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The expression of hunger and appetite by patients receiving total parenteral nutrition

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THE EXPRESSION OF HUNGER AND APPETITE BY PATIENTS RECEIVING TOTAL PARENTERAL NUTRITION

by

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DISSERTATION

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THE EXPRESSION OF HUNGER AND APPETITE BY PATIENTS RECEIVING TOTAL PARENTERAL NUTRITION A DESCRIPTIVE STUDY

CHAPTER ONE

THE STUDY PROBLEM

The use of TPN has made possible the maintanence, solely by the intravenous route, of individuals who would have otherwise died of malnutrition (Dudrick, 1968). This technique has been well established, and its metabolic and mechanical complications well documented, but the human responses to it have received little attention.

Patients are affected by situational stresses which result in adaptive responses and the goal of nursing is to promote the patient's optimum adaptation in health and illness. This goal is accomplished either by directly altering, or assisting the patient to alter, the stress and/or the response to stress (UCSF School of Nursing, 1979).

In order for the nurse to attain the goal of altering either the stress or the patient's response to it, the sources of stress with TPN must be described and correlated with the clinical pictures; the factors must be isolated and addressed. To intervene requires a knowledge of the origin of the possible internal or external stresses and the approach needed to help the patient cope. This is an essential prerequisiste to a research-based practice.

Total parenteral nutrition presents the patient with a tremendous burden of changes in activities of daily living superimposed on an

alteration in normal eating. Hunger and appetite are clusters of sensations which stimulate the acquisition and ingestion of food. When these sensations are present, they signal the need or desire for nutrients or oral gratification, but if the person is unable to respond to them, these sensations may become distressing.

The loss of the ability to eat has been reported to be a major life stress for patients on home parenteral nutrition (Gulledge, Gipson, Steiger, Hooley, & Srp, 1980; Jedlicka, Davis, & Johnson, 1977; Malcolm, Robson, Vanderveen, & O'Neil, 1980; Perl, Peterson, Dudrick, & Benson, 1981; Price & Levine, 1979; Robinovitch, 1981), but only one study has examined hunger in hospitalized patients receiving TPN (Jordan, Hamilton, MacFayden, Jr., & Dudrick, 1974). Although some authors mention the expression of hunger and appetite as an area of concern with hospitalized patients on TPN (Bayer, Bauers, & Kapp, 1983; Grant, 1980; Levenson, 1982), these concerns have not been supported by systematic study. Recently published major textbooks on clinical nutrition made no reference to these alterations (Deitel, 1985; Rombeau & Caldwell, 1986).

Most of the literature has focused on patients on long-term parenteral nutrition where hunger and/or appetite are viewed as psychosocial responses. In reality, however, it is not known whether these feelings are primarily due to psychological factors associated with feelings of oral deprivation or whether they have a physiological basis such as inadequacy of caloric intake.

Contributing to this confusion is the fact that the terms

"hunger" and "appetite" are often used interchangeably and with a variety of meanings (Castonguay, Applegate, Upton, & Stern, 1984). While hunger and appetite are often used synonymously to indicate a need or craving for food, there may be an important distinction between the two states for the patient on TPN who is unable to take anything by mouth.

The Problem

It is well accepted that humans maintain their weight with remarkable consistency over long periods of time. The most obvious evidence of a physiologic mechanism regulating caloric intake is that food intake is adjusted to energy requirements with such precision that body stores of fat over the age continuum vary only slightly (Janowitz, 1958). Yet, when people are fed artificially, their internal regulators are not considered.

An accurate assessment of the energy needs of the patient on TPN is necessary to provide caloric requirements and promote anabolism, while avoiding excessive energy intake. Several methods of estimating a patient's energy requirement have been developed, but controversy exists as to which is the most appropriate (Paauw, McCamish, Dean, & Ouellette, 1984; Quebbeman, Ausman, & Schneider, 1982). The most accurate methods of estimating caloric requirements require elaborate equipment to estimate metabolic rate and invasive techniques to estimate body composition.

When people are fed artificially, little attention is given to their physiological regulators and a patient's expression of hunger

is viewed as a pychological response. Feelings of hunger often accompany TPN, and caretakers are instructed to assure the patient that these feelings are normal (Bayer, 1984; Jedlicka, Davis, & Johnson, 1977). An extensive review of the literature revealed no studies supporting this approach.

A statement of appetite may reflect an oral craving or desire (Nicolaidis & Rowland, 1976). A statement of hunger, on the other hand, may be a danger signal indicating a physiological deficit (Grossman, 1955); a sign of inadequate repletion (Hansen, et al, 1977). Patients may indeed be indicating a psychological appetite ("I'd love a banana split right now") as opposed to a physiological need ("I'm hungry"), but the failure to recognize or acknowledge the distinction between hunger and appetite may allow a clinical problem to go untreated.

The increasing use of TPN requires explication of the totality of the human response to this potentially disruptive and distressing situation about which so little is now known. It is this totality which confounds investigations into the expression of hunger and appetite. Human beings are biological, psychological, and sociocultural in nature and are in constant interaction with their environment. The ingestion of nutrients meets a biological demand but also serves psychological and sociocultural purposes and has implications for the body, mind and spirit.

In a report of the psychiatric aspects of TPN, Levenson (1982) stated that "Depression is the most common and prominent

psychological disturbance found during [TPN]" (p. 38), yet all of the supporting references concerned patients on long-term TPN. Although no studies have examined the incidence of depression in hospitalized patients on TPN, it would seem that they would be especially vulnerable. Thus, depression as the etiology of alterations in expressions of hunger and appetite in patients on TPN must be ruled out before it is assumed that these alterations are a direct result of therapy.

The Purpose

The purposes of this study were to determine in a sample of hospitalized patients receiving TPN as their sole nutritional intake.

1. Whether there are differences in the magnitude of expression of hunger and appetite.

2. The relationship between the magnitude of the expression of hunger and the adequacy of caloric intake.

3. The relationship between the magnitude of the expression of appetite and the adequacy of caloric intake.

4. Whether there is a relationship between depression and the magnitude of hunger.

5. Whether there is a relationship between depression and the magnitude of appetite.

Significance of the Study

Patients maintained solely on TPN, who are unable to eat in response to their feelings of hunger and appetite, depend on others to determine their nutritional needs and assure adequate intake. Studies investigating TPN and its effect on hunger and satiety in animals have demonstrated that animals adjust their voluntary oral food intake to compensate for the adequacy of calories infused parenterally (McHugh & Moran, 1978; Hansen, Jen, & Kripps, 1981; (Martyn, Hansen, & Jen, 1984). It is plausible that patients on TPN would respond to a low caloric intake in a like manner if they had the capacity for oral intake.

Because of its relative abundance, food intake in the contemporary Western world is guided more by appetite (tempting sights and odors, cravings for certain favorites, social and religious mores, and the habit of eating at certain times) than by physiological hunger (Cannon, 1929; Deitel, 1985). In most instances, the amount of food ingested in response to these appetitive cues is sufficient to provide adequate nourishment. When patients are unable to respond to these sensations for themselves, caretakers must elicit this information and intervene for them. If hunger is "that set of internal signals that stimulate the acquisition and consumption of food," (Castonguay, et al, 1984, p. 19), and appetite is "the incentive to eat aroused by external food-related stimuli, e.g., taste" (Sclafani, 1976, p. 281), exploring the meaning of a patient's expression of hunger is of paramount

importance. The presence of hunger sensations may indicate inadequate nutritional intake which, if unchecked, will lead to malnutrition.

The meaning of the expression of hunger and appetite by hospitalized patients on TPN is ambiguous at best. Is a patient complaining of hunger expressing a wish to return to normal eating habits or reflecting a physiological deficit? If it were known that patients could interpret their internal signals and distinguish between hunger and appetite, then the meaning of each could be explored and the appropriate intervention selected.

CHAPTER TWO

THEORETICAL FRAMEWORK

The theoretical framework for this study involves the distinction between hunger and appetite. It posits that the etiology of hunger and the etiology of appetite are separate and distinct entities, not two poles of one continuum. The nature of these psychic or subjective signals for food ingestion has been of interest to scientists and philosophers for centuries (Bolles, 1980), and debate over semantics of these terms has raged equally long (Grossman, 1955).

In their classic study of the nature of hunger signals, Cannon and Washburn (1912) refuted the then commonly-held view that appetite was the first stage of hunger. They concluded that the two were fundamentally different, in etiology, localization of sensations, and in psychic elements. "Hunger may be satisfied while the appetite still calls" (Cannon, 1929, p. 270).

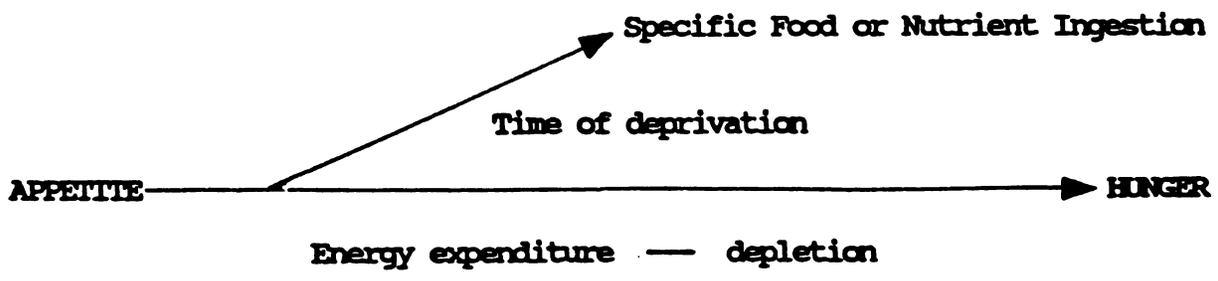
Grossman (1955) differentiated between hunger and appetite, relating hunger to a physiological need, and appetite to an affective desire. Janowitz (1958) wrote that, although many authors had advocated the banishment of the terms hunger and appetite as separate sensations, "Their (the terms') stubborn longevity appears to this observer to mean that there is some basis of reality for the distinction drawn between hunger and appetite in every day speech" (p. 327). Mayer (1955) lamented that the distinction between hunger and appetite had been so generally accepted that "desirable

though it may be to reject it, it seems a little late in the day to do so" (p. 15).

The controversy continues. Booth (1981) stated that "it is a confusion of phenomenological and physiological categories to redefine 'hunger' as physiological drive and 'appetite' as dietary craving" (p. 147). Mayer (1968, & 1976) distinguished between hunger and appetite, yet adopted the earlier view of a continuum. He defined appetite as the first, pleasant, complex of sensations by which one is aware of a need for food; and hunger as the complex of unpleasant sensations, felt after a period of prolonged deprivation, which impels the organism to seek food. "The passage from appetite to hunger is dependent on duration of deprivation, rate of energy expenditure, etc." (Mayer, 1976, p. 474).

Figure 1 depicts the view of those authorities who view appetite and hunger on a continuum.

Figure 1. Appetite and Hunger Viewed on a Continuum



Appetite, a cluster of pleasant sensations is presented as the first, gentle urge to eat; selected food may be ingested at this stage. If deprivation continues over time, the feelings intensify and appetite becomes hunger, a cluster of unpleasant sensations. In the beginning stages, one is more particular about the food selected, but as deprivation continues, one becomes less particular and will seek any food to eat.

Hunger, as viewed in this study, is a subconscious and involuntary instinct resulting from biochemical and physiological responses; appetite is a function of a person's mind, a learned mechanism expressed according to personal preferences of taste, social conditioning, mores, and taboos. Ingestive behaviors are complex and are controlled by a multitude of mechanisms that depend jointly upon physiological and psychological factors as well as the overriding ultimate control of the higher brain centers, the cognitive, cerebral factors. The ability to consciously and voluntarily restrict food intake in spite of urges may well be the feature that most clearly distinguishes the eating behavior of humans from that of animals (Stunkard, 1981). Satiety is related to hunger and appetite as an opposing affective state signifying a lack of desire to eat or, more precisely, a desire not to eat (Grossman, 1955). It is not simply a decrease in food intake but a cluster of sensations reflecting satisfaction and fulfillment. Satiety cannot be implied simply because one stops eating or does not eat when presented with food. Animals and humans can consciously avoid

ingestion when hungry or with appetite or may be incapable of eating even though hungry (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). For example, some survivors of the airplane crash in the Andes (Read, 1974) were unable to eat another human being and, in spite of extreme physical deprivation, consciously refused to eat food that was abhorent to their conditioning and chose to starve to death. Also it is possible to have feelings of hunger without an appetite for food (Bruch, 1955), when, for instance, one knows that hunger exists but "nothing sounds good." Conversely, although hunger may be satisfied, a tempting dessert stimulates the appetite. One may eat in response to social or cultural conditioning even in the absence of hunger or appetite. Or, hunger may occur to such an intensity that one eats food which would otherwise be distasteful, food which does not stimulate the appetite. Table 1 summarizes the relationships between hunger, appetite, and satiety.

Clearly, there is not a consensus in the literature regarding hunger and appetite. Authorities who distinguish between hunger and appetite have defined hunger as the drive for food aroused by internal stimuli which reflect physiological deficits accumulated during food deprivation and body weight loss; and appetite as an incentive to eat aroused by external food-related stimuli (e.g., taste and texture) or cultural conditioning (Sclafani, 1976; Durrant 1981). This is the basis for the present study and is depicted in Figure 2.

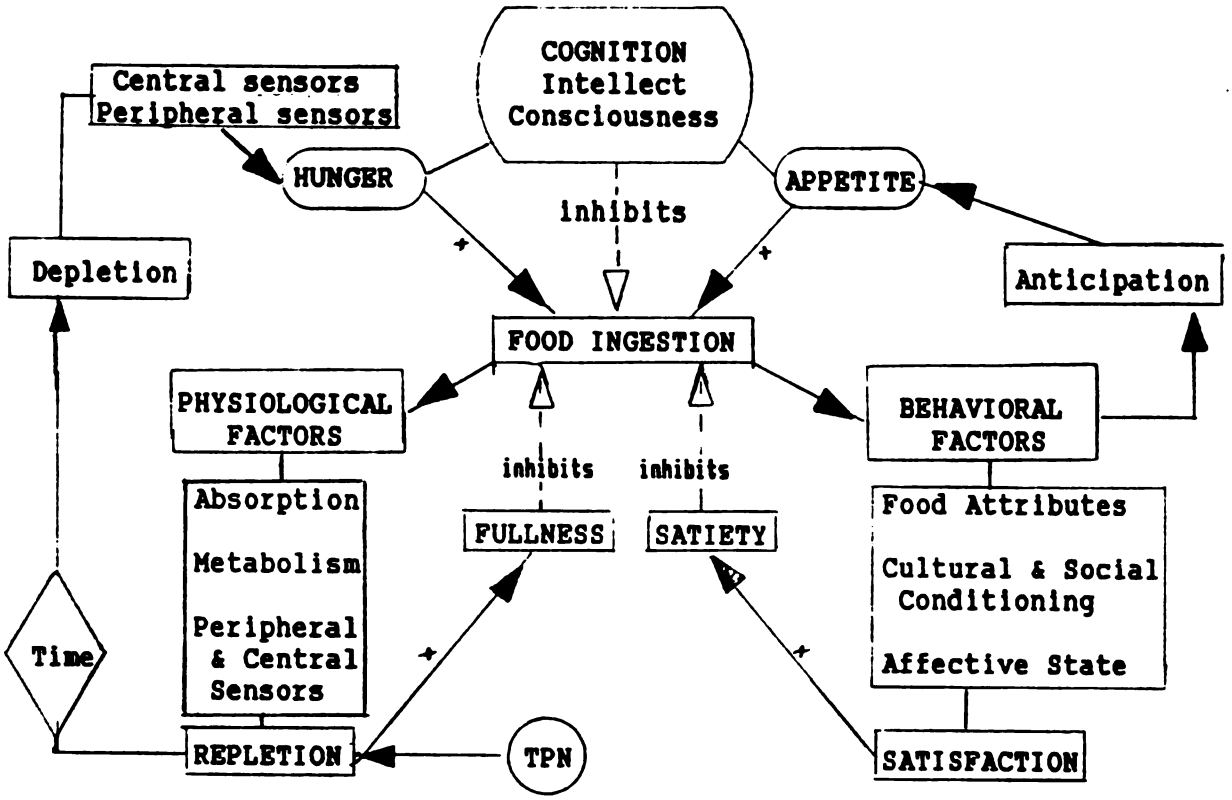
Table 1. Relationships between Hunger, Appetite, and Satiety[1]

Concept	Subjective experience	Conscious feelings	Observed behavior
HUNGER	Physical symptoms (individualized) -visceral -mouth and throat -cerebral -generalized	Urge to eat	Eating begins
APPETITE	Mental urge for specific food	Craving or desire	Specific food selected
	Perceived pleasantness		
	Food attributes -smell -taste -texture		Eating begins
	Cultural & social preferences		
SATIETY	Physical fullness; cessation of hunger	Fullness	Eating ceases
	Mental satisfaction; cessation of appetite	Satisfaction	Eating ceases

[1]Note: Eating may begin in response to hunger and/or appetite and may cease in response to physical fullness and/or mental satisfaction. Alternatively, eating may begin in the absence of hunger or appetite or cease without physical fullness or mental satisfaction.

Affective states or moods may cause alterations in hunger, appetite, or eating. Depression results in a lack of interest, desire or pleasure; this may extend to a lack of desire for food, a lack of interest in seeking food, or a lack of pleasure in eating food so that ingestion ceases. Some depressed people, however, respond with increased food intake. The motivation for the observed behavior must be sought from the individual's subjective experience, and not assigned to the individual.

Figure 2. Conceptual Model of Hunger and Appetite[1]



[1]Hunger and appetite are different in physiolgical basis and in psychological elements. Both have the same function of initiating food ingestion; they may operate in combination or singly.

Note that TPN operates only via repletion of energy stores and metabolites to inhibit food ingestion. Rather than an observed inhibition of food intake, which is not allowed in most patients on TPN, this would be reported as a lack of hunger or a disinhibition to ingest food should it be available. Although TPN bypasses the gastrointestinal (g.i.) tract structurally, some g.i. factors may still be operational due to hormonal responses. TPN does not participate in the appetitive structures, but patients on TPN may cognitively deny hunger or appetite, if they believe they are being adequately nourished.

The overriding factor in the contol of food intake is the cognitive element which can prevent food intake despite overwhelming and intense feelings of hunger and appetite. Even though feelings of hunger and/or appetite may be intense the conscious control of choice of food, guided by likes and dislikes and social restraints, prevents one from eating "just anything" to prevent starvation.

When one is able to respond to individual signals for food ingestion, the distinction between hunger and appetite may be just a confusion of terms. When one is not able to respond to these sensations, the expression of these feelings must be sought out and interpreted by others, especially if the sensations of hunger and appetite are regulatory systems.

It is not known to what extent the sensations of hunger and appetite are merely an awareness that the regulatory processes are operating or to what extent they participate in, and control, the regulation of food intake. In lower forms of animals, the urge to eat is probably a primitive instinct that exists at all times unless inhibited, and regulation is mainly involved in inhibiting this signal (Morley & Levine, 1980). The higher the organism on the phylogenetic scale, the more important the sensations of hunger and appetite become (Lepkovsky, 1973). Even though these sensations seem to be dispensible in regulating intake, the unrequited feelings of hunger and/or appetite may be distressful to those who are not allowed oral intake due to disease or anatomical structural abnormalities.

LITERATURE REVIEW

Most authors, in the review of the literature which follows, use the terms appetite and hunger interchangeably to denote eating behavior itself, the size of the meal reflecting the intensity of the feelings (Bolles, 1980). Measurements of hunger ratings in humans often have been equated with appetite, and appetite in turn

has been equated with quantitiy consumed (i.e. meal size). These inferences should, however, be treated with caution (Smith, 1982).

Rowland and Carlton (1986) in a comprehensive review of the literature regarding pharmacological appetite suppressants, suggest caution when reviewing research studies which used appetite and hunger interchangeably. They concluded that until the normal mechanisms of hunger and appetite are understood, research into the causes and controls of ingestive behavior will remain elusive.

The effects of the caloric density of infused liquid diets on voluntary food intake has been examined in a number of studies. Investigators using animals as research subjects have noted that the animals will eat more to compensate for a reduction in calories infused either parenterally or enterally (McHugh & Moran, 1978; Hansen, Jen, & Kripps, 1981; Koopmans & Maggio, 1978; Leibling, Eisner, Gibbs, & Smith, 1975; Share, Martyniuk, & Grossman, 1952, Campbell & Davis, 1974; Martyn, Hansen, & Jen, 1984). There is some evidence that humans also will compensate by eating more when given a diluted test diet with reduced calories (Campbell, Hashim, & Van Itallie, 1971; Porikos, Hesser, & Van Itallie, 1982).

DeSomery & Hansen (1978) investigated the effects of parenteral nutrition on the number of calories orally ingested by two monkeys. After recovery from surgery to implant intravenous catheters and gastric strain gauges, baseline determinations of each monkey's usual caloric intake were made, using a complete liquid diet delivered through a feeding machine to which the monkeys had become

accustomed. TPN was given at varying levels of the subject's baseline intake while the subjects were allowed access to the liquid oral diet. In the two monkeys, voluntary intake was reduced in response to the increasing levels of the TPN infusion. The total caloric intake of one monkey was generally above baseline for all levels of infusion while the other monkey over-ate only at the lower infusion levels and ceased oral feedings completely when 100% of his baseline intake was being given as TPN. Motility data (reported only on the second monkey) showed that the motility pattern usually associated with hunger was absent at the high levels of TPN infusion. Suppression of oral intake continued after cessation of TPN for nine and 21 days. In another report of this study, the authors hypothesized that hunger might be a danger signal indicating inadequate repletion (Hansen, DeSomery, Hagedorn, & Kalnasy, 1977).

Nicolaidis and Rowland (1976) studied the effect of various intravenous (IV) nutrients on the oral (PO) intake of regular chow in rats and found a direct correlation between less nutritionally complete infusions and the the largest oral intake. These authors concluded that the residual oral food intake resulted from a specific appetite for a missing element in the diet. They cited the results of Adair, Miller, & Booth (1968) who offered rats the same diet PO that was being infused IV and found marked hypophagia. Had Nicholaides and Rowland offered the rats a varied diet PO, the rats' intake might have increased. Nicolaides & Rowland extended their work (1977) and devised a system where rats learned to press a

lever for intravenous self-injection of liquid diet when oral food was not available. Intakes were low but regulated, and were sufficient to balance energy expenditures, although at a lower body weight. They concluded their report by stating: "Systemic receptors alone are thus adequate to motivate feeding behavior and meter the caloric yield of the intravenous injections" (p.589).

There is some evidence to support the hypothesis that humans also respond to varying levels of caloric intake. Walike, Jordan, and Stellar (1969) studied the effects of disguised oral preloads from a hidden reservoir on subsequent meal intake and subjective ratings of hunger in 17 normal volunteers. All caloric preloads reduced intake of the test meal and the greater the preload, the greater was the depression of intake. The accuracy of the compensation was inconsistent, however, and some subjects overate their baseline at the test meal. Hunger, rated from 0 (not hungry) to 9 (hungriest ever), varied with the caloric value of the preload; the lowest caloric preload produced the greatest hunger rating, which diminished as the caloric value increased, but hunger was never completely eliminated. Again, hunger and appetite were not differentiated, and the subjects may have been indicating an appetite for solid food during the liquid meals, rather than a physiological hunger.

Geliebter (1979) administered varying caloric preloads to 12 healthy men after a 13 hour fast. Subjects were asked to indicate their feelings of hunger on a scale from 0 (not at all hungry) to

100 (extreme hunger) and satiety on a similar scale. The amount of a liquid diet taken through a straw from a hidden container was measured one hour later. The higher caloric preloads tended to suppress intake more than the non-caloric ones, but there were no differences in hunger ratings before or after the preload or between the caloric and non-caloric preloads. The investigator did not differentiate between hunger and appetite and defined appetite as "the subjective evaluation of hunger and satiety" (p. 271). Subjects gargled with a lidocaine solution for seven minutes before the preload and wore a noseclip during ingestion to disguise taste and consistency of the preload. It is conceivable that these would adversely affect a subject's feeling of appetite, in that nothing would seem tasty. However, feelings of hunger may not have been as affected as appetite by the dulled sensations of taste and smell. Had the investigator differentiated between hunger and appetite, different conclusions may have been drawn.

In the single study in which the subject's expressions of hunger and appetite rather than food intake was the dependent variable, Durrant and Royston (1979) reported increased claims of hunger in obese patients when given a disguised low calorie preload compared to a high calorie preload. They gave preloads of either 100 or 300 calories one hour before a meal to 18 obese subjects on a metabolic unit. The subjects were asked to indicate the degree of hunger (physiological signals) and appetite (mental signals) they were feeling immediately after the preload and again one hour later. The

subjects were unable to estimate the energy content of the preload but were significantly more hungry with the low preloads than the high ones ($P<0.02$). Appetite ratings did not correlate with the energy content of the preloads. It would have been interesting had intake of subsequent meals been measured, to see if subjects compensated for the preload and whether the magnitude of intake correlated with hunger ratings. The results of this study are significant in that the subjects were able to distinguish between hunger and appetite and that hunger, a physiological signal, did vary with the energy load while appetite, a mental urge, did not.

In the only reported study of hunger in patients on TPN, Jordan, Moses, MacFayden, and Dudrick (1974) administered a questionnaire to assess hunger and appetite. They observed 18 patients on TPN from 10 to 85 days (M= 31) to assess the degree of hunger felt during therapy and its relationship to the amount of food ingested during the transition to oral feedings. The investigators found that most patients reported hunger during TPN in spite of daily intakes as high as 4800 kcal and even the hungriest patients had difficulty eating when oral diets were introduced. The authors concluded that the patients' reports of hunger "reflected a need for some oral stimulation, or satisfaction rather than a need to actually ingest the food" (p.153).

There are several methodological difficulties in interpreting Jordan's data. The questionnaire used to assess hunger and satiety is open to criticism in that some items contained more than one

condition or feeling but required a "yes" or "no" response, making it unclear to which item the answer referred. Also, assessments of hunger were not obtained concurrently with measurements of metabolic state (e.g., blood sugar levels) or correlated with the adequacy of caloric intake. The subjects in this study had diagnoses that included pelvic abscesses with sepsis enterocutaneous fistulas and inflammatory bowel disease, and perhaps had caloric requirements greater than 4800 per day. In their conclusion, the authors in essence doubted the validity of the patients' expression of hunger when they were able to consume only a small amount of food. However, TPN leads to hypoplasia of the stomach and small intestine, with decreased absorptive abilities (Feldman, Dowling, McNaughton, & Peters, 1976; Johnson, Copeland, Dudrick, Lichtenberger, & Castro, 1975; Morin, Ling, & Bourassa, 1980; Ryan, Dudrick, Copeland, & Johnson, 1979;). These anatomical and physiological changes occurring with TPN may account for early satiety in spite of feelings of appetite or hunger.

The study is of interest, however, because it is the only study of hunger in humans on TPN. It points out the magnitude of the problem in that 16 of the 18 patients reported hunger during therapy. Based only on patients' inability to eat normal amounts when presented with foods, the authors considered the patients' complaints psychologically induced and advocated the practice of reassuring patients that there is no need to worry about nutrition. Further research is required before such feelings are dismissed as a

natural occurrence with TPN, especially when the adequacy of intake is unknown or undetermined.

Oral deprivation with artificial feeding has been considered a psychological phenomenon. Padilla, et al, (1979) reported findings from a study of 30 patients on nasogastric tube feedings. The three most commonly reported distressing psychosensory experiences were related to oral deprivation: experiencing an unsatisfied appetite for food; being deprived of tasting, chewing, or swallowing food or drinking fluids; and being deprived of regular food. Physiological aspects of hunger and appetite were not pursued. According to several other authors, the inability to respond to feelings of hunger and/or appetite would seem to be distressing to the hospitalized TPN patient (Bayer, Bauers, and Kapp, 1983; Colley and Phillips, 1973; Grant, 1980; Salmond, 1981) as well as the patient on enteral tube feedings or home TPN, but no studies have explored this issue.

The only study in which the psychological effects of TPN in hospitalized patients were investigated was conducted by Malcolm, Robson, Vanderveen, and O'Neil in 1980. They followed 59 patients over a period of one year and found that during short-term TPN (less than one week), psychological alterations seemed to have a biological etiology and neither the patient nor the family viewed TPN as anything beyond another hospital treatment. During intermediate-term TPN (one to 12 weeks), as control became more of an issue, the catheter and infusion apparatus became more important

to the patient but the family still seemed to view it as just another treatment. During this time, anxiety and apprehension were the predominant alterations in psychological states. The patients on long-term, home TPN were subject to more severe psychological disturbances such as phobias, which required the involvement of family or other care-givers. The investigators did not address the issue of hunger or appetite.

The realization that one is hungry is a private process based on the recognition and integration of various sensations. In only one study has the array of sensations of hunger been examined. Mayer, Monello and Seltzer (1965) used a self-administered, structured questionnaire consisting of eight single and 70 multiple-choice questions "designed to help the subjects recollect their sensations or moods" (p. A-98).

Eight hundred subjects, including children and adults, were asked to describe their physical sensations and moods at six different times: 1) two hours before a meal; 2) 1/2 hour before a meal; 3) during a meal; 4) 1/2 hour after a meal; 5) two hours after a meal; and 6) during extreme hunger (as recalled). The subjects were separated into five groups, according to age and gender. Each group included between 100 and 200 subjects. There were two groups of adult college graduates 20 to 67 years of age; one group of adolescent girls; one group of adolescent boys; and one group of obese adolescent girls from a summer camp.

Manifestations of hunger reported in this study were grouped

into 1) gastric sensations (emptiness, rumbling, ache, pain, tenseness, nausea); 2) mouth and throat sensations (emptiness, dryness, salivation, pleasant or unpleasant taste or sensation); 3) cerebral sensations (headache, dizziness, etc.); and 4) general over-all sensations (weakness, fatigue, restlessness, coldness, warmth). Moods were categorized as 1) negative active mood (irritable, nervous, tense); 2) negative passive mood (depressed, apathetic); 3) positive passive mood (calm, relaxed, contented); and 4) positive active mood (cheerful, excited). Preoccupations with thoughts of food were graded as 1) not at all preoccupied (no thoughts of food); 2) mildly preoccupied (only occasional thoughts of food); 3) moderately preoccupied (many thoughts of food but can easily concentrate on other things); and 4) quite preoccupied (most thoughts are on food and find it difficult to concentrate on other subjects).

The authors concluded that gastric sensations appeared to be the most consistent indicators of hunger. Other investigators have attempted to discover a single sensation for the operational definition of hunger, namely gastric "hunger pangs", emptiness, or dull aches. Cannon & Washburn (1912) recognized other affective states as appearing in hunger but stated that these are individualized and negligible and that "the dull, pressing (epigastric) sensation is the constant characteristic, the central fact..." (p.442).

Although gastric sensations have been found to be the most

common indicator, it is well known that humans can still recognize hunger after gastrectomy or gastric denervation (Grossman & Stein, 1948; Wangensteen & Carlson, 1931). Expression of hunger, therefore, is motivated by a complex of sensations and behavioral responses. The private aspects of this complex may include gastric or other specific sensations, but these are highly variable and individual.

In an attempt to find an objective, valid, and sensitive physiological measure of hunger, salivation response to food has been proposed and tested (Wooley & Wooley, 1981). This method involves weighing cotton dental rolls to quantify the degree of changes in the subject's salivation. However, for salivation to be a valid measure, the subjects must be sufficiently hungry, be able to view, recognize and want the food, and think that they will be able to eat it. This method works well in the laboratory setting but would not be feasible with patients who are allowed nothing by mouth [nil per os (NPO)] and are unable to eat. In addition, salivation is not specific to the desire for food but is influenced by thirst or emotional states having no consistent relationship to ingestive behaviors (Booth & Fuller, 1981).

Other objective measurements such as body weight, laboratory test results, and anthropometric measurements are reflective of the consequences of unmet hunger (malnutrition) rather than hunger per se. In the final analysis, determination of the presence of hunger or appetite relies on the individual experiencing some sensation(s)

or process(es), integrating these, and defining them as hunger or appetite according to the individual's own experience. Thus, measurement and quantification of the degree of hunger must rely on self-report.

While few of the above studies distinguished between hunger and appetite and none investigated the possiblity of hunger as a physiological phenomenon, they reflect the recognition of hunger as a legitimate health concern. The results and paucity of these studies indicate that there is a need for further investigation into the expressions of hunger and appetite in patients receiving TPN.

Theories of the Controls of Hunger and Appetite

The precise mechanisms involved in signalling hunger, i.e. what event(s) impel an organism to seek out and ingest food, are unknown. Several theories have been proposed and some have been scientifically tested, but there does not appear to be any one single event that heralds the sensations and expression of hunger. There are numerous control mechanisms and built-in redundancies in the system; if any one of the control sites is bypassed or altered, other mechanisms take over and it appears that normal regulation ensues.

In this paper, the theories of the control of hunger and satiety have been divided into four subheadings. In the first two parts, the physiological centralist and peripheralist theories regarding the sites of control are reviewed with the inclusion, where

appropriate, of possible control mechanisms. The behavioralist theories, which deal with psychological, emotional, and conditioned learning aspects of hunger and satiety are presented in the third section. Lastly, these theories are integrated.

Centralist Theories

Centralist theories focus upon the role of the central nervous system as the center for regulation of hunger and satiety. The traditional view emphasizes the role of the hypothalamus as the control site. This remains a viable hypothesis, although data indicate that input to the hypothalamus may come from other centers in the brain.

Ventromedial Hypothalamus

It had long been noted that brain tumors led to obesity, (Brobeck, 1943) but the area of the brain responsible for this development, whether the pituitary or the hypothalamus, was unknown. Hetherington and Ranson (1939 & 1940) reported the results of studies that first presented the role of the ventromedial hypothalamus (VMH) as the satiety center.

Although it had been shown that VMH damage would produce obesity the pathogenesis of this obesity was undetermined. Evidence that these lesions produced overeating leading to obesity was presented in 1943 by Brobeck, Tepperman, and Long.

This group induced electrolytic lesions in the VMH of rats and compared their intake to a control group of rats offered the same

diet. The team then studied 12 groups of pair-fed rats, with one rat of each pair being given electrolytic lesions of the VMH. They found that the free-feeding rats with VMH lesions ate more than the control group and became obese while the pair-fed, lesioned rats gained only as much weight as their mates. Anesthetization of the VMH with procaine hydrochloride (HCl) led to hyperphagia and provided additonal evidence for the VMH as the satiety center (Epstein, 1960). This role of the VMH was further supported when electrical stimulation of the VMH suppressed intake.

Lateral Hypothalamus

The role of the lateral hypothalamus (LH) as the feeding center was proposed by Anand and Brobeck (1951) with the finding that electrolytic lesions of the LH in rats produced aphagia and adipsia. Stricker, Swerdloff, and Zigmond (1978) injected kainic acid to destroy selectively the cell bodies of the LH in rats while preserving the axons (fibers of passage through the LH). They found that these rats ate less food in response to acute glucoprivation than did a control group. Epstein (1960) found that anesthetization of the LH with procaine HCL suppressed eating while hypertonic saline injections elicited eating.

The Dual Center Hypothesis

The dual center hypothesis posits that the LH, which initiates feeding, is inhibited by the VMH as a result of unspecified body changes after feeding. When these signals are depleted, the LH

becomes active again. The "feeding center" in the LH is responsible for the urge to eat, while the VMH, or some "structure in its neighborhood", exerts an inhibitory control (Anand & Brobeck, 1951). Morley & Levine (1980) postulated that there is a tonic signal impelling animals to eat and that appetite regulation is mainly involved in inhibiting this signal.

Refutation of the LH and VMH as Control Centers

The majority of arguments against hypothalamic control of hunger center around the fact that alterations in other sensory-motor behaviors are also produced by lesion or stimulation, and that these "side effects" play a more important role in the ingestive behavior observed. Brobeck (1946) suggested the possibility that lesions of the hypothalamus resulted in the interruption of fibers passing to other areas of the brain. Behavioral changes seen in LH damage are not confined to ingestive behavior but extend to most aspects of motivation (Krasne, 1962). Lesions of the amygdala produce behavioral changes and alterations in feeding patterns similar to hypothalamic damage (Fonberg, 1981). It has been suggested that the decreased intake seen with LH lesions may be due in part to damage or interruption of trigeminal or olfactory cortex nerve fibers and, possibly, other sensory input (Ziegler & Karten, 1974). However, several investigators have selectively damaged the LH neurons, producing aphagia without other observable behavioral changes (Grossman, Dacey, Halaris, Collier, & Routtenberg 1978; Keesey,

Boyle, Kemmits, & Mitchel, 1976; Stricker, Swerdloff, & Zigmond, 1978).

Controversy remains about the site of CNS regulation, but evidence mounts for more diffuse excitatory and inhibitory systems rather than specific anatomical areas. This appreciation has led to studies attempting to identify the substances and pathways between these excitatory and inhibitory systems and the method of communication with the periphery.

Central Neurochemical Mediators

Monamines. Studies of the effects of monamines on feeding behavior have used various techniques to alter the concentration including: intra-cerebral injections, selective depletion of the putative transmitter, destruction of the monamine pathway with lesions or neurotoxins, increasing the availability of the amino acid precursor, or by inhibiting the monamine oxidase system which catabolizes the monamine (Krasne, 1962).

Studies in which the role of the brain concentration of precursors on monamine synthesis has been examined have involved the use of monamine oxidase inhibitors or large doses of the amino acid (Keesey, et al, 1976). Whether the availability of these substrates is subject to variations with the ingestion of normal meals has been questioned. Peters and Harper (1981) studied animals to determine the relationship between protein and calorie intake, plasma ratios of tryptophan and tyrosine to other neutral amino acids, and brain concentrations of serotonin, dopamine, and norepinephrine. They

failed to find any correlation. Fernstrom (1982) also found no effect on brain concentrations of neurotransmitters with the ingestion of a mixed diet.

Just how the brain receives input resulting in an increase or decrease of these mediators to affect feeding is undetermined. Normal dietary intake probably does not have a great influence on brain concentrations of serotonin, norepinephrine, epinephrine, and dopamine (Wurtman, 1982) but it has been shown that acute alterations in diet can influence these levels. The amino acid precursors to these neurotransmitters compete with other neutral amino acids for active transport across the blood brain barrier (Bender, 1978). Tryptophan is present in small amounts in foods and an increase in protein intake does little to increase serotonin synthesis. But the insulin stimulated by a meal rich in carbohydrate lowers the concentration of the other amino acids that causes a relative increase in the blood level of tryptophan, thus facilitating its transport into the brain, and increasing serotonin levels (Fernstrom, 1974). Tyrosine is the precursor amino acid for norepinephrine, epinephrine, and dopamine. When the plasma level of tyrosine is increased, brain catecholamine synthesis is increased (Wurtman, Larin, Mostafapour, & Fernstrom, 1974).

Even though the brain concentration of neurotransmitters probably does not vary in normal individuals, certain disease states may alter the blood-brain barrier and the competitive uptake of precursors. In one group of rats, Jeppson, et al (1981) induced

sepsis by puncturing the cecum. Sham-operated rats served as the control group. When they compared the two groups of rats, the investigators found that the brain uptake of labelled neutral amino acids was increased in the septic rats. Krause, James, Humphrey, and Fischer (1979) found elevated concentrations of brain tryptophan and increased levels of serotonin in tumor-bearing rats. Because serotonin is the neurotransmitter which signals satiety in the VMH, these findings may account for the relative anorexia that accompanies fever or cancer. "Synthesis of important substances in the brain is probably at the mercy of changes of food intake only in extreme circumstances" (Curzon, 1978).

Peptides. Until it was demonstrated that many peptides are synthesized by and widely distributed in the brain (Leibowitz, 1980), the interest in peptides as modulators of brain activity was largely pharmacological. Peptides normally do not cross the blood-brain barrier. Numerous regulatory peptides have been identified in the mammalian central nervous system (Roberts, Crow, & Polak, 1981).

The peptide cholecystokinin (CCK) has received the most attention and has been administered centrally as well as peripherally. Studies have concluded that CCK and its active fragment, CCK-8, suppress intake when given as a continuous infusion either intraventricularly, intravenously, or intraperitoneally. Central administration required only five percent of the peripheral dose to suppress intake (Maddison, 1977; Anika, Houpt, & Houpt, 1980; Gibbs, Falasco, & McHugh, 1976; Pi-Sunyer, Kissileff,

Thornton, & Smith, 1981). Della-Fera and Baile (1980) hypothesized that the mechanism of the effect of centrally administered CCK is separate from that of peripheral administration.

Additionally, endogenous opiates have been studied as influencing control mechanisms for hunger and satiety. Opiate agonist injections increased intake while opiate antagonists decreased intake (Baile, Keim, Della-Fera, & McLaughlin, 1980; Trenchard & Silverstone, 1983; Morley & Levine, 1982). Morley and Levine (1982) offered a teleological explanation for the role of endogenous opiates in inducing feeding, stating that, "It would be of use to an animal when hungry and forced to encounter danger to find food to have some protection against pain" (p. 760).

However, nurses anecdotally report that the administration of exogenous opiates results in a decreased desire for food; patients who are in pain and receive narcotic relief often forego or reduce their next meal intake. Whether this is due to pain, the sedative effect of the drug, or its effect on feeding mechanisms is undetermined, but experiments with rats have supported this observation (Reid, 1985). This point helps to illustrate the importance of the differential effects of pharmacological and physiological dosages of putative regulators.

Peripheralist Theories

The peripheralist theories of the control of food intake focus upon the origin of signals of hunger and satiety arising outside the

central nervous system, namely the gastrointestinal (g.i.) tract, adipose tissue, and the liver. Some theories maintain that purely peripheral events are responsible, whereas other theories suggest a peripheral sensor with input into the central nervous system.

Gastrointestinal Tract

The earliest theory of the mechanisms of hunger and satiety began in ancient Greece (Novin and VanderWeele, 1977). It was posited that hunger pangs arising from contractions of an empty stomach initiated feeding and the sensation of fullness terminated it. The first experimental testing of this hypothesis in humans was performed by Cannon & Washburn (1912). Washburn swallowed a tube that had pressure balloons attached. To correlate his gastric motility with hunger, he indicated when he experienced the sensation of hunger. The authors found that, invariably, hunger correlated with increased gastric contractions. They concluded that the periodic activity of the gastrointestinal tract is the sole source of hunger pangs. However, citing a case study of a patient with a total gastrectomy, Wangensteen and Carlson (1931) provided evidence that the stomach is not essential in the expression of hunger.

In more recent times, the study of the g.i tract has focused more on its role in producing satiety than on its role in signalling hunger and initiating food intake. This approach is consistent with the idea that the urge to eat is dominant and would exist at all times unless inhibited by some satiety factor.

Pregastric Phase: Visual, Olfactory and Orosensory. Studies in which the role of the pregastric segment in feeding in animals has been examined have concluded that normal feeding is enhanced by these sensations but that they are neither sufficient nor essential. Epstein & Teitelbaum (1962) eliminated orosensory input by means of direct gastric infusions in rats and found that they were able to regulate their food intake. However, in humans, direct intragastric infusions or varying amounts and caloric dilutions of oral preloads led to overeating during a test meal (Walike, Jordan, & Stellar, 1968). The overeating, according to the authors, was due to insufficient oral stimulation. This view is supported by Rodin (1980) who stated, "internal signals do not guide eating behavior unless cognitive or external cues are present" (p. 231).

The subjects in the Walike, Jordan, and Stellar (1968) study received no cues, visual or tactile, because the intragastric preloads were out of the subjects' view and the caloric value of the oral preloads was disguised. Over a longer period of time, both humans and animals are able to adjust their oral intake when given direct gastric infusions of varying percentages of their baseline intake (Jordan, 1969; McHugh & Moran, 1978). However, the observation that overeating occurs initially when oral feedings are combined with intragastric feedings demonstrates the importance of oropharyngeal sensations for proper metering of intake (Kraly & Smith, 1978). It has been well documented that the sight, smell, and taste of food induces significant physiological events (Powley &

Berthoud, 1985), including the release of insulin, which will be discussed later.

Gastric Phase. Several studies have concluded, and it is generally accepted, that gastric distention induces satiety and meal termination in both humans and laboratory animals (Burks & Villar, 1980; Davis & Campbell, 1973; Deutsch, Young, & Kalogers, 1978; Janowitz & Ivy, 1949; Koopmans & Maggio, 1978; Kraly & Smith, 1978). The specific mechanism responsible has not been identified. There appears to be a different mechanism operating which signals gastric distention as opposed to that which monitors the nutrient load of the intake. Kraly and Gibbs (1980) concluded that the signal for gastric distention was not mediated by the vagus when a bilateral subdiaphragmatic vagotomy failed to block the satiating effect of food in the stomach of rats. In contrast, Gonzales and Deutsch (1981) reported that subdiaphragmatic vagotomy did abolish the satiating effect of gastric distention in rats. With the finding that vagotomized rats were able to compensate as well as controls for the removal of nutrients from the stomach, Deutsch and Ahn (1986) concluded that "The signals concerning distention travel to the central nervous system via the vagus. The route taken by nutrient signals remains unknown except that it cannot be the vagus." In a study in which rats were fed into a denervated stomach that had been transplanted from another rat and yet demonstrated satiety, Koopmans and Maggio (1978) concluded that neural signals were not responsible for gastric satiety. They theorized that "Some

humoral signal must be involved" (p. 52). They further proposed that release of this hormone requires a combination of chemical stimulation of the gastric surface and gastric distention for release, the mechanisms of which are also unknown.

Although the role of gastric distention, especially with nutritive substances, in eliciting satiety is well accepted, it is also recognized that gastrectomized humans and animals are able to regulate their intake and eat normally. Nicolaides & Rowland (1977) demonstrated that rats were able to regulate their intake by pressing a lever for self-injection of an intravenous liquid diet, and concluded that "systemic receptors alone are thus adequate to motivate feeding behavior and meter the caloric yield of the intravenous injections."

These findings point out the probable redundancy in the regulatory system for food intake. There seems to be no single mechanism which controls food intake and if one signal is bypassed or non-functional, other signals become operational, or become more dominant, and are sufficient for normal regulation.

Intestinal Phase. The role of the intestines in eliciting satiety seems to be centered in the upper duodenum but whether satiety is signalled by the osmotic or nutrient load of the ingestate, or both, is inconclusive. Yin and Tsai (1973) concluded that the preabsorptive signals from the osmotic load presented to the duodenum were responsible for signalling satiety. They infused iso- and hypertonic solutions of glucose (5, 15, or 30%), hypertonic

sodium chloride (5.4%), or xylose 25% into the stomach and upper intestine of rats. The hypertonic glucose and xylose solutions in the duodenum suppressed oral intake, while the same solutions infused into the jugular vein had no effect on intake. Campbell and Davis (1974) reported that the nutritional load, rather than the osmotic load, contributed to satiety, when infusions of an isotonic nutrient load induced satiety in rats. Leibling, et al. (1975) instilled a liquid diet, in varying amounts and osmolalities, into the duodenum of rats. She found that the combination of both osmotic and nutrient factors in the intestinal load (concentration times volume, rather than either concentration or volume alone), was the stimulus for intestinal satiety.

Combined Pregastric, Gastric, and Intestinal Phases. Smith and Gibbs (1981) suggest that pre-gastric and intestinal mechanisms for satiety are synergistic, over and above the function of pre-gastric stimuli acting as a cue to prepare the intestines. Moran and McHugh (1981) suggest that the rate of gastric emptying into the duodenum provides the link between these two phases.

Adipose Tissue

The role of adipose tissue in the regulation of food intake was first proposed by Kennedy (1953) and expanded by Brobeck (1975). This theory states that the clearance rate of circulating metabolites by fat depots influences hunger and satiety. If fat is being synthesized, the satiety signal produced by feeding will be increased; if fat is not being synthesized then the signal will be

diminished and feeding will continue for a time.

VanderWeele, Haraczkiewicz, & Van Itallie (1982) tested this theory in relation to insulin as the satiety signal in rats. They found a decrease in food intake with infused insulin, which effected the clearance of nutrients from the blood into the adipose tissue. Woods, Lotter, McKay, & Porte, Jr. (1979) also tested Kennedy's theory, using baboons as the experimental subjects. They supported the role of insulin by citing the fact that insulin is secreted in direct relationship to adiposity. They hypothesized that insulin in the cerebral spinal fluid signals the state of adiposity and influences food intake via a central mechanism. In this study, insulin was infused intraventricularly, at varying intervals for several weeks, with a significant reduction of food intake and body weight without an effect on plasma insulin or glucose levels.

The role of fat stores is also implicated in the long-term control of control of body weight. This is discussed in the section on the integration of theories.

Liver

The hepatostatic theory, which places the liver as the site of regulation of food intake, was first proposed by Russek, Rodriguez-Zendejas, & Pina (1968). This theory states that there are hepatic receptors monitoring glucose availability, relating both to glucose delivered to the liver and liver glycogen stores. Thus, when absorption from the intestine is reduced and liver glycogen is

depleted, hunger is signalled. Absorption will restore liver glycogen and liver pyruvate and induce satiety (Russek, 1981).

Campbell and Davis (1974) infused glucose into the portal vein of rats and found decreased intake of an oral glucose solution. They concluded that this effect was specific to glucose when isovolumetric infusions of urea or saline produced no effect. Friedman and Stricker (1976) supported this hypothesis: "Hunger usually is associated with a decreased supply of fuels from the intestines [and adipose tissue]...when the availability of fat is relatively low, the need for exogenous fuels increases. The liver is the organ that is most responsive to differences of this kind in the supply of metabolic fuels from both endogenous and exogenous sources" (p. 428). However, support is not unanimous and, therefore, this theory remains controversial (Bellinger, 1981; Friedman, 1981; Louis-Sylvestre, 1981; Niijima, 1981).

Postabsorptive Signals and Mechanisms

Postabsorptive mechanisms could be mediated by one of the several hormones secreted by the stomach, intestines, or pancreas. The most studied of these are CCK, bombesin (BBS) and insulin.

Cholecystokinin, as reviewed above, has been demonstrated to decrease intake when injected intra-peritoneally (IP) or IV. This decreased intake is enhanced by other satiety factors such as force of habit or central mechanisms (Schallert, Pendergrass, & Farrar, 1982). CCK seems to act locally, rather than on the CNS, since abdominal or gastric vagotomy blocks its effects while lesions of

the VMH do not (Smith, Jerome, Cushin, Eterno, & Simansky, 1981).

Bombesin injected intra-peritoneally (IP) in rats decreased intake (Gibbs, et al., 1979), but controversy exists whether this decreased intake was due to malaise rather than actual satiety (Deutsch, 1980). Other hormones studied (gastrin, secretin, and gastric inhibitory peptide) have demonstrated no effect on satiety (Schanzer, Jacobson, & Dafny, 1978; Smith, et al., 1981).

The effects of insulin are equivocal. Insulin injections produced hunger sensations in normal human subjects but only those with abnormal hypoglycemia (42mg%) and hunger subsided when blood sugar reached 61 mg%, still below normal levels (Janowitz & Ivy, 1949). Increased insulin responses to sights and sounds of food have been implicated in the development of obesity in humans, leading to increased intake to balance the increased level of insulin (Rodin, 1980).

Inoue and Bray (1981) found that hyperinsulinemia resulting from VMH lesions in rats led to excessive fat deposition and obesity. However, it is <u>not</u> thought that insulin causes obesity by leading to overeating because pair-feeding in rats failed to block the obesity seen with VMH lesions (Cox & Powley, 1981). The rats developed hyperinsulinemia but, although allowed to overeat, still gained weight.

Still other researchers theorize that insulin is a satiety hormone. Anika, Houpt, and Houpt (1980) infused insulin intravenously in pigs in doses that approximated physiologic

responses, according to the authors, and found it to depress food intake. Woo, Kissileff, and Pi-Sunyer (1979) infused insulin intravenously in human volunteers while maintaining blood glucose levels and concluded that a rise in plasma insulin levels without a fall in glucose levels does not influence food intake or satiety. The prevailing hypothesis, despite these conflicting results, is that insulin is a satiety signal because its inhibitory effects on satiety appear only after severe hypoglycemia and this effect is nonphysiological (Hernandez & Hoebel, 1980; Powley & Berthoud, 1985; Woods, et al, 1985). This is in keeping with the observations that insulin increases serotonin levels in the brain and that serotonin induces satiety (Fernstrom, 1985).

Energostatic Theory

It has been well established that animals and humans regulate and defend their body weight with remarkable consistency over long periods of time (Novin & VanderWeele, 1977; Jequier & Schutz, 1981). The most easily appreciated evidence of a physiological mechanism which regulates caloric intake, is that food intake is adjusted to energy requirements with such precision that body weight varies only slightly over the age continuum. Such regulation must be both complex and subtle because it is able to integrate information from the volume of a meal, the number of meals eaten daily, and the nutritive density and completness of the diet. Therefore, it is not surprising that this complex device has not been fully mapped and

only an incomplete account of it can be given at present. The main problem with both the pure peripheralist and centralist theories is that none of the identified control sites seems essential to regulation. There are many redundancies in the system, and backup mechanisms exist to replace those that are lost. The elimination of information from peripheral sites (with the exception of adipose tissue, which has not been scientifically tested) has had little long-term effect on feeding. Normal regulation of food intake apparently ensues.

The discrepancies among the findings of the various studies may be reconciled if the control of hunger and satiety is viewed as a system whose main goal is to regulate the body's energy supply (energostatic theory). This involves adjustments in ingestion as well as adjustments in the expenditure of energy. How, where, or when the defended body weight is established is undetermined. Various investigators hypothesize a "ponderostat" or "set point" (Hansen, Jen, & Kripps, 1981; Keesey, 1980, see also studies discussed above).

Animals with LH or VMH damage do eventually recover and maintain their body weight, although at a different level. Keesey, Boyle, Kemmitz, and Mitchel (1976) reported experimentally induced shifts in the regulated body weight of rats with LH lesions. When allowed control of its own intake, each animal successfully maintained a stable body weight, although at a different level than before the operation. After dietary-induced shifts in weight occurred, an

animal increased or decreased intake and returned to pre-diet, baseline weight (Keesey, et al., 1976).

Body weight is maintained at a stable level when energy input equals energy expenditure. "The control of food intake and the modulation of energy expenditure cannot be considered separately" (James, Trayhurn, Daview, Crisp, & Ravenscroft, 1981, p. 147). Keys, et al. (1950) reported that humans responded to chronic caloric restriction with a decreased metabolic rate. In an extensive review of the literature, Landsberg and Young (1981) concluded that the metabolic rate of animals and humans decreased with underfeeding, and increased with overfeeding, under both acute and chronic conditions. Keesey (1980) cited a classic study conducted by Neuman in 1902, in which he, Neuman, overate for three years, consuming approximately 400,000 additional calories during that time. Theoretically, he should have gained about 100 pounds, yet reported a gain of only several pounds. "Clearly, such stability of body weight in the face of such large differences in intake can be accounted for only if marked changes in the rate of energy expenditure have taken place" (p. 155).

The energostatic theory does not account for all aspects of food intake, however. "While it appears that the behavioral event of feeding is essentially controlled by ischymetric (power production of substrates monitored at intracellular level) mechanisms, these are modulated by both specific appetite and 'oral need' factors" (Nicolaidis & Rowland, 1976).

Although physiological events have been identified, their precise causation (input) and mode of action (output) have yet to be determined. Canon and Washburn (1912) stated that "Habit no doubt plays an important role" (p. 452). The control of food intake in humans is more complex than that of other animals because of the greater influence of non-nutritional factors such as psychological and emotional states, sociocultural and religious background, economic status, the amount of work required to obtain food, and the esthetic appeal of the food and environment. These behavioral aspects of the control of hunger and appetite are discussed in the next section.

Behaviorialist Theories

Clearly, hunger and satiety are signaled by physiological mechanisms but the findings of the studies regarding these mechanisms are inconsistent. "The basic mistake in the physiologists' approach was to supppose that some singular stimulus could be found that would tell the animal's brain what it needed to know about the animal's body. Ingestive behaviours really are complex and are controlled by a multitude of mechanisms that depend jointly upon both physiological and psychological factors" (Bolles, 1980 p. 5).

Behavioral (psychological, sociological and cultural) aspects play a significant role in regulation of food intake even in laboratory animals. Friedman and Stricker (1976) have observed that

"animals with nutritional needs may not choose to eat, and that animals with no such needs may eat anyway" (p. 424). Perhaps behavioral aspects could account for the inconsistent findings in a group of four retriever dogs that received the same type of vagotomy; all dogs were tested for completeness of vagotomy yet "...these four dogs, having undergone apparently identical denervation, showed a range of response to food ranging from completely normal to completely abnormal; moreover, these responses were specific to each dog and consistent throughout the study period" (Reverdin, et al., 1980).

Fonberg (1981) also cautioned that social factors play a significant role not only in humans but in animals. In humans, it is well known that a party or social gathering improves appetite and one may eat even though not hungry because the social stimuli add to the reward of eating. In dogs who are accustomed to humans providing their food, these social relations probably also have an alimentary component. "This may influence the results of many experiments performed on ingestive behavior in which social factors are not taken into account; these factors should not be omitted but incorporated into the study" (p. 26). None of the experimental studies reviewed had done so.

Much of the work on non-physiological aspects of human feeding behavior has been reported in the obesity literature*. The prominent theories of obesity are interrelated: The "externality" theory embraces features of the "restrained-eating" theory, and both

are related to the "set-point" theory. The hypotheses underlying these theories will be reviewed below, with the admonition to the reader to bear in mind that numerous nonphysiological factors are involved in the regulation of food intake, including psychological, sociocultural, emotional, environmental, cognitive, and ethnological (Psychological Aspects of Feeding Group Report, 1976) and that there is probably not one theory that explains all aspects of feeding behavior.

Externality

Although almost everyone responds to external cues such as the appearance, aroma, and taste of food, numerous investigators have observed that obese individuals are more affected by these nonnutritional factors than leaner individuals. This "externality" theory was first proposed by Schachter (1968), based on results of studies in which obese subjects ate more than lean subjects in response to visual and sensory cues. The criteria for determining obesity were not presented. Pliner (1978) also found that obese people appeared to be more responsive to external factors and ate more when presented with palatable food served in an appealing

*NOTE: The definitions of obesity used in these studies were predicated on reference to published height-weight tables; the term "statistical overweight" refers to these actuarial tables. These tables are open to criticism on a number of points and must be viewed with these limitations in mind (see next section).

manner whereas lean people tended to respond more to their internal, physiological, states.

Stunkard (1959) corroborated this theory when he found that obese subjects seemed to be unaware of their internal signals. He placed tubes with pressure balloons in the stomachs of obese subjects and, even though frequent and large gastric contractions were recorded reflecting "hunger pangs" the subjects did not express hunger, and denied it when questioned. The results of the study by Monello, et al (1967) found that, while gastric contractions were a frequent symptom in hunger, it was not universal; Stunkard's subjects may not have been hungry, rather than denying it.

The responsiveness of the obese to external events was also found by Rodin (1973 & 1976). However, she cautioned that the Schachterian hypothesis is too simplistic in many respects. The factors leading to weight gain may be quite different than those which determine the level of body weight one finally reaches and maintains, the set-point (Rodin, 1980). Not all overweight people are overly responsive to external cues and lean people, as well, are often tempted by aromas, sights, and thoughts of food (Rolls, 1981).

Restrained Eating

Schachter's hypothesis that the obese tended to eat on the basis of external cues and ignore their internal, physiological, signals was tested by Herman and Polivy (1975). Under the pretense of a test of tactile stimulation on taste, subjects were seated near an electrical apparatus, presented with three flavors of ice cream,

told to taste as much as they wanted, and were left alone for ten minutes. Anxiety was created in half of the 42 subjects with the suggestion that the tactile stimulation would be an electric shock. A restraint scale, developed by the authors, assessing the individual's concern for dieting and keeping their weight down, was correlated with the amount eaten. They found that restrained eaters ate more than unrestrained eaters, whether obese or lean. Unrestrained normal-weight individuals ate significantly less when anxious than restrained eaters.

Set-Point

Nisbett (1972) posed the theory that the difference in eating behavior between the obese and the lean had more to do with the degree of underweight than the degree of overweight. He hypothesized that obese people might be statistically and socially overweight but biologically underweight; their increased number of adipocytes generate hunger signals, while social and cultural pressures tend to inhibit weight gain. Thus, these obese individuals exhibit the behaviors of people who are food-deprived (Keys, et al, 1950). Normal-weight individuals who are constantly dieting to maintain their weight resemble the obese who limit their intake. They would gain weight if they would "let themselves go". Once this self-control is breached, the restraint is removed, and eating continues.

According to the above theories, anxiety would decrease eating

in normal-weight individuals by inhibiting gastric contractions and releasing glucose into the circulation; obese individuals who are relatively insensitive to their internal signals would be unaffected. Restrained-eaters, when presented with tempting food, respond to anxiety with increased intake.

Depression

Depression also affects ingestive behaviors. Weight loss has been recognized as a feature of depression since 1904 (Weissenburger, Rush, Giles, & Stunkard, 1986), while weight gain has been recognized more recently. The effect of depression is usually in the direction of decreased desire for and enjoyment of food (Zung, 1965). Many of the self-report inventories for assessing depression include items about weight loss (Beck, 1961, Zung, 1965).

Frost, Goolkasian, Ely, and Blanchard (1982) found that depression was usually accompanied by a reduction of appetite. They experimentally induced various mood states in 68 student volunteers and counted the number of M & M's eaten during the sessions. Prior to the procedures, subjects were given the restrained-eating questionnaire; their responses to the three different moods (depressed, neutral, or elated) were classified according to their level of restraint.

High-restraint subjects who were induced into a depressed mood ate more than high-restraint subjects induced into neutral or elated moods, or low-restraint subjects in a depressed mood. The authors

stated that these results provided strong support for the restrained-eating theory; that depression interferes with self-control of the high-restraint person and once the dietary restraint was removed, the subject's eating behavior was uninhibited. The major drawback of this study was that the moods were artificially induced; the investigators read a series of statements which were either "depressing, neutral, or cheerful". This presupposes that all people would respond in a like manner to the same statement. Also, using only one food item as the dependent variable might have skewed the results if subjects did not find the item palatable.

Polivy and Herman (1976) suggested that certain people may gain rather than lose weight when depressed. They administered a questionnaire regarding symptoms of depression, especially weight changes, and their restraint questionnaire to 12 moderately depressed outpatients. As discussed above, high-restraint persons are thought to be characterized by an over concern with dieting and food related issues, and by substantial weight fluctuations (Herman and Mack, 1975, Herman and Polivy, 1975). Polivy and Herman (1976) found that depressed, low-restraint subjects experienced typical weight loss during the course of their depression, while high-restraint subjects recalled weight gain during depression. Using the same research strategy and a larger sample, Zielinski (1978) replicated these results.

Paykel (1977) examined the relationship of appetite and depression in 208 psychiatric patients. Appetite was rated on a

scale of 0 to 12; each gradation was labelled, with 12 being "severe, little food eaten" to 0 being "severe increase in intake and preoccupation with food". Level of depression was assessed with an interview by the author and rated on a scale from 1 to 7. Sixty-six percent of the 208 subjects reported a decrease in appetite, 20% reported no change, while only 14% reported an increase. He concluded that the severity of depression increased as appetite decreased.

Some linkages between the results of physiological and behavioral studies have been made. Stunkard (1975) and Booth (1976, 1977, & 1981) argued that satiety is a conditioned response. This explanation attempts to reconcile the inadequate mechanisms proposed in purely physiological studies. The role of food intake is to assure adequate energy for metabolic functioning but a distended stomach cannot sense calories and satiation occurs before adequate digestion and absorption into the blood could signal metabolic events. Therefore, there must be some conditioning that has taken place in response to eating similar foods on other occasions. This conditioned response theory does not address the issue of initiation of eating, i.e., what signals the hunger or appetite.

Fonberg (1981) compared the results of hypothalamic damage with those from studies of amygdalar lesions in feeding and emotional states of various species. She found that the more social the animal (e.g. dogs and cats as compared with rats), the more effective amygdalar lesions were in producing behavioral and food

intake changes. The role of the amygdala in contributing to this conditioning, which varies from species to species as well as from person to person, was suggested (Fonberg 1981).

Thus, although the presence of physiological determinants and signals of hunger has been acknowledged, there are numerous ways in which behavioral events contribute to, or influence, the regulation of food intake. Despite the overwhelming evidence offered in support of specific mechanisms or an interplay of mechanisms, the overriding role of conscious control of food ingestion cannot be denied. As an example, hunger strikers have consciously refused to eat regardless of internal physiological and emotional states as well as external pressures, regardless of their degree of hunger or appetite. Many patients with anorexia nervosa experience an intense desire for food but suppress their expression of hunger (Bruch, 1978). When questioned, these individuals still feel and experience hunger sensations but refuse to eat. Thus, it is possible to elicit feelings of hunger and appetite, even though the person would refuse to eat when presented with food, or is unable to eat, such as a patient on TPN who is not allowed oral intake.

Total Parenteral Nutrition

Total parenteral nutrition, the provision of nutrients intravenously, is indicated in situations where there is an inability to process nutrients via the g.i. tract. Whereas wasting and eventual

death were the inevitable results of a non-viable digestive system, the advent of TPN has made it possible to replete and maintain the nutritional status of patients wholly by the parenteral route. In this section, the background and development of TPN and its metabolic alterations are reviewed.

Background/Development

The need for various nutrients and the importance of adequate nutrition as a crucial aspect of treatment, preoperative and postoperative care, and convalescence were well accepted (Coleman and Dubois, 1915; Cuthbertson, 1930; Studley, 1950), but the method of providing nutrition to those patients unable to tolerate adequate nutrition via the gastro-intestinal tract remained elusive.

By the 1950's, intravenous (IV) therapy with sterile dextrose and normal saline solutions was standard treatment. But, the amount of calories provided by this method was limited, due to the large volume needed with a 5 percent (%) dextrose solution. The increased tonicity of more concentrated solutions often led to thrombosis of peripheral veins.

Elman demonstrated the ability to administer protein IV to humans as the hydrolysate of casein by acid (1937) and by enzymatic hydrolysis (1939). In 1955, Rose, Wixom, Lockhart and Lambert published the final summary of their 12 year study to determine the amino acid requirements of humans. Thus, the technology and knowledge to provide amino acids IV to humans was established.

The earliest reported attempt to infuse fat parenterally was in

1679 in dogs (Geyer, 1960). Fat emulsions were marketed for use in humans in the 1950's but removed by the United States Food and Drug Administration in the mid 1960's due to a high incidence of adverse effects such as fever, nausea and vomiting, and liver damage (Wretlind, 1981). A fat emulsion suitable for IV use in humans was developed in response to the realization that fat yields nine calories per gram and would furnish a more concentrated caloric source. Contributing little osmolality, the emulsion could be used peripherally without the thrombosis seen with more concentrated solutions and would furnish essential fatty acids (Wretlind, 1981).

Even with the components of carbohydrate, protein and fat available, there was a problem with the large amount of solution needed to infuse the required calories at a tonicity that would not cause thrombosis of the peripheral veins. To circumvent this, two approaches were taken.

In the first approach, diuretics were given along with the peripheral parenteral solution, making it possible to infuse up to five or seven liters of less concentrated solution per day (Rhoads, 1962). This use of diuretics produced vast electrolyte losses and required almost constant monitoring and has been superceded by the delivery of more concentated solutions into central veins (Rhoads, Dudrick, & Vars 1986). Peripheral parenteral nutrition is limited in the number of calories and amount of protein delivered and is considered only a supportive therapy for short-term administration or for use in combination with other feeding methods (Walker, 1981).

The second method approached the large central vein transcutaneously. In 1949, Rhode, Parkins, Tourtellotte and Vars devised a technique for providing continuous IV administration of hypertonic nutritive solutions into the superior vena cava of dogs, via the jugular vein.

Rhode, Parkins and Vars (1949) infused 50% glucose with and without protein in order to study nitrogen metabolism in dogs. The infusions lasted from four to 20 weeks with no apparent vessel pathology at the site of infusion. With this technique, Dudrick, Vars and Rhoades (1967) demonstrated growth and maturation in treated puppies as compared with their normally fed littermates. In 1968, using the subclavian approach to the superior vena cava that had been used to monitor central venous pressures, Dudrick, Wilmore, Vars and Rhoades administered total parenteral nutrition to adult humans (1968). In 1969, this group was able to demonstrate growth and maturation in a human infant as well (Dudrick, et al., 1969).

Thus, central venous hyperalimentation was developed. It has evolved into widespread use, with rapid gains in technology of delivery equipment and solutions. With the introduction of fat emulsions and commercially prepared trace element solutions for intravenous use, total parenteral nutrition, the provision of total nutritional needs via the parenteral route, has become a reality. But, because of the potential for complications, experts agree that TPN should be reserved for those patients who are unable to process adequate amounts of food or nutrients via the g.i. tract.

Metabolic Alterations

Some of the gastroenteropancreatic (GEP) hormones may still be operating in the control of hunger with TPN, although the g.i. tract is bypassed. Several studies have found that the GEP hormones are maintained in patients on TPN.

Motilin release in humans with PO and IV nutrients was studied by Christofides, Bloom, Besterman, Adrian, and Ghater (1979). Baseline plasma motilin levels were measured in 32 normal human subjects. A standard oral test meal was given to one group while other subjects received either PO fat, protein, or glucose. In addition, four subjects received isocaloric IV infusions of glucose, amino acids, or fat for 30 minutes in random order on separate days. After the test meal or infusion, plasma motilin levels again were determined. The authors found that there was a consistent response regardless of the route of administration. Glucose and amino acids decreased motilin release while fat caused an increase. With a balanced meal, only a small net change resulted.

Greenberg, Wolman, Christofides, Bloom, and Jeejeebhoy (1981) studied the effects of adequate nutritional support with "luminal starvation" on gut hormone response in two groups of patients with Crohn's disease. The subjects in this study received one gram of amino acids per kilogram of body weight with 40 nonprotein calories per kilogram of body weight divided equally between lipids and glucose. The first group (n = 9) had been on TPN and NPO for a mean of 25 days. Each patient received a test breakfast before discharge

from the hospital. Blood was drawn before, during, and after the meal for serum hormone determinations. After a period of refeeding (mean = 156 days; range = 38-395 days) gut hormone release was again studied before, during, and after the test meal. The results are discussed below. Gut hormone release was studied in eight other patients three days before, during, and three days after TPN (mean = 22 days, range = 18-25 days). The authors found that the endocrine response of the gut in humans is well-maintained during TPN and NPO. They found no significant difference in plasma levels of the gut hormones except for increased secretion of enteroglucagon (seen with TPN but not with refeeding) and decreased insulin levels with TPN. Nevertheless, plasma glucose levels were identical, perhaps indicating a peripheral insulin hypersensitivity that occurs during TPN. The authors concluded that the results of their study "would suggest that TPN may not necessarily require supplemental oral feeding to preserve the integrity of the gut" (p. 993).

Levine, Mullen, and O'Neill (1980) studied the effects of TPN on gastric acid secretion in 10 patients who had been on TPN for 3-6 weeks. Five of these patients were restudied after one month of refeeding. Subjects were given the TPN solution at full rate (130 ml per hour) and half rate (65 ml per hour), with and without pentagastrin stimulation. Controls were saline infusions with and without stimulation. Total parenteral nutrition significantly increased gastric acid secretion compared with saline. However, the acid secretion was low during TPN, approximately 30% of the maximum

output. There was no difference in the basal or stimulated acid output between the two groups. Serum gastrin levels also were similar in both groups. The solution used in this study contained 5.5% amino acids; other components were not indicated. These results are in opposition to Johnson, Schanbacher, Dudrick, and Copeland (1975) who found decreased weight of the oxyntic area in rats.

Kotler and Levine (1979), reported a case study of a patient who had been on TPN for two years. During TPN administration, they found gastric and pancreatic hyposecretion that quickly returned to normal when an oral diet was resumed.

In discussing these results, Levine, Mullen, and O'Neill (1980) stated that it was unknown whether the increased basal secretion of gastric acid seen with TPN is deleterious or not, citing the results of Oram-Smith and Rosato (1976). The latter group induced gastric lesions in three groups of rats: one on TPN; one on rat chow; and one on a hypocaloric, semi-starvation regimen. They concluded that adequate intravenous nutrition alone protects against gastric lesions as effectively as PO chow, when compared to the semistarved rats after seven days. Gastric secretory studies were not reported by this group, however, and as other investigators (Johnson, et al., 1975) have found decreased gastric secretions with TPN in rats, the decrease in lesions seen in this study may have been due to decreased gastric secretions. The results of these studies demonstrate that TPN alone seems to lead to atrophy and hypoplasia

of the gastrointestinal tract, but GEP endocrine responses apparently remain intact. Thus, it may be possible, via endocrine responses, for gastrointestinal events to influence hunger in patients maintained totally on parenteral nutrition.

Determining Energy Requirements

Parenteral nutrition usually is prescribed by using an estimate of the patient's metabolic rate with an additional factor sufficient to meet or exceed energy expenditure. The energy needs of an individual are the sum of the basal metabolism, the calories expended in physical activity, stress and disease, plus any additional calories needed for anabolism.

The ideal method for determining basal energy expenditure (BEE) is direct calorimetry where the patient is placed in an insulated chamber and heat loss is calculated by temperature change, reflecting the amount of calories utilized. The elaborate equipment for this test can not be taken to the bedside, and so it requires the patient or subject to go to the testing site, thus rendering direct calorimetry unsuitable for routine patient care.

Indirect calorimetry (using oxygen and carbon dioxide exchange and urinary nitrogen excretion) may be performed at the bedside and has been shown to measure the BEE accurately when compared with direct calorimetry. However, even though it is more simplified than direct calorimetry, indirect calorimetry is not always a simple task, especially in the day-to-day care of the patient. To obtain accurate values, special equipment for measuing gas exchange is

necessary and few institutions own this equipment.

To find a less cumbersome method of determining basal energy expenditure (BEE), Harris and Benedict (1919) performed indirect calorimetry in 236 subjects and correlated the results with combinations of several other parameters. Multiple regression and correlation statistics were performed by hand to derive the constants and formulas of the classic Harris-Benedict Equation (HBE) that are still valid today. It is "meticulous in design, execution, and description" (Daly, et al., 1985, p. 1172).

Roza and Shizgal (1984) used Harris and Benedict's original data to examine the relationship between the measured BEE, sex, age, and body cell mass (BCM). The subjects' BCM were calculated from body surface area, oxygen consumption, and carbon dioxide production values from the original studies. Using multiple correlation statistics, alternate formulas were derived to predict the subjects' BEE. Although they recalculated the subjects' BEE in three different ways, using various other parameters such as BCM, CO_2 and O_2, they found that the original HBE results were virtually identical to their values and concluded that the HBE was valid for both younger and older individuals.

The results of the HBE reflect basal energy requirements; to determine daily caloric needs, it is necessary to add to this value the amount of calories expended during physical activity, the stress of disease or injury, and additional calories as needed for anabolism. The metabolic response to injury and illness result in

increases in energy expenditure and nitrogen excretion, as protein is metabolized.

Rutten, Blackburn, Flatt, Hallowell, and Cochran (1975) conducted a retrospective study of 13 patients on TPN. Using the HBE, nitrogen balance studies, serum albumin, net protein utilization and other clinical nutritional parameters with regresssion analysis, they determined that it was necessary to give 1.75 times the calculated BEE to promote anabolism with TPN.

Long, Schaffel, Geiger, Schiller, and Blakemore (1979) studied 20 normal volunteers and 39 patients. Their metabolic determinations included nitrogen balances, indirect calorimetry during various levels of activity, and derived factors to adjust the BEE for for these increased requirements. Use of the BEE equation multiplied by the additional factors as determined by Long, et al., (1979) appropriate for the patient's level of activity and stress provides an estimate of the daily required caloric intake. This method has had widespread acceptance and use (Barot, Rombeau, Feurer, & Mullen, 1982; Rainey-MacDonald, Holliday, & Well, 1982).

The use of the BEE equation plus additional calories as needed for the patient's level of activity and stress provides an easy-to-use method of estimating caloric requirements.

ASSUMPTIONS

Assumptions of this study were;

1. The subjects will answer the questionnaires honestly, to the best of their ability.

2. The amount of solution as charted by the nurses is the amount of solution actually received by the patient.

RESEARCH HYPOTHESES

Specific hypotheses addressed in this study were: In hospitalized patients being fed exclusively by TPN

1. There will be no relationship between the magnitude of hunger and and the magnitude of appetite.

2. There will be a negative correlation between the magnitude of the expression of hunger and the adequacy of caloric intake.

3. There will be no correlation between the magnitude of the expression of appetite and the adequacy of caloric intake.

4. There will be no relationship between the degree of depression and the magnitude of the expression of hunger.

5. There will be a negative correlation between the degree of depression and the magnitude of the expression of appetite.

DEFINITION OF TERMS

Hunger

Hunger is a subjective reflection of a physiological need for

nutrient intake which is evoked by various individualized sensations, moods, and feelings. Hunger is controlled not just by one mechanism, but by the sum of the internal events which impel an individual to seek out and ingest food. The degree of hunger was quantified by measuring the distance between the left hand edge of the hunger visual analog scale (VAS) to the patient's mark, expressed in millimeters.

Appetite

Appetite is a subjective reflection of a psychological desire, urge or craving for a specific food or nutrient. It is based on prior learning, with social and cultural conditioning, with preferences of taste and smell or remembered feelings and moods that accompanied previous ingestion. The degree of appetite was quantified by measuring the distance between the left hand edge of the appetite VAS to the patient's mark, expressed in millimeters.

Depression

Depression is a broad spectrum of feelings ranging from disappointment, sadness, and pessimism, to a disabling loss of interest or pleasure in life. Alterations in eating habits are commonly associated with alterations in moods such as depression (Fonberg, 1981), and center around the desire for food or changes in the expression of hunger and appetite. The alterations are highly individual. Some people react with increased intake, while others find it "impossible to eat anything". Depression is usually

associated with a decreased enjoyment and lack of pleasure with eating (Paykel, 1977). The degree of depression was quantified by scoring the subject's responses to the Beck Depression Inventory. The greater the score, the greater the degree of depression.

Total Parenteral Nutrition

Total parenteral nutrition is the delivery of all of the patient's nutritional intake by the parenteral route, via a central vein.

Energy Requirements

Energy requirements are based on energy losses and are expressed in kilocalories [(kcal) but more commonly called "calories"] per unit of time, usually per 24 hours (Daly, et al., 1985). The two main components of energy loss are the basal energy expenditure and physical activity. Actual basal energy expenditure may be measured by direct calorimetry where actual heat production is monitored in a whole-body chamber or by indirect calorimetry where a person's expired air is anlyzed to measure oxygen consumption and carbon dioxide production. Factors that contribute to a person's basal energy expenditure are body weight, height, age, and gender. Energy requirements for subjects in this study were caluculated with the Harris-Benedict Equation with the adjustments for activity and injury, as determined by Long, et al. (1979).

The activity factor refers to whether the patient was on bedrest or ambulatory. The injury factor is determined by the patient's

diagnosis and surgical procedure.

Caloric Intake

Protein, fats, and carbohydrates are interchangeable as a source of needed calories, and the contribution of each was included in the calculation of calories infused. Protein yields 4 kcal per gram of amino acids, fat metabolism yields 1.1 kcal per ml of 10% lipid emulsion, and carbohydrates yield 3.6 kcal per gram of dextrose in an aqueous solution. The total caloric intake received by the patient was calculated by summing the caloric contribution of each of the major fuel sources.

Adequacy of Caloric Intake

The subject's caloric intake for the 24-hours before data collection was compared to requirements as determined by the HBE, adjusted for activity and degree of illness (Long, et al. (1979). The activity factor refers to whether the patient was on bedrest or ambulatory. The injury factor was determined by the patient's diagnosis and surgical procedure. The factor for operation was used if the patient had undergone a surgical procedure, while the trauma factor was used with patients admitted with skeletal trauma following motor vehicle accidents, with surgical procedures in some cases. Gunshot injuries were classified as trauma. Patients with sepsis were excluded and none of the subjects had burn injuries.

CHAPTER THREE

METHODS

In this study, the expressions of hunger and appetite were examined in hospitalized patients receiving TPN as their sole source of nourishment. Subjects were asked to indicate their degree of hunger (defined as a physical sensation) and appetite (defined as oral cravings or a mental urge to eat) on separate visual analog scales (VAS). The Beck Depression Inventory was used to examine the effects of depression on the expression of hunger and appetite.

Research Setting

Three hospitals of the University of California, San Francisco (UCSF), and two hospitals of the Kaiser Permanente Medical Care System, in Oakland and Walnut Creek, were the settings for this study. The UCSF hospitals include the Veteran's Administration (VA) Hospital in San Francisco, 368 beds; San Francisco General Hopsital, 580 beds; and Long Hospital, on the UCSF campus, 450 beds. San Francisco General Hospital is the major trauma center for the San Francisco Bay Area and serves the City and County of San Francisco; the VA hospital serves veterans of the military services. The UCSF hospitals are major teaching and research centers and serve as referral centers for Northern California.

Kaiser Hospitals are part of a major health maintenance organization in the Bay Area. The Oakland facility is a 350 bed teaching hospital employing medical interns and residents. The

Walnut Creek facility has 150 beds and utilizes only Kaiser employees.

Sample

Subjects were non-randomly selected from the hospitalized patients who met the criteria for this study. Adult patients with non-neoplastic disease who were receiving TPN, for at least three days, were the subjects in this study. Criteria for inclusion in this study were: (a) hospitalized adults receiving TPN; (b) ability to read, understand, and speak English; and (c) NPO for nutritive substances.

Excluded were subjects with: (a) metastatic cancer; (b) alcoholics with hepatitis; (c) alcoholics with cirrhosis; and (d) body temperature above 99 degrees Farenheit in the 24 hour period preceding data collection, in order to control for the effect of fever and its concomitant anorexia and for the increased caloric requirements produced by a fever (Murray, Murray, & Murray, 1978). Patients with metastatic cancer were excluded because of the known concomitant anorexia with this disease (DeWys, 1977). Alcoholics with hepatitis or cirrhosis were excluded due to the effect of these diseases on nutritional processes.

Protection of Human Rights

Approval from the Committee on Human Research at each institution was obtained. Subjects signed the appropriate consent

form and the rights of these subjects were protected during the study. Anonymity and confidentiality were maintained with the use of code numbers.

Instruments

Hunger and Appetite

Visual Analogue

The visual analogue scale (VAS) is a reliable, valid, and sensitive tool to assess the degree of hunger and appetite (Rosen, Hunt, Sims, & Bogardus, 1982; Silverstone, 1975 & 1980; Stacher, Bauer & Steinringer, 1979). The visual analogue scale is a self-reporting device using a straight line 100 mm long, for ease of measurement, with polar statements of the extremes of the concept being measured at either end of the line (Appendix A). The subjects were asked to place a mark at the point which corresponded to their feelings at the time they were filling out the questionnaires.

Reliability. The VAS to assess hunger has been shown to have both test-retest and between-subject reliability. Robinson, McHugh and Folstein (1975) used the VAS to measure appetite in 18 normal volunteers and 10 patients in a psychiatric hospital at one-hour intervals during an unspecified fasting period. Raw scores were not given, but the correlation coefficients for test-retest ratings was .92 ($p < .001$) for both groups.

Silverstone and Fincham (1978) found the VAS to have between-subject reliability. They used the VAS to measure hunger changes in

three separate groups of fasting normal subjects after administration of three different anorectic compounds. One study compared the changes in hunger ratings over an eight-hour period in two separate groups of subjects (n = 5 and 6), given the same anorectic agent, with changes in hunger during a similar period following administration of a placebo. Another study compared the changes in hunger ratings of eight subjects after receiving two different anorectic agents and a placebo on three separate occassions. Their findings, presented in a line graph, demonstrated a consistent pattern of changes in hunger ratings with the anorectic agents and no changes in hunger ratings with the placebo. No correlations were given.

Validity. To assess validity of a measurement there must be some objective standard to which to compare the results; as there is no objective measurement of hunger and appetite the next best correlation is to the amount of food ingested by a subject after VAS ratings. Robinson, et al. (1975) and Silverstone and Fincham (1978) found a consistent relationship between the intensity of ratings and the amount of food subsequently ingested by the subject. Robinson, et al. (1975) utilized a liquid diet while Silverstone and Fincham (1978) used a vending-type machine to record the amount of solid food each subject removed from the dispenser.

The VAS is simple for the subject to mark, requires little time, and is less likely to induce extinction than more lengthy or complicated questionnaires. Rosen, et al. (1982) utilized 16 visual

analogue scales twice a day for 50 days without the subjects tiring of the procedure and obtained consistent and valid results.

Depression

Beck Depression Inventory

This tool was used to quantify the degree of depression experienced by the subjects (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The scale consists of 21 multiple choice items evaluating symptoms characteristic of depression and its results register varying degrees of depression along a continuum.

The Beck Depression Inventory (BDI) is a self-report questionnaire with 21 multiple choice items evaluating various symptoms characteristic of depression. The symptoms are not labelled on the Inventory given to the subject, each item is identified only by a letter, A - U, and the scoring weights are not evident to the subject. (See Appendix B for the BDI given to the subjects and Appendix C for the BDI with symptom labels and scoring weights). Under each item is a set of statements, graduating in severity, and scored on a scale of 0 to 3, note that some responses are given equal weight. The subjects chose the statement in each set that most closely described current feelings. The total score depended both on the number of symptoms acknowledged and on the severity of each.

Scales which measure depression were devised to rate the severity of psychiatric depression; the subject was assumed to be otherwise healthy. The data may be skewed when subjects respond to

items relating to deterioration in their physical condition when these changes result from injury or disease rather than the subject's mental outlook.

To circumvent this problem, the subject's responses to the BDI were divided into physical and non-physical (mental) subscales for further examination. This subscaling was validated by Plumb and Holland (1977) who studied depressive symptoms in 97 patients hospitalized with advanced cancer. The responses of these patients were compared to those of two groups of control subjects. One group consisted of 66 of the patients' next-of-kin and the other group contained 99 psychiatric patients, without significant physical illness, who had been hospitalized for a suicide attempt. The investigators found that patients with cancer scored in the same low range ($\underline{M}$ = 3.7) as their relatives, on the scale items rating mental symptoms (Items A-M, Appendix C). All of the scores on these items were high in the attempted-suicide group ($\underline{M}$ = 10.6). The depressive symptoms on which the cancer patients scored as high as the suicide attempt patients were the physical symptoms of depression, which are also physical symptoms of advanced cancer, i.e., changes in body image; work and sleep disturbances; fatigability; somatic preoccupation; and loss of libido, appetite, and weight (Items N-U, Appendix C).

The physical items sub-scale was further modified by deleting the response to Item R (Loss of Appetite) from the totals. This extraction prevents any artificial inflation which would occur by

including the item being tested in the total and has been utilized by several investigators (Harris, Young, & Hughes, 1984; Weissenburger, Rush, Giles, & Stunkard, 1986).

Procedure

Subjects were selected consecutively, utilizing the pre-set criteria, and their consent for the study was obtained. Attempts were made to collect data between the hours of two and four o'clock in the afternoon to control for variations in hunger and appetite with the sights and odors of food being distributed to patients on the units. If this was not possible, data were collected at times other than mealtime.

A Research Assistant (RA), who was the Clinical Nurse Specialist on the institution's Nutrition Support Service, collected data from some subjects at Long's Hospital and San Francisco General Hospital. The author collected data at the Kaiser hositals and from some of the subjects at the other hospitals.

An initial interview with each subject was conducted by either the RA or the author to elicit permission and explain the protocol, methods of data collection, and use of the instruments. After the consent form was signed, and any questions the subject may have had were answered, the instruments were left for the patient to complete; the order in which they were to be completed was not specified. Data were recorded from the patient's medical record while the patient responded to the instruments. Subjects generally

took no more than 10-20 minutes to fill out the questionnaires, although no records were kept regarding the time taken.

Data Analysis

The coefficient of correlation, Pearson product moment r, was calculated to investigate the degree of relationship between the variables in this study. Scatter graphs were used to evaluate the individual relationships.

Data Used

Hunger and Appetite

Subjects' scores on the hunger and appetite visual analogue scales were quantified by measuring the distance between the left edge of the scale and their mark, in millimeters (mm). The larger the score, the greater was the degree of hunger or appetite.

Degree of Depression

Degree of depression was quantified by scoring the Beck Depression Inventory according to the specified formula (Appendix C). The greater the score was, the greater the subject's degree of depression.

Caloric Adequacy

Caloric intake was calculated by summing the calories contributed by carbohydrate, protein, and fat in the TPN solution received by the subject in the 24 hours prior to data collection. Basal energy requirements were calculated with the HBE, and the 24

hour caloric requirements were determined multiplying the BEE first by the activity factor and then by the injury factors outlined by Long, et al. (1979).

Caloric requirements were calculated with the HBE using the subject's actual weight. The patient's height was recorded from the medical record; body weight was recorded from the nurses' notes and reflects the subjects' weight the day of data collection.

The Harris-Benedict Equation is:

females: $655.10 + 9.56W + 1.85H - 4.68A$

males: $66.47 + 13.75W + 5.00H - 6.76A$ where

W = weight (kg); H = height (cm); A = age(years)

The activity factor and the injury factor (Long, et al. 1979) are:

Activity factor:	HBE multiplied by
a) confined to bed	1.2
b) out of bed	1.3
Injury factor:	
a) minor operation	1.20
b) skeletal trauma	1.35

The adequacy of each subject's caloric intake was quantified by calculating the amount of calories infused as a percent of calories required by the individual subject as determined by the Harris-Benedict equation, adjusted for activity and injury factors.

Quetelet's Index

Quetelet's Index, the body mass index (wt/ht^2) was calculated by first converting the patient's height to meters, squaring that value, then dividing the body weight (kg) by the height (m) squared.

CHAPTER FOUR

RESULTS OF THE STUDY

The sample consisted of 28 adults, 12 females and 16 males, who were receiving total parenteral nutrition as their sole source of nutritional support. Their ages ranged from 22 to 78 years (M = 47, SD = 16). Twenty subjects were patients in a university medical center and two of its satellite hospitals. Eight subjects were obtained from two hospitals of a major health maintenance organization.

Nine of the subjects had a diagnosis of inflammatory bowel disease (IBD); six were trauma victims [stab wound, motor vehicle accident (MVA), or gun shot wound]; two had paralytic ileus due to a ruptured appendix; two had diverticulitis; and the remaining nine had various other diagnoses, including congestive heart failure, alkaline gastritis, recto-vaginal fistula, pancreatic pseudocyst, gastrointestinal bleeding, small bowel obstruction, cholangitis, refractory diarrhea, and gastro-bronchial fistula. None of the subjects had a malignant disease and all were afebrile. The length of time the subjects had been receiving TPN prior to data collection ranged from three to 62 days (M = 14, SD = 13). (Table 2).

Subject's height ranged from 150 to 193 cm (M = 170, SD = 10) and body weight ranged from 40 to 105 kg (M = 63, SD = 15). Quetelet's Index ranged from 15 to 32 (M = 21, SD = 3). (Table 3).

Table 2.

Age, Sex, Diagnosis, and Length of Time on Total Parenteral Nutrition by Subject

Subject No.	Sex[1]	Age	Diagnosis[2]	No. Days on TPN
1	F	26	Ruptured appendix, ileus	9
2	M	48	IBD	3
3	F	68	IBD	12
4	F	32	Recto-vaginal fistula	7
5	F	38	Pancreatic pseudocyst	15
6	M	78	Cholangitis	5
7	M	36	Multiple stab wounds	3
8	M	45	MVA, blunt trauma	25
9	M	28	SBO	5
10	M	37	Gun shot wound	28
11	M	27	Refractory diarrhea	13
12	M	57	Gun shot wound	26
13	M	57	G.I. bleed	23
14	F	51	MVA, blunt trauma	5
15	F	67	CHF	14
16	F	55	Ruptured appendix, ileus	5
17	F	34	IBD	10
18	F	22	IBD	6
19	F	77	Alkaline gastritis	27
20	M	66	IBD	5
21	F	42	IBD	4
22	F	36	IBD	37
23	M	60	Gastro-bronchial fistula	4
24	M	34	IBD	5
25	M	29	IBD	18
26	M	31	SBO, trauma	62
27	M	60	Diverticulitis	8
28	M	63	Perforated diverticulum	7

[1] F = Female
M = Male

[2] IBD = Inflammatory Bowel Disease; SBO = Small Bowel Obstruction; MVA = Motor Vehicle Accident; CHF = Congestive Heart Failure

Table 3.

Height, Weight, and Quetelet's Index[1], by Subject

Subject No.	Height (cm)	Weight (Kg)	Quetelet's Index
1	170	61	21
2	175	73	24
3	152	50	22
4	168	65	23
5	163	50	19
6	168	63	23
7	180	66	20
8	178	87	28
9	173	59	20
10	183	75	22
11	193	86	23
12	165	65	24
13	175	70	23
14	161	52	20
15	159	43	17
16	163	65	24
17	169	59	21
18	155	45	19
19	150	40	18
20	183	79	24
21	168	60	21
22	168	45	16
23	170	63	22
24	166	42	15
25	173	65	22
26	185	71	21
27	180	61	19
28	180	105	32

[1]Quetelet's Index = weight/height^2

Hunger and Appetite

Hunger scores ranged from 2 to 96 (*M* = 32, *SD* = 28). Appetite scores ranged from 1 to 99 (*M* = 46, *SD* = 30). (Table 4).

Table 4.

Hunger and Appetite Scores by Subject

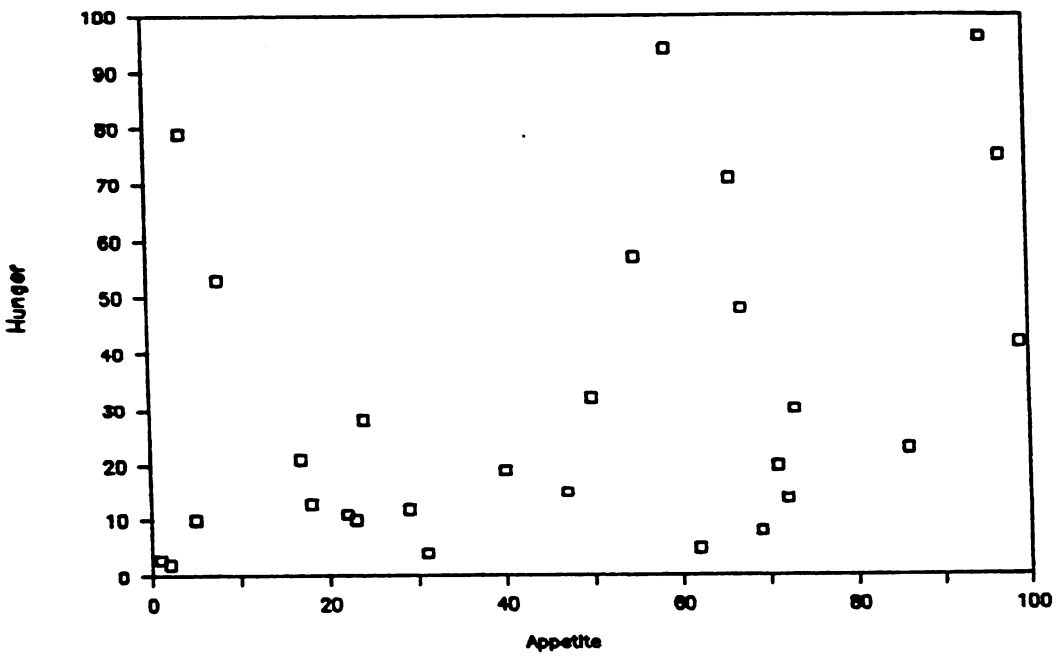
Subject No.	Hunger	Appetite
1	94	59
2	10	23
3	79	4
4	14	72
5	21	17
6	96	95
7	5	62
8	71	66
9	48	67
10	53	8
11	42	99
12	20	71
13	10	5
14	4	31
15	13	18
16	11	22
17	57	55
18	15	47
19	3	1
20	12	29
21	23	86
22	75	97
23	8	69
24	28	24
25	30	73
26	19	40
27	2	2
28	32	50

To test the null hypothesis that there is no relationship between the magnitude of hunger and the magnitude of appetite, the Pearson product moment correlation coefficient was calculated. The hypothesis that there will be no relationship between the magnitude of hunger and the magnitude of appetite was not supported ($r = .39$, $p < .05$).

To further examine the question of whether hospitalized patients can differentiate between hunger and appetite, the subject's scores on each were plotted on a scatter graph (Figure 3). Although there is a wide scatter of data points, all but one of the subjects (No. 27, Table 3) were able to differentiate between hunger and appetite and report different intensities of each. One subject (No. 3) had the third highest hunger score (79) and the third lowest appetite score (4). These data indicate that subjects were able to distinguish between hunger and appetite and indicate the intensity of their feelings.

Figure 3

HUNGER AND APPETITE



Caloric Adequacy

The number of calories infused ranged from 1209 to 5215 (*M* = 2903, *SD* = 845). Caloric requirements ranged from 1231 to 3250 (*M* = 2250, *SD* = 514). The adequacy of caloric intake ranged from 56% to 222% (*M* = 133%, *SD* = 41%). (Table 5).

Hunger

To test the null hypotheses that there is no relationship between the adequacy of caloric intake and the degree of hunger, the Pearson product moment correlation coefficient was calculated (Table 6). The correlation coefficient of -.017 was not statistically significant ($p > .05$) and the null hypothesis was not rejected. The hypothesis that there will be a negative correlation between the magnitude of the expresion of hunger and the adequacy of caloric intake was not supported by statistical analysis.

Appetite

To test the null hypotheses that there is no relationship between the adequacy of caloric intake and the degree of appetite, the Pearson product moment correlation coefficient was calculated (Table 6). The correlation coefficient of -.188 was not statistically significant ($p > .05$) and the null hypothesis was not rejected. The hypothesis that there will be no relationship between the magnitude of the expression of appetite and the adequacy of calorie intake was supported.

Table 5.

Basal Energy Expenditure (BEE[1]), Activity Factor[2], Injury Factor[2], Caloric Requirements and Caloric Adequacy Expressed as a Percent of Required, by Subject

Subject No.	BEE	Activity Factor	Injury Factor	Caloric Requirements	Calories Received	Caloric Adequacy
1	1427	1.3	1.2	2226	2136	96%
2	1622	1.3	1.2	2530	2190	87%
3	1089	1.3	1.2	1699	2586	152%
4	1428	1.3	1.2	2228	3210	144%
5	1245	1.3	1.0	1619	3570	221%
6	1249	1.3	1.2	1949	2497	128%
7	1638	1.2	1.55	3046	4110	135%
8	1852	1.3	1.35	3250	5215	160%
9	1552	1.3	1.0	2018	4110	204%
10	1762	1.3	1.2	2749	4110	150%
11	2032	1.3	1.0	2641	4110	156%
12	1403	1.3	1.35	2463	2666	108%
13	1514	1.3	1.2	2363	3036	129%
14	1202	1.3	1.0	1563	3468	222%
15	1036	1.3	1.0	1347	2502	186%
16	1309	1.3	1.2	2042	1934	95%
17	1367	1.3	1.2	2132	2681	126%
18	1261	1.3	1.2	1968	2026	103%
19	947	1.3	1.0	1231	2420	197%
20	1617	1.3	1.2	2522	2590	103%
21	1334	1.3	1.2	2081	2874	138%
22	1219	1.3	1.35	2218	2402	108%
23	1378	1.3	1.2	2150	1209	56%
24	1246	1.3	1.2	1944	1939	100%
25	1628	1.3	1.2	2539	3018	119%
26	1760	1.3	1.35	3204	3210	100%
27	1401	1.3	1.2	2185	2568	118%
28	1987	1.3	1.2	3100	2900	94%

1
BEE calculated with the Harris-Benedict Equation

2
Activity and injury factors according to Long, et al. (1979)

Table 6.

Pearson Product Moment Correlation Coefficients with Levels of Significance between Adequacy of Caloric Intake and Hunger and Appetite

Hunger	-.017	(NS)
Appetite	-.188	(NS)

NS = Non-significant ($p > .05$)
df = 26

To aid in the interpretation of the quantitative data analysis, scatter graphs were constructed for visual inspection of the data for trends and relationships (Figures 4 and 5). The weak negative relationship between appetite and adequacy of caloric intake is shown along the diagonals on the far right of Figure 5, but the cluster of data points in the lower left reveal low appetite scores with less than 100% caloric adequacy.

Figure 4

HUNGER AND ADEQUACY OF CALORIC INTAKE

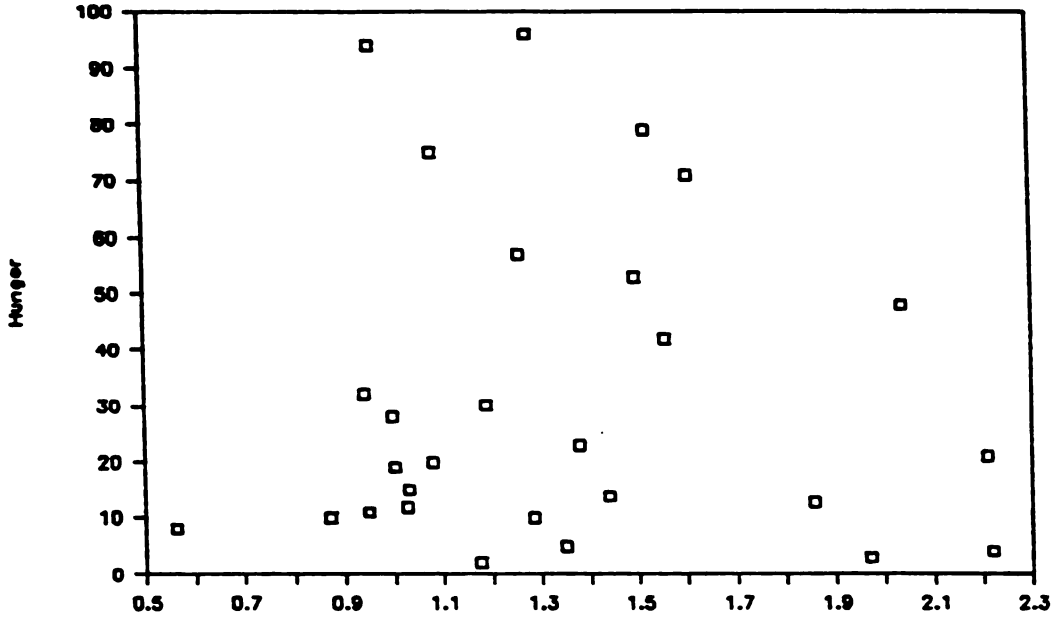
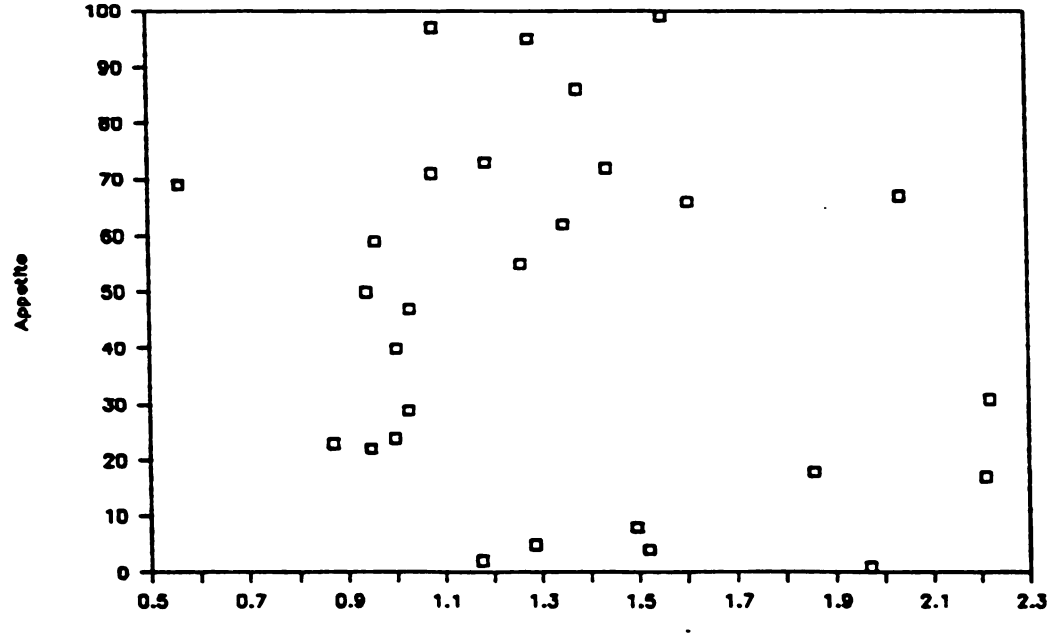


Figure 5

APPETITE AND ADEQUACY OF CALORIC INTAKE



Degree of Depression

The total scores for all items on the BDI ranged from 4 to 24 and the (M = 13, SD = 5). Mental items responses ranged from 0 - 13 (M = 4, SD = 3). The range of scores for the physical items was 4 to 15 (M = 9, SD = 3); with Item R (Loss of Appetite) deleted, the modified physical-items scores ranged from 4 to 21 (M = 12, SD = 5). (Table 7).

Table 7.

Scores on the Beck Depression Inventory Mental, Physical, and Modified-Physical Sub-scales by Subject

Subject No.	Mental (Items A-M)	Physical (Items N-U)	Modified-Physical (Items N-Q, S-U))
1	1	5	5
2	1	13	11
3	0	14	13
4	3	6	5
5	0	5	5
6	4	7	7
7	5	4	3
8	8	7	7
9	10	9	8
10	3	8	7
11	1	8	7
12	13	7	7
13	1	10	9
14	4	8	6
15	9	13	12
16	3	10	8
17	1	7	6
18	4	9	8
19	3	15	12
20	0	4	4
21	3	8	8
22	4	6	6
23	1	6	5
24	3	15	14
25	3	14	12
26	10	12	10
27	9	15	12
28	4	11	11

Hunger and Degree of Depression

The hypothesis that there will be no relationship between the degree of depression and the magnitude of the expression of hunger was supported when compared with the total scores on the BDI ($r = -.215$). To explore whether the relationship between hunger and the level of depression would vary with the physical and mental subscales, a correlation was performed for each subscale. Neither the mental items sub-scale ($r = -.109$, $p > .05$) nor the modified physical items sub-scale ($r = -.102$, $p > .05$) reached statistical significance (Table 8). The total score on the Modified BDI ($r = -.138$, $p > .05$) also failed to reach statistical significance (Table 8).

Table 8.

Pearson Product Moment Correlation Coefficients with Levels of Significance between Hunger and Beck Depression Inventory (BDI) and Subscale Scores

BDI (Total Score)	-.215	(NS)
BDI (Physical Items)	-.220	(NS)
BDI (Mental Items)	-.109	(NS)
Modified BDI (Physical Items Minus Item R)	-.102	(NS)
Modified BDI (Total Score) Minus Item R)	-.138	(NS)
BDI Item R (Loss of Appetite)	-.516	($p < .01$)

NS = Non-significant ($p > .05$)
df = 26

The lower correlation coefficient between hunger and score on the Modified BDI - Physical Items (with Item R extracted) demonstrates the influence this item had on the correlation coefficients of the total score.

A post-hoc analysis between the magnitude of hunger and Item R (Loss of Appetite), revealed a negative correlation of -.516, which was significant at the .01 level. This finding tends to substantiate the use of the VAS to measure hunger, and to extract the item from the BDI to avoid artificial inflation of the results.

The scattergraphs of the data points of the degree of hunger and scores on the Beck Depression Inventory are displayed in Figures 6 through 10.

Figure 6

HUNGER AND BECK DEPRESSION INVENTORY

Total Score

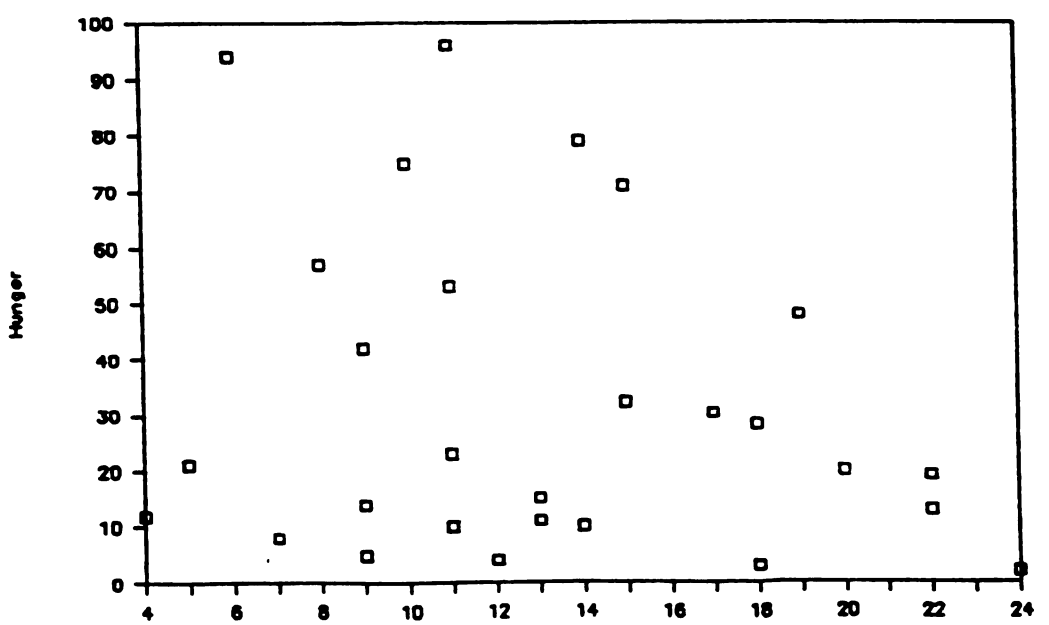


Figure 7

HUNGER AND BECK DEPRESSION INVENTORY

Physical Items Score

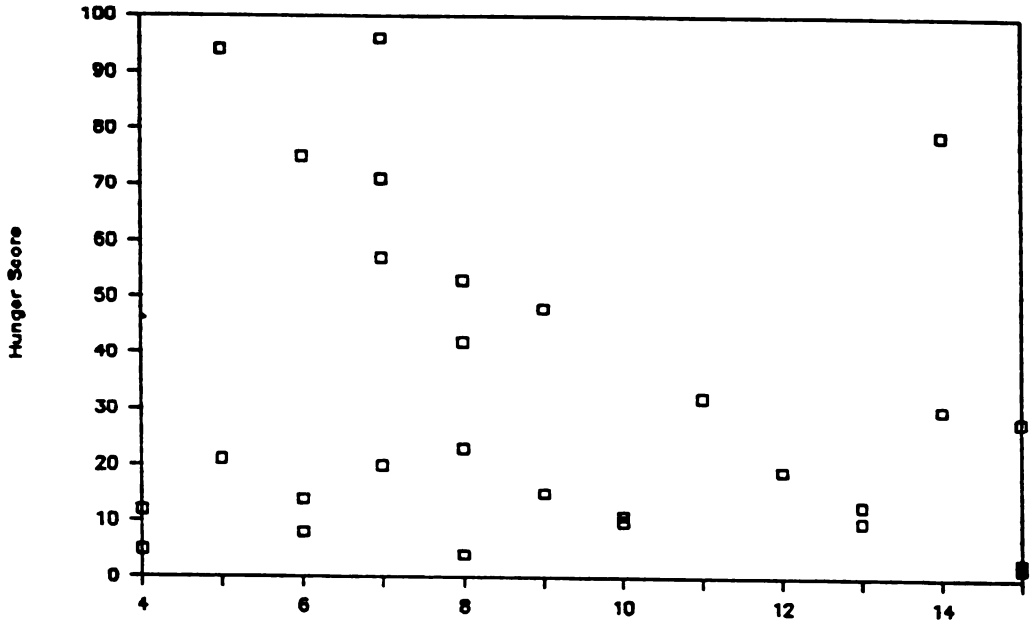


Figure 8

HUNGER AND BECK DEPRESSION INVENTORY

Mental Items Score

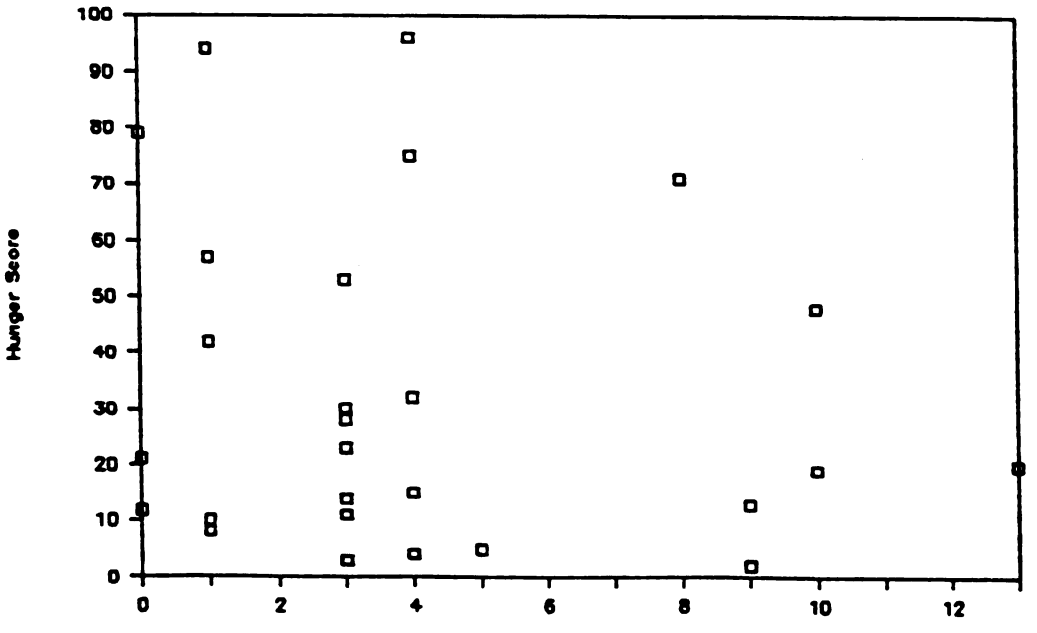


Figure 9

HUNGER AND BECK DEPRESSION INVENTORY

Modified Physical Items Score

Item R Extracted

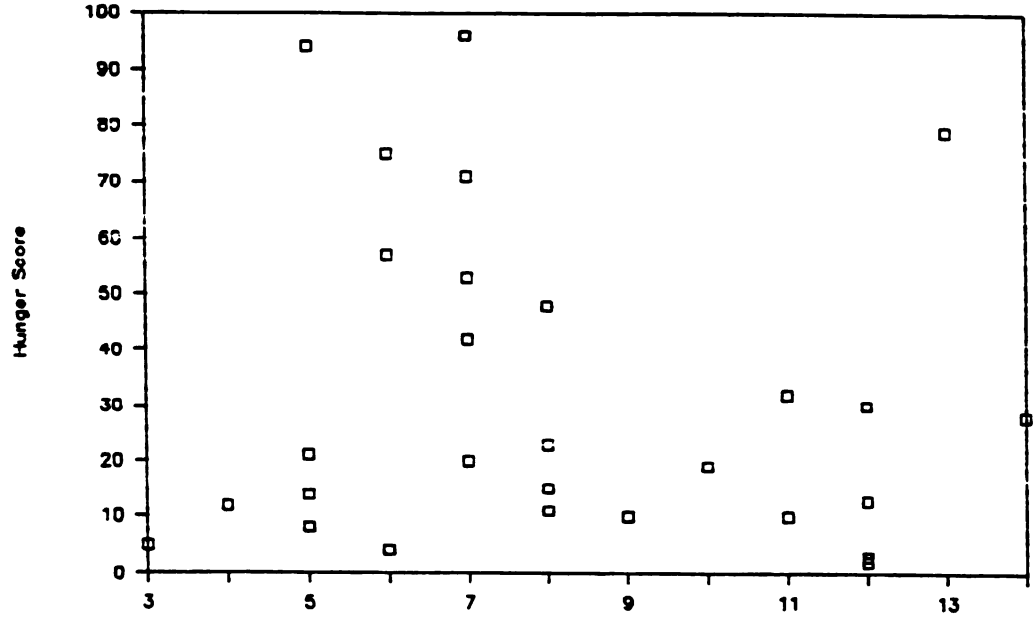
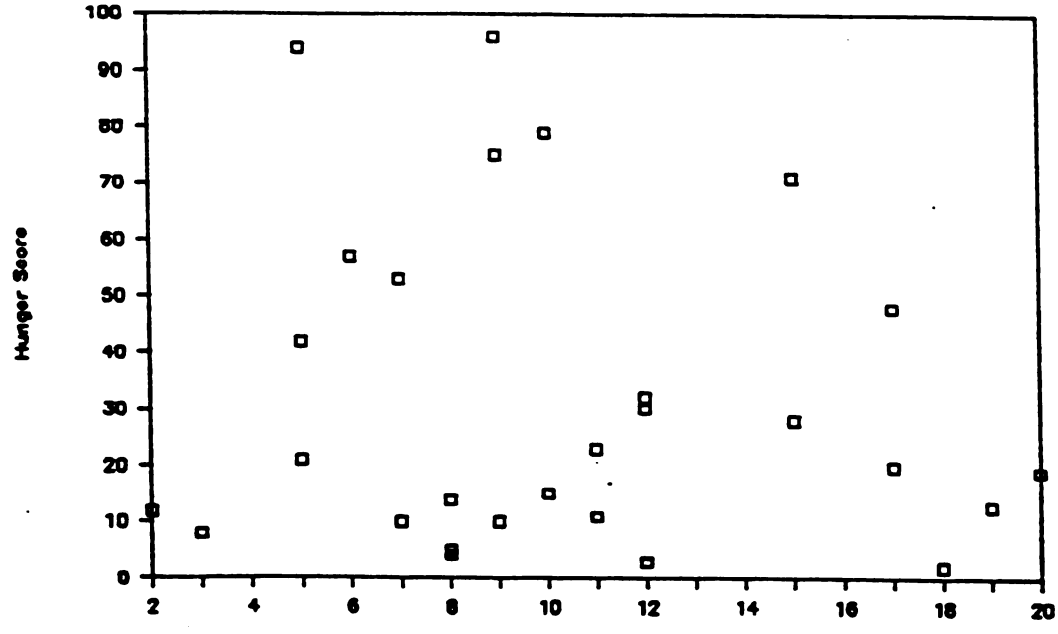


Figure 10

HUNGER AND BECK DEPRESSION INVENTORY

Modified Total Score



Appetite and Degree of Depression

The hypothesis that there will be a negative correlation between the degree of depression and the magnitude of the expression of appetite was not supported when compared to the total score on the BDI or the mental items subscale. The Pearson product moment correlation coefficient for appetite and the total score on the Beck Depression Inventory was -.259, which reflects little or no relationship (Colton, 1974) and was not significant ($p > .05$). There was no relationship between appetite and the mental items subscale ($r = -.115$; $p > .05$). The coefficient for appetite and the physical items subscale was -.509, which reflects a moderate to good relationship, and was statistically significant ($p < .05$). The Pearson product moment r of -.448 showed a fair relationship between appetite and the modified physical items subscale with Item R (Loss of Appetite) extracted, and reached statistical significance ($p < .05$). There was no relationship between appetite and the total score on the modified BDI ($r = -.189$, $p > .05$). (Table 9).

Table 9.

Pearson Product Moment Correlation Coefficients with Levels of Significance between Appetite and Beck Depression Inventory (BDI) and Subscale Scores

BDI (Total Score)	-.259	(NS)
BDI (Physical Items)	-.509	(p <.01)
BDI (Mental Items)	.115	(NS)
Modified BDI (Physical Items Minus Item R)	-.448	(p <.05)
Modified BDI (Total Score) Minus Item R)	-.189	(NS)
BDI Item R (Loss of Appetite)	-.496	(p <.01)

NS = Non-significant (p > .05)
df = 26

The scattergraphs of the appetite scores and scores on the Beck Depression Inventory are diplayed in Figures 11 through 15.

Figure 11

APPETITE AND BECK DEPRESSION INVENTORY

Total Score

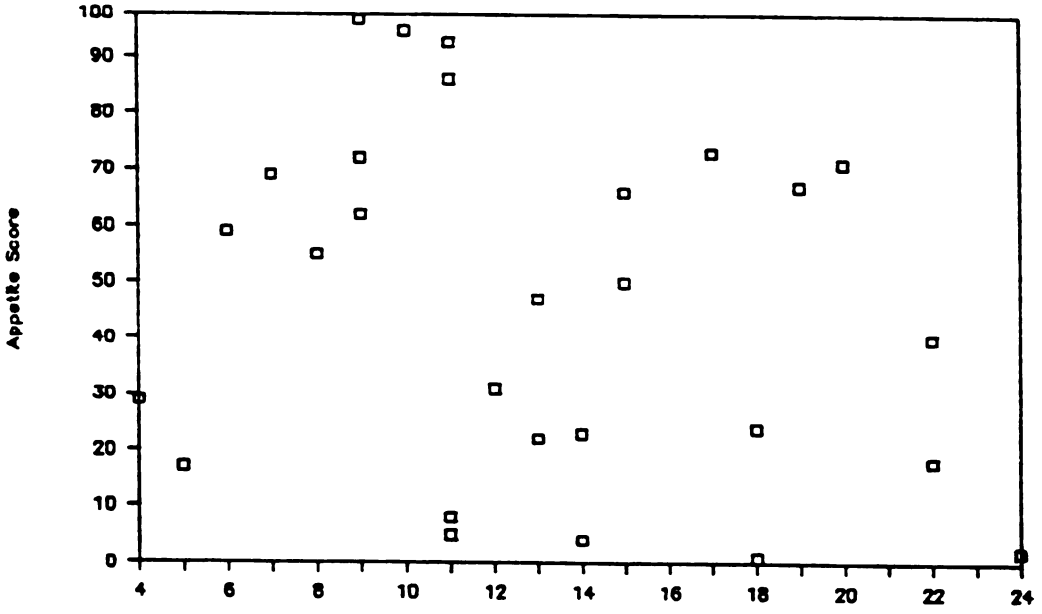


Figure 12

APPETITE AND BECK DEPRESSION INVENTORY

Physical Items Score

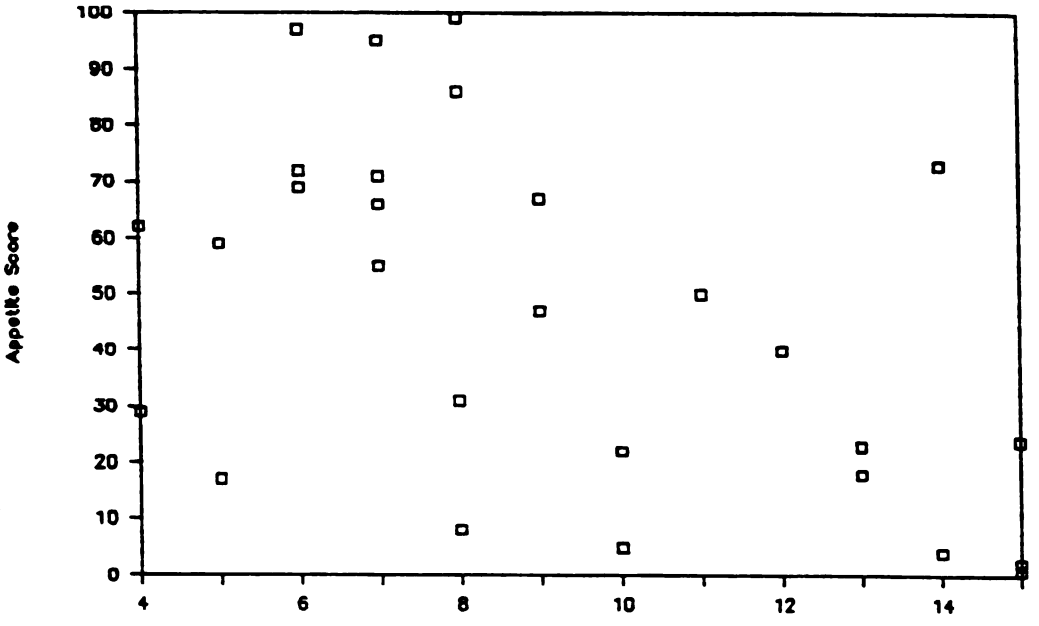


Figure 13

APPETITE AND BECK DEPRESSION INVENTORY

Mental Items Score

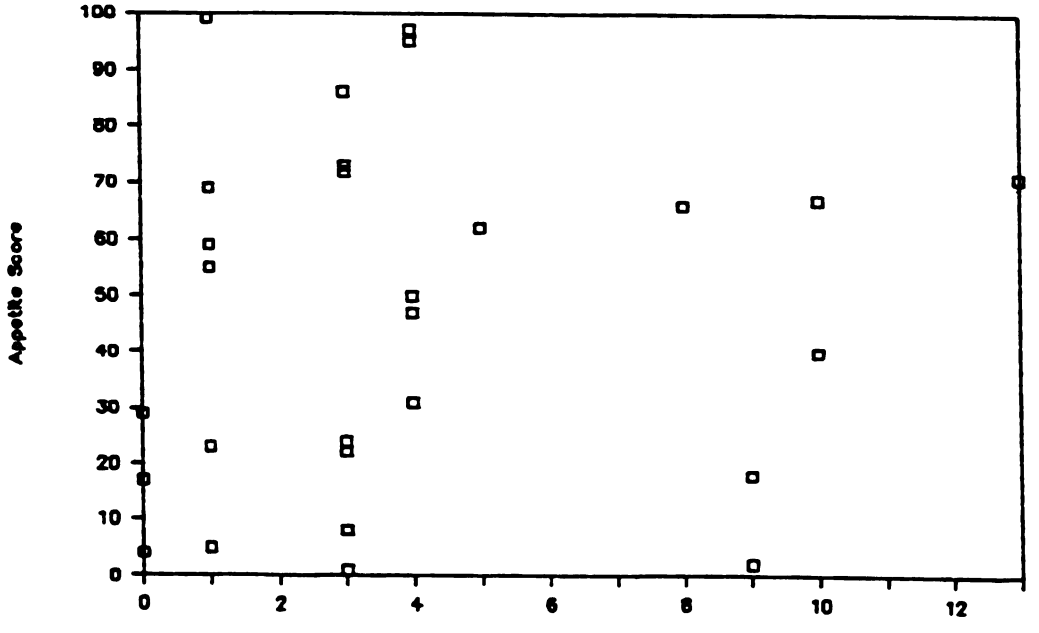


Figure 14

APPETITE AND BECK DEPRESSION INVENTORY

Modified Physical Items Score

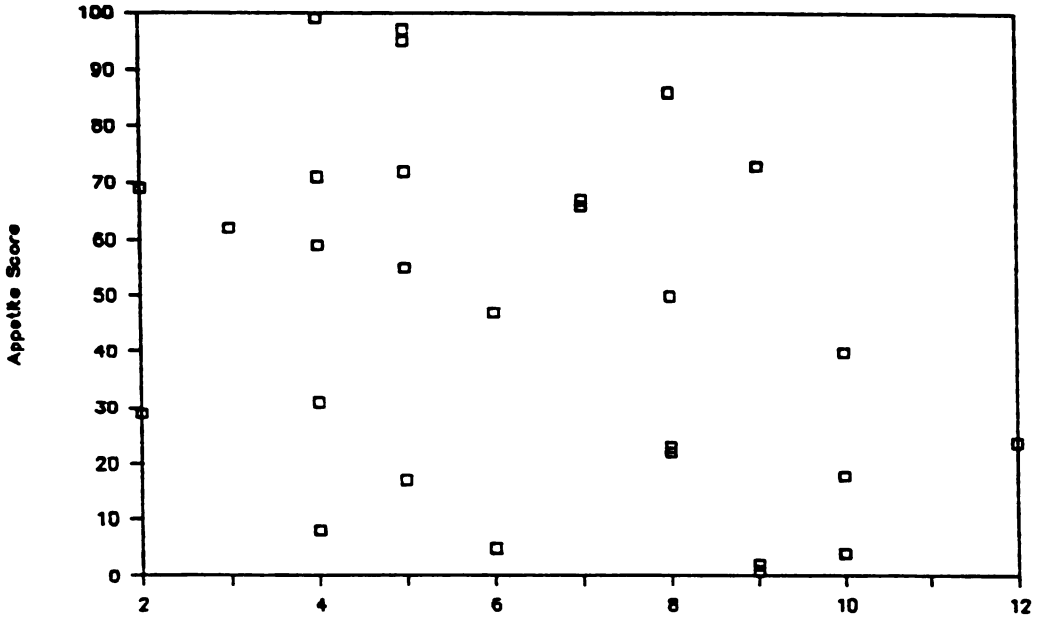
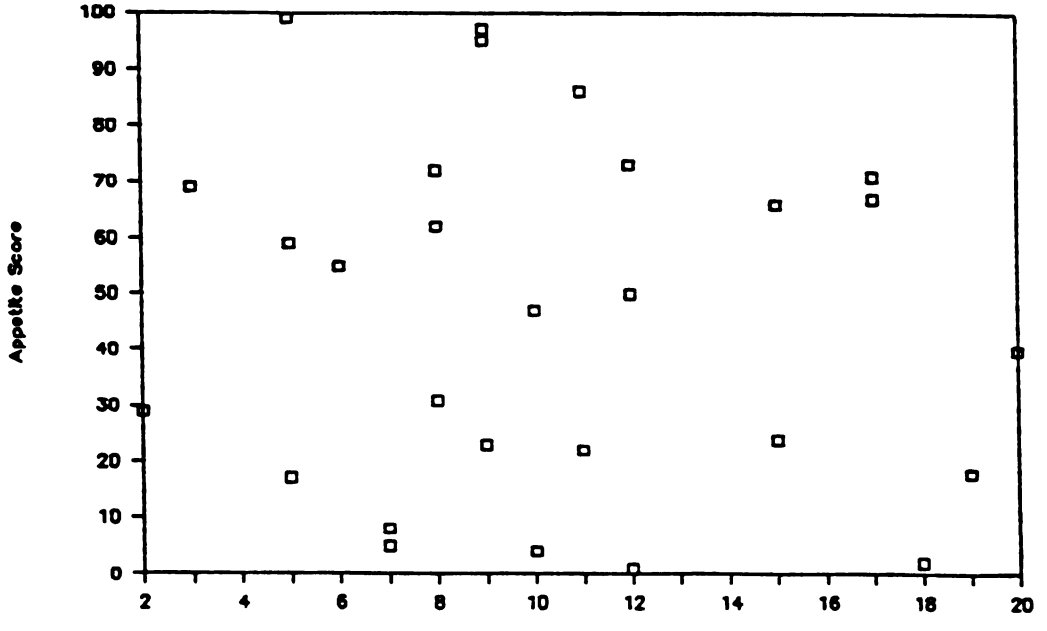


Figure 15

APPETITE AND BECK DEPRESSION INVENTORY

Modified Total Score



Post-hoc Analysis

Age.

To examine relationships between the age of the subject and the degree of hunger and appetite, scatter graphs were plotted (Figures 16 and 17). There was no relationship between hunger and age ($r = -.114$, $p > .05$). The correlation coefficient between appetite and age was -.357 which shows a mild to moderate relationship, but did not reach statistical significance ($p > .05$) (Table 10).

Length of Time on TPN.

To examine the relationships between the length of time subjects had been receiving TPN and the degree of hunger and appetite, data points were plotted on scatter graphs (Figures 18 and 19). There was no relationship between hunger and and length of time on TPN ($r = .105$, $p > .05$). The Pearson product moment correlation coefficient between appetite and number of days on TPN was .169 ($p > .05$), also showing no relationship (Table 10).

Quetelet's (Body Mass) Index.

Table 10 also presents the Pearson product moment correlation coefficients between the body mass index and hunger ($r = .100$, $p > .05$), and appetite ($r = .169$, $p > .05$). This lack of relationship is revealed by the wide scatter of the data points in Figures 20 and 21.

Table 10.

Pearson Product Moment Correlation Coefficients with Levels of Significance between Hunger and Appetite and Age, Days on TPN, and Quetelet's Index.

	Hunger		Appetite	
Age	-.114	(NS)	-.357	(NS)
Days on TPN	.105	(NS)	-.045	(NS)
Quetelet's Index	.100	(NS)	.169	(NS)

NS = Non-significant ($p > .05$)
df = 26

Figure 16

HUNGER AND AGE

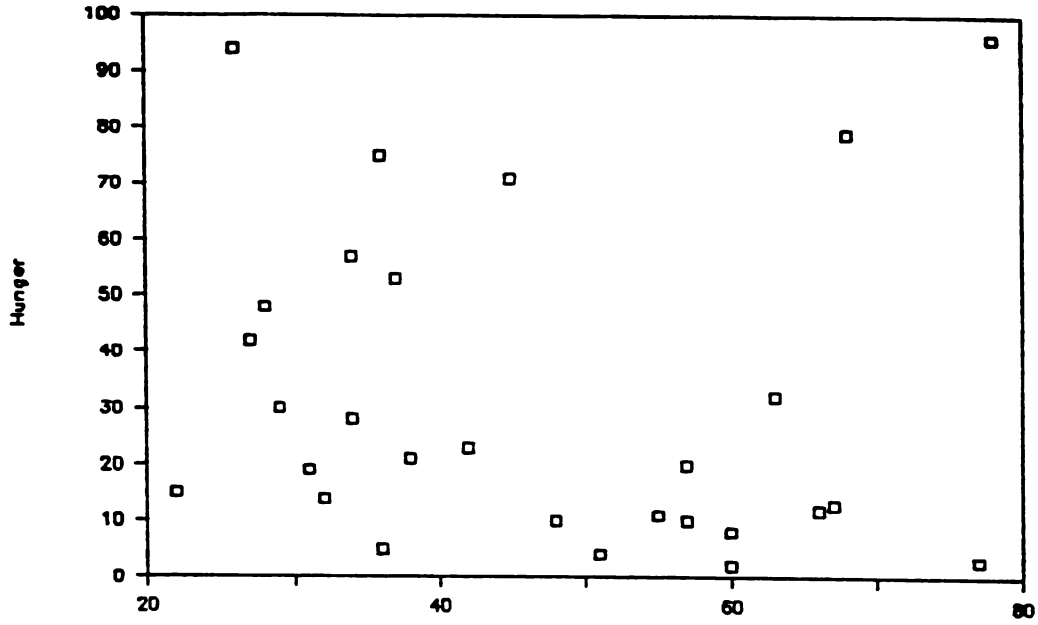


Figure 17

APPETITE AND AGE

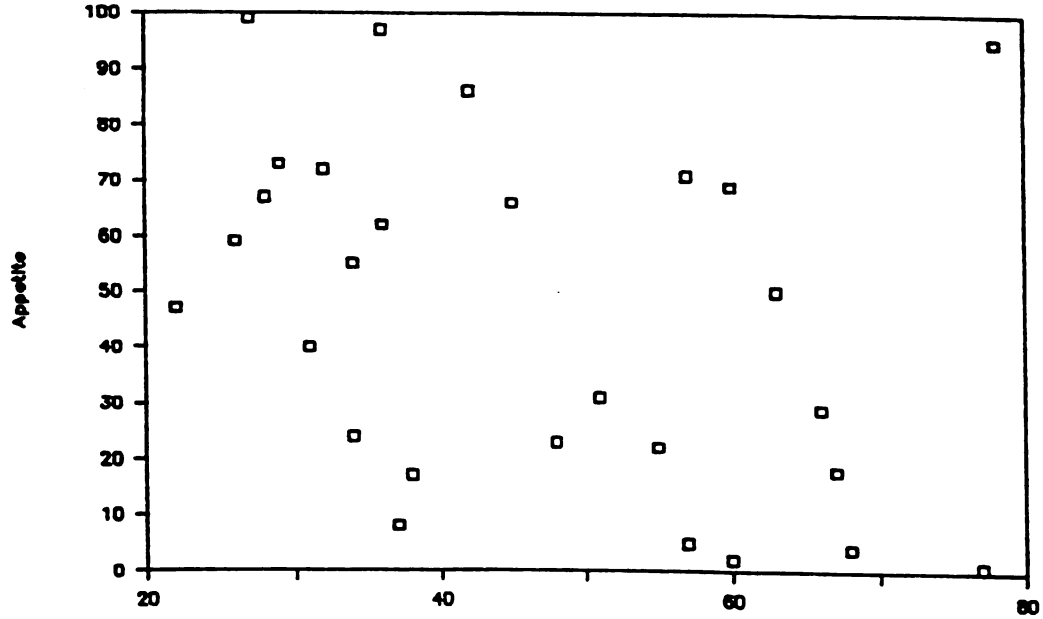


Figure 18

HUNGER AND LENGTH OF TIME ON TPN

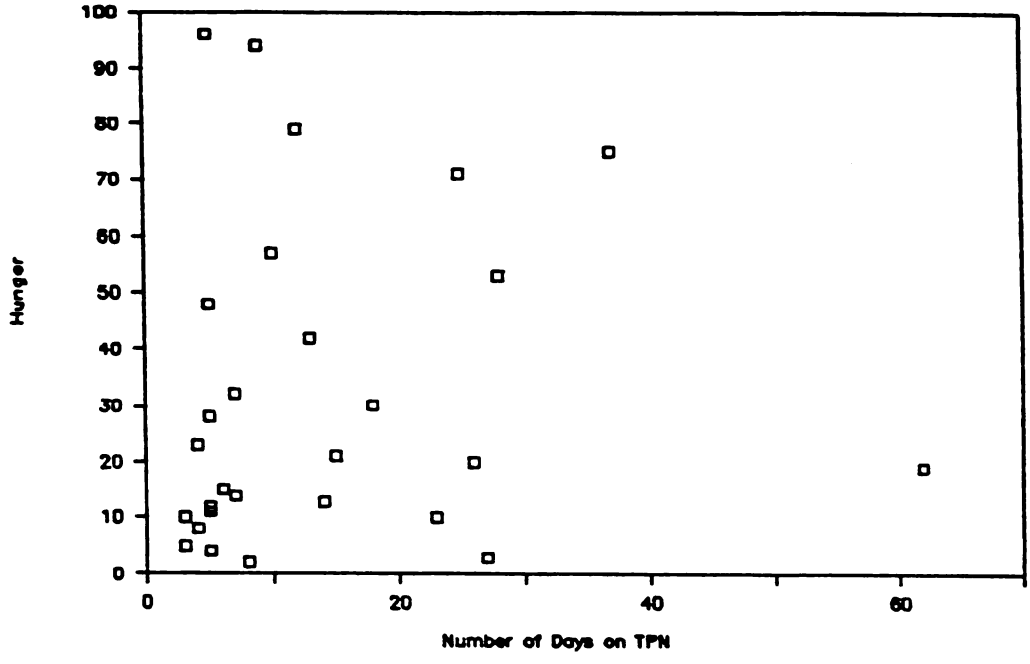


Figure 19

APPETITE AND LENGTH OF TIME ON TPN

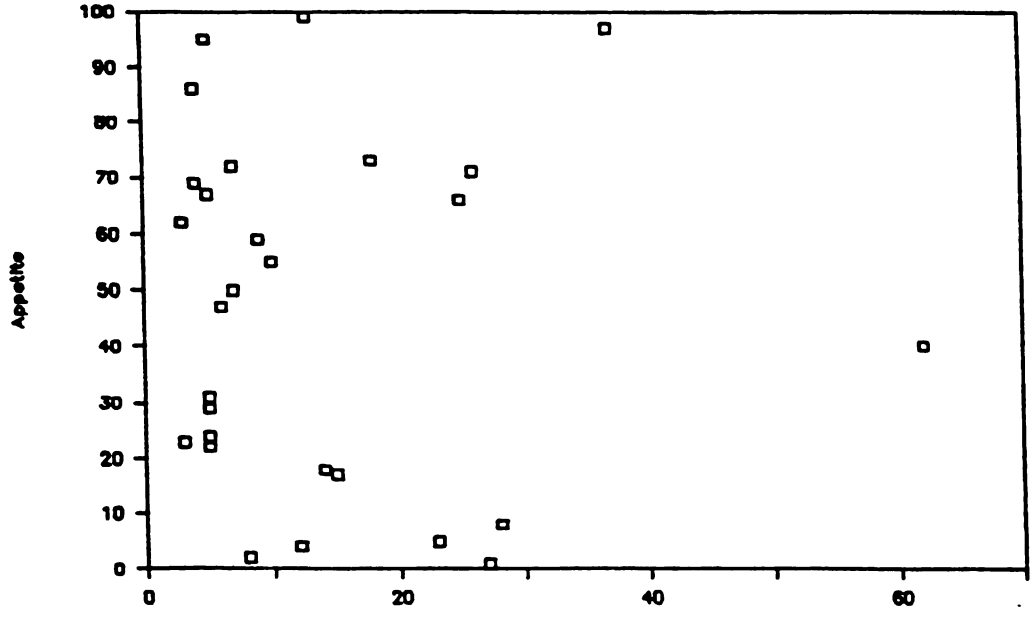


Figure 20

HUNGER AND QUETELET'S INDEX

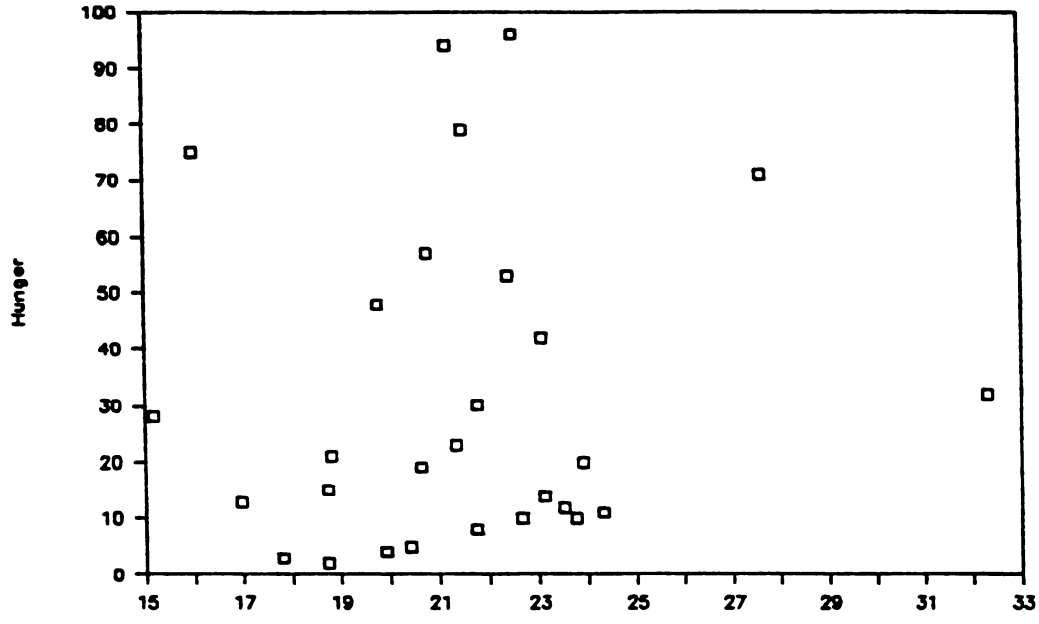
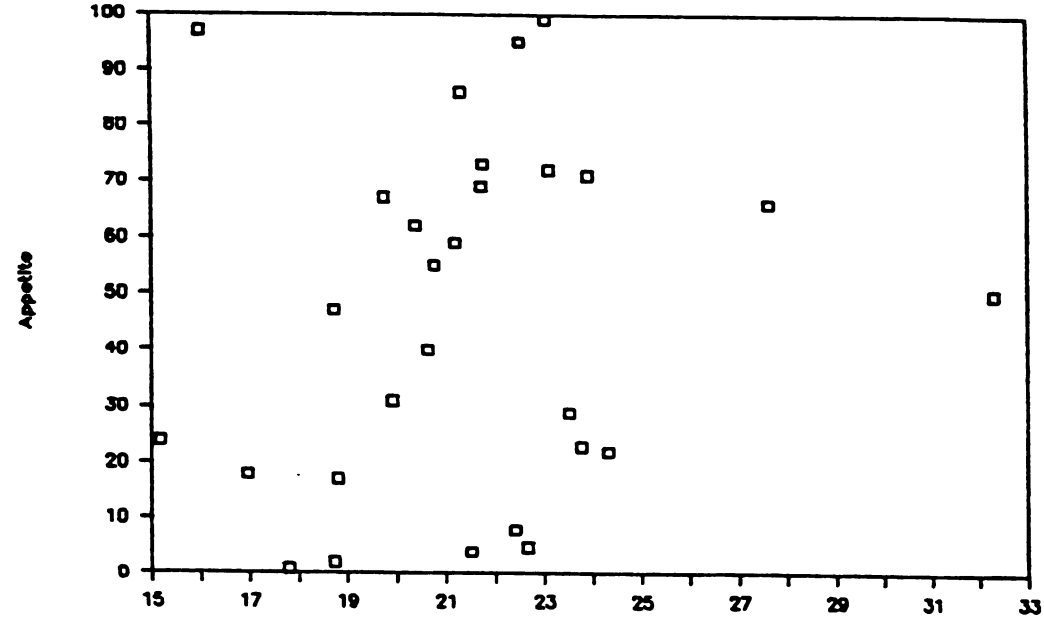


Figure 21

APPETITE AND QUETELET'S INDEX



Categorization of Hunger and Appetite Scores

To further examine the relationship between hunger and appetite the scores were categorized by scores on the visual analog scale: 0-25 representing no to very little hunger or appetite, 26-50 mild, 51-75 moderate, and 76-100 being severe hunger or appetite.

With this categorization of hunger, 57% of the subjects reported no to very little hunger, 16% had mild hunger, 15% reported moderate hunger, while 12% had severe hunger. Categorization of appetite scores showed 36% of the subjects reported no to very little appetite, 18% had mild appetite, 32% had a moderate appetite and 14% reported severe appetite. Table 10 summarizes these results.

Within each category of hunger and appetite, the mean Quetelet's Index, age, number of days the patient had been receiving TPN, and the mean of the total scores on the modified Beck Depression Inventory were calculated. The 16 subjects who reported no to very little hunger had a mean QI of 21, a mean age of 50 years, had been receiving TPN for an average of 14 days, and had a mean score of 12 on the modified BDI. The 10 subjects who reported no to very little appetite had a mean QI of 20, had an average age of 54 years, and had been receiving TPN for an average of 14 days; their mean score on the modified BDI was 14. Mean scores within the other categories of hunger and appetite are displayed in Table 12.

Table 11.

Number of Subjects in Each Category of Hunger/Appetite[1].

Severe/ None 1	Severe/ Mild 0	Severe/ Moderate 1	Severe/ Severe 1	12%
Moderate/ None 1	Moderate/ Mild 0	Moderate/ Moderate 2	Moderate/ Severe 1	15%
Mild/ None 1	Mild/ Mild 1	Mild/ Moderate 2	Mild/ Severe 1	16%
None/ None 7	None/ Mild 4	None/ Moderate 4	None/ Severe 1	57%
36%	18%	32%	14%	

Hunger

Appetite

[1] Based on scores on the Visual Analogue Scale for Hunger and Appetite: 0- 25 = none to very little
26- 50 = mild
51- 75 = moderate
76-100 = severe

Table 12.

Number of Subjects (n), Mean Quetelet's Index (QI), Mean Age (in years), Mean Number of Days on TPN, and Mean Total Score on the Modified Beck Depression Inventory (BDI) within Each Category of Hunger and Appetite.

	n	QI	Age	No. Days on TPN	Total Score Modified BDI
HUNGER					
None	16	21	50	14	12
Mild	5	22	36	10	15
Moderate	4	22	44	22	11
Severe	3	22	52	7	9
APPETITE					
None	10	20	54	14	14
Mild	5	23	47	17	12
Moderate	9	22	39	12	11
Severe	4	21	46	15	10

Based on scores on the Visual Analogue Scale for Hunger and Appetite:

0 - 25 = none to very little
26 - 50 = mild
51 - 75 = moderate
76 - 100 = severe

CHAPTER FIVE

DISCUSSION OF FINDINGS

Hunger and Appetite

The hypothesis that there is no relationship between the magnitude of hunger and the magnitude of appetite was not supported by statistical tests; a relationship was found between hunger and appetite. A possible explanation may be provided by looking again at the controversy in the literature. The correlation between hunger and appetite means that they are related, which is not surprising, because both sensations stimulate food ingestion. The theoretical basis for the hypothesis in this study was derived from data that indicate that the etiology of hunger and the etiology of appetite are separate and distinct entities, not two poles of one continuum.

Appetite has been presented as the first, gentle, urge to eat and, as deprivation continues, the feelings intensify and appetite becomes hunger (Mayer, 1976). In this view, appetite, as the earlier impetus for food ingestion, is related to the selection of specific foods guided by personal preferences. If food is not eaten in response to the feeling of appetite at this point, and as the length of time of deprivation continues, the sensation intensifies and becomes hunger. If hunger and appetite were on a continuum as depicted in Figure 1, the presence of hunger would preclude a sensation of appetite as shown in Figure 22. Depending upon the point along the continuum at which appetite becomes hunger, the

diagramatic representation may be as shown in Figure 23 with the sensations of appetite and the sensations of hunger over-lapping.

Figure 22.

Hunger and Appetite Viewed on a Continuum.

Appetite----------Hunger

Figure 23.

Appetite and Hunger Viewed as Overlapping Sensations.

Appetite----------a----------b

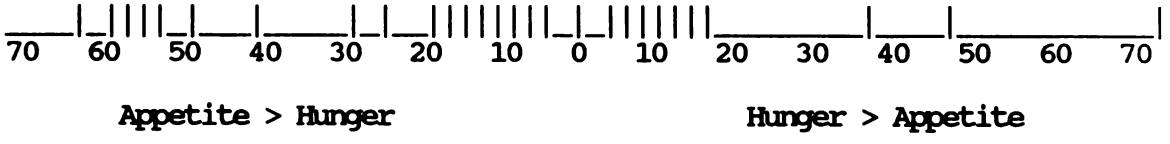
a----------b--------Hunger

A major consideration with the conceptualization in Figure 23 is the point, or area, where appetite and hunger overlap. It is tempting to view the area of overlap at the mid point of appetite (point "a") such that appetite continues to exist along with hunger for a period of time, influencing the selection of food, even when eaten in response to hunger signals. In this view, the scores of the subjects who report the presence of both appetite and hunger would have to be within the area of overlap (i.e., between point "a" and point "b", in the diagram).

However, an examination of the scores in this study revealed that 17 subjects had higher appetite scores than hunger scores and 10 subjects reported more hunger than appetite (one subject reported hunger and appetite as equal, with a score of 2). The differences between hunger scores and appetite scores ranged from 0 to 75 and are depicted in Figure 24.

Figure 24.

Difference between Hunger Scores and Appetite Scores of Subjects[1]

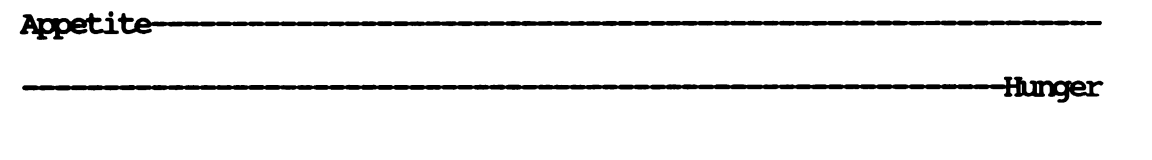


[1]Each vertical line represents one subject. Subjects with greater appetite scores than hunger scores (appetite > hunger) are on the left of the diagram, while those subjects with greater hunger scores than appetite scores (hunger > appetite) are shown to the right.

These results imply that although hunger and appetite are related, they are not on a continuum. Figure 25 represents the view of the relationship between hunger and appetite as revealed in this study.

Figure 25.

Diagramatic Relationship between Hunger and Appetite as Revealed in Present Study.



Additional support is provided for this conceptualization by the fact that only one subject had the same intensity of hunger and appetite (No. 27, Table 3), while all the other subjects were able to differentiate between the two and report different intensities of each.

The significant finding in this study was that the subjects were able to distinguish between hunger and appetite and indicate the intensity of their feelings. Although the terms are often used interchangeably, once the terms were defined, subjects were able to distinguish between the two and indicate the intensity of each of the sensations. This finding opens the door to future studies exploring the meaning of these feelings to the patient.

Hunger and Appetite vs Adequacy of Caloric Intake

The hypothesis that there will be a negative correlation between the magnitude of the expression of hunger or appetite and the adequacy of caloric intake was not supported. The hypothesis that

there will be no correlation between the magnitude of the expression of appetite and the adequacy of caloric intake was supported. There are inherent difficulties in accurately predicting an individual's caloric requirements. The use of the Harris-Benedict Equation (HBE) for determining basal energy expenditure (BEE) is the accepted standard. However, the particular body weight used in the formula will alter the results. Weight may be expressed in a variety of ways including a) current body weight; b) ideal body weight from published charts; c) usual body weight; or d) the patient's stated preferred body weight, the weight at which the patient felt the best.

The studies which established the validity of the HBE used direct or indirect calorimetry, which reflects the subject's current physiological state and have compared their findings with the calculated predictions using the HBE. Yet, if only the calculated amount of calories using the HBE were given, it would be clearly inadequate to provide for the increased demands of activity or anabolism. Also, a malnourished patient presenting with a depleted body weight would be maintained at that level if caloric intake were guided solely by results of the HBE using the patient's current weight. If the calculated basal requirements are too low, even with the adjustments for the activity and injury factors, the predicted caloric requirements may be less than required. Thus, even though a subject received more than 100% of calculated requirements, the baseline may have been inadequate and the amount infused may still

be less than the patient actually required, leading to feelings of hunger.

Even if the adjustments provided an actual reflection of the patient's physiological needs at the time, it may be that the patient was at the proper weight, statistically, but was biologically underweight and still had feelings of hunger, as the set-point theory (Nisbit, 1972) and the theory of restrained eating (Herman & Polivy, (1975) propose. These theories provide a case for calculating caloric requirements with the the weight at which the person feels and functions the best, esthetics and social acceptance of body size aside.

The issue of "ideal body weight" is questionable; there are inherent problems with using body weight from published standard tables. Although the health hazards due to excessive obesity and excessive leanness are multiple and diverse, the weight recommendations from actuary tables are based solely on the risk of dying (Andres, 1985). The weight recommendation tables in nearly universal usage have been derived from the experience of the life insurance industry.

The "desirable weights" in the Metropolitan Life Insurance height-weight tables (1983) were based on data from 25 life insurance companies in the United States and Canada and show the prevalence of mortality from 1954 to 1972 of approximately 4.2 million insured persons. Applicants with major disease conditions at the time of policy insurance were excluded from the study. The

terms 'ideal' or 'desirable' body weight were not even used in the new height and weight tables because of previous semantic misinterpretations (Schultz, 1986).

A seemingly potential strength of the 1983 tables was that they included a means of assessing frame size. Although both tables had made use of the small, medium, and large frame designations, the 1959 version did not provide a method by which this was to be measured. The 1983 revision gave an explanation for elbow breadth measurements based on the National Health and Nutrition Examination Survey, 1971-1974. It must be kept in mind, however, that frame size was not actually measured on policy holders, but was statistically assigned after the data were accumulated on heights and weights.

The division of the overall recommended weight range into separate ranges for three body frames was not based on actual measurements of body frame in the insured population. Neither the rationale for nor the detail of the computation of the ranges for the three frames or indeed for the overall ranges in weight for individual heights was presented. The 1983 tables provided two cutpoints for elbow breadth for men and for women at different height ranges that placed subjects into one of the three frame categories. However, because elbow breadth was not measured in the insured population, the weight adjustments for frame may have been set by unexplained empirical rules.

Differences found between actual metabolic rates and estimates

from the HBE may be due to differences in body composition and problems associated with estimating metabolic rates. Quantification of the body cell mass (BCM), the amount of lean, metabolically-active tissue, is obtainable only by istope dilution with measurements of exchangeable potassium and sodium (Kinney, Lister, & Moore, 1963).

Roza & Shizgal (1984) concluded that the HBE underestimated energy expenditure in malnourished patients but was accurate with well-nourished patients. Indirect calorimetry and body composition studies with isotope dilution were performed in 74 hospitalized patients to assess the accuracy of the HBE. They found that the HBE accurately predicted basal energy expenditure in the normally-nourished individuals with about 14% accuracy, but was unreliable in the malnourished patient. Malnutrition results in a loss of body fat, a loss of the BCM with expansion of the extracellular mass. The changes are reflected in changes in body weight, but because of the variable loss of body fat, body weight is not an accurate reflection of the loss of BCM. The use of the HBE assumes a normal body composition, which is not always a valid assumption in the hospitalized population.

Depression

Total scores on the Beck Depression Inventory did not correlate with the degree of hunger or appetite in this study. As expected, the presence of Item R (Loss of Appetite) in the Inventory skewed

the results when compared to the magnitude of appetite and hunger. This finding tends to validate the use of the VAS to measure hunger and to extract the item from the BDI to avoid artificial inflation of the results.

The results of the present study tend to support the theoretical framework presented in Chapter 2. Figure 2 (p. 13) presents hunger and appetite as two separate entities: hunger as the expression of physiological signals and appetite as reflecting psychological or mental urges or cravings. Although neither hunger nor appetite was significantly correlated with the Mental Items Subscale of the BDI. the expression of appetite was negatively correlated with the Physical Items Subscale (with and without Item R - Loss of Appetite). There was no significant relationship between hunger and depression scores on the BDI. These findings lend credence to the practice of distinguishing hunger from appetite. More research is needed to further explore the meaning of these sensations and their corresponding clinical and psychological states.

An interesting finding was that hunger correlated with Item R to a greater degree than did appetite. This raises the question of the definition used by the subjects in this study. Logs were not kept regarding the order of response to the tools; it is possible that those subjects who responded to the VAS prior to the BDI used the definition given in the VAS directions, the operational definition for this study. Those subjects responding to the BDI initially may have used their own definintion, or have used hunger and appetite

interchangeably.

To further examine the degree of depression subjects were grouped according to the cutting scores suggested by Beck et al. (1963): not-depressed (0-13), moderately depressed (14-24), and severely depressed (over 25). Using the total score on the BDI, 16 subjects were not depressed; 12 were moderately depressed; and no subject's score reached the severely- depressed category. When only the scores on the mental item sub-scale were used, none of the subjects were depressed. Although the statement made by Levenson (1982) that "the great majority of patients [on TPN] manifest depressive symptoms" (p. 38), may be true, they are not depressed, when the influence of the physical alterations are removed as suggested by Plumb and Holland (1977).

Age

As age increased there was a vague tendency for hunger to decrease, although the highest hunger score (96) was in the oldest subject (78 years) and the youngest subject (22 years) had a hunger score of 15. Gazzaniga (1978) felt that the use of age in the HBE was a liability since the older the patient, the larger the value subtracted from the energy requirement estimate. This may result in the potential to underestimate energy needs in the older population. If that were the case, then hunger would be expected to increase with age, as the adequacy decreases even though the calculated adequacy appears sufficient.

Overall, the hunger scores were lower than the appetite scores for all ages in this study. While appetite scores did seem to relate to age more than hunger scores did, no conclusions can be drawn from this data. The oldest subject had the highest hunger and appetite scores, while the youngest subject had one of the lower hunger scores.

Length of Time on TPN

The wide range of the length of time patients had been receiving TPN prior to data collection was due to the exclusionary criteria for this study. Patients were not eligible for admission into until they were able respond to the data collection tools. Many patients, especially those admitted with trauma, were critically ill at admission, in the intensive care unit, and physcially unable to complete the forms. Patients with hepatic or pancreatic disease were excluded until resolution of the active phases of the disease. The exclusion of febrile patients further limited data collection, especially those patients with an inflammatory process who were febrile in the initial stages. There was no significant effect of the length of time the subjects had been receiving TPN on the variables in this study.

Quetelet's Index

Because the present study lacked the facilities to determine BCM, the subject's body size was expressed in terms of the body mass

index to examine these effects. An easily obtained value, the body mass index (weight/height squared) was pioneered by Quetelet over 150 years ago (Keys, Fidanza, Karvonen, Kimura, & Taylor, 1972). Quetelet's Index (QI) has recently been found to be a valid and reliable indicator of body composition and relative fatness (Garrow & Webster, 1985). It has been used mainly in the obesity literature with 19-24.9 representing desirable index, 25-30 mild overweight, and over 30 being clinically relevant as an indicator of obesity (Garrow & Webster, 1985). Although desired ranges are still recommended, without supporting rationale, the use of the body mass index removes some of the cautions inherent in interpreting the published height/weight tables.

Keys, et al (1972) raised the question as to why the body weight, which is roughly the equivalent of a volume, which is three-dimensional, should be standardized in terms of a single linear dimension such as height. "Obviously, if the body had the same form at different heights, weight would tend to be proportional to the third power of the height" (p. 330). Consultation with a statistitian has confirmed these equations (Barr, 1987). After an extensive review of the literature, Keys, et al., concluded that Quetelet's Index, showed the highest correlation with independent measures of body fatness and removed the dependence on height.

Quebbeman, Ausman, and Schneider (1982) also found a difference in the predicted and actual energy expenditure with varyied body sizes. After comparing the predicted BEE from the HBE with measured

energy expenditure with indirect calorimetry in 44 patients, they concluded that use of the HBE underestimated the caloric requirements of subjects with smaller body sizes and smaller energy expenditure values. Measurement of body size was not stated, but the use of QI may have increased the study's generalizability.

Four subjects in the present study had a QI below 19, 22 were within the desirable range, one was mildly overweight, while one subject had a QI of over 30 and would be considered obese. When the hunger and appetite scores of the subjects were related to the BMI, no firm conclusions were reached. The theory proposed by Russek (1981), that hunger is stimulated by reduction in fat stores, should have produced an increased hunger with decreased body mass. If actual body composition studies had been performed in this study, it is possible that a correlation would have been found.

While the HBE correlates well with actual energy expenditure, individual metabolic rates may differ significantly and alter the results. Geissler, Miller, & Shah (1987) studied sixteen post-obese women, eight of whom had maintained their target weights for at least 6 months ($\underline{M}$ = 2.5 years). An additional eight subjects were still losing weight but were closely approaching their target weights; six were within the acceptable weight range. The control group consisted of 16 lean female volunteers with no history of a weight problem. Each post-obese subject was individually matched with her lean control by age, weight and height. Metabolic rate was calculated with the HBE and measured with a respirometer. Metabolic

rate was lower in the post-obese than the lean subjects whether at rest or during exercise.

It would have been interesting to have asked the 16 lean subjects in this study if they had ever been obese. A perplexing thought in attempting to separate the contributing factors in this study is the subject's pre-hospital lifestyle and eating habits. Closer scrutiny of the sample, by setting, reveals at least eight subjects who had come from a high-risk indigent population. This group may have had dietary restriction imposed by economic factors and had become accustomed to denying their hunger or appetite, much as the "restrained eating" theory of conscious control for weight reduction for cosmetic or health reasons (Herman & Polivy, 1975)

Implications for Nursing Practice

The results of this study tend to support the practice of distinguishing hunger from appetite. While the data did not support the hypothesis that hunger may be a danger signal reflecting inadequate caloric repletion, the results demonstrate that patients are able to sort out their feelings, interpret them as either hunger or appetite, and relate the intensity of their feelings.

The ability of patients to distinguish between hunger and appetite may be of value to the nurse whose goal is to modulate the human responses to disease processes or therapeutic interventions. This goal can be enhanced if feelings of hunger and appetite are elicited from patients on TPN who are not allowed to take anything

by mouth. Oral deprivation is problematic to patients who have no control over their ingestive behavior; this would be compounded if they also felt that their basic needs were being ignored, and that their symptoms were being dismissed out of hand. Further studies are needed to more fully explore the sensations of hunger and appetite and the meaning of each, to the patient and as a reflection of internal events.

More research is needed to discover simpler and more accurate methods of measuring hunger and distinguishing it from appetite to better administer to the patient's needs. The surface has barely been scratched. Until research studies provide specific guidelines, using the VAS as a rough estimate of the presence of hunger and appetite can be recommended. Patients should be monitored over time, with weight change and nitrogen balance studies to assure adequate caloric intake. Because the visual analog scale has only been used in research studies, the critical values for patients are unknown. Research studies using patients as subjects are needed to provide guidelines for this important concept.

The Beck Depression Inventory warrants further research for use in hospitalized patients. Development of categorization of the severity of depression, using the mental and physical subscales may reveal critical values which would identify those patients who would benefit from planned nursing interventions.

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APPENDIX A

Visual Analogue Scales

INSTRUCTIONS:

On the next sheet of paper, you will find two "visual analogue scales" which are used to measure people's feelings, something usually very hard to determine. Only you know how much hunger or appetite you are feeling; these scales provide a means of communicating this.

Please indicate the amount of hunger and appetite you are having at this time by making a mark, a short straight line, through the lines of the scales at the point which reflects the way you feel now. (Make your mark like this: ——|——).

HUNGER: Refers to the physical feelings you have when you need to eat. Hunger is a subjective (personal) indication of a physiological (biological) need for food. Everyone feels different things when they are hungry; some people feel an emptiness or rumbling in their stomach, some get a headache or feel lightheaded or grouchy. You may feel some of these or may feel other sensations. Only you can tell when you are hungry or how hungry you are.

APPETITE: Refers to the psychological (mental) desire or craving for food in general or for a specific food. Appetite is a subjective (personal) desire or urge to eat because of a mental urge. Everyone feels different things when they have an appetite; it is based on personal likes and dislikes of certain foods and taste, smell or texture. Maybe you just like something or it reminds you of things you like, such as a special occasion). Appetite also refers to the desire just to chew something (an oral craving). Only you can tell when you have an appetite or how strong that appetite is.

A mark all the way on the left side of the hunger line means you are not hungry at all; a mark all the way to the right indicates that you are as hungry now as you've ever been. Feel free to put your mark anywhere along the line to show the amount of hunger you feel now. Repeat this with the appetite scale. THERE ARE NO RIGHT OR WRONG ANSWERS. It is possible to be hungry but have no appetite, or to have an appetite but not be hungry, or to be hungry and have an appetite at the same time. The purpose of this study is to find out whether patients on total parenteral nutrition and enteral tube feedings are hungry, or have cravings, or both and we don't know the answers yet.

VISUAL ANALOGUE SCALE

Please indicate your level of hunger and appetite below:

HUNGER

Not at all physically hungry		Hungriest ever felt

APPETITE

No appetite (no cravings)		Severe appetite (Severe cravings)

Do you have an appetite for specific foods? Yes_____ No_____

If yes, please indicate the particular foods:

(Investigator will fill in this part)

Subject No._____

Time_____

Date_____

APPENDIX B

Beck Depression Inventory

Please use the attached sheet to indicate your responses. For each item, decide which statement best describes your feelings AT THIS TIME and write its number in the space provided for each category. Please respond to all items, if at all possible. There are 21 items and each item has four, five, or six options from which you can choose. Please select one statement from each category. There are no right or wrong answers. This is only to see if feelings of depression, not uncommon in hospitalized patients, affect statements of hunger and appetite.

ITEM A

1. I do not feel sad
2. I feel blue or sad
3. I am blue or sad all the time and I can't snap out of it
4. I am so sad or unhappy that it is very painful
5. I am so sad or unhappy that I can't stand it

ITEM B

1. I am not particularly pessimistic or discouraged about the future
2. I feel discouraged about the future
3. I feel I have nothing to look forward to
4. I feel that I won't ever get over my troubles
5. I feel that the future is hopeless and that things cannot improve

ITEM C

1. I do not feel like a failure
2. I feel I have failed more than the average person
3. I feel I have accomplished very little that is worthwhile or that means anything
4. As I look back on my life all I can see is a lot of failures
5. I feel I am a complete failure as a person (parent, husband, wife)

ITEM D

1. I am not particularly dissatisfied
2. I feel bored most of the time
3. I don't enjoy things the way I used to
4. I don't get satisfaction out of anything any more
5. I am dissatisfied with everything

ITEM E

1. I don't feel particularly guilty
2. I feel bad or unworthy a good part of the time
3. I feel quite guilty
4. I feel bad or unworthy practically all the time now
5. I feel as though I am very bad or worthless

ITEM F

1. I don't feel I am being punished
2. I have a feeling that something bad may happen to me
3. I feel I am being punished or will be punished
4. I feel I deserve to be punished
5. I want to be punished

ITEM G

1. I don't feel disappointed in myself
2. I am disappointed in myself
3. I don't like myself
4. I am disgusted with myself
5. I hate myself

ITEM H

1. I don't feel I am any worse than anybody else
2. I am very critical of myself for my weaknesses or mistakes
3. I blame myself for everything that goes wrong
4. I feel I have many bad faults

ITEM I

1. I don't have any thoughts of harming myself
2. I have thoughts of harming myself but I would not carry them out
3. I feel I would be better off dead
4. I have definite plans about committing suicide
5. I feel my family would be better off if I were dead
6. I would kill myself if I could

ITEM J

1. I don't cry anymore than usual
2. I cry more now than I used to
3. I cry all the time now. I can't stop
4. I used to be able to cry but now I can't cry at all even though I want to

ITEM K

1. I am no more irritated now than I ever am
2. I get annoyed or irritated more easily than I used to
3. I feel irritated all the time
4. I don't get irritated at all at the things that used to irritate me

ITEM L

1. I have not lost interest in other people
2. I am less interested in other people now than I used to be
3. I have lost most of my interest in other people and have little feeling for them
4. I have lost all my interest in other people and don't care about them at all

ITEM M

1. I make decisions about as well as ever
2. I am less sure of myself now and try to put off making decisions
3. I can't make decisions any more without help
4. I can't make any decisions at all any more

ITEM N

1. I don't feel I look any worse than I used to
2. I am worried that I am looking old or unattractive
3. I feel that there are permanent changes in my appearance and they make me look unattractive
4. I feel that I am ugly or repulsive looking

ITEM O

1. I can work about as well as before
2. It takes extra effort to get started at doing something
3. I don't work as well as I used to
4. I have to push myself very hard to do anything
5. I can't do any work at all

ITEM P

1. I can sleep as well as usual
2. I wake up more tired in the morning than I used to
3. I wake up -2 hours earlier than usual and find it hard to get back to sleep
4. I wake up early every day and can't get more than 5 hours sleep

ITEM Q

1. I don't get any more tired than usual
2. I get tired more easily than I used to
3. I get tired from doing anything
4. I get too tired to do anything

ITEM R

1. My appetite is no worse than usual
2. My appetite is not as good as it used to be
3. My appetite is much worse now
4. I have no appetite at all any more

ITEM S

1. I haven't lost much weight, if any, lately
2. I have lost more than 5 pounds
3. I have lost more than 10 pounds
4. I have lost more than 15 pounds

ITEM T

1. I am no more concerned about my health than usual
2. I am concerned about aches and pains or upset stomach or constipation or other unpleasant feelings in my body
3. I am so concerned with how I feel or what I feel that it's hard to think of much else
4. I am completely absorbed in what I feel

ITEM U

1. I have not noticed any recent change in my interest in sex
2. I am less interested in sex than I used to be
3. I am much less interested in sex now
4. I have lost interest in sex completely

Time 1 or 2 (circle) Time_____Subject No._____

BECK DEPRESSION INVENTORY

Response Sheet

Please use this sheet to record your responses to each item on the Beck Depression Inventory. Write the number of the statement which best describes your feelings (at this time) in each category.

A. _____

B. _____

C. _____

D. _____

E. _____

F. _____

G. _____

H. _____

I. _____

J. _____

K. _____

L. _____

M. _____

N. _____

O. _____

P. _____

Q. _____

R. _____

S. _____

T. _____

U. _____

APPENDIX C

BECK DEPRESSION INVENTORY SCORING KEY

The numbers on the left of each option indicate the relative value assigned to each option for scoring purposes (in some cases two responses have the same weight and are designated a, b, c to indicate this.) The name of each item is given in parentheses.

A (Mood)

0 I do not feel sad
1 I feel blue or sad
2a I am blue or sad all the time and I can't snap out of it
2b I am so sad or unhappy that it is very painful
3 I am so sad or unhappy that I can't stand it

B (Pessimism)

0 I am not particularly pessimistic or discouraged about the future
1a I feel discouraged about the future
2a I feel I have nothing to look forward to
2b I feel that I won't ever get over my troubles
3 I feel that the future is hopeless and that things cannot improve

C (Sense of Failure)

0 I do not feel like a failure
1 I feel I have failed more than the average person
2a I feel I have accomplished very little that is worthwhile or that means anything
2b As I look back on my life all I can see is a lot of failures
3 I feel I am a complete failure as a person (parent, husband, wife)

D (Lack of Satisfaction)

0 I am not particularly dissatisfied
1a I feel bored most of the time
1b I don't enjoy things the way I used to
2 I don't get satisfaction out of anything any more
3 I am dissatisfied with everything

E (Guilty Feeling

0 I don't feel particularly guilty
1 I feel bad or unworthy a good part of the time
2a I feel quite guilty
2b I feel bad or unworthy practically all the time now
3 I feel as though I am very bad or worthless

F (Sense of Punishment)

0 I don't feel I am being punished
1 I have a feeling that something bad may happen to me
2 I feel I am being punished or will be punished
3a I feel I deserve to be punished
3b I want to be punished

G (Self Hate)

0 I don't feel disappointed in myself
1a I am disappointed in myself
1b I don't like myself
2 I am disgusted with myself
3 I hate myself

H (Self Accusations)

0 I don't feel I am any worse than anybody else
1 I am very critical of myself for my weaknesses or mistakes
2a I blame myself for everything that goes wrong
2b I feel I have many bad faults

I (Self-punitive Wishes)

0 I don't have any thoughts of harming myself
1 I have thoughts of harming myself but I would not carry them out
2a I feel I would be better off dead
2b I have definite plans about committing suicide
2c I feel my family would be better off if I were dead
3 I would kill myself if I could

J (Crying Spells)

0 I don't cry anymore than usual
1 I cry more now than I used to
2 I cry all the time now. I can't stop
3 I used to be able to cry but now I can't cry at all even though I want to

K (Irritability)

0 I am no more irritated now than I ever am
1 I get annoyed or irritated more easily than I used to
2 I feel irritated all the time
3 I don't get irritated at all at the things that used to irritate me

L (Social Withdrawal)

0 I have not lost interest in other people
1 I am less interested in other people now than I used to be
2 I have lost most of my interest in other people and have little feeling for them
3 I have lost all my interest in other people and don't care about them at all

M (Indecisiveness)

0 I make decisions about as well as ever
1 I am less sure of myself now and try to put off making decisions
2 I can't make decisions any more without help
3 I can't make any decisions at all any more

N (Body Image)

0 I don't feel I look any worse than I used to
1 I am worried that I am looking old or unattractive
2 I feel that there are permanent changes in my appearance and they make me look unattractive
3 I feel that I am ugly or repulsive looking

O (Work Inhibition)

0 I can work about as well as before
1a It takes extra effort to get started at doing something
1b I don't work as well as I used to
2 I have to push myself very hard to do anything
3 I can't do any work at all

P (Sleep Disturbance)

0 I can sleep as well as usual
1 I wake up more tired in the morning than I used to
2 I wake up -2 hours earlier than usual and find it hard to get back to sleep
3 I wake up early every day and can't get more than 5 hours sleep

Q (Fatigability)

0 I don't get any more tired than usual
1 I get tired more easily than I used to
2 I get tired from doing anything
3 I get too tired to do anything

R (Loss of Appetite)

0 My appetite is no worse than usual
1 My appetite is not as good as it used to be
2 My appetite is much worse now
3 I have no appetite at all any more

S (Weight Loss)

0 I haven't lost much weight, if any, lately
1 I have lost more than 5 pounds
2 I have lost more than 10 pounds
3 I have lost more than 15 pounds

T (Somatic Preoccupation)

0 I am no more concerned about my health than usual
1 I am concerned about aches and pains @u(or) upset stomach or constipation or other unpleasant feelings in my body
2 I am so concerned with how I feel or what I feel that it's hard to think of much else
3 I am completely absorbed in what I feel

U (Loss of Libido)

0 I have not noticed any recent change in my interest in sex
1 I am less interested in sex than I used to be
2 I am much less interested in sex now
3 I have lost interest in sex completely