; Nerenz et al., 1983; Yasko, 1985). However, reports failing to find this relationship are just as common (e.g., Andrykowski, 1985; Dolgin et al., 1985; vanKomen & Redd, 1984; Weddington et al., 1982). Andrykowski (1985) argues that the tendency for younger patients to be reported as being more susceptible to ANV may not be a function of their age, but rather may actually be a confound due to the increased severity of their drug treatment programs (e.g., Zeltzer et al., 1983). Marital status was not found to be associated with ANV development by Morrow (1982) or Duigon (1986); however, Fetting et al. (1983) did find such a relationship. Further research with regards to the possible effects of an individual's social support network is necessary before the importance of this component can be established.

Motion Sickness: Morrow (1984b, 1984c) has suggested that an individual's susceptibility to motion sickness may be a predisposing element for those experiencing ANV. Morrow also notes that this may indicate a vestibular component is involved in the development of nausea and that the current ineffectiveness of antiemetics is due to their failure to address this aspect of development. This finding has not been replicated by other investigators (e.g., Dolgin et al., 1986; Nesse et al., 1980).

Physical Symptoms: Individuals with ANV have also been characterized by a variety of pretreatment measures. Those with ANV have been shown to exhibit significantly more fatigue during the 24 hours prior to their session (Dobkin et al., 1984); however, contradictory evidence has been reported by vanKomen and Redd (1984). There have also been reports that individuals with ANV can be characterized by their experiences of severe diarrhea (Morrow, 1985), dry skin or itching (Nicholas, 1982), constipation or extreme fatigue (vanKomen & Redd, 1984). Despite significant findings in each of these studies, there have been no consistent replications of these results. Additionally, the failure to develop a theoretical basis for establishing these individual factors as mediators of response development prevents them from being considered as reliable predictors of ANV.

Delay of PCNV: Morrow (1982) has reported that patients with ANV experience PCNV within the first four hours after treatment whereas those without ANV do not develop PCNV until four to eight hours after treatment. On the basis of these results Morrow attempted to show the importance of temporal contiguity in ANV development. Unfortunately, Dobkin (1985) failed to find any significant differences between individuals who developed ANV and those who did not with respect to when they first experienced PCNV. As it is currently stated, the respondent learning model requires a

close temporal relationship between the UCS and the UCR. Until such time as the respondent learning model can account for the occurrence of conditioning despite the presence of the large temporal gap, its ability to function as a precise model for ANV development remains in question.

Previous Illness: There have been no differences reported between individuals and their susceptibility to ANV on the basis of their type of cancer or previous experience with chemotherapy, surgery or radiation treatments (Duigon et al, 1986; vanKomen & Redd, 1985; Weddington et al., 1982). As a result, most studies have not precluded enrolling cancer patients with previous histories of the disease as subjects in their research.

Coping Strategies: Previous cancer patients with long histories of the disease may have developed coping strategies which aid them in suppressing certain responses. Research by Altmaier et al. (1982) and Schwarz, Michel and Hornburg (1985) has suggested that patients with ANV have inadequate coping strategies for the stresses to which they are exposed. Additionally, it is possible that the effectiveness of the behavioral interventions for ANV may be due to the development of a coping strategy capable of dealing with the conditioned response. Although it may be important to evaluate a patient's style of coping with certain stressors in order to estimate the potential for

developing ANV, it is not clear how such a model would fit within the respondent learning model, if at all.

Prospective Studies of ANV Development

Few prospective studies have been conducted to predict the occurrence of ANV, thereby making it difficult to differentiate between potential causes for and responses to ANV. Two studies (Andrykowski et al., 1985; Nerenz et al., 1986) have attempted to measure personality characteristics that may predict which patients are at risk for developing ANV.

Andrykowski et al. (1985) followed 71 patients for a period of six months. After initial baseline measures, patients were questioned concerning their severity of nausea and vomiting and their state anxiety levels, before and after each treatment session for a period of six months. Patients responded to questionnaire measures of state-trait anxiety, the Eysenck Personality Inventory, the severity of PCNV and the presence of a variety of physical symptoms. The results indicated that patients with ANV experienced more severe PCNV and greater state anxiety than did those without ANV. These findings supported the respondent learning model of ANV development. The authors state that these findings provide important information concerning the developmental process of ANV and thus permit a better understanding of which intervention strategies

should prove effective. There is, however, no further discussion concerning the person-variables that may put an individual at risk for the development of ANV.

Nerenz et al. (1986) attempted to identify those factors that were predictive of the development of ANV. Data from 192 patients were collected immediately prior to their first treatment and subsequently prior to their second, third, fourth and sixth treatment sessions. Measures of anxiety before injection, the occurrence of taste during injection, the severity and duration of PCNV and the occurrence of ANV were obtained. Of the 192 patients interviewed 38.5% experienced ANV. The development of ANV was associated with higher levels of anxiety prior to injection as well as the occurrence of taste during the injection. However, the variables with the greatest significance related to the severity and duration of a patient's PCNV. In fact, when the effects of this variable were accounted for statistically, anxiety and taste were no longer significant ($p < .05$). Taste was, however, significantly related to the patient's reported severity of PCNV. In accordance with a conditioning model, the authors state that taste may facilitate the development of ANV due to the increased salience of the UCR (PCNV).

To date these are the only two prospective studies which attempt to outline the person and situation variables that may be mediating ANV development. The present study

was designed to replicate and extend these two studies and to further define the nature of the population at risk for the development of ANV.

Unassessed Variables of Conditioning

A variety of potentially useful variables have not been adequately assessed in previous studies of ANV.

Social Network: An individual's social network has been indirectly implicated as an element which may have a mediating capacity for the development of ANV (Fetting et al., 1983). The finding of a difference in ANV development between married and single patients suggests there is a difference in patients' supportive networks (Fetting et al., 1983). Berkman and Syme (1979) indicate that "social support may be protective against the harmful health consequences associated with stressful life events" (p.186). Whereas marital status has been considered, a direct investigation of social networks has yet to be conducted.

Socio-Economic Status: At the present time only one study has addressed the element of an individual's socio-economic status (Morrow, 1986). Morrow was uable to report any significant differences between patients with or without ANV on the basis of their socio-economic status. Although we have no basis for hypothesizing any differences concerning the process of ANV development with respect to

this dimension, its importance as a potential mediating variable remains to be accurately assessed.

Physiological Arousal: One individual difference which has received only a cursory investigation is the level of physiological arousal. A common element in each of the behavioral interventions proven effective in reducing the occurrence of ANV is its ability to also reduce the patient's subjective level of physiological arousal (Burish & Lyles, 1981). Paul (1969) reported that lowered levels of physiological arousal are incompatible with the feelings commonly associated with nausea and/or vomiting. Lumsden and Holden (1969) found that by lowering levels of physiological arousal it is possible to interrupt the muscle sequence that functions during vomiting.

Whereas high levels of anxiety are insufficient to accurately identify those individuals at risk for ANV, it is possible that the physiological arousal commonly associated with anxiety is capable of eliciting the response (e.g., Burish & Lyles, 1981; Cotanch, 1983; Redd et al., 1982). Ahles et al. (1984) reported that patients who developed ANV had increased levels of physiological arousal. Morrow (1982) stated that increased levels of physiological arousal may predispose some individuals to gastrointestinal upset and nausea.

Although low levels of arousal are associated with a lower frequency of nausea and/or vomiting, high levels have

not been shown to be reliable predictors of ANV (Frytak & Moertel, 1981; Harris, 1978; Redd et al., 1982). The lack of supportive data may be due to the fact that the majority of studies have primarily been concerned with actual rather than perceived levels of physiological arousal (Katkin, 1984).

Mandler, Mandler and Uviller (1958) argued that in stressful situations, subjects' cognitive perceptions of their own physiological arousal, rather than actual physiological measures, correlated with their true level of disturbance (e.g., Costello, 1971). In order to measure an individual's perceived level of physiological arousal in an anxiety-provoking situation Mandler et al. (1958) developed the Autonomic Perception Questionnaire (APQ). Mandler (1984) stated that those individuals who exhibit high levels of awareness concerning their autonomic activity, as measured by the APQ, are more reactive to stressful stimuli and also overestimate their true level of physiological arousal. Mandler argued that individuals will respond to stimuli in a manner which corresponds to their perceived level rather than their actual physical level of arousal. Borkovec (1977b) argued that the APQ characterizes perception of an autonomic response rather than an exact estimate of that event. A study conducted by Olafsdottir et al. (1986), stated that the APQ, a measure of one's

perception of autonomic arousal, was significantly correlated with measures of anxiety and nausea. It is suggested that due to this differential mode of responding to stressful stimuli the APQ may be a useful tool for differentiating, and thus predicting, whether or not an individual will be at risk for the development of ANV (Olafsdottir et al., 1986).

Although the APQ is hypothesized to increase the ability to prospectively identify the nature of the group at risk for ANV, there remains the question of identifying the cognitive components which mediate autonomic perception. The determination of the cognitive elements involved in both the activation of anticipatory reactions and one's autonomic perceptual ability should allow for a precise delineation of those individuals who will or will not develop ANV.

Absorption: Absorption refers to the cognitive ability or predisposition to become highly involved in sensory and imaginative experiences and an openness to self-altering experiences (Tellegen & Atkinson, 1974). Questionnaire measures of absorption assess an individual's tendency to become absorbed in such imaginative activities as day-dreaming, watching movies and listening to poetry (Finke & MacDonald, 1978; Tellegen & Atkinson, 1974). The Absorption Scale developed by Tellegen and Atkinson measures an individual's "disposition for having episodes

of total attention that fully engage one's representational (i.e., perceptual, enactive, imaginative and ideational) resources" (p.268).

A host of studies have indicated that absorption is a reliable, valid and unique component of personality that cannot be accounted for under typical over-arching personality constructs such as extraversion-introversion or stability-neuroticism (e.g., O'Grady, 1980; Pekala, Wenger & Levine, 1985; Tellegen & Atkinson, 1974). Nor is this measure correlated with scores on social desirability, locus of control or state-trait anxiety (O'Grady, 1980). Furthermore, it has been found to correlate with such variables as hypnotic susceptibility (Tellegen & Atkinson, 1974; Spanos & McPeake, 1975; Spanos, Stam, Rivers & Radtke, 1980), the ability to recall dreams and the frequency with which an individual reports dreams (Spanos, Stam, Radtke & Nightingale, 1980), skills in meditation (Greenfield, 1977), response to EMG biofeedback (Qualls & Sheehan, 1981) and self-reported changes associated with marijuana use (Fabian & Fishkin, 1981).

One component which appears common amongst those patients who develop ANV is their self-reported cognitive activity prior to treatment. Dobkin et al. (1985) stated that of those patients exhibiting the response of ANV, 40% reported ruminating about their upcoming treatment. Redd et al. (1982) implied that a variety of personal factors

including cognitive stimuli are functioning to provoke ANV. Yasko (1985) also reported that those individuals experiencing ANV could be characterized by having significantly more thoughts related to their chemotherapy (e.g., Duigon, 1984; Weddington et al., 1982). Additionally, van Komen and Redd (1985) stated that 75% of their patients claimed that their anticipatory responses were elicited by thoughts about the treatment session and its possible side effects.

The ability of those patients who develop ANV to become absorbed in imaginative activities related to their treatment might account for a number of shortcomings in the respondent learning model. The question of how conditioning persists given the long temporal gap between UCS presentation and UCR could be due to the ability of absorbers to maintain, cognitively, specific components of the UCS thereby functionally bridging the time interval.

Absorption could also account for both the rapidity with which the response of ANV can develop and the extensive stimulus generalization that occurs. First, individuals who score high on measures of absorption may be able to vividly and clearly imagine various components of the UCS-UCR pairing. It is possible that this ability increases the salience of the UCS and UCR and thereby facilitate conditioning. In support of this notion, Mathews (1971) found that a conditioned response can be

reliably produced while an individual who is high in absorption simply imagines the conditioned stimuli. Thus, the sub-group of cancer patients who are high absorbers should be at the highest risk for the development of ANV. Subsequently, the more negative the event is perceived to be, the more salient the conditioned stimuli surrounding the treatment session will become, and thus, the stronger the conditionability of the patient.

If it is true that absorption is capable of mediating a conditioned response such as ANV, the relationship between absorption and state anxiety needs to be clarified. The increased salience of the UCS-UCR pairings, as a direct result of high absorption levels, may also be functioning to elicit high levels of pretreatment state anxiety. The common finding that levels of state anxiety are elevated prior to treatment may therefore be a function of a patient's level of absorption. High levels of absorption lead to greater state anxiety and thus, ultimately, to the development of ANV.

Although the relationship between autonomic perception and measures of absorption has yet to be analyzed, it is likely that these two elements will be positively and moderately correlated with each other. Given that autonomic perception requires some evaluative cognitive functioning in order to assess one's level of physiological

arousal, it is hypothesized that high levels of absorption will facilitate this task.

Summary

It is hypothesized that individuals who are characterized by high levels of absorption will also obtain high scores on the APQ measure. The presence of these two cognitive variables should function to increase their conditionability and thus their susceptibility to the development of ANV. The measurement of these two, stable, abilities should ultimately allow for an accurate, prospective identification of those individuals who will develop the conditioned response of ANV. Although the respondent learning model will be accepted as the process through which this response develops, it is argued that the occurrence and maintenance of ANV is a far more complex phenomenon that cannot be accounted for by this model alone. Cognitive and imaginative elements are believed to mediate both the development and amelioration of ANV.

Hypotheses

The first hypothesis is that absorption and perceived levels of arousal will be weakly correlated with measures of trait anxiety and depressive symptoms. In other words, these two cognitive measures are hypothesized to be tapping unique personality dimensions.

The second hypothesis is that when controlling for the toxicity of drugs used in therapy and the number of treatment sessions, high absorption subjects will demonstrate higher levels of pretreatment anxiety than will low absorption subjects.

The third hypothesis concerns the prediction of ANV from a respondent learning model. Specifically, the presence of higher levels of prechemotherapy state anxiety, more toxic drugs, greater severity and duration of PCNV and a greater number of treatment sessions are expected to predict ANV.

The presence of the individual cognitive components of perceived physiological arousal and absorption are believed to facilitate an individual's ability to acquire the conditioned response of ANV. The fourth hypothesis is that absorption and perceived arousal will predict a greater proportion of patients who develop ANV than will state anxiety, toxicity of drugs and PCNV severity and duration.

METHOD

Subjects

Seventy oncology patients receiving I.V. chemotherapy, 21 males and 49 females, were recruited over a one-year period from the Tom Baker Cancer Centre, Calgary, Alberta. All new cancer patients were contacted through the primary nurses in the Day Care Unit at this centre. A new patient was defined as having received no chemotherapy treatments within the past year. Patients were required to be 18 years of age or older at the time of the study and to be receiving I.V. chemotherapy as their only form of treatment. Subjects must also have been scheduled for a minimum of five treatment sessions to ensure a sufficient number of UCS-UCR pairings for the response of ANV to develop. Patients were interviewed at home after their first yet prior to their second treatment session. At this time they were required to respond to questionnaire measures of anxiety, depression, autonomic arousal and absorption (see Materials section). Patients were then seen immediately prior to a minimum of four to a maximum of six of their subsequent chemotherapy treatment sessions at which time they responded to questionnaire measures of anxiety, depression and a variety of physical symptoms.

Materials

The Absorption scale (Tellegen & Atkinson, 1974) is a 40-item true or false questionnaire which measures the extent to which an individual is capable of becoming involved in everyday imaginative pursuits (e.g., Do you like to watch cloud shapes change in the sky?). Of the 40 items on this questionnaire 20 are scored and 20 are distractors. This measure was shown to have only a .15 correlation with Spielberger's state anxiety measure indicating that absorption accounts for a significant proportion of the variance not accounted for by state anxiety (O'Grady, 1980). Additional research by O'Grady provides support for the discriminant validity of this scale. According to O'Grady (1980) the absorption scale measures a distinct dimension of an individual's personality that is neglected by other personality scales.

The Autonomic Perception Questionnaire (APQ; Mandler, et al., 1958) is a 21-item scale which measures the patients' perceptions of their autonomic responses when in an anxious situation (e.g., Does your heart beat faster?). Subjects indicate on a ten-point scale the extent to which they typically experience each of the items on the scale. According to Olafsdottir et al. (1986), the APQ is a useful tool for the establishing whether or not an individual is likely to experience anxiety and/or nausea. Hodges (1976) reported that the APQ is significantly correlated with the

Taylor Manifest Anxiety Scale (r = .27). However, Mandler, Mandler, Kremen & Sholiton (1961) reported that this correlation was insignificant (r = .15) when individuals were tested in stressful situations. The APQ has also been shown to have a good test-retest reliability (r = 0.71) (Borkovec, 1974). Whereas initial research by Mandler et al. (1958) indicated that the APQ was an accurate measure of autonomic reactivity, subsequent research has failed to support this finding (e.g., Whitehead, Drescher & Blackwell, 1976). Borkovec (1976), however, has suggested that individuals who score high on this measure can be characterized by their increased attention to their levels of physiological arousal. High scorers tend to overestimate the extent of this arousal and appear to respond, behaviorally, in accordance with their perceived rather than actual level of physiological arousal (Mandler, 1984). As Borkovec points out, high scorers appear more reactive to stressful stimuli than do low absorbers.

The Berkman Social Network scale (BSN; Berkman & Syme, 1979) is a 4-item questionnaire that establishes an individual's social network on the basis of marital status, number of friends and relatives, and church and group memberships. On the basis of this scale, Berkman and Syme were able to identify two distinct groups of individuals, those with either high or low social network. In a further study (Berkman & Syme, 1983) the authors found that there

were significant differences between individual mortality rates on the basis of scoring either high or low on the social network scale. Individuals with a low social network score were likely to die sooner than individuals scoring high on the social network scale.

The Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977) is a 20-item measure in which subjects are asked to rate their experiences and feelings over the previous week on a four-point scale (e.g., I felt I could not get "going"?). Devins and Orme (1985) established that the CES-D was appropriate for use in adult populations regardless of when (morning or evening) or how (in-vivo, by telephone, self-administered) the test was administered. Additionally it was shown to be a reliable measure across age, sex and socio-economic status. Acceptable levels of convergent and divergent validity, as well as the lack of focus on somatic symptomatology, has made this scale a valuable measure of depression for investigations involving medical patients (Devins and Orme, 1985).

The Cognitive-Somatic Anxiety Questionnaire (CSAQ; Schwartz, 1978) is a 14-item measure which asks subjects to indicate on a five-point scale the frequency of occurrence for each of the items under anxiety provoking conditions. Seven of the questions are concerned with anxiety which is experienced somatically (e.g., Do you perspire?) while the

other seven items reflect anxiety which is experienced cognitively (e.g. Do you imagine terrifying scenes?). Research has indicated (Davidson & Schwartz, 1976; Norton, Rhodes, Hauch & Kaprowy, 1985; Schwartz, Davidson & Goleman, 1978) that anxiety may be experienced both cognitively and somatically. Both of these components of the CSAQ are significantly correlated with Spielberger's State-Trait Anxiety Inventory ($\underline{r}$ = .67, $\underline{r}$ = .40 respectively). Although the cognitive and somatic elements are moderately correlated amongst themselves, "their shared variance is significantly low" indicating that they may be accounting for separate components of anxiety (Schwartz et al., 1978, p.325).

The Hollingshead Four Factor Index of Social Status (1975) was used to obtain a rating of each patient's status in society. The four factors of education, occupation, age and sex are used to determine a score ranging from a high of 66 to a low of 8. "It is assumed that the higher the score of a family or nuclear unit, the higher the status its members are awarded by other members of our society" (Hollingshead, 1975, p.23).

The Morrow Assessment of Nausea and Emesis (MANE; Morrow, 1982) and the MANE follow-up (MANE-FU) are concerned with the patients' experiences with nausea and vomiting both prior and subsequent to their chemotherapy treatment sessions. The MANE is administered during the

first pretreatment interview whereas the MANE-FU is given during each subsequent meeting. The MANE consists of five questions, four dealing specifically with the duration, frequency and severity of PCNV and ANV; the fifth question asks if the patient is susceptible to motion sickness. The MANE-FU also asks four questions concerning the duration, frequency and severity of PCNV and ANV, but the fifth question asks whether they took any anti-nausea or antiemetic drugs after their last treatment, and if the drugs proved to be effective. Research by Morrow (1984) found that the MANE and the MANE-FU had significant 7-month test-retest reliabilities ranging from .61 to .78. Additionally, results indicate that these scales possess good convergent and divergent validity and thus appear to "assess chemotherapy related nausea and vomiting in a consistent and reliable manner" (Morrow, 1984, p. 2274).

The Pre-illness Nausea and Vomiting Pattern questionnaire (PINVP; Farber, personal communication, 1985) is a 5-item measure, using a six-point scale, which determines the patient's experience, frequency and perceived unpleasantness of nausea and vomiting prior to the diagnosis of their cancer. Additionally, this scale determines those situations which may, in the past, have caused the patient to vomit (e.g., anxiety, (non)prescription drugs, illness). This measure establishes patients' attitudes towards nausea and

vomiting, and whether they have a predisposition or are easily susceptible to the development of these responses.

The Physical Symptoms Checklist (PSC; Andrykowski & Redd, 1985) is a true or false measure which determines the absence or presence of any of eight physical symptoms during the past 24 hours (e.g., dizziness, sleep loss, appetite loss, diarrhea). This scale has already been used in a study on ANV (Andrykowski & Redd, 1985) and was implemented in our study with only minor changes. In addition to the eight questions already on this scale, two questions concerning changes in the patient's taste and/or smell were asked. The previous literature which outlined the apparent importance of both of these factors warranted their inclusion.

The State Trait Anxiety Inventory (Spielberger, 1966) consists of two 20-item scales which measure both the state and trait components of anxiety. The trait measure assesses how the subject generally or typically feels, whereas the state reflects how the subject feels precisely at that moment. The state-trait inventory has been consistently shown to be a valid measure of both of these components of anxiety (e.g., Dreger, 1978).

In order to determine the toxicity of the drug treatment used by any patient enrolled in this study, an 11-point scale was devised. Each drug or drug combination was listed on the scale and given to the attending

oncologists, primary Day Care Unit nurses and pharmacists at the Tom Baker Cancer Centre. Toxicity was defined as the quality of being poisonous, or the degree of virulence which a specific drug combination may produce in an individual. Each professional rated the toxicity of the drug on a 0 to 10 scale with "0" indicating "no toxicity" and "10" indicating "extreme toxicity" (see Appendix A).

Three 10-cm visual analogue scales (VAS) were used to assess the severity of, (a) anticipatory nausea, (b) anticipatory vomiting and (c) anticipatory anxiety. Each scale was anchored at one end with "none" and the other end with "extreme". Patients were asked to give an indication of the severity of these responses at three specified time periods, (a) last evening before treatment, (b) this morning before treatment and (c) now, immediately before treatment (see Appendices B and C).

All information concerning a patient's illness, prior medical history and their current treatment was obtained from their medical records with the attending physicians' permission.

Procedure

In this study the following definitions of nausea and vomiting were adopted. Nausea was defined as "an awareness of the urge to vomit and is accompanied by loss of gastric tone and peristalsis with contraction of the duodenum and

reflux of intestinal contents into the stomach" (Seigel & Longo, 1981, p.352). Vomiting is the "forceful expulsion of gastrointestinal contents through the mouth and is associated with powerful sustained contractions of the abdominal muscles and opening of the cardia" (Seigel & Longo, 1981, p.352). Patients were categorized as experiencing either anticipatory nausea (AN), vomiting (AV) or both (ANV) if they developed any of these conditions at any time within the 24 hours prior to treatment. Patients were classified as experiencing post-chemotherapy nausea (PCN), vomiting (PCV) or both (PCNV) if they reported any of these conditions as a direct result of their treatment session.

At the time of their first treatment session, each eligible patient was given a letter of introduction by their primary nurse (see Appendix D). This letter outlined the requirements of the study and informed the patient that they would be contacted in the immediate future by the investigator to discuss both the details of the study and their possible participation.

Subsequent to each patient's first treatment session, the author contacted him/her by phone. At this time subjects were informed that the study was being conducted in an attempt to understand how and why some of the side effects of chemotherapy develop. They were also informed that their participation in the study was voluntary and

would in no way interfere with their treatment. It was made clear that their time committment would consist of the following: (1) a one-hour interview to be conducted at a time and place of their convenience prior to their next treatment session and (2) a ten-minute meeting prior to each of their next six treatment sessions. If they agreed to participate a convenient time and location was chosen for the initial one hour interview.

The majority of initial interviews were conducted at the patient's home 85.7% (60). Due to time and/or travel restrictions some patients were interviewed at the Tom Baker Cancer Centre 10% (7), and some were given the questionnaires to complete at home 4.3% (3). The results of multiple t-test comparisons showed that there were no significant differences between these three groups on any of the demographic or baseline measures regardless of where their baseline interviews took place.

At the start of the initial one hour interview (baseline) patients were asked to sign a consent form indicating their willingness to participate (see Appendix E). They were then asked a number of questions concerning basic demographic information, their social and economic status (Hollingshead), and their social network (BSN). Each subject was then asked to respond to questions from the PSC, the PINVP, the CSAQ, the Absorption Scale, the APQ, the STAI and the CES-D. The data were collected

through structured interviews, thereby minimizing intrusion on the patient. To aid the patient in answering the questionnaires, each was given a card listing the possible responses for each question. All answers were recorded on standardized forms and subsequently coded to preserve anonymity.

After the initial meeting, subjects were met for a minimum of four to a maximum of six brief interviews (approximately 5-10 minutes) prior to each of their subsequent treatment sessions. The second treatment session coincided with the first pre-treatment interview, the third treatment with the second pre-treatment interview, etc. The last pre-treatment interview was therefore conducted prior to the seventh treatment session. At these interviews the patient was asked to respond to the PSC, the state component of the STAI, the CESD, and the MANE/MANE-FU scale. Patients were also asked to rate their pre-treatment anxiety on a visual analogue scale at three specified times (the evening before treatment, the morning before treatment, and immediately before treatment). Patients who responded "true" on the PSC to having experienced nausea or vomiting within the past 24 hours (AN,AV) were also required to complete a visual analogue scale rating the severity of their ANV at the three times outlined above.

At the end of the last session, patients were thanked for their participation and informed that the results of this study would be forwarded to them upon completion of all data collection.

The toxicity of each patient's drug regimen was determined through the use of a toxicity scale. The Pharmacists, attending oncologists and Primary Day-Care nurses were asked to rate the toxicity of each drug combination. All results from these professionals were totalled and mean toxicity ratings were established.

RESULTS

Ninety-eight cancer chemotherapy patients were contacted for participation in this study. Of these 78.6% (77) agreed to take part. Of the 21 patients who refused, most stated that they were too ill to take part and that participation in the study would be "too much" for them (17); two patients said that if the study were to be without direct personal benefit they would rather not participate; one patient felt that the study would be an intrusion on his time; and one patient died prior to the initial baseline interview.

The inability of seven patients to continue in this study for the minimum of four interviews, due primarily to changes in their chemotherapy programs (5), or death (2), forced the removal of their results from our totals. Seventy subjects (71.4% of those initially contacted) were therefore used in the final analyses.

Of the 70 patients used in this study, most (84%) were seen for the full complement of 6 interview sessions. Although included in the results, 16% (11) of the patients were forced to drop out of the study after their fifth treatment (fourth interview session) due to changes in their treatment program (10), or death (1). No significant differences existed on any of the questionnaire measures between individuals who were interviewed only four times prior to treatment versus those interviewed six times.

Patients ranged in age from 20 to 74 years old ($\underline{M}$ = 50.6), the majority of whom were female 70% (49) and married 65.7% (46). Table 1 presents the results of the demographic variables of the sample (e.g., marital status, gender).

Diagnosis/Treatment

The prevalence of each cancer diagnosis in this study is presented in Table 2 along with the corresponding rank as estimated by the National Cancer Institute (American Cancer Society, 1986).

A listing of each possible drug combination that was administered to patients in this study (omitting all antiemetics and antidepressants) can be seen in Appendix A.

Patients with ANV

Of the 70 patients eligible for participation in this study 30% (21) experienced AN. Of those patients who developed AN, 14.3% (3) also experienced AV. Since AV did not occur without AN, these two responses will be collapsed into one category and subsequently referred to as ANV. Although the incidence of ANV is commonly reported using only those patients who experienced PCNV (e.g., Andrykowski et al., 1982) our rate is based upon the entire sample of 70 patients. This incidence rate accurately reflects the true prevalence of this response throughout this sample of

Table 1

Demographic Information

Participation	**N = 98**
Refusals	21
Incomplete (< four interviews)	7
Four interviews	11
Six interviews	59
Place of Baseline Interview	**N = 70**
Home	60
Cancer Centre	7
Self-administered	3
Gender	
Males	21
Females	49
Marital Status	
Married	46
Single	14
Widowed	10
Age (years)	
Range	20-74
Mean	50.62

Table 2

Rankings of the Prevalence of Cancer

	Present Study	National Cancer Institute
Cancer	Rank /(Freq)	Rank in Population (U.S.)
Breast	1 (29)	3
Lung	2 (9)	1
Lymphoma	3 (8)	7
Lymphocytic leukemia	4 (5)	16
Hodgkin´s	5 (4)	23
Brain	6 (3)	13
Colon/rectal	7 (3)	2
Kidney	8 (2)	11
Ovary	9 (2)	12
Larnyx	10 (1)	17
Mouth	11 (1)	18
Respiratory	12 (1)	29
Tongue	13 (1)	24
Uterus	14 (1)	5
	N = 70	

cancer patients. If we had assessed the occurrence of ANV only on the basis of those patients who developed PCNV (64 patients), then 32.8% of our sample experienced at least one episode of ANV.

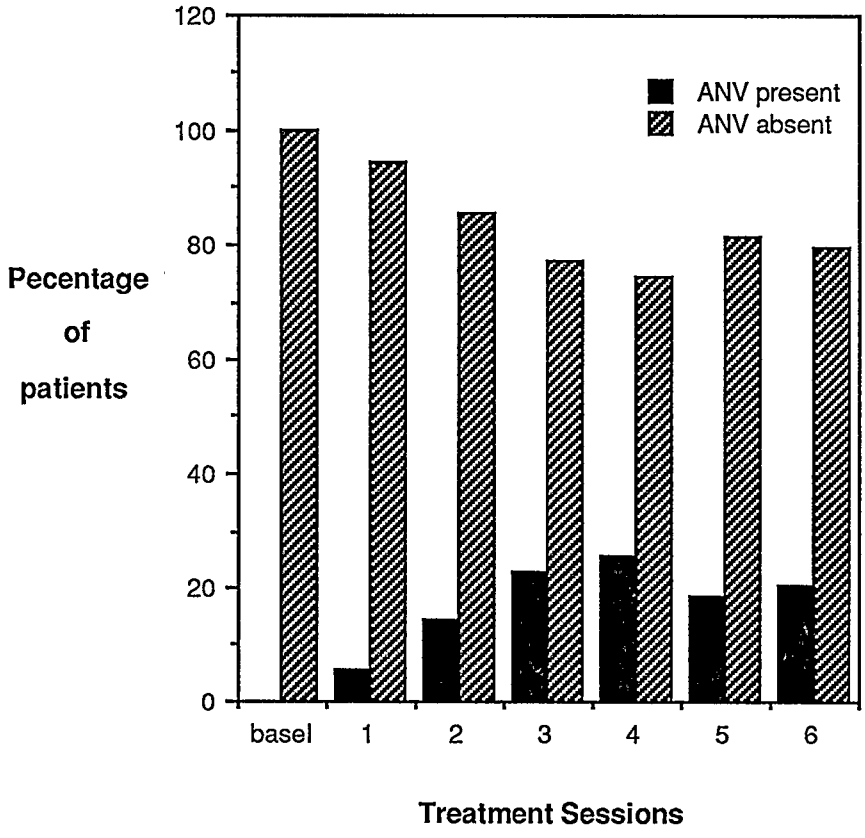
No patient experienced ANV without first experiencing PCNV. Of those patients who developed ANV (48%) had experienced the response by the start of their third treatment session (second pretreatment interview), 76% had developed ANV by their fourth treatment (third interview), and all patients had experienced at least one occurrence of ANV by the time of their fifth treatment session (fourth interview). On average, patients developed ANV after 3.4 treatment sessions. Figure 1 plots the non-cumulative frequency of patients with ANV by treatment session.

Group Differences (ANV, no-ANV)

Gender: Despite that fact that the majority of patients who developed ANV were female (85.7%) chi-square analyses revealed that the relationship between gender and ANV was not significant. Table 3 presents the results of the person and demographic variables for all subjects.

Age: Patients with ANV were, on average, not significantly younger than patients without ANV (M = 47.9 vs. M = 53.5, respectively) (see Table 3).

Patients with and without ANV at each session



Note: N = 70 for baseline through session 4. N = 59 for sessions 5 and 6.

Figure 1

Table 3

Group Differences (ANV vs. no-ANV)

Demographics	ANV	no-ANV	t-value
Age	47.95	53.31	<1
Socio-economic status	35.95	37.37	<1
Social support network	2.76	2.55	<1
Gender	**N = 21**	**N = 49**	**X^2-value**
Males/females	3/18	18/31	2.08
Marital Status			
Single/married	6/15	18/31	<1
Motion sickness			
Yes/no	8/13	18/31	<1
Patients with Taste Changes			
Baseline	7	17	<1
Interview 1	4	13	<1
Interview 2	3	10	<1
Interview 3	2	8	<1
Interview 4	2	7	<1
Interview 5	0	6	1.19
Interview 6	2	5	<1

Note: No significant differences found for any of these measures ($p < .05$).

Marital Status: There were no significant differences between the two groups on the basis of their marital status (see Table 3).

Demographics: Demographically, patients with ANV could not be differentiated on the basis of their socio-economic status or their social networks (see Table 3).

Motion Sickness: Contrary to Morrow (1982), chi-square analyses did not show a significant relation between ANV and susceptibility to motion sickness. Only 38.1% of those patients with ANV reported experiencing motion sickness. This is comparable to that of patients who failed to develop ANV (36.7%) (see Table 3).

Taste: Results which would indicate that taste is capable of differentiating between who will or who will not develop ANV were not replicated. There was no significant relation between the two groups when asked if they noticed any changes or anything unusual in their sense of taste either at baseline or over the six treatment sessions (see Table 3).

Previous Illness: In order to establish that patients with ANV were not predisposed to nausea and/or vomiting, their responses to the PINVP scale were analyzed. There were no significant differences between the two groups (ANV, no ANV) on the basis of the following measures: rated unpleasantness of nausea or vomiting, frequency of occurrence of nausea or vomiting prior to their present

illness, or whether nausea consistently precedes the occurrence of vomiting.

Additionally there was no significant relation between these groups with respect to the situations which may have caused them to vomit in the past (i.e., anxiety, pregnancy, prescription drugs, non-prescription drugs, illnesses, allergies or food poisoning). Table 4 presents the findings and chi-square analyses for each of these factors.

Trait Anxiety: In contrast to previously published reports there were no significant differences found between the two groups (ANV, no-ANV) on the basis of their baseline trait anxiety scores (see Table 5).

Cognitive-Somatic Anxiety: Individuals who developed ANV could be identified by significantly higher scores of somatic anxiety ($t(68) = 2.60$, $p < .05$) and on the overall CSAQ ($t(68) = 2.70$, $p < .01$). There were no significant differences between groups on cognitive measures of anxiety (see Table 5).

Physical Symptoms Checklist: Although some of the factors assessed by the PSC indicated the presence of a significant chi-square relation this significance was not maintained across the treatment sessions. Appendices F and G present a listing of these symptoms and their respective frequencies for the presence and absence of ANV.

Toxicity: The measures of drug toxicity obtained from the oncologists, Day Care nurses and pharmacists were combined

Table 4

Pre-Illness Nausea and Vomiting

	Mean rating		
Unpleasantness[a]	ANV	no-ANV	t-value
Nausea	2.03	2.46	1.93
Vomiting	3.33	3.67	1.89
Frequency[a]			
Nausea	2.05	2.21	1.22
Vomiting	3.29	3.95	1.35
	Frequency of Patients		
	Responding Yes		X^2-value
Anxiety	1	2	<1
Fever	1	4	<1
Other Illness	3	8	<1
Pregnancy	10	20	<1
Food poisoning	3	9	<1
Prescription drugs	1	3	<1
Non-prescription drugs	2	4	<1
Allergies (food)	0	0	<1
Anaesthesia	1	5	<1
	N = 21	N = 49	

Note: No significant differences found for these measures ($p < .05$). [a]Scored on a 1 (low) to 5 (high) scale.

Table 5

Baseline Group Differences (ANV vs. no-ANV)

	Means		
Variables	ANV	no-ANV	t-value
Trait anxiety	37.00	32.76	1.71 n.s.
Cognitive anxiety	18.48	16.20	1.91 n.s.
Somatic anxiety	18.29	15.92	2.60 *
Cognitive-somatic	36.77	32.12	2.70 **

* $p < .05$

** $p < .01$

Note: Not significant ($p < .05$).

to form a single measure of each drug's toxicity. The accuracy of our toxicity measures was established by examining the correlation between the rated toxicity of the drugs and the patient's reported severity and duration of PCNV. Morrow (1984) reported that the MANE/FU scale's estimation of a patient's severity and duration of PCNV was significantly correlated (r = .33) with the clinical rating of toxicity. Our results indicate that patients' responses on the MANE/FU were significantly correlated with the toxicity ratings by the health care professionals (severity, r = .28, p < .01; duration r = .19, p < .05).

Antiemetics: As expected, patients' ratings of the effectiveness of antiemetics for controlling the side effects of treatment were relatively low. In response to questions on the MANE, patients reported that the antiemetics "helped a little" to reduce the effects of PCNV (rated as two on a four-point scale). There were no significant differences (alpha = .05) found on measures of the effectiveness of these drugs between those who experienced ANV and those who did not (t(68) = .69).

PCNV: The respondent learning model predicts that patients with ANV should experience greater illness subsequent to their treatment sessions (PCNV) than were patients without ANV. Results indicate that those patients with ANV reported significantly more severe postchemotherapy nausea (PCN) than those patients without ANV (t(68) = 3.31, p <

.05). However, patients with ANV did not experience significantly (alpha = .05) more postchemotherapy vomiting (PCV) than did non-ANV patients ($\underline{t}(68) = 1.67$).

In accordance with the respondent learning model's predictions concerning the salience of the UCR, it is interesting to note that of those patients who reported their PCNV as severe or very severe (25), 60% subsequently developed ANV. Only 29% (13) of those patients who rated their PCNV as very mild, mild or moderate developed ANV ($\underline{X}^2(68) = 8.76$, $\underline{p} < .005$). Table 6 presents the results relating to the severity and duration of PCNV. Figure 2 plots severity and duration of PCNV as a function of the occurrence of ANV.

Although we were not able to measure when, subsequent to treatment, PCNV was first noted to occur, we were able to establish when PCNV was rated as being most severe. Results indicate that patients with ANV report the most severe PCN 8 to 12 hours after treatment whereas patients without ANV report PCN as being the most severe 4-8 hours after treatment ($\underline{t}(68) = 4.12$, $\underline{p} < .001$).

Morrow (1982) indicated that patients with ANV were most likely to experience PCNV within the first four hours after treatment and those without ANV between 4 and 8 hours after treatment. Thus, compared to patients without ANV, those patients experiencing ANV not only develop PCNV

Table 6

Severity and Duration of PCNV by ANV

PCNV	ANV	no-ANV	t-value
Severity[a]			
Nausea	71.10	39.80	4.85 **
Vomiting	41.80	26.80	1.78 n.s.
Duration[b]			
Nausea	119.55	59.02	1.76 n.s.
Vomiting	87.61	50.13	1.06 n.s.

** $p < .001$

Note: Not significant ($p < .05$). [a]Severity rating is on a scale of 0 (low) to 100 (high). [b]Duration is measured in hours.

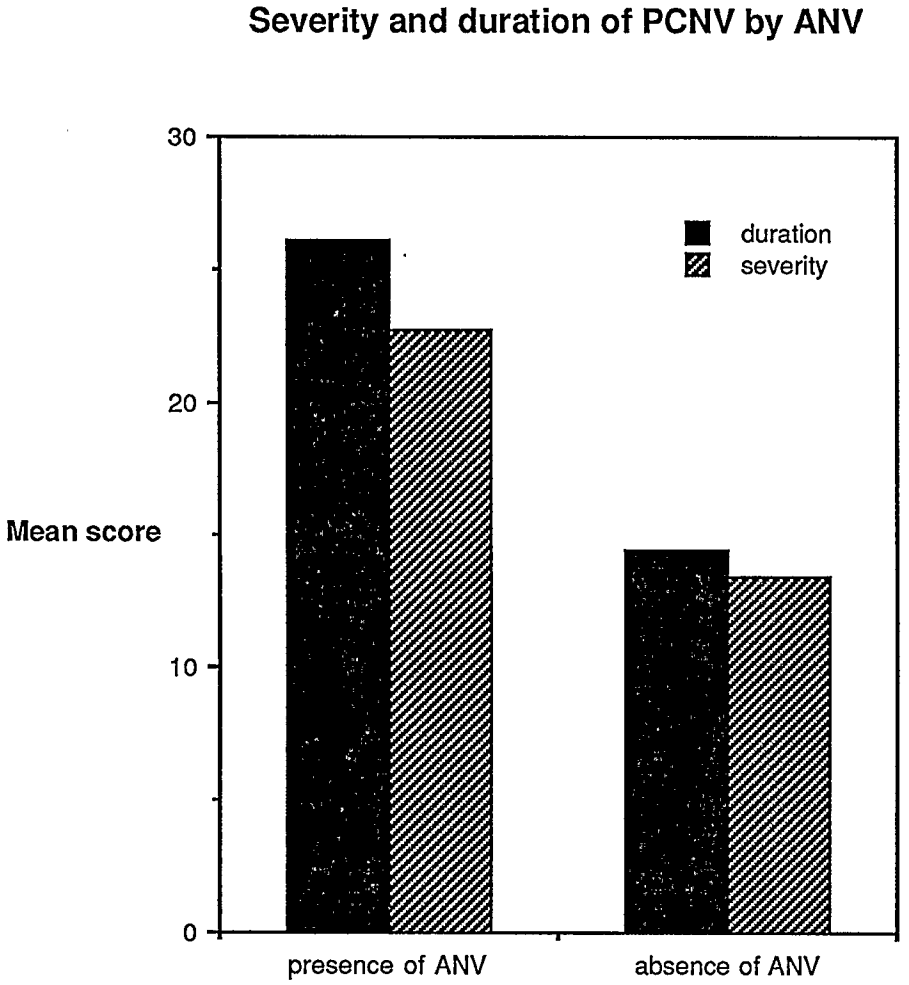

Severity and duration of PCNV by ANV
30
20
10
0
Mean score
duration
severity
presence of ANV
absence of ANV

Figure 2

sooner after treatment, but also experience significantly more severe PCN up to 12 hours post-treatment.

ANV

Whereas Duigon (1986) reported that patients reported the occurrence of ANV, on average, 17 hours prior to treatment, our results suggest that these patients experienced ANV, on average, 14 hours before treatment. Figure 3 plots the time of onset of ANV during the 24 hours pretreatment.

In response to the MANE and MANE-FU, patients experiencing AN reported, on average, a severity of three (moderate) on a five-point scale. Patients with AV reported an average severity of two (mild) on the five-point scale. Unfortunately, it is difficult to accurately assess the severity of AV due to the small number of patients (3) who developed this response.

Results from the 10 point visual analogue scale indicate that, on average, patients reported the severity of their ANV the night before treatment as being 5.3, the morning before as 5.3 and immediately before as 4.6.

Depression

Levels of depression were assessed at the time of the initial interview as well as prior to each treatment session. An Analysis of Covariance (2x7 ANCOVA) with

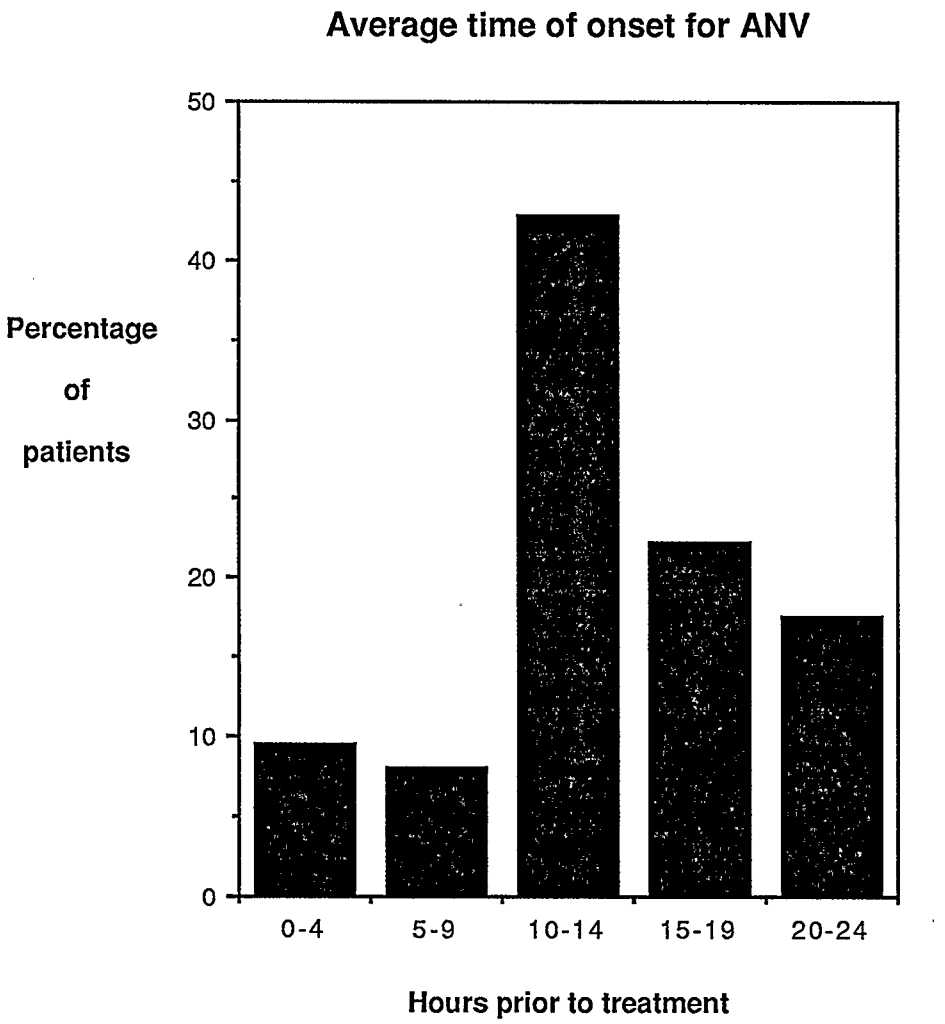

Average time of onset for ANV
Percentage of patients
50
40
30
20
10
0
0-4
5-9
10-14
15-19
20-24
Hours prior to treatment

Figure 3

toxicity as a covariate and one between-subjects variable (ANV) and one within-subjects variable (sessions) was conducted on CES-D scores. No significant main effect was found for the presence or absence of ANV and there was no significant interaction. There was, however, a significant main effect for measures of CES-D across treatment sessions ($F(6,342) = 2.74$, $p < .05$).

The Newman-Keuls post-hoc analysis follows the concept of an error rate based upon a stepwise approach to significance testing. This test of significance "is sensitive to the number of means being tested and thus reduces the size of the critical difference depending on the number of steps separating the ordered means" (Kirk, 1982, p.125). Post-hoc (Newman-Keuls) analyses of the sessions main effect revealed that there were significant differences (alpha = .05) between baseline measures of depression and interview session two, three, four and six. Patients reported significantly higher levels of depression at baseline than at the above specified treatment sessions.

Table 7 presents the adjusted mean of depression across each session as a function of the presence or absence of ANV. The adjusted means are presented due to the fact that they represent the patient's mean score of depression once the covariate (toxicity) has been removed. An ANCOVA Summary Table can be found in Appendix H.

Table 7

Adjusted Mean Levels of Depression for Session Main Effect (absence/presence of ANV)

Sessions	Means*	Sd.
Baseline	15.65a	10.36
Interview 1	13.26ab	10.15
Interview 2	12.02b	9.40
Interview 3	11.28b	8.60
Interview 4	11.75b	10.37
Interview 5	12.40ab	9.50
Interview 6	11.24b	8.14

Adjusted Mean Levels of Depression for Session Main Effect (high/low absorption)

Sessions	Means*	Sd.
Baseline	14.81a	9.92
Interview 1	12.38ab	9.41
Interview 2	10.91b	9.45
Interview 3	10.67b	8.43
Interview 4	11.31b	9.87
Interview 5	11.19b	8.75
Interview 6	10.49b	7.66

Note: (*) Means sharing the same subscript fail to differ significantly ($p < .05$).

Continued...

Table 7 continued

Actual Mean Levels of Depression

Sessions	Means	Sd.
Baseline	14.73	9.75
Interview 1	12.17	9.13
Interview 2	10.85	9.38
Interview 3	10.59	8.62
Interview 4	11.19	9.79
Interview 5	11.02	8.41
Interview 6	10.30	7.45

Similar results were obtained when individuals were differentiated by their levels of absorption. Patients were separated into two groups (using a high and low median split; Md = 22.167) on the basis of their absorption scores. A 2x7 ANCOVA was again used with one between-subjects variable (absorption), one within-subjects variable (sessions) and toxicity as the covariate. Although there was no significant main effect for levels of absorption or for the interaction, there was a main effect for sessions ($F(6,342) = 3.28$, $p < .001$).

Post-hoc (Newman-Keuls) analyses of the sessions main effect revealed significant differences (alpha = .05) between baseline measures of depression and interview session two, three, four, five and six. As with the previous example, patients experienced significantly higher depression at baseline than at any of the above specified interview sessions. Table 7 presents the adjusted mean levels of depression across each session as a function of absorption. An ANCOVA Summary Table can be found in Appendix I.

State Anxiety

A 2x7 ANCOVA with toxicity as the covariate and one between-subjects variable (ANV) and one within-subjects variables (sessions) was conducted on state anxiety scores. There was a significant main effect for the occurrence of

ANV (M = 39.72 ANV, M = 33.72 no-ANV; $F(1,56) = 5.22$, $p < .05$). There was also a significant interaction ($F(6,342) = 2.16$, $p < .05$), as well as a significant main effect for sessions ($F(6,342) = 5.24$, $p < .001$).

Post-hoc (Newman-Keuls) analyses of the sessions main effect revealed a significant difference (alpha = .05) on measures of state anxiety between baseline and session one, with patients exhibiting significantly higher state anxiety at session one.

Using the correction for unequal n's (Glass & Hopkins, 1984; Kleinbaum & Kupper, 1978) post-hoc (Newman-Keuls) analyses of the ANV main effect revealed signficant differences (alpha = .05) between the two groups (ANV, no-ANV) at session one, two, three, five and six. Patients with ANV reported significantly higher levels of state anxiety than did patients without ANV. Table 8 presents the adjusted means for state anxiety as a function of the presence or absence of ANV. Figure 4 presents the relationship between state anxiety and the absence or presence of ANV. An ANCOVA Summary Table can be found in Appendix J.

A comparison of the results of the visual analogue scale for state anxiety revealed that patients with ANV reported significantly higher levels of state anxiety the night before treatment ($t(68) = 3.18$, $p < .005$), the

Table 8

Adjusted Mean Levels of State Anxiety for Session Main Effect (absence/presence of ANV)

Sessions	Means*	Sd.
Baseline	31.90a	10.77
Interview 1	39.19b	13.04
Interview 2	38.26ab	9.49
Interview 3	37.56ab	9.87
Interview 4	37.71ab	11.40
Interview 5	36.47ab	10.27
Interview 6	35.96ab	9.21

Note: (*) Means sharing the same subscript fail to differ significantly ($p < .05$).

Relationship between state anxiety and ANV

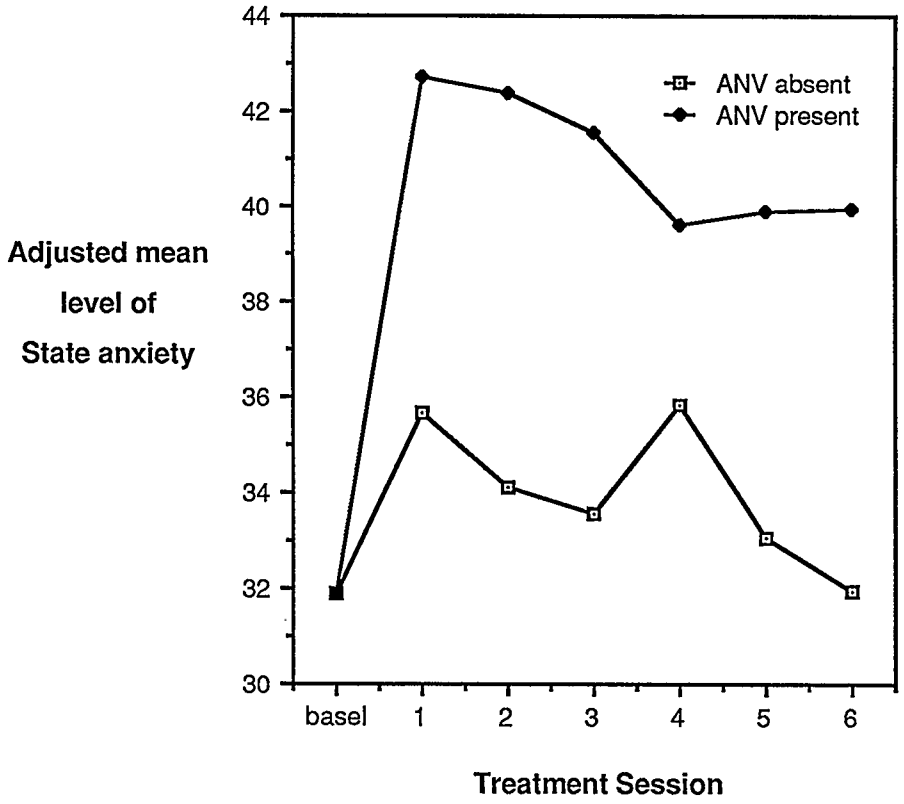


Figure 4

morning before treatment ($t(68) = 3.51$, $p < .001$) and immediately before treatment ($t(68) = 3.18$, $p < .005$).

Absorption/APQ

There were significant differences between ANV and no-ANV patients' scores on both the absorption scale and the APQ. Patients who subsequently developed ANV scored significantly higher ($t(68) = 9.27$, $p < .001$) on measures of absorption. Additionally, this same group scored significantly higher ($t(68) = 5.50$, $p < .001$) on measures of the APQ. Figure 5 presents the relationship between ANV and absorption and autonomic perception. A positive correlation was found between measures of autonomic perception and absorption ($r = .59$, $p < .01$).

Hypotheses

Hypothesis 1: The first hypothesis was tested using correlation coefficients. Absorption was not significantly correlated with trait anxiety ($r = .18$) or with measures of depression ($r = .15$). This indicates that absorption accounts for unique variance in our measures that does not overlap with anxiety and depression. The APQ, however, was significantly correlated with trait anxiety ($r = .31$, $p < .01$) and depression ($r = .28$, $p < .05$). This indicates that the measures of trait anxiety and depression overlap

Relationship between ANV and Absorption and Autonomic Perception

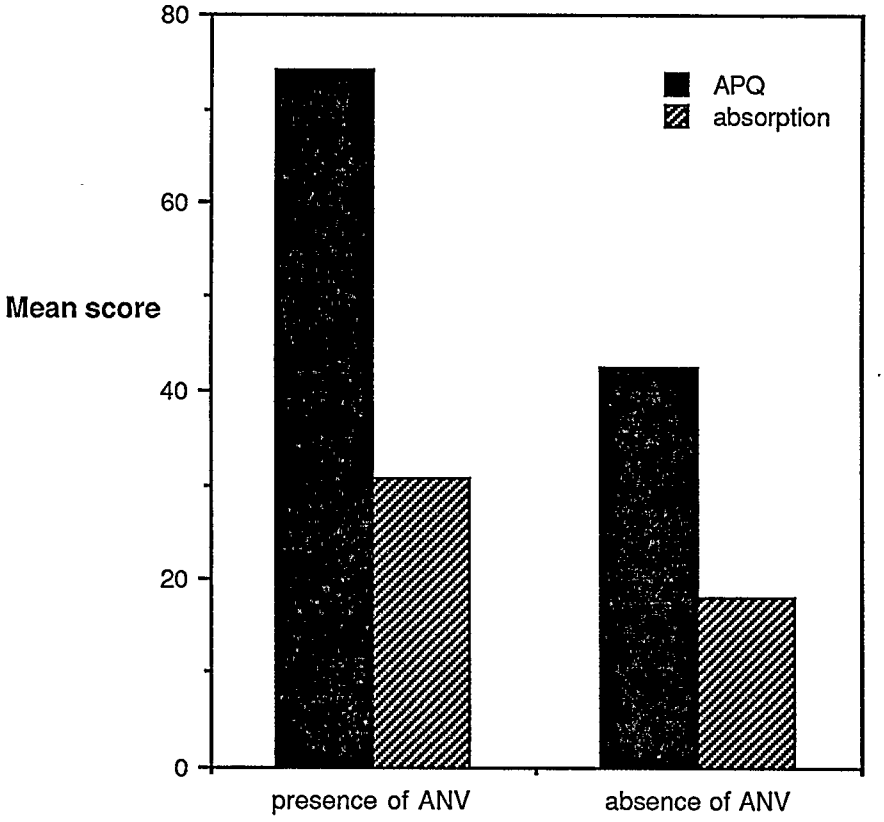


Figure 5

the APQ but the correlations are relatively small and thus the variance in common is minimal.

Hypothesis 2: The second hypothesis was tested using an 2x7 ANCOVA with one within-subjects variable (7 sessions), one between-subjects variable (high/low absorption), and toxicity as the covariate. Patients were divided into groups on the basis of either high or low absorption scores (using the median split; Md = 22.167). There was a significant main effect for absorption (M = 37.86 high, M = 33.23 low; F(1,56) = 4.05, p < .05), and a significant main effect for sessions (F(6,342) = 4.65, p < .001). The interaction approached significance (F(6,398) = 1.95, p < .075).

Post-hoc (Newman-Keuls) analyses of the sessions main effect revealed significant differences (alpha = .05) between baseline measures of state anxiety and session one, baseline and session two, and baseline and session five. Patients reported significantly higher levels of state anxiety at each of these sessions as compared to their baseline scores.

Using the correction for unequal n's (Glass & Hopkins, 1984; Kleinbaum & Kupper, 1978) post-hoc (Newman-Keuls) analysis of the interaction showed significant differences (alpha = .05) between the two groups (high-low absorption) for session one, two and five. Patients with high levels of absorption reported significantly higher levels of state

anxiety than did patients without ANV at those interview sessions specified above. Table 9 presents the adjusted means for state anxiety as a function of absorption. Figure 6 presents the relationship between state anxiety and absorption. An ANCOVA Summary Table can be found in Appendix K.

The visual analogue measures of state anxiety obtained prior to each treatment session were averaged across sessions to yield a single score for each patient at each of the three time periods; the night before, the morning before and immediately before treatment. A median split for absorption (Md = 22.167) was used to differentiate groups on these anxiety scores. Patients with high absorption reported significantly higher levels of pretreatment anxiety than did low absorbers the night before treatment ($t(68) = 3.12$, $p < .005$), the morning before treatment ($t(68) = 3.63$, $p < .001$) and now, immediately before treatment ($t(68) = 3.16$, $p < .005$).

Hypotheses 3 & 4: A hierarchical discriminant function analysis was performed to predict membership of the two groups on the basis of six learning variables (as outlined by the respondent learning model; hypothesis 3) and again after the addition of two personality variables (hypothesis 4). The groups were patients with ANV or without ANV. Patients were classified as having developed ANV if they reported AN and/or AV prior to at least one chemotherapy

Table 9

Adjusted Mean Levels of State Anxiety for Session Main Effect (high/low absorption)

Sessions	Means*	Sd.
Baseline	31.90a	10.22
Interview 1	37.96b	12.36
Interview 2	36.58b	10.03
Interview 3	35.93ab	10.43
Interview 4	37.01ab	11.55
Interview 5	35.12b	10.55
Interview 6	34.31ab	9.76

Note: (*) Means sharing the same subscript fail to differ significantly ($p < .05$).

Relationship between state anxiety and absorption

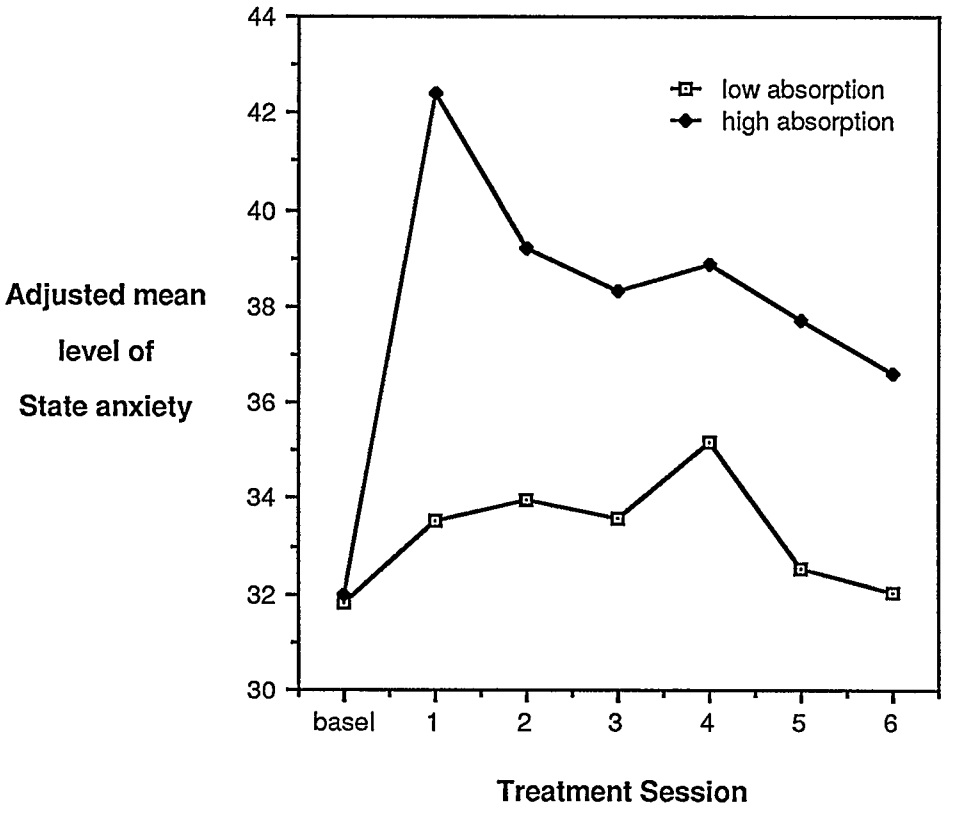


Figure 6

session. The six learning variables were drug toxicity, state anxiety, the severity of PCN and PCV and the duration of PCN and PCV. The values of PCN and PCV were established by determining the mean severity rating and the mean duration of both these measures over the six treatment sessions. The two personality variables were absorption and autonomic perception.

The significance of the relative contributions of each of these variables to the discriminant function was tested using Wilk's lambda. According to Pedhauzer (1982) Wilk's lambda is the most widely used statistical test of significance for discriminant function analysis (see also, Olson, 1976).

Hypothesis 3: The severity of PCV was removed from the last step of the analysis due to an insufficient F level. The results with the five remaining learning variables indicate that there is a statistically significant discrimination between the two groups (ANV, no-ANV) ($\underline{F}(5,64) = 10.31$, $\underline{p} < .001$). These learning variables correctly classified 85.71% of the cases into their appropriate groups. This finding provides support for the respondent learning paradigm as an accurate model for ANV development. Table 10 presents a Summary Table of the discriminant function analysis for hypothesis 3. The percentage of correctly and incorrectly identified cases can be seen in Appendix L.

Table 10

Discriminant Function Analysis -- Summary Table

Variables	Wilk's Lambda	Rao's V	Change in V
Severity of nausea	.779	19.61 ***	19.61 ***
Severity of vomiting	.700	29.07 ***	9.86 *
Toxicity of drugs	.638	38.60 ***	9.53 **
State anxiety	.614	42.69 ***	4.09 *
Duration of vomiting	.578	49.53 ***	6.84 **
Duration of nausea	.556	54.19 ***	6.41 *

* $p < .05$

** $p < .005$

*** $p < .001$

Hypothesis 4: A discriminant function analysis was conducted using the measures of absorption and autonomic perception, alone. The results indicated that there is a statistically significant discrimination between the two groups (ANV, no-ANV) ($F(2,67) = 28.75$, $p < .001$). These two variables correctly classified 84.29% of the cases. This result suggests that absorption and the APQ can accurately discriminate group membership. Table 11 presents a Summary Table of the discriminant function analysis. The percentage of correctly and incorrectly classified cases can be seen in Appendix M.

The addition of the two personality variables to the learning variables (see hypothesis 3) also yielded a significant effect ($F(8,61) = 15.87$, $p < .001$). With all eight of the predictor variables included, 95.71% of the cases were correctly classified. This indicates that the addition of absorption and APQ results in an even higher level of discrimination.

A McNemar's chi-square test (Tabachnick & Fidell, 1984) for change in proportion of correct identification with the addition of the personality variables revealed a statistically significant gain in classification performance above and beyond the learning variables (X^2 (1) = 4.00, $p < .05$). Table 12 presents a Summary Table of the discriminant function analysis for hypothesis 4. The

Table 11

Discriminant Function Analysis -- Summary Table

Variables	Wilk's Lambda	Rao's V	Change in V
Absorption	.588	47.62 ***	47.62 ***
Autonomic perception	.538	58.36 ***	10.74 ***

*** $p < .001$

Table 12

Discriminant Function Analysis -- Summary Table

Variables	Wilk's Lambda	Rao's V	Change in V
Toxicity	.837	13.28 ***	13.28 ***
State anxiety	.774	19.91 ***	6.63 **
Severity of nausea	.696	29.74 ***	9.82 **
Severity of vomiting	.614	42.69 ***	12.95 ***
Duration of nausea	.595	46.37 ***	3.68 *
Duration of vomiting	.549	55.94 ***	9.57 **
Absorption	.351	126.00 ***	70.05 ***
APQ	.326	140.70 ***	14.74 ***

* $p < .05$

** $p < .005$

*** $p < .001$

percentage of correctly classified cases can be seen in Appendix N.

Structure Coefficients: The structure coefficients for each of the eight predictor variables were determined. Structure coefficients indicate the extent to which a variable and the function are related. When the absolute magnitude of the coefficient is very large the function carries almost the same information as the variable (Klecka, 1984). Structure coefficients are simple bivariate correlations and thus are not biased (unlike the standardized coefficients) by the multicollinearity that is present between predictors (Thompson, 1984). Absorption and autonomic perception add the largest contribution to the discriminant score ($\underline{r}$ = .58 and $\underline{r}$ = .47 respectively). The structure coefficients for each of the eight predictors variables can be seen in Table 13.

Accuracy of Prediction by Session: Discriminant function analyses were also conducted for each of the six treatment sessions. The predictors were toxicity, severity and duration of nausea and vomiting, and the state anxiety score for the specific session in question. The two personality variables were absorption and autonomic perception. Since only one person experienced ANV at the time of the first pretreatment interview, the analysis could not be conducted for this session. Discriminant function analyses were therefore conducted for the last

Table 13

Structure Coefficients

Variables	Coefficient
Absorption	.58
Autonomic perception	.47
Severity of nausea	.37
Toxicity	.31
State anxiety	.27
Duration of nausea	.16
Severity of vomiting	.15
Duration of vomiting	.09

five treatment sessions. At the second pretreatment interview, a significant discriminant function was found ($F(8,61) = 64.83$, $p < .001$). All eight predictor variables correctly classified 90.00% of the cases.

In accordance with the respondent learning model, prediction of group membership should improve as a result of an increased number of UCS-UCR pairings (more treatment sessions). By studying the results of the sixth pretreatment interview we are able to provide support for this model. A significant discriminant function ($F(8,61) = 67.42$, $p < .001$) was found for these predictors, indicating an even higher level of group differentiation than at session two. Additionally, these eight predictors were able to correctly classify 98.57% of the cases. A McNemar's change test revealed that group classification improved significantly as a result of an increased number of UCS-UCR pairings (X^2 (1) = 4.17, $p < .05$).

DISCUSSION

These results provide important new information regarding the nature of the patient variables, as well as the environmental factors that may put an individual at risk for the development of ANV. Strong support is also provided for a variety of previously reported findings regarding the developmental processes of ANV.

Hypotheses

Hypothesis 1: The findings related to the first hypothesis indicate that absorption is capable of accounting for unique variance that is not accounted for by measures of trait anxiety or depression. The autonomic perception questionnaire was significantly related to both trait anxiety and depression. However, the relatively small correlations found between trait anxiety and the APQ (r = .31, which accounts for 9.6% of the variance in trait anxiety) and depression and the APQ (r = .28, which accounts for 7.8% of the variance in depression) indicates that the APQ is still able to account for variance not accounted for by trait anxiety or depression.

Hypothesis 2: Levels of state anxiety, as measured by both the Spielberger State questionnaire and the visual analogue scale, were significantly different for those individuals who developed ANV than for those who did not. In terms of the learning model, state anxiety adds significantly to the

prediction of ANV. Unfortunately, measures of pretreatment anxiety do not allow for a prospective determination of who is at risk for this response. Due to the fluctuating nature of this particular measure, a patient's level of pretreatment anxiety, and thus their potential for ANV, can only be measured immediately before treatment, making behavioral intervention at this time relatively difficult. It may be that the high levels of state anxiety which have been recorded are not so much predictors of ANV as they are responses to the occurrence of this response. Thus, although adding to the accuracy of the respondent learning model by functioning to increase the salience of the UCS-UCR pairings, it is impossible to establish whether state anxiety facilitates the development or is a direct result of ANV.

The lack of significant reductions in reported state anxiety over the six treatment sessions within either the ANV or no-ANV group indicate that little or no adjustment or effective coping with the anxious response is occurring. The reduction of high levels of state anxiety through the implementation of behavioral techniques would aid the patient in coping with the psychological side effects of the treatment sessions. Although previous research (Morrow, 1982) has indicated that the reduction of anxiety does not necessarily result in a corresponding reduction in

the occurrence of ANV, it remains likely that the comfort of the patient would be improved.

Hypothesis 3: The verification of our third hypothesis provides additional support for the utility of the respondent learning model as an explanation of the developmental process of ANV. As previously stated, however, the respondent learning model may be necessary but not sufficient to account for the development of ANV. Although numerous variables have been proposed that mediate the development of ANV, our results indicate that none of these factors is capable of predicting its occurrence with any greater accuracy than the respondent learning model.

Hypothesis 4: Our fourth hypothesis indicates that the inclusion of absorption and autonomic perception into the respondent learning model provides significantly more information about the prediction of ANV. The significant increase in the percentage of cases correctly classified as a result of the inclusion of absorption and autonomic perception indicates that these variables accurately assess the presence of cognitive mediators in the development of ANV.

An important aspect of these measures, above and beyond their accuracy in defining group membership, is that both scales represent stable traits which can be used prospectively to identify individuals at risk for the development of ANV. Prospective identification avoids the

confounds that are present when one attempts to establish those variables that are predictors of ANV rather than reactions to its occurrence.

Absorption may be capable of bridging the temporal gap that exists between the occurrence of the UCS and the UCR. Individuals high in absorption are more capable than low absorption individuals in experiencing imaginary activities as though they were present and real. Ruminations concerning the treatment and its side effects function to maintain the salience of the UCS in the patients' cognitive representation.

Additionally, absorption may account for the observation that anticipatory symptoms can occur up to 24 hours prior to treatment. Individuals high in absorption are capable of clearly imagining the cues associated with the upcoming treatment session. Pennebaker (1971) suggested that individuals who focus their attention on a negative event develop a more severe representation of that event. It is likely that this functions to increase the salience of the cues surrounding treatment, thereby facilitating conditioning.

According to the literature, individuals who score high on the APQ tend to overestimate their true level of autonomic arousal (Mandler, 1984). These same high perceivers respond in accordance with their perceived rather than their actual levels of arousal. As a result,

when individuals perceive themselves to be highly aroused, they will then respond behaviorally in accordance with these perceptions.

The effectiveness of the behavioral interventions for controlling ANV may also be explained in terms of the two personality factors of absorption and perceived autonomic arousal. It is possible that, due to its effects upon the cognitive activity of absorption, distraction may be functioning to ameliorate the occurrence of ANV. Guided imagery and hypnosis each function to focus one's attention away from those environmental and cognitive cues which are capable of eliciting the conditioned response. It should therefore be expected that the implementation of these behavioral procedures should function to eliminate the probability of occurrence of ANV.

With respect to the APQ, it is possible that relaxation procedures function to lower an individual's level of physiological arousal. It is assumed that this reduction in actual physiological arousal also functions to reduce the patient's own perceived level of arousal. Such a reduction should result in the patients assessing their physiological arousal as being lowered and thus responding accordingly.

These two variables, within the constraints of the respondent learning model, permit for a prospective identification of those individuals at risk for the

development of ANV. It is hoped that this prospective identification will lead not only to a better understanding of the conditioning process as it relates to noxious stimuli, but also will permit earlier behavioral interventions with cancer patients that will ultimately lead to the prevention of the occurrence of ANV.

Incidence of ANV: The presence of ANV in 30% of our sample is well within the range of the incidence rates reported by others (e.g., Morrow, 1982). Although only 3 (4%) patients reported experiencing AV, a number of studies have indicated that the occurrence of AV is considerably less than AN. VanKomen and Redd (1984) found that only 10% of their sample experienced AV. Nicholas (1982) has also commented on the rarity of occurrence of AV as compared to AN, finding an incidence of only 18% for AV. Our results do confirm, however, that the responses of AN and AV occur in a sufficient proportion of cancer chemotherapy patients to warrant concern for their physical and psychological well-being.

Gender: As mentioned previously, reports indicating that there are differences in susceptibility to ANV on the basis of gender are not consistently supported in the literature. Our results also fail to provide any support for a significant difference between males and females with respect to ANV development.

Age: Patients with or without ANV were not significantly different on the basis of age. A number of authors have commented upon the finding that younger patients seem more susceptibile to ANV (e.g., Morrow, 1982; Nerenz et al., 1983). Andrykowski (1985) argued that these age differences may be due entirely to the differences in severity or toxicity of the treatments which younger individuals experience. The relatively mature nature of our sample (78.6% over the age of 40) makes it difficult to speculate on this argument and its applicability to younger populations other than to say that there were no significant age differences between those who developed ANV and those who did not.

Motion Sickness: Morrow's (1984) findings regarding one's susceptibility to motion sickness and the increased risk for developing ANV were not supported by our data. It may be that the relatively few individuals who indicated that they were susceptible to motion sickness (8 of those with ANV) precluded the possibility of finding significant differences. However, this same low occurrence prevents motion sickness from being considered a practical or reliable predictor of ANV.

Demographics: There were no significant findings with respect to differences in socio-economic status or social network. The lack of a significant difference amongst those patients with either low or high levels of social

networks was of particular interest. Although there has been a lack of research on this variable with respect to ANV, it had been suggested that an impoverished social network (as reflected by an increased prevalence of ANV in unmarried individuals) may be an important factor in the development of ANV. Since there were no significant differences for measures of social network the importance of this factor is once again brought into question.

Previous Illness: These results indicate that those patients with ANV had not previously experienced nausea or vomiting due to elevated levels of anxiety. Also, there were no significant differences between these two groups and their susceptibility to nausea and/or vomiting on the basis of previous illnesses, food poisoning, previous experience with prescription or non-prescription drugs, anaesthesia or pregnancy.

No differences were found between groups regarding the frequency or perceived unpleasantness of either nausea and/or vomiting experienced prior to their present illness. This finding suggests that individuals who subsequently develop ANV are no more predisposed to experiencing nausea and/or vomiting than those who did not develop the response.

Physical Symptoms: There were no differences found on any of the factors evaluated by the PSC. Previous research has indicated that many of these factors may be present in

patients with ANV prior to their treatment sessions and it has been argued that the presence of these elements may be functioning to predispose individuals to ANV development (Morrow, 1984b; Redd et al., 1982). The presence of headaches, fatigue and dry mouth, although occurring in a large proportion of patients (both ANV and no-ANV), did not differentiate groups on the basis of their subsequent development of ANV.

Trait Anxiety: Patients who developed ANV were not significantly different on trait anxiety from those who failed to develop this response. The relative importance of this variable in terms of the conditionability of patients is therefore debatable.

Depression: Patients with ANV were not significantly different on measures of depression than were those patients without ANV. There were, however, significant differences across treatment sessions for the combined scores of the two groups. Baseline measures of depression were significantly higher than subsequent, pretreatment measures. The recency of diagnosis or the uncertainty that surrounds one's treatment and prognosis may have added significantly to levels of depression at baseline. As suggested by Altmaier et al. (1982), this uncertainty may abate once treatment begins and the patient begins to accept the diagnosis and obtains additional information concerning the treatment process.

Respondent Learning Variables: The toxicity of the drugs used in treatment is of crucial importance in establishing the rate and probability of ANV development. In fact, each study in this field, ours included, has shown that the toxicity of the drugs used in therapy is essential for ANV to develop. In line with the respondent learning model, the toxicity of the treatment drugs functions to increase the salience of the UCR. Drug regimens that produce severe side effects, most notably nausea and/or vomiting, therefore increase the probability of a patient's conditioning.

As mentioned previously, the relative ineffectiveness of most pharmacologically-based antiemetic procedures has led clinicians and investigators to seek behavioral methods of treatment. In this study, two important aspects of the patients' responses to antiemetics are worth noting. First, the lack of any significant differences between patients who did or did not develop ANV on measures of the effectiveness of antiemetic medications indicates that this factor is unable to account for differences in susceptibility rates to ANV. Second, in both groups the effectiveness of the antiemetics was rated as helping a little. This finding indicates that present antiemetic procedures are viewed by the patients as being only slightly effective in relieving some of the side-effects of treatment (see also, Burish & Carey, 1986; Morrow, 1986).

The number of treatment sessions has also provided support for the learning model of ANV response development. In addition to verifying this model these results also support the claim that the wide range of incidence rates in the literature for the occurrence of this response may be a result, in part, of the point in treatment when the patients were asked if they had experienced ANV. In this study, if we had interviewed patients subsequent to their second treatment session our rate of occurrence would have been only 14%; if asked after their third treatment the rate would have been 23%. It is essential that future studies ensure that measurement of ANV prevalence rates not be conducted until each patient has received a minimum of four treatments.

Summary

Our results indicate that the respondent learning model, although accurate for describing the process through which ANV develops, fails to account for its low incidence rate. Despite the fact that numerous mediators of conditioning have been suggested to account for the incidence of ANV our results have failed to provide support for their inclusion in a theory of ANV development.

The finding that absorption and APQ are capable of predicting ANV both alone, and above and beyond the learning variables, provides important information

regarding the role that cognitions play in ANV response development. ANV can no longer be described in terms of a simple learning model but must now take into account the cognitive processes of the individual.

The ability to accurately and prospectively identify those patients who will develop ANV opens the door for a reorganization of current behavioral and psychological treatment processes. Earlier intervention, as well as a greater understanding of those factors that function to mediate conditioning, should permit more effective intervention by the health care professional and thus result in an improved outcome for the patient.

CONCLUSION

In conclusion, our results show that the relation between increased severity and duration of PCNV, levels of state anxiety and the greater toxicity of drug regimens for those patients who experience ANV, replicates previous research on the respondent learning model of anticipatory nausea and vomiting.

Absorption represents a unique person variable that cannot be accounted for by measures of trait anxiety or depression, but that significantly predicts state anxiety in cancer patients.

Measures of absorption and autonomic perception alone significantly differentiate between those individuals who do or do not develop ANV.

Measures of absorption and autonomic perception add significantly to the prediction of ANV, even after the variance accounted for by the learning variables is entered.

These data indicate that ANV is a much more complex phenomenon than is commonly described by the respondent learning model. Cognitive and imaginative abilities play an important role in the development of ANV and thus, treatment efforts aimed at its amelioration should take these variables into account.

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APPENDICES

Appendix A

Anticipatory Nausea and Vomiting Study

As part of our data collection procedures concerning the prediction of anticipatory nausea and vomiting we would like to obtain a rating of the toxicity of each patients treatment program. Toxicity is defined as the quality of being poisonous, or the degree of virulence which a specific treatment may produce in an individual. As the people who prescribe treatment we feel that the physicians are most able to assess toxicity. Could you please take a moment and rate the regimens we have listed. It would add important information to our analyses of the data.

Please rate on a scale of 0 to 10, the toxicity of each of the following combinations of drug treatments. On this scale, 0 represents no toxicity and 10 represents extreme toxicity.

1. Methotrexate 200 mg.
   5-FU 500 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

2. Methotrexate 30 mg.
   Procytox 400 mg.
   5-FU 600 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

3. Cytoxan 60 mg.
   Procytox 600 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

4. Adriamycin 100 mg.
   Procytox 700 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

5. Cisplatinum 140 mg.
   Cytoxan 1150 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

6. Epirubicin 40 mg.
   Procytox 400 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

7. Adriamycin 85 mg.
   Cisplatinum 110 mg.
   Cytoxan 1150 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

8. Epirubicin 90 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

9. Epirubicin 70 mg.
   Cytoxan 600 mg.

   none 0 1 2 3 4 5 6 7 8 9 10 extreme

10. Methotrexate 60 mg.
    5-FU 500 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

11. Vinblastine 10 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

12. Methotrexate 30 mg.
    Cytoxan 500 mg.
    5-FU 500 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

13. Epirubicin 40 mg.
    Cyclophosphamide 400 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

14. 5-FU 500 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

15. Vincristine 2 mg.
    Adriamycin 90 mg.
    Cytoxan 1350 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

16. Epirubicin 100 mg.
    Cyclophosphamide 1200 mg.

    none 0 1 2 3 4 5 6 7 8 9 10 extreme

17. Adriamycin 75 mg.
Cuclophosphamide 500 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

18. Methotrexate 40 mg.
Procytox 600 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

19. Vincristine 2 mg.
Procytox 1500 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

20. Vincristine 2 mg.
Adriamycin 75 mg.
Cyclophosphamide 1500 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

21. Vincristine 2 mg.
Adriamycin 75 mg.
Procytox 1000 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

22. Bleomycin 16 units
Adriamycin 55 mg.
Solu-cortef 100 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

23. Vincristine 1 mg.
Adriamycin 30 mg.
Cytoxan 50 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

24. Methotrexate 400 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

25. Nitrogen Mustard 10 mg.
Solu-cortef 100 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

26. Vincristine 1 mg.
Adriamycin 40 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

27. Interferon 5,000,000 units

none 0 1 2 3 4 5 6 7 8 9 10 extreme

28. CDDP 150 mg.
Cyclophosphamide 1000 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

29. Asparaginase 10,000 units

none 0 1 2 3 4 5 6 7 8 9 10 extreme

30. Vincristine 2 mg.
Adriamycin 120 mg.
Cyclophosphamide 2400 mg.

none 0 1 2 3 4 5 6 7 8 9 10 extreme

Appendix B

VAS - ANV

Please give an indication of the extent to which you have felt nauseous or the severity of vomiting which you may have experienced at each of the following times. The left edge of the line indicates "none", while the right edge indicates "extremely".

1. Last evening

none _____ extremely

2. This morning before coming into the clinic

none _____ extremely

3. Now, immediately before treatment

none _____ extremely

Appendix C

VAS - ANX

Please give an indication of the extent to which you have felt anxious at each of the following times. The left edge of the line indicates "none", while the right edge indicates "extremely".

1. Last evening

none _____ extremely

2. This morning before coming into the clinic

none _____ extremely

3. Now, immediately before treatment

none _____ extremely

Appendix D

Side Effects of Chemotherapy Study

Preliminary Information

The Department of Psychosocial Resources is conducting a study on the side effects of chemotherapy. We are interested in your experiences with chemotherapy and if you are willing, we would like to ask you a number of questions about yourself and your response to treatment. The initial interview should not take more than 30-40 minutes and will be conducted at a time and place that is convenient for you. Prior to each chemotherapy session we would also like to ask you a few questions about your experiences, which would require 5-10 minutes.

We will be telephoning you in the near future to ask if you would be willing to participate. Please note that whether or not you participate will in no way affect your treatment here in the Cancer Centre.

Thank you for your consideration.

Dr. Hank Stam
Mr. Gary Challis
Department of Psychosocial Resources

Appendix E

INFORMED CONSENT

I, _____, do hereby give permission to be interviewed by Gary Challis, a M.Sc. student in the Department of Psychosocial Resources at the Tom Baker Cancer Centre. I understand that this research is aimed at explaining some of the factors related to the side effects of cancer chemotherapy.

I also understand that at the time of a personal interview I will be asked to respond to a number of questions concerning myself, my disease, my treatment, and how I feel about them. Additionally, I will be required to respond to a number of brief questions prior to a maximum of 6 of my chemotherapy sessions. I understand that the initial interview will not exceed one hour in length, and that each of the subsequent testing times will require only 10 to 15 minutes to complete. I am aware that my participation in this study is voluntary and that I may withdraw from this study at any time. I may also refuse to answer any question I so desire.

I know that my answers will be kept confidential and anonymous and that I can ultimately decide whether the researcher may use my responses in his report. I also know that my withdrawal from this study will have no effect upon the care or treatment which I am to receive from the Centre.

_____
Signature

_____
Name (please print)

_____
Witness

_____
Date

Appendix F

FREQUENCY OF POSITIVE RESPONSES TO THE PSC

PATIENTS WITH ANV

	Interview Sessions					
Symptom	1	2	3	4	5	6
Appetite loss	3	4	3	4	2	3
Dizziness	2	3*	3	2	1	0
Dry mouth	4	6	7	12	5	4
Fatigue	3	5	8	11	9*	6
Headache	2	3	5	5	3	7*
Sleep loss	2	6	3	6	5	5*
Nausea	----	----	----	----	----	----
Vomiting	----	----	----	----	----	----
Changes in Smell	3*	2	0	1	1	1
Changes of Taste	4	3	2	2	0	2
Number of cases:	4	10	16	18	11	12

* $p < .05$.

Note: (*) indicates a significant chi-square for the frequency of occurrence of this symptom for patients with and without ANV (see Appendix G).

Appendix G

FREQUENCY OF POSITIVE RESPONSES TO THE PSC

PATIENTS WITHOUT ANV

	Interview Sessions					
Symptom	1	2	3	4	5	6
Appetite loss	25	16	13	9	6	8
Dizziness	16	3*	9	7	3	1
Dry mouth	29	21	17	7	12	12
Fatigue	35	20	28	19	15*	12
Headache	20	16	11	17	8	9*
Sleep loss	24	17	13	15	12	5*
Nausea	----	----	----	----	----	----
Vomiting	----	----	----	----	----	----
Changes in Smell	9*	6	3	3	2	3
Changes of Taste	13	10	8	7	6	5
Number of Cases:	60	55	54	52	48	47

* $p < .05$.

Note: (*) indicates a significant chi-square for the frequency of occurrence of this symptom for patients with and without ANV (see Appendix F).

Appendix H

ANCOVA SUMMARY TABLE -- DEPRESSION

Source	SS	df.	MS	F
ANV	1230.59	1	1230.59	3.65 n.s.
Toxicity (covar.)	9.61	1	9.61	0.03 n.s.
Error	18857.28	56	336.74	
Depression	670.52	6	111.75	2.74 *
ANV-Depression	106.18	6	17.70	0.43 n.s.
Error	13962.14	342	40.82	

* $p < .05$

Note: Not significant ($p < .05$)

Appendix I

ANCOVA SUMMARY TABLE -- DEPRESSION

Source	SS	df.	MS	F
Absorption	841.08	1	841.08	2.45 n.s.
Toxicity (covar.)	63.59	1	63.59	0.19 n.s.
Error	19246.80	56	343.69	
Depression	800.73	6	133.46	3.28 *
Absorb-Depression	168.09	6	28.02	0.66 n.s.
Error	13900.23	342	40.64	

* $p < .01$

Note: Not significant ($p < .05$)

Appendix J

ANCOVA SUMMARY TABLE -- STATE ANXIETY

Source	SS	df.	MS	F
ANV	2514.23	1	2514.23	5.22 *
Toxicity (covar.)	636.97	1	636.97	1.32 n.s.
Error	26950.86	56	481.27	
State anxiety	1586.05	6	264.34	5.24 **
ANV-State Anxiety	652.76	6	108.79	2.16 *
Error	17244.21	342	50.42	

* $p < .01$

** $p < .001$

Note: Not significant ($p < .05$)

Appendix K

ANCOVA SUMMARY TABLE -- STATE ANXIETY

Source	SS	df.	MS	F
Absorption	1986.11	1	1986.11	4.05 *
Toxicity (covar.)	1013.53	1	1013.53	2.07 n.s.
Error	27478.97	56	490.70	
State Anxiety	1412.17	6	235.36	4.65 **
Abs.-State Anxiety	588.25	6	98.04	1.94 n.s.
Error	17308.71	342	50.61	

* $p < .01$

** $p < .001$

Note: Not significant ($p < .05$)

Appendix L

CLASSIFICATION RESULTS -- HYPOTHESIS 1

Actual Group	Number of cases	Predicted	
		no-ANV	ANV
no-ANV	49	43 87.8%	6 12.2%
ANV	21	4 19.0%	17 81.0%

Percentage of grouped cases correctly identified: 85.71%

Appendix M

CLASSIFICATION RESULTS -- ABSORPTION/APQ

Actual Group	Number of cases	Predicted	
		no-ANV	ANV
no-ANV	49	39 79.6%	10 20.4%
ANV	21	1 4.8%	20 95.2%

Percentage of grouped cases correctly identified: 84.29%

Appendix N

CLASSIFICATION RESULTS -- HYPOTHESIS 4

Actual Group	Number of cases	Predicted	
		no-ANV	ANV
no-ANV	49	46 83.9%	3 6.1%
ANV	21	0 0.0%	21 100.0%

Percentage of grouped cases correctly identified: 95.71%