FACTORS RELATING TO DRUG-NUTRIENT INTERACTIONS IN LONG TERM CARE PATIENTS

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FACTORS RELATING TO DRUG-NUTRIENT INTERACTIONS

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LIST OF ABBREVIATIONS

INH - Isoniazid

L-dopa - Levodopa

PRN - As needed

mg - Milligrams

gm - Grams

ADA - American Dietetic Association

RDA - Recommended Dietary Allowance

HIEFSS - Hospital, Institutional, Educational Food Service Society

b.i.d. - Twice a day

t.i.d. - Three times a day

q.i.d. - Four times a day

Reg - Regular

Low Res - Low Residue

Lo Na - Low Sodium

AS - Arteriosclerosis

Fx - Fracture

ASHD - Arteriosclerotic heart disease

COPD - Chronic obstructive pulmonary disease

GI - Gastrointestinal

CVA - Cerebrovascular accident

CVI - Cardiovascular insufficiency

OBS - Organic brain syndrome

MR - Mental regardation

ca - Cancer

CV - Cardiovascular

MI - Myocardial infarct

CP - Cerebral palsy

ASCVD - Arteriosclerotic cardiovasulcar disease

L - Left

CHF - Congestive heart failure

TUR - Trans uretheral resection

CNS - Central nervous system

TIA - Transient ischemic attack

MAO - Monoamine oxidase

CBS - Chronic brain syndrome

UTI - Urinary tract infection

PVD - Peripheral vascular disease

BPH - Benign prostatic hypertrophy

TB - Tuberculosis

CHAPTER I

INTRODUCTION

The purpose of this study was to look at drug-nutrient interactions from the standpoint of the consulting dietitian dealing with nursing home residents. Nursing home residents may be old or sick or both. It is often difficult to detect by appearance a drug-nutrient interaction because the older person's appearance is already altered due to age. The emotional state of the older patient may contribute to this change due to a feeling of no longer being wanted or useful and this in turn may affect his or her appetite. Many times the older patient prefers desserts to protein foods. They are easier to chew and have a sweet taste, which may appeal to the older individual. In dealing with drug-nutrient interactions, the importance of proper nutrients and adequate fluids cannot be underestimated in that the older patient is often the one who is taking large amounts of drugs.

Becoming aware of drug-nutrient interactions will require the combined effort of both the dietitian and the pharmacist. The dietitian is often not familiar with the many drug categories and trade names and the pharmacist is often unfamiliar with nutritional implications of drug usage. The combined efforts of the pharmacist and the dietitian plus proper charting of those efforts will aid the physician, in treating the patient, to be aware of specific problems.

The objectives of this study were:

1. to determine the number and kinds of drugs prescribed and the time of administration for approximately 200 nursing home patients,

2. to relate the drugs being used to drug-nutrient interactions reported in the literature,

3. to assess aspects of nutritional status of subjects through determination of several health related factors, and

4. to make recommendations regarding health care of patients in nursing homes based on these findings.

CHAPTER II

REVIEW OF LITERATURE

Drug Absorption and Metabolism

Goodman and Gilman (1) define a drug as any chemical agent that affects living processes. Martin (2), in Hazards of Medication, defines a drug as a physiologically active substance used for diagnosis, prevention or treatment of disease. A dictionary definition is a chemical substance administered to a person or animal to prevent or cure disease or otherwise enhance physical or mental welfare (3). Martin (2) states:

> . . . there are no harmless medications. All are potentially hazardous to some extent and all must be prescribed and administered with caution. Otherwise, patients may be seriously injured (p. 17).

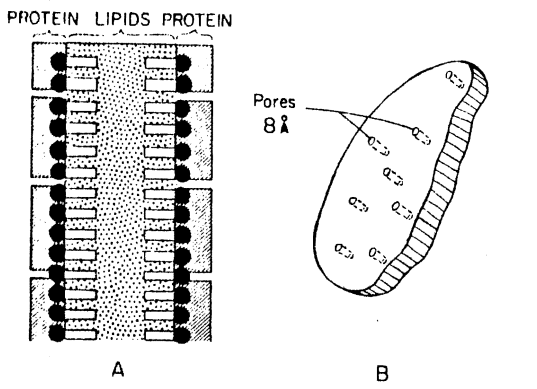
In order to produce its intended effects, a drug must be present at its site of action in the proper amount. This involves the amount of drug administered. It also involves how the drug is absorbed and distributed. Five factors which influence efficacy of a drug are:

1. Form--The drug is an appropriate biologically active form (salt, ester, ether, metabolite or other derivative).
2. Place--The drug reaches the appropriate site of drug action in the patient (gastrointestinal or urinary tract, blood, lymph or specific cells, tissue, organ or system) and therefore the drug must be suitably released from its dosage form and absorbed and delivered to the site of action within an appropriate period of time.
3. Quantity--The drug permeates that site in an appropriate concentration in an unbound pharmacologically active form.
4. Time--The drug remains at that concentration for an appropriate length of time by controlling input (gastrointestinal, parenteral or dermatomucosal) to offset the

output (metabolic destruction and excretion).
5. Response--The patient responds to the drug in an appropriate manner (2, p. 17).

The absorption of drugs is involved with the passage of a drug across cell membranes. Drugs generally pass through rather than between cells. Thus, the common barrier is the cell plasma membrane. The cell membrane is composed of proteins and lipids (1). The postulated structure is a central layer of lipids covered on either side by protein (Figure 1). The presence of protein on the surface supposedly makes water adhere easily to the membrane. The lipid or fat center makes the membrane impermeable to fat-insoluble substances. The lipids are about 65 per cent phospholipids, 10 per cent other lipids and 25 per cent cholesterol. The small knobbed structures are phospholipid molecules. The fat portion of the phospholipid molecule is dissolved in the lipid part of the cell membrane and the polar (ionized portion) projects outward with the protein lining the outer surface of the membrane (4).

Drugs cross cell membranes by passive processes, active transport, or pinocytosis (1). Active transport is a mechanism in which enzyme systems and special carrier substances carry the substance through the membrane (4). Passive processes refers to the simple process of diffusion (4). Pinocytosis is a process which causes the cell membrane to first turn back within itself and then to pinch off inside the cell to form a pinocytic vesicle. The pinocytic vesicle either dissolves inside the cell and discharges the contents into the intracellular space or combines with digestive enzymes before distributing them intracellularly (4).



Source: Arthur C. Guyton, Function of the Human Body (1969).

Figure 1. Cell Membrane--(A) Postulated Molecular Organization of the Cell Membrane, (B) Pores in the Cell Membrane

Absorption of drugs is affected by many variables. One variable is drug solubility. A drug given in solid form is not absorbed as readily as one given in solution. Conditions at the site of absorption affect solubility. Acidity is an important alkalinity variable. Acidic drugs tend to precipitate in the stomach fluids if the pH of the gastric juice is low. Dissolution then occurs very slowly. Drugs given in solutions of high concentration tend to be absorbed more rapidly than drugs in low concentration (2).

Absorption of a drug is increased or slowed by blood circulation at the site of absorption. If circulation is slow due to decreased blood flow, absorption is slow. If circulation is increased, such as by massage or local application of heat, absorption is more rapid (2).

The size of the area of the absorbing surface is important in absorption of a drug. Large surface areas cause the drug to be absorbed more rapidly (2). The absorbing surface is determined mostly by the route of administration. The oral route has been used for the longest period of time and is also the safest, most economical and most convenient. There are some disadvantages to the oral route of ingestion. These include vomiting which may be caused by irritation of the gastrointestinal mucosa of the drug. Some drugs may be destroyed by the digestive emzymes. Other drugs combine with food to form substances that cannot be absorbed. Using the oral route of drug administration also means the patient must cooperate in taking and swallowing the drug (1).

If gastric emptying time is retarded, drug absorption may be reduced. The small intestine provides a large surface area for absorption of a drug. If the drug is held in the stomach due to delayed gastric emptying time, absorption of the drug will be slowed because of the longer time before reaching the larger surface area of the intestine. Ingestion of food at the same time a drug is given will delay absorption time (1).

Sustained release pharmaceutical preparations are based on the factor of the dissolution rate in gastrointestinal fluids. Tablets within tablets, pellets in capsules, slowly dissolving salts in solids, provide a drug effect for eight hours or longer (1).

Drugs may also be given by injection. Absorption may be more predictable and more rapid. The dose which would be effective could be more accurately selected. It is necessary to give drugs by injection if the patient is vomiting and unable to retain the drug by mouth, or is

unconscious. The disadvantage of giving a drug by injection includes pain for the individual, strict asepsis must be followed to avoid infection and the patient cannot perform the injection himself (1).

Certain areas of the body serve as drug storage sites. Plasma proteins are one storage depot outside the cell. The binding is usually to albumin. Other plasma proteins can also bind drugs if the albumin is saturated and cannot bind any additional amount of a drug or drugs. The amount is dependent on the kind of drug involved. If phenylbutazone is given, binding may be as high as 98 per cent. If antipyrine is given, binding may be practically nil. If the drug has an affinity for albumin, the albumin may serve as a large storage depot. Some drugs are bound to ionic groups for the mucopolysaccharides and are stored in connective tissue. Bone may act as a storage for heavy metals and tetracylines (1).

Some drugs accumulate in cells. Active transport systems may be involved in drug storage within a cell. The binding within a cell occurs with tissue constituents such as nucleoproteins, phospholipids or proteins. This process is usually reversible, since almost all drugs finally disappear from the body. One example of a drug which is stored in the cell is quinacrine (an antimalarial agent). Four hours after a single dose, the liver concentration is 2,000 times greater than the plasma level. If the drug continues to be administered the liver accumulation may amount to 22,000 times the plasma level (1).

Many drugs are stored in the solid fat since they have a high lipid solubility. If an individual is obese, the fat content of the body may be as high as 50 per cent. Even in starvation, fat makes up 10 per cent of the body weight. In some instances, three hours after administration,

as much as 70 per cent of a highly lipid soluble drug may be present in body fat.

Drugs may accumulate in the gastrointestinal tract. If a drug is not very soluble in gastrointestinal fluids, it will be absorbed slowly. This will prolong the length of time the drug is in the body (1).

The cerebrospinal fluid does not generally store drugs since there is no protein present for binding (1).

The thyroid serves as a storage area for iodine and can also store some drugs such as perchlorate and other ions.

Drug effect is usually terminated by biotransformation and then excreted from the body. A drug may also be redistributed from its site of action. If it is distributed to other tissues, it is stored in active form and must still have its effects terminated by the usual means (1).

Two reactions by which biotransformation of drugs occurs are classified as synthetic and nonsynthetic. The nonsynthetic include oxidation, reduction or hydrolysis. The synthetic reaction is also called conjugation. This means a coupling between the drug and its metabolite plus a substrate in the body such as a carbohydrate or an amino acid.

Drayer (5, p. 927) states drug metabolism usually takes place in two steps:

$$\text{Drug} \xrightarrow[\text{enzymes}]{\text{Phase I}} \begin{cases} \text{Oxidation} \\ \text{Reduction} \\ \text{Hydrolysis} \\ \text{Metabolites} \end{cases} \xrightarrow[\text{enzymes}]{\text{Phase II}} \text{Conjugated metabolites}$$

Groups such as OH, CO_2H, and NH_2 are added to the drug molecule in step one. The metabolite reacts with a substrate in the body in step two, such as an amino acid or glucoronic acid, which results in a

conjugated metabolite that is usually excreted from the body. Cohen (6, p. 2) shows this reaction with the following diagram:

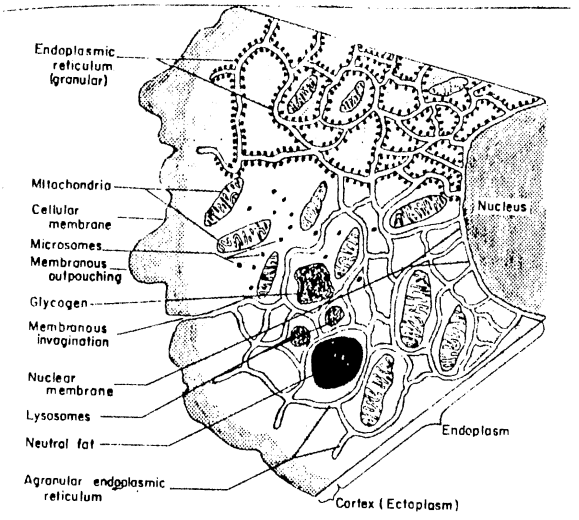
$$\text{Substance} \xrightarrow[\text{Glucuronyl Transferase}]{\text{Glucuronic Acid}} \text{Substance--}\underset{\text{(inactive)}}{\overset{\text{Glucuronide Complex}}{}}$$

If the drug is lipid soluble, the portion reabsorbed in the kidney tubule cells by diffusion is greater than if the drug is not lipid soluble (5).

The main site for biotransformation of drugs is in the liver. Some may occur in the plasma or kidneys. The enzyme system involved in the liver is located in the endoplasmic reticulum of the cells. More specifically, activity seems to occur in the microsomes (1). Microsomes are minute corpuscles embedded in the protoplasm. Great numbers of these corpuscles contribute to the granular appearance of protoplasm (3). The microsomal enzymes cause the reactions to occur for both synthetic and nonsynthetic biotransformations (1).

The endoplasmic reticulum extends through the cell like a canal system (Figure 2).

The endoplasmic reticulum may transport substances within the cell. Through the electron microscopy small ribonucleoprotein particulaes, which are called "palade granules" have been identified. These cause the endoplasmic reticulum to have a rough surface. The rough surface microsomes are thought to be concerned with protein synthesis. Only the smooth surface microsomes have enzymes that metabolize drugs. Drugs must be lipid soluble to be metabolized by liver microsomes. Some microsomal enzymes change lipid soluble substances to more water soluble substances (1).



Source: Guyton, Function of the Human Body (1969).

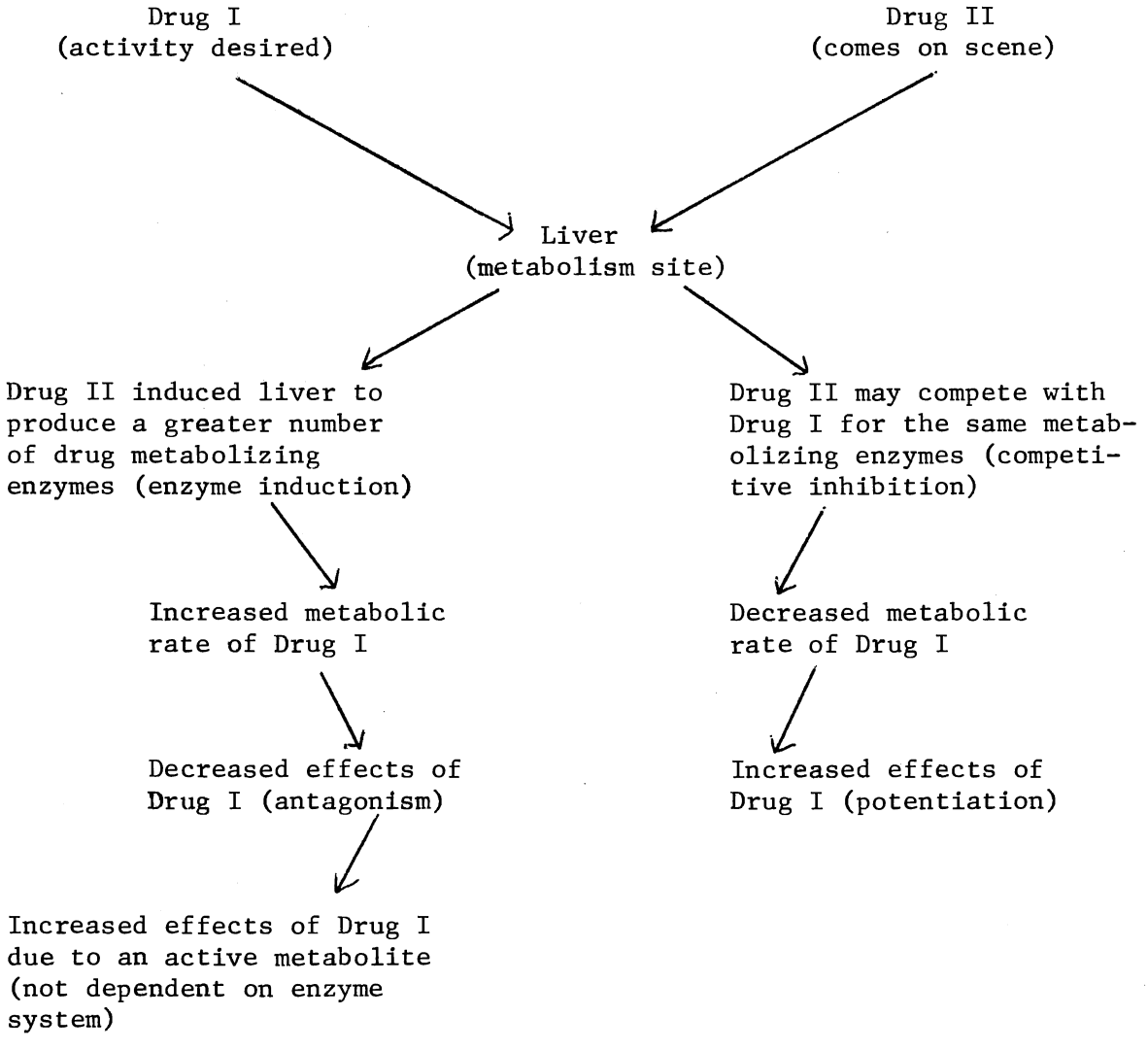
Figure 2. Organization of the Cytoplasmic Compartment of the Cell

Many things seem to affect the activity, structure and amount of smooth surfaced or drug metabolizing microsomes of the endoplasmic reticulum. Some of these are age, sex, temperature, nutritional status, stress or strain, pathological status, various drugs and hormones (1).

Observations have been made that a drug can stimulate its own metabolism or can stimulate the metabolism of another drug. The pharmacological effects of a second drug or second dose can be modified by the first drug or first dose since its metabolism can be affected by stimulation of the drug-metabolizing enzymes. The following diagram shows how the interaction may occur (1) (Figure 3).

Excretion of Drugs

The organ in the body of most importance for drug excretion is the



Source: M. S. Cohen, Drug Metabolism Class Notes.

Figure 3. Pathways of Interaction

kidney. Some drugs may be excreted in the feces if they are not absorbed in the digestive tract. Some drugs are eliminated from the body unchanged, some are eliminated as metabolites. The drugs which are eliminated unchanged are usually the more polar compounds. The drugs which are lipid-soluble and less polar are usually not eliminated until they are metabolized to be more polar. Only non-ionized or unchanged parts of a drug cross the lipid-like membranes of the cells in the kidney. Drug molecules are filtered at the glomerulus in the kidney and return to the blood stream by diffusion across the tubular cells. If compounds are more polar they cannot penetrate the membrane and so they are given off in the urine. Transfer of molecules across the membrane of the cell are pH dependent. If pH of the luminal fluid is changed, the excretion of the drug is influenced (1).

Three processes are involved in the excretion of drugs: (1) passive glomerular filtration, (2) active tubular secretion, and (3) passive tubular diffusion (1). Drugs which are organic acids may be moved through the body by the same system that transports substances like uric acid. Organic bases may be transported in the body by a system which takes care of naturally occurring bases in the body (1).

Weak acids and bases in non-ionized forms are reabsorbed or execreted by passive diffusion. Passive diffusion can be bidirectional. Drugs can diffuse in either direction across tubular cells, this depends upon the pH and the concentration of the drug. The excretion of a drug can be increased by changing the alkaline or acid content of the urine. The weak acids are excreted slower if the urine is more acid than plasma. The weak acids are excreted faster if the urine is more alkaline than plasma (1).

Some metabolites which have been formed in the liver are excreted via the bile. This may result in the drug being excreted in the feces but usually the drug is reabsorbed into the blood and then is excreted via the urine (1).

When discussing drugs, the terms "action" and "effect" have different meanings. Drug effects are produced by combining with enzymes or cell membranes. The action of drugs is related to the chemical structure. The chemical structure of a drug may have a minor modification which can result in a major change in pharmacological properties (1).

Drugs may act on particular cells or organs and produce a localized effect. Other drugs can have very general effects and act in or on most cells of the body. Drugs can also produce effects by increasing or decreasing the action of another substance in the body (1).

Drug effects may differ with one patient as compared to another patient. A drug may differ with even the same patient on different occasions and under different circumstances. All drugs produce some effect. The margin of safety of a drug is determined by the relationship of the desired effects versus the undesired effects (1).

Factors Which Predispose a Patient to a Drug Interaction

Many factors may influence an individual to a drug interaction. Some of these factors originate in the individual and others are related to the drug or drug product (8). Some of these factors are discussed in the following paragraphs.

Age

Age is an important factor in that studies show there is increased danger of adverse drug reactions in the geriatric individual and in the very young. The risk of a drug reaction is almost double in adults 60 to 70 years of age as compared to adults 30 to 40 years old (8).

Gastric secretion may be lower in older individuals. Goodman and Gilman (1) suggest one-third of older persons between 60 and 90 years of age may have achlorhydria. This affects certain drugs which are usually destroyed to a significant degree by gastric acid. As an example, penicillin G is usually destroyed to a significant degree by gastric acid and the result will be that penicillin is likely to have a greater effect in older people. Low gastric acid secretion may also be present during the first month of life (8).

Renal function may be slowed with age. This may result in a drug that would ordinarily be excreted accumulating in the body with possible toxic effects (8). Rosenberg and Mann (7) report one study of 35 elderly patients in which 46 per cent had reduced renal function, even though they did not have a kidney disease. The suggested cause was vascular insufficiency to certain areas of kidney tissue which results in reduced tubular function. When the glomerular filtration rate is decreased, the half-life of many drugs is increased. It is, therefore, necessary to decrease the dosage of the drug to prevent toxicity.

One study concerning digoxin compared blood levels and urine excretion levels of elderly men between 73 and 81 and men between 20 and 33 years of age. It was found the older men had blood levels of digoxin nearly double that of the younger men (7).

Factors which accentuate toxic effects of drugs in the infant are listed by Rosenberg and Mann (7) as follows:

1. Drug enzyme processes are immature or not formed.
2. There are different absorption, distribution and excretion patterns.
3. Immature tissues respond to drugs differently than mature tissues.
4. Drugs in this age group alter distribution of naturally occurring substances of the body, such as hormones and bilirubin, in a manner that does not occur in other age groups.
5. Drugs actually alter a developmental process that is occurring. In infants, oxidative pathways are deficient, particularly mocrosomal enzymes which metabolize drugs, e.g., barbiturates, acetanilid, amphetamines, codeine, and chlorpromazine. Reductive processes are less deficient and hydrolytic reactions are the least deficient in the newborn compared to the adult (p. 34).

One example of a drug which may produce death in an infant is chloramphenicol. The drug is detoxified by glucoronide formation but the young lack the enzyme glucoronyl transferase which forms glucoronide (7).

The blood brain barrier in infants is poorly developed. Some drugs displace naturally-occurring bilirubin from its bound form. Examples of these are salicylates and sulfonamides. The free bilirubin will pass through the blood brain barrier since it is poorly developed. The result can be degenerative changes that would not happen to an older child who has a developed blood brain barrier (7).

Tetracyclines cause discoloration or other damage to the teeth in young children if they are given when teeth are being formed. It is recommended that use of the drug be avoided during the first half of pregnancy, infancy and up to the age of eight (8).

Individual Differences

Different individuals metabolize drugs at different rates. Factors

which can vary with the individual patient are pH of the fluids, bile flow, gastric emptying time, volume and composition of gastrointestinal fluids, intestinal mucosal surface area (7).

Hussar (8) states that some drugs can be metabolized by different individuals at widely varying rates. One example is the coumarin anticoagulants such as warfarin and bishydroxycoumarin. Resistance to warfarin has been shown to be related to genetic variations.

Another inherited characteristic is a deficiency of the enzyme glucose-6-phosphate dehydrogenase. This appears more frequently in black persons, especially male, and among ethnic groups of the Mediterranean area. When drugs such as primaquine, phenacetin or sulfonamides are given to such individuals hemolysis can develop, causing a hemolytic anemia (8).

Ethnic Background

Black people seem to be more susceptible to hypertension. Antihypertensive therapy may be more likely to result in a drug interaction since hypertension is likely to develop at a young age and frequently be more severe (8).

Weight

It is recommended drug dosage be adjusted according to body weight. A person weighing 100 lbs. would need a smaller dosage than an individual weighing 220 lbs. The ratio between the body weight and the amount of drug administered generally influences the concentration of the drug at its site of action. However, if the body weight of an obese person is the same as that of a muscular football player, a problem would develop

since many drugs are distributed in the water and lean mass and not in the fat. For example, the same dosage of a drug, such as digoxin, would act differently. The obese person may develop a toxicity, as there would be a higher concentration of drug at the site of action. It is possible that women have a higher incidence of drug reaction than men because of the difference in weight, lean mass and subjective response (7).

Temperament

Easily excitable, nervous and unstable individuals require smaller amounts of stimulants and larger amounts of depressants. On the other hand slow, sluggish individuals require large amounts of stimulants and smaller amounts of depressants. Individuals who are neurotic, sensitive and high strung report more placebo reactions and have a tendency to experience more actions and reactions from medications (7).

Renal Function and Liver Function

Hussar (8) states the renal status of a patient should be known, particularly if the drug is primarily excreted by the kidney. In one study the adverse reactions in patients with impaired renal function were two and one-half times that of patients with normal renal function (9). A prolonged effect is seen if there is renal impairment and as additional doses are given, the blood levels will increase resulting in toxicity.

Only the portion of a drug in the blood bound to plasma proteins is available for glomerular filtration. Any agent that alters the protein binding may alter its filtration rate. If a second drug displaces plasma

protein binding sites, there is an increased amount of "free" drug present and the filtration rate will increase (8).

Urinary pH may alter activity of some drugs. One example is the administration of sodium bicarbonate with the older, less soluble sulfonamides to provide a more alkaline urine in which the drugs and their metabolites are more soluble, thus reducing the risk of crystal formation. The pH of the urine will influence the ionization of weak bases and weak acids and affect the extent to which drugs are reabsorbed or excreted. If the drug is in its non-ionized form it will more readily diffuse back into the blood from the urine. Erythromycin and Novobiocin are generally thought to be effective against gram-positive organisms. If the pH is made more alkaline, they may also be effective against a number of gram-negative bacterial infections (8).

Metabolism of drugs is concentrated in the liver. It is a triple system which consists of an uptake system (Y and Z proteins), an oxygen insertion system centered on cytochrome P-450, and a conjugation system. This triple system is adapted to removal of foreign compounds. Albumin transports lipophilic molecules, and the biliary apparatus secretes conjugated drugs into the bile. Drug metabolism is not a single entity but a family of enzymatic processes (14).

If there is hepatic damage, drugs may be metabolized at a slower rate and exhibit a prolonged effect. Some drugs may alter the amount or activity of the liver microsomal enzymes. Some substances are known to stimulate the activity of liver microsomal enzymes. The result is a more rapid metabolism and excretion of a drug. For example, phenobarbitol stimulates the activity of liver enzymes which can then increase the rate at which warfarin is metabolized. The result is that

warfarin is more rapidly excreted resulting in a decrease in anti-coagulant activity. The opposite effect could occur where one drug inhibits the activity of liver enzymes, which could prolong and intensify the effect of other drugs (8).

Protein Alteration

Many drugs are extensively bound to plasma proteins, only the unbound fraction of a drug is active. A dietary reduction of either quantity or quality causes a depression of hepatic microsomal mixed function oxidase activities. In persons whose dietary protein is low, the availability of drugs and thus their activity could be affected (11) (12) (13) (14).

In the ill, the binding of drugs to serum proteins can be different. When a pathological condition exists albumin may be reduced which may decrease the number of drug binding sites available (7).

Smoking

Jick (10) provided evidence that smokers behave differently than non-smokers when certain drugs are given. The data reinforced the hypothesis that cigarette smoke induces certain liver microsomal enzymes which increase the metabolism of some drugs. One drug studied was propoxyphene, a widely used oral analgesic. The drug was rated ineffective in 10.1 per cent of non-smokers, 15.0 per cent of light smokers and 20.3 per cent of heavy smokers. Another group of drugs studied were the benzodiazepines. There was a strong association between adverse reactions to diazepam and chlordiazepoxide and smoking. Adverse reactions were correlated with age and dose.

Environmental Factors

Liver microsomal enzyme activity may be increased by trace amounts of DDT and chlordane in foods which would significantly increase the metabolism of many drugs. The organic phosphorus insecticides may inhibit the hydroxylation of drugs by affecting liver microsomes (7).

Sunlight can be an environmental factor. Some drugs in combination with sunlight may increase the individual's susceptibility to dermatitis. These drugs are potent photosensitizers: barbituates, diuretics, certain steroids, tetracyclines, Dilantin, certain antidepressants, and the phenothiazines (7).

The temperature in different climates has an effect on drug reactions. In tropical climates, preliminary excitement and delirium are more readily produced by narcotics. Increase in temperature increases the rate at which drugs are metabolized and excreted. Temperature modifies individual response to drugs by effecting the intensity of sensory stimulation (7).

Lower body temperature decreases neuromuscular blockage produced by succinylcholine. The phenothiazines are more depressant at 13 to 18° C as compared to 25 to 30° C (7).

High altitude with associated oxygen deprivation increases sensitivity to many agents. Increased dosages of barbituates are necessary when there is intense noise of long duration. Crowding or isolation alters the response to drugs (7).

Diet

The body's adaptation to low energy and protein intake causes profound biochemical change. If protein is deficient, the body conserves

nitrogen by increased recycling of amino acids resulting in decreased urea formation. In this manner, nitrogen balance may be achieved but if the individual develops an infection, the balance is easily upset. Drugs which antagonize protein synthesis may also upset the balance. When energy intake is deficient, energy is provided by a breakdown of body proteins.

Clinical malnutrition may increase with infection and nutritional anemia (11).

Basu and Dickerson (12) state secondary malnutrition commonly results from some disease states. In developing countries where there is a shortage of food, primary malnutrition is common. Malnourished subjects are treated with drugs and both the drugs and the disease may effect metabolic processes. The metabolism of the drugs may be altered by the patient's nutritional status. The drugs themselves may change the requirement for particular nutrients.

Campbell and Hayes (13, p. 171) discuss the role of nutrition in the drug metabolizing enzyme system. They state "experiments with both adequate and deficient diets have shown impressive differences in the activities of drug metabolizing enzymes." Most of the information concerning diet and drug metabolizing enzymes has been from animal studies rather than clinical data. The lack of clinical data on drug and nutrition interaction may be based on the following:

1. The separation of cause and effect for the toxicity may be far removed in time.
2. The information on pathological symptoms characteristic of environmental toxins is not yet complete.
3. The expected systems are rather subclinical, nondescript and chronic in nature (13, p. 171).

Diet may affect the gastrointestinal absorption rate and consequently the concentration of certain drugs in the blood. As an

example, tetracycline and penicillin derivatives should be given one hour before or two hours after meals for optimal effect. Milk and other dairy products which contain calcium may decrease the absorption of tetracycline derivatives by forming a complex with them in the gastrointestinal tract (8).

If tetracyclines and milk are given together, plasma levels of the antibiotic only reach 19 to 75 per cent of what should be expected. Tetracyclines inhibit protein synthesis in man. They should not be used in renal failure. Tetracyclines can cause negative nitrogen balance in patients with normal renal function. Normal clinical doses of tetracyclines may inhibit the utilization of amino acids for protein synthesis. Prolonged use of tetracyclines must be considered in postoperative and injured patients who require effective tissue rebuilding (15).

Drugs may be mixed with various juice and beverages to try to mask unpleasant taste or to assist patients who have difficulty swallowing. This is especially true in the pediatric or geriatric patient. Mixing drugs with juices may precipitate a problem with acid labile substances where absorption might be impaired in the stomach because of decreased gastric pH or inactivation in acid media. Acid labile antibiotics such as ampicillin, erythromycin base and penicillin G potassium should not be mixed or allowed to stand in the beverages listed in the following table. The ingestion of large volumes (in excess of eight ounces) of such beverages with any acid labile substance should be avoided whenever possible (16).

It is generally thought that extreme shifts in urinary pH (well below 5 and 8) are difficult to achieve by dietary patterns alone.

TABLE I

pH RANGE OF SELECTED COMMERCIALLY CANNED JUICES AND OTHER BEVERAGES

Juices and Beverages	Approximate pH Range
Canned Juices	
Cherry	3.4 - 3.6
Cider	2.9 - 3.3
Cranberry	2.5 - 2.7
Currant	3.0
Grapefruit	2.9 - 3.4
Grape	3.5 - 4.5
Lemon	2.2 - 2.6
Lime	2.2 - 2.4
Pineapple	3.4 - 3.7
Prune	3.7 - 4.3
Tomato	3.9 - 4.4
Other Beverages	
Milk (cow's)	6.4 - 6.8
Milk (evaporated)	5.9 - 6.3
Beers	4.0 - 5.0
Wines	2.3 - 3.8

Source: P. G. Pierpaoli, Drug Therapy and Diet, Drug Intel. Clin. Pharm. (1972).

TABLE II

APPROXIMATE pH OF CARBONATED BEVERAGES

Beverages	pH
Club soda	4.7
Cream soda	3.9
Cherry soda	3.0
Cola	2.4
Ginger ale (pale dry)	2.7
Grape	3.0
Grapefruit	3.0
Lemon	2.9
Lemon-lime	3.1
Orange	3.2
Quinine	2.5
Raspberry	3.1
Root beer	4.0
Sarsaparilla	4.0

Source: Pierpaoli, Drug Intel. Clin. Pharm. (1972).

However, urinary changes have been observed with ingestion of acid-ash or alkaline-ash diets and urinary acidifying or alkalinizing drugs. Urinary pH can have clinical significance, one cited case was caused by ingestion of antacids, an alkaline-ash diet and quinidine causing quinidine intoxication. The patient was consuming eight tablets daily of an antacid preparation containing 200 mg. of magnesium, 20 mg. of simethicone, 200 mg. of dried aluminum hydroxide gel. He was also consuming one quart daily of fresh orange-grapefruit juice (1:1 ratio). The fruit juice mixture would be equal to about 50 mEq. of bicarbonate ion daily. The combination of an excessive alkaline-ash diet intake plus antacid therapy produced a consistent alkaline urine with a resulting decrease in quinidine excretion. The patient developed a serious abnormal sinus rhythm requiring hospitalization (16).

A strict vegetarian diet or excessive and chronic use of alkaline-ash foods could more than likely produce alkaline urine. Tables III and IV show examples of acid-ash foods and alkaline-ash foods. This should be considered in drug histories with patients receiving acidic or alkaline drugs. Excretion may be altered by changes in urinary pH which could result in drug toxicity (16).

Time of Administration

Time of administration of a drug is also of importance to the patient. The following table (Table V) has been compiled by Milne (15).

Shore (17) compiled an extensive table of drugs by both non-proprietary and trade name. The tables list times to take the drug in relationship to other foods and antacids.

TABLE III

POTENTIALLY ACID OR ACID-ASH FOODS

Meat	Fat
Meat, fish, fowl, shellfish Eggs Cheese (all types) Peanut butter	Bacon Nuts (Brazil, filberts, peanuts, walnuts)
Vegetable	**Bread**
Corn and lentils	Breads (all types), crackers Macaroni, spaghetti, noodles
Fruit	**Dessert**
Cranberries, plums, prunes	Cakes and cookies, plain

Source: Pierpaoli, Drug Intel. Clin. Pharm. (1972).

TABLE IV

POTENTIALLY BASIC OR ALKALINE-ASH FOODS

Milk, cream and buttermilk	Vegetables All types (except corn and lentils)
Nuts Almonds, chestnuts, coconut	Fruit All types (except cranberries, prunes, plums)

Source: Pierpaoli, Drug Intel. Clin. Pharm. (1972).

TABLE V

TIMES TO TAKE DRUGS

Drugs to be taken on an empty stomach (one hour before or three hours after meals)

- Ampicillin
- Erythromycin base drugs
- Penicillin G (ammonium and potassium)
- Triethylene melamine
- Oxacillin
- Lincomycin
- Triacetylolcandomycin
- Phenmetrazine
- Tetracycline and its derivatives except doxycycline
- Sulfonamides
- Sulfisoxazole
- Sulfadimethoxine

Drugs to be taken one-half hour before meals

- Atropine Sulphate
- Belladonna Tincture
- Phenobarbitol and Belladonna extract

Drugs to be taken immediately before, with or immediately after meals or with food or drink

- Aspirin
- Chloropropamide-Chloramide
- Chlorinase
- Diabinase, Stabinol
- Ferrous fumerate-Ferroton, Ferrofume, Fernamel, Fumiron, Irofum, Novofuma, Palifer, Tolifer
- Other ferrous compounds
- Phenoformin
- DBI
- Potassium Drugs
- Prednisone
- Tolbutamide

Drugs not to be taken with milk or milk products

- Potassium chloride tablets and solutions
- Potassium iodide
- Tetracyclines and its derivatives
- Biscacodyl (Ducolax)
- Digitalis
- Digoxin

Drugs not to be taken with fruit juice (Fruit juice is acidic and may cause premature breakdown of these acid labile drugs, or the juice may increase the urinary excretion of the drug. This includes <u>all</u> juices, beer, wine, and soft drinks.)

- Ampicillin
- Erythromycin base drugs--E-mycin
- Erythromycin, Llotycin
- ASA and other salicylates
- Lasix
- L-Thyroxin

TABLE V (Continued)

Drugs not to be taken with fruit juice (Continued)

- Penicillin G (ammonium and potassium)
- Atropine
- Aldomet
- Demerol
- Elavil
- Neg Gram Tabs (Nalidixic acid)
- Quinidine
- Warfarin
- Amobarbitol
- Benadryl

Drugs absolutely contra-indicated with alcohol

- Apresoline
- Chloral hydrate
- Codeine
- Chloromycetin
- Compazine
- Darvon
- Dilantin
- Demerol
- Diuril
- Elavil
- Flagyl
- Forhistol
- Guanethidine
- Hydrodiuril
- Insulin
- Lasix
- Librium
- Morphine
- Meprobamate
- Nitroglycerine
- Neg Gram Tabs
- Seconal
- Quinol
- Tetracyclines
- Valium
- Natulan
- Procarbazine
- Methyl hydrazine
- Tolbutamide

Drugs not to be taken with antacids

- Bisacodyl
- Ferrous gluconate, lactate
- Succinate
- Sulphate
- Ferrous Fumerate
- Tetracyclines and all its derivatives

Drugs not to be taken with mineral oil

- All vitamin preparations

Foods high in pressor amines should not be eaten while taking these drugs and should not be eaten for three weeks following cessation of the drug

- Tranylcypromine (Parnate)
- Isocarbazid (Marplan)
- Nialamide (Niamid)
- Morphine
- Phenylzine sulphate (Nardil)
- Natulan
- Amphetamines
- Procarbazine
- Digitalis and digitalis glycosides
- Methyl hydrazine
- Phenylephrine (neo-synephrine)
- Guanethidine
- Insulin
- Reserpine
- Furazolidone
- Isoniazid

TABLE V (Continued)

Drugs not to be taken with vitamins
Anticoagulants and vitamin C
Anticoagulants and vitamin B complex
Vitamin C SHOULD be taken with ferrous compounds as they are better absorbed in the presence of ascorbic acid.

Source: A. A. Milne, Food and Drug Interactions, J. Can. Dietet. A. (1973).

Drug-Nutrient Interactions

Drugs are one class of a group of substances which are of no nutritive value and are chemically foreign to the body. This group of substances are known as xenobiotics, anutrients or foreign compounds. Anutrients include food and cosmetic additives such as benzoic acid, azo dyes and butylated hydroxytoluene; natural anutrients in foods such as alkaloids, flavonoids, ethers; food contaminants such as pesticides and industrial chemicals (12).

Chemicals normally regarded as foreign to the body are metabolized by a group of non-specific enzymes located in hepatic microsomes. These are referred to as "microsomal drug metabolizing enzymes" (12).

Pierpaeoli (16) categorizes pharmacologically active substances present in foods:

1. Foods of Plant and Animal Origin
   In this class, one can cite the presence of 5-hydroxy-tryptamine in pineapples and bananas, 3-4-dihydroxy-phenylalanine (DOPA) in broad beans, oxalates in spinach, rhubarb and celery. Various metals such as selenium, potassium, calcium, magnesium and sodium in grains and

other foods. Also, the presence of tyramine in various cheeses and chicken livers as well as fatty acids and lipids in meats.

2. Foods of Marine Origin
   Neurotoxins have been found in species of poisonous fish and paralytic toxins in polluted shellfish. Pesticide and heavy metal residues also have been detected in several species of edible fish.
3. Food Additives and Contaminants
   Increasing quantities of intended food additives such as preservatives, antioxidants, sequestrants, surface-active agents, stabilizers and thickeners, bleaching and maturing agents, buffers, acidulants, food colors, non-nutritive and special dietary sweeteners, flavors and so forth are used in food processing technology to enhance the taste, structure or storage life of food. Examples of naturally occurring contaminants are the potent mycotoxins and bacterial toxins which result from fungal or bacterial contamination of foods. Man-made contaminants include antibiotics, pesticides, radionuclides, metals and processing degradation products.
4. Water, Soft Drinks and Alcoholic Beverages
   These dietary constituents may contain various metals, xanthines, histamines, alcohol and congeners (p. 89).

The absorption of drugs and foods is not similar. The absorption of the majority of drugs is dependent on lipid solubility, pH of the medium, particle size, physical form, and rate of dissociation. The more lipid-soluble the drug, the greater the extent of absorption. Most drugs transported across the gastric or intestinal mucosa are transported by passive non-ionic diffusion. Active transport is believed to be present with the digitalis glycosides and the pyrimidine compounds. Weak acids are usually absorbed in the stomach (pKa less than 2) and weak bases (pKa less than 8) are usually absorbed in the upper part of the intestine. It is believed that digestive enzymes are not factors in drug absorption (16).

Absorption of food depends on gastrointestinal secretions, pH and enzyme activity. Lipid solubility is only important in lipid absorption. Transport of food is not limited to passive diffusion (16).

Most drugs that interfere with nutrients and impair absorption are given for long-term treatment. An exception to this is the anti-infective agents (16).

Drugs can increase vitamin excretion, interfere with absorption of vitamins or with vitamin utilization. This can occur even when the diet is adequate under normal circumstances. Drugs produce deficiency when compounds are chemically and functionally related to a vitamin, or they may be unrelated in structure and have a common side effect such as malabsorption (18).

According to Roe (19), it can be generalized that with the exception of certain vitamin antagonists, drugs in therapeutic dosage interfere to a limited extent with nutrient utilization, having least effect over short periods of time or where the intake of a nutrient exceeds demand. A person who is a chronic drug user may have the most effect. The drugs will emphasize a condition where nutrients are lacking either because of the disease condition or by marginal intakes of nutrients.

Malabsorption of nutrients can occur because of drug intake. Abuse of cathartics can result in severe malabsorption problems. Malabsorption can occur through a number of mechanisms. A drug may provide a solution for nutrients such as in the case of mineral oil and interfere with absorption of fat soluble vitamins. Cathartics may greatly induce intestinal transit time and cause the nutrients to pass too rapidly through the small intestine for optimal absorption (19).

Some drugs may bind bile salts and impair absorption of fats and fat soluble vitamins. Some evidence exists that absorption of vitamin B_{12} may be decreased when a drug binds bile salts (19).

Some drugs cause cellular damage in the small intestine. This may be interference with the mitotic activity of the intestinal epithelium or destruction of the villous epithelium. If the microvilli are destroyed, there may be a loss or inhibition of intestinal enzymes (disaccharidases and peptidases) located along the brush border. Certain drugs may depress nutrient uptake from the small intestine. A drug may have a particular affinity for a nutrient. Roe (19) has prepared the following table (Table VI) of primary intestinal absorptive defects induced by drugs.

Drugs can cause secondary malabsorption by affecting nutrient metabolism so the active transport system is inhibited. Examples that have been documented are related particularly to vitamin D and calcium transport (19).

Drugs differ greatly in their chemical structure which have an effect on carbohydrate absorption. Because carbohydrate digestion and absorption are extremely efficient, mild malnutrition or a single drug used in a patient is probably insufficient to produce clinical signs of malabsorption. One group at risk is the elderly patient who may be poorly nourished and also taking several medications at the same time. The drugs may interact to produce malabsorption and further increase the malnourished state (20).

Drug-Vitamin Interactions

Some compounds act as antivitamins. Synthesis of antivitamins has either been on purpose or by chance. Metabolites that participate in enzyme reactions can be antagonized by a compound having a similar structure, chemically or physically. It must be similar enough that the

TABLE VI

PRIMARY INTESTINAL ABSORPTIVE DEFECTS INDUCED BY DRUGS

Drug	Usage	Malabsorption or Fecal Nutrient Loss	Mechanism
Mineral oil	Laxative	Carotene, Vit. A, D, K	Physical barrier Nutrients dissolve in mineral oil and are lost Micelle formation
Phenolphthalein	Laxative	Vitamin D, Ca	Intestinal hurry K depletion Loss of structural integrity
Neomycin	Antibiotic to "sterilize"	Fat, nitrogen, Na, K, Ca, Fe, lactose, sucrose, vit. B_{12}	Structural defect Pancreatic lipase Binding of bile acids (salts)
Cholestyramine	Hypocholesterolemic agent Bile acid sequestrant	Fat, vit. A, K, B_{12}, D, Fe	Binding of bile acids (salts) and nutrients (e.g., Fe)
Potassium chloride	Potassium	Vitamin B_{12}	Ideal pH
Colchicine	Anti-inflammatory agent in gout	Fat, carotene, Na, K, vit. B_{12}, lactose	Mitotic arrest Structural defect Enzyme damage

TABLE VI (Continued)

Drug	Usage	Malabsorption or Fecal Nutrient Loss	Mechanism
Biguanides: Metformin Phenformin	Hypoglycemic agents in diabetes)	Vitamin B_{12}	Competitive inhibition of B_{12} absorption
Para-amino salicylic acid	Anti-tuberculosis agent	Fat, folate, vitamin B_{12}	Mucosal block in B_{12} uptake
Salicylazo-sulfapyridine (Azulfidine)	Anti-inflammatory agent in ulcerative colitis, and regional enteritis	Folate	Mucosal block in folate uptake

Source: Daphne A. Roe, Drug Induced Nutritional Deficiencies (1976).

enzyme is deceived into taking the foreign molecule in place of the substrate (19).

Substances which are folic acid antagonists are the following:

1. Methotrexate--used in the treatment of malignant and benign diseases.
2. Pyrimethaime--used in the treatment of chloroquine resistant malaria and ocular toxoplasmosis.
3. Pentamidine--used in the treatment of African trypanosomiasis.
4. Triamterene--diuretic.
5. Trimethoprim--potentiator of sulfonomides in the therapy of bacterial infection.

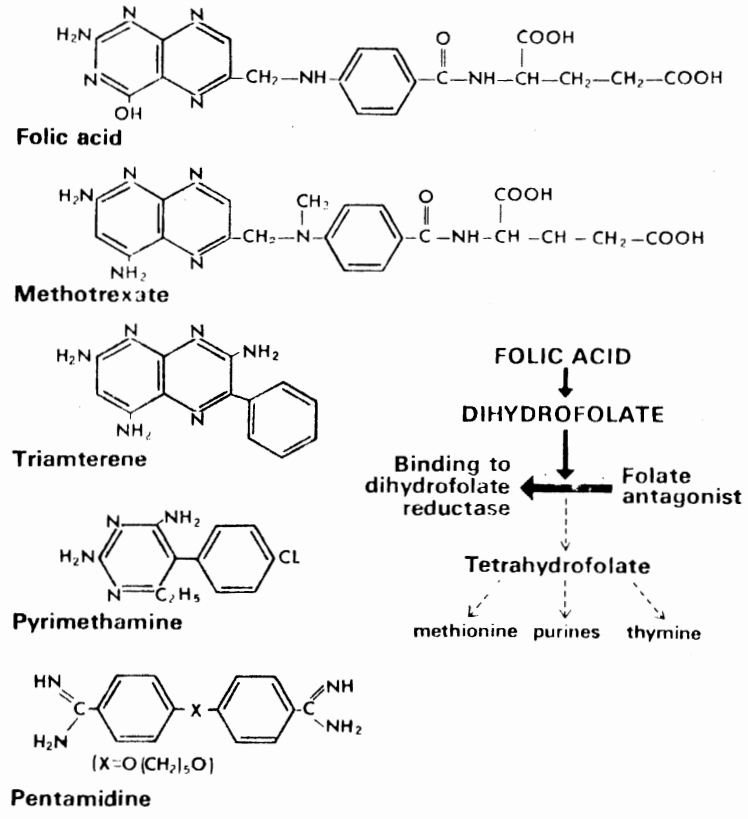
Roe (19) shows the chemical structure and site of action of folate antagonists in Figure 4.

The symptoms of folate deficiency due to drugs are weight loss, megoblastic anemia, glossities, diarrhea. Ankle edema, hyperpigmentation of the skin, splenomegaly, and hepatomegaly may also be seen (19).

In the body, there are vitamin B_6 dependent reactions which are of concern when discussing the effects of drugs. Vitamin B_6 is important to the metabolism of tryptophan and its conversion to niacin. Roe (19) shows the importance of B_6 in the body in the following diagram and the step which is blocked by B_6 antagonists (Figure 5).

It is obvious why drugs which are vitamin B_6 antagonists are able to produce niacin deficiency (19).

Vitamin B_6 is important for the synthesis of several neurotransmitters and neurohormones. Roe (19) has shown the following relationships (Figure 6). These compounds are necessary for neuronal function at many levels. This explains the fact that there are



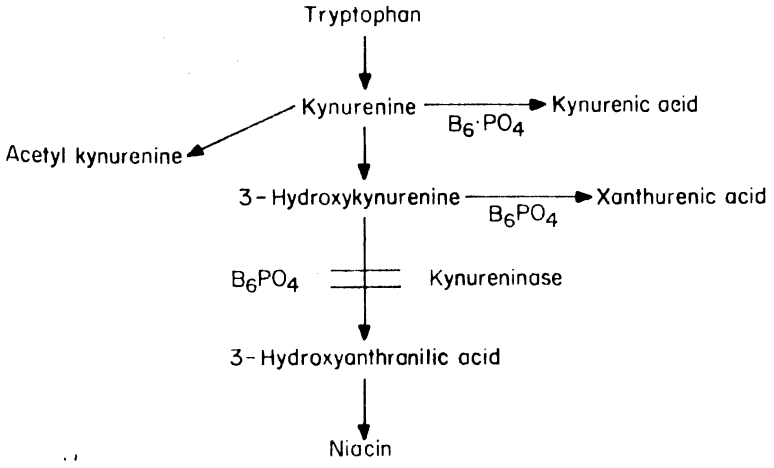
Source: Roe, Drug Induced Nutritional Deficiencies (1976).

Figure 4. Structures and Site of Action of Folate Antagonists

neurological symptoms associated with vitamin B_6 deficiency associated with drug uptake.

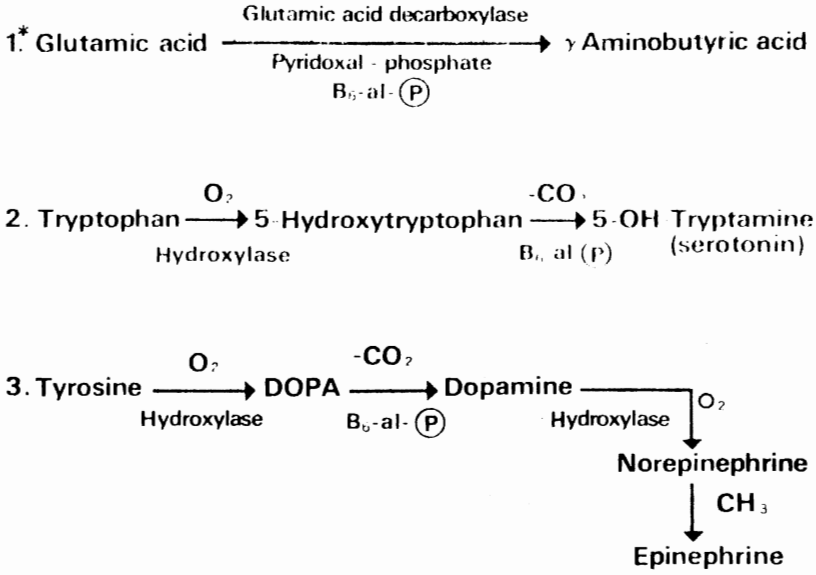
Vitamin B_{12} may also be affected by certain drugs. The vitamin is protein bound as it occurs in animal products. The pepsin in the stomach and the acid pH split off vitamin B_{12} from its protein binding sites. The stomach acid enhances the binding of B_{12} to the gastric intrinsic factor. Absorption of vitamin B_{12} is dependent on calcium. Vitamin B_{12} functions as a coenzyme (19).

There is data to suggest that vitamin B_{12} is a necessary requirement for the cell uptake of the coenzyme form of folate. Figure 7 shows sites of drug interactions.



Source: Roe, Drug Induced Nutritional Deficiencies (1976).

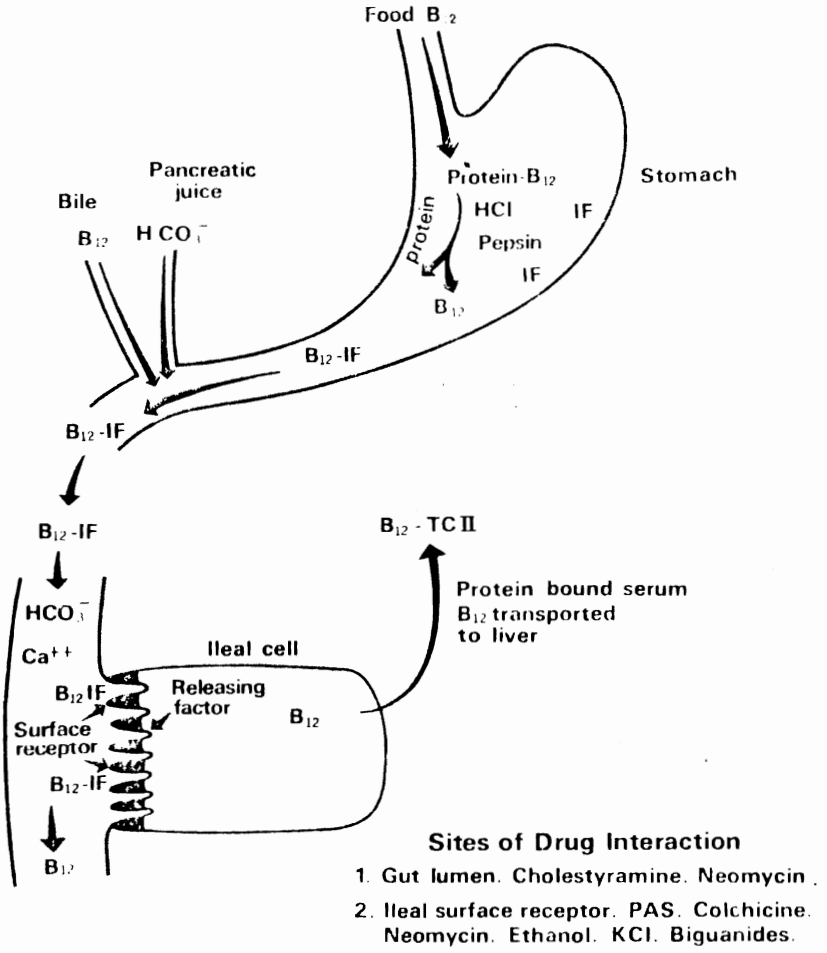
Figure 5. Endogeneous Pathway for Niacin Biosynthesis From Tryptophan Showing Vitamin B_6-Dependent Reactions and Step Preferentially Blocked by B_6 Antagonists



*Convulsions in drug induced B_6 deficiency may be associated with decreased function of B_6 dependent, glutamic acid decarboxylase

Source: Roe, Drug Induced Nutritional Deficiencies (1976).

Figure 6. Vitamin B_6 and the Synthesis of Neurotransmitters



Source: Roe, Drug Induced Nutritional Deficiencies (1976).

Figure 7. Absorption of Vitamin B_{12}

Metabolic interactions are present between vitamin B_{12} and folate. If methyltetrahydrofolate is to be converted to other folate coenzymes, the vitamin B_{12} dependent methyltransferase reaction must be present. When there is a deficiency of vitamin B_{12} the activity of the necessary enzyme is decreased. The result is lowered tissue levels of certain folate enzymes. These enzymes are necessary for purine and pyrimidine biosynthesis and, therefore, normal DNA biosynthesis. In both cases of folate and vitamin B_{12} deficiency, abnormal DNA synthesis results in megoblastic cells which are unable to mature in a normal manner or enter mitosis. This results in a condition known as megoblastic anemia (19).

The largest proportion of all nutritional deficiencies caused by drugs fall into the category of absorption or utilization of vitamin B_6, folate and vitamin B_{12} (19).

Five major groups of drugs function either as vitamin B_6 antagonists or increase the turnover of vitamin B_6 in the body:

1. Antituberculous drugs--isonicotinic acid hydrazide, cycloserine.
2. Hydralazine--used in treatment of hypertension
3. Penicillamine--a metal chelator.
4. L-Dopa--treatment of Parkinson's disease.
5. Oral contraceptives (19, pp. 15-16).

Other major groups of drugs have been shown to affect folate, either by affecting absorption or acting as a folate antagonist or increasing the loss of folate from the body:

1. Cytotoxic agent--methotrexate.
2. Anti-malarial--pyrimethamine.
3. Anti-convulsants--diphenylhydantion, phenobarbitol, primidone.
4. Diuretic--triamterene.
5. Oral contraceptives.
6. Antituberculous drug--cycloserine.
7. Anti-inflammatory drugs--aspirin, salicylaxosulfapyridine, pentamidine.
8. Anti-infective--aromatic diamidine (19, p. 16).

Four major groups of drugs affect absorption of vitamin B_{12}:

1. Biguanides--metformin and phenformin.
2. Antituberculous drug--paramino-salicylic acid.
3. Bile acid sequestrant--cholestyramine.
4. Potassium chloride (19, p. 17).

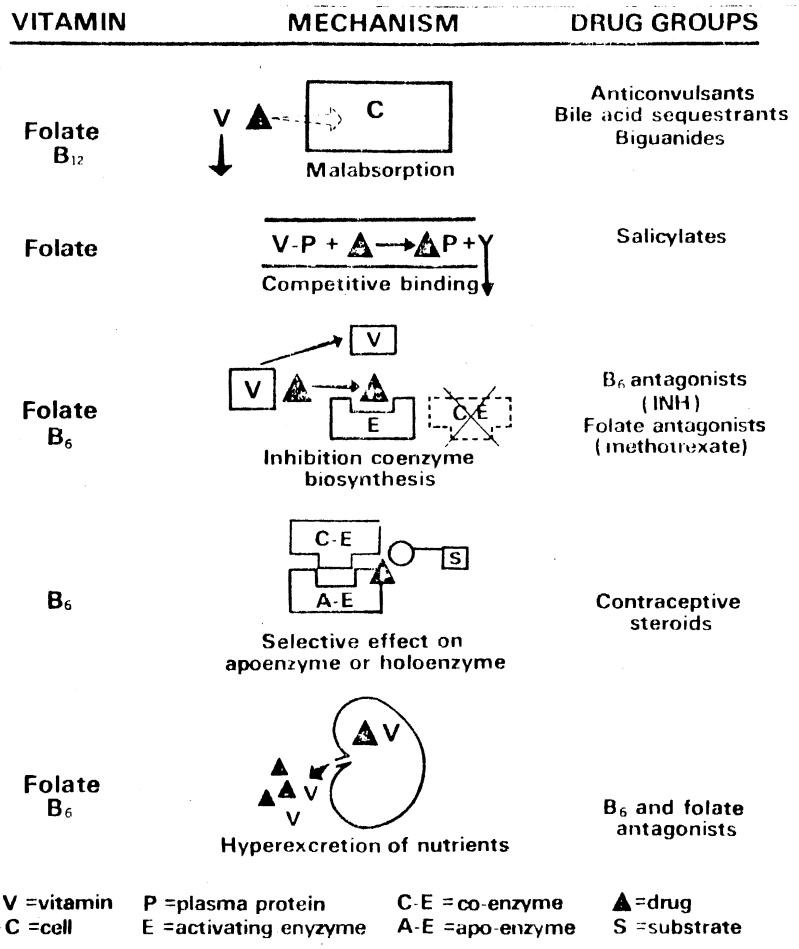
Alcohol affects all three vitamins because it causes a direct toxic effect on the gastrointestinal tract, on the liver and the hemopoietic system (19). Figure 8 shows a comparison of mechanisms for production of vitamin B_6, folate and vitamin B_{12} depletion by drugs.

Niacin is a water-soluble vitamin. Tuberculosis patients on isonicotinic hydrazide have been reported to have a pellagra-like illness. The niacin deficiency may be secondary to the pyridoxine deficiency caused by the drug. Pyridoxine is necessary in the conversion of tryptophan to niacin (18).

The coenzymes NAD (nicotinamide adenine dinucleotide) and NADP (nicotinamide adenine dinucleotide phosphate) are involved with hydrogen transport. Many dehydrogenases require one or the other for activity. NAD and NADP are necessary for carbohydrate metabolism, in protein metabolism and in fat metabolism. Nicotinamide coenzymes are important for microsomal mixed function oxidations. NADPH is necessary for microsomal drug oxidation reactions (19).

Niacin deficiency not only affects endogenous metabolic systems but may also impair drug metabolism because of inactivation of biotransformation systems (19).

Riboflavin in food is mainly in the form of two coenzymes--FMN (Flavin mononucleotides) and FAD (Flavin adenine dinucleotide). Free riboflavin is present in breads and cereals when they are enriched with B vitamins and iron. When riboflavin and FMN are absorbed from the



Source: Roe, Drug Induced Nutritional Deficiencies (1976).

Figure 8. Comparison of Mechanisms for Production of Vitamin B_6, Folate and Vitamin B_{12} Depletion by Drugs

intestines, a large part of the vitamin and coenzyme is bound to albumin. Protein binding sites for drugs may displace riboflavin (19).

Boric acid forms a complex with the ribityl side chain of riboflavin. Absorbed boric acid causes hyperexcretion of riboflavin, either as the borate complex or as the free vitamin (19).

Riboflavin is converted in the liver to FAD and FMN. Conversion of riboflavin to FMN involves the enzyme flavokinase and ATP. FMN is converted to FAD by the enzyme FAD pyrophosphorylase and ATP (19).

Riboflavin deficiency was found in one study in 50 per cent of a group of hospitalized alcoholic patients. The deficiency of riboflavin may be due to deficient intake; to impaired coenzyme synthesis if the patient has cirrhosis or to defective absorption of the vitamin (19).

Absorption of riboflavin is increased in the presence of food in the intestinal tract. Investigators who made this observation thought it might be due to a decrease in intestinal transit rate; thereby, leaving the vitamin longer in the area of the absorption sites (19).

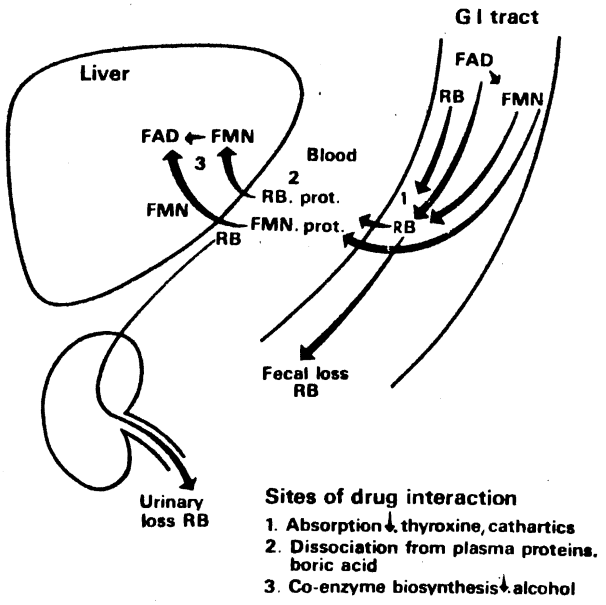
Hyperthyroidism or administering thyroxine decreases riboflavin absorption. Riboflavin absorption is increased in hypothyroidism (19).

Findings suggest that long term drug intake which requires increased activity of the drug metabolizing enzymes may increase riboflavin requirements (19).

Figure 9 shows Flavin absorption, transport, biotransformation and excretion in man.

Thiamine is important in the enzyme catabolism of pyruvic acid. It also functions in the decarboxylation of alpha-ketoglutaric and other a-keto acids. The co-enzyme form of thiamine is thiamine pyrophosphate. Blood pyruvate levels and blood levels of a-ketoglutarate are elevated

in severe thaimine deficiency. Thiamine pyrophosphate is required for the enzyme transketolase which functions to catalyze the production of ribose from glucose. Ribose is necessary for nucleotide formation. A useful test to detect minor degrees of thiamine deficiency is a test for erythrocyte transketolase activity (19).



Source: Roe, Drug Induced Nutritional Deficiencies (1976).

Figure 9. Flavin Absorption, Transport, Biotransformation and Excretion in Man

Thiamine deficiency in alcoholics may be due to impaired absorption, impaired function of the co-enzyme form of thiamine, or a deficient intake of thiamine in the diet (19).

Thiamine needs may be increased if a patient is receiving digitalis alkaloids. In patients with digitalis intoxication, increased serum pyruvate levels have been found (19).

Vitamin C is important in the oxidation-reductions reactions in the body. It is also necessary for normal synthesis of collagen and elastin. Vitamin C may function in mixed function oxidase systems. The synthesis of vitamin C may be increased by drugs which stimulate drug-metabolizing enzymes. Drugs which may cause tissue desaturation of ascorbic acid are alcohol, anorectic agents, anticonvulsant drugs, tetracycline and aspirin. Aspirin is the most important drug shown to cause tissue depletion. It causes a reduction in platelet levels of ascorbic acid (19).

The first changes in vitamin C deficiency are decreased urinary excretion and decreased plasma volumes. Clinical signs which develop are follicular hyperkeratosis of the backs of backs of the arms, valves of the legs, thigh and buttocks. Further clinical signs are bleeding and swollen gums, conjunctival hemorrhages, and perifollicular hemorrhages (19).

Subclinical deficiency of vitamin C has been noted among drug addicts. There may be some detrimental effects to massive doses of vitamin C. A group of paraplegics taking large doses of vitamin C (1000 mg.) daily were shown to have low vitamin B_{12} levels in the blood. It has been suggested that large amounts of vitamin C should not be taken with meals since it may destroy vitamin B_{12} in food. A high intake may also effect the utilization of betacarotene (19).

Vitamin D is necessary for calcium uptake from the intestine. Recent studies have shown that sterols within the vitamin D class

function similar to steroid hormones. A deficiency of vitamin D in children results in rickets and in adult osteomalacia. Normal adults who have exposure to sunlight are assumed to get enough vitamin D without considering dietary sources. Adults who are screened from the sun require vitamin D in the diet. Intestinal absorption is enhanced by the presence of bile salts and dietary lipids (19).

Many drugs have been shown to interfere with absorption and metabolism of vitamin D. The drugs are corticosteroids, diphosphonates, anticonvulsants, antacids, laxatives, and certain sedatives such as gluthethimide. Mineral oil and phenolphthalein used as laxatives have caused clinical signs of vitamin D deficiency in adults and children. Aluminum hydroxide, anticonvulsants, and gluthethimide also have caused development of rickets or osteomalacia (19).

Drugs and other foreign compounds which induce the drug metabolizing microsomal enzymes in the liver cause an accelerated degradation of vitamin D_3 and 25-HCC. Some of the drugs which cause increased activity of the drug metabolizing microsomal enzymes are sedatives, oral antidiabetic agents, muscle relaxants and anticonvulsants (19).

Four drugs which have been shown to accelerate the degradation of 25-HCC severe enough to cause rickets or osteomalacia are phenobarbitol, primidone, gluthethimide and diphenylhydantoin. Diphosphonates have been shown to block production of 1,25-DHCC in the kidney and induce osteomalacia (19).

The major occurrence of drug-induced deficiency of vitamin D is found within institutionalized groups of patients who are kept indoors and may not be getting adequate vitamin D in the diet while on anticonvulsant drug therapy (19).

Osteomalacia is found in adults and has symptoms of pain localized to the back and thighs. It may also include the shoulder region or ribs. Deformities may occur of the long bones, pelvis and thorax. Osteomalacia may be suspected in drug using persons who have symptoms of bone pain, weakness of the proximal limb muscles and progressive difficulty in walking. Biochemical findings include elevated serum alkaline phosphatase and changes in calcium and phosphorus levels in the serum (19).

Vitamin K is important for normal blood coagulation. The following clotting factors are vitamin K dependent: Stuart-Prower factor (x), Christmas factor (IX), proconvertin (VII), and prothrombin (II). A deficiency of vitamin K in man has not been produced by a vitamin K free diet. The assumption is that the human needs for vitamin K can be supplied by the intestinal synthesis of vitamin K_2 (19).

A synthesized form of vitamin K_3 is used therapeutically. The chemical structure of vitamin K_3 is closely related to menaquinone. Vitamin K_1 is found in green leafy vegetables such as spinach, kale, cabbage and collard greens. Vitamin K is found in large amounts in pork liver (19).

Broad spectrum antibiotics can decrease vitamin K_2 synthesis in the intestine. Mineral oil and the bile acid sequestrant, cholestyramine can decrease vitamin K absorption. The coumarin anticoagulants can cause vitamin K deficiency. Aspirin and other salicylates can induce hypoprothrombinemia (19).

Vitamin A occurs naturally as retinol (vitamin A_1 alcohol), dehydroretinol (vitamin A_2 alcohol) or aldehyde derivative of these forms. The most valuable precursor of vitamin A is B-carotent. A dietary

deficiency is most likely to occur when the diet is mainly cereal sources lacking carotene sources and milk, liver and fish (19).

The presence of dietary fat is important for optimal absorption of vitamin A. Drugs can interfere with vitamin A absorption, mineral oil, neomycin, cholestyramine and alcohol. Neomycin inhibits pancreatic lipase, may inactivate bile salts and may cause mucosal damage. Cholestyramine interferes with vitamin A absorption by absorbing bile salts (19).

Vitamin A is necessary for specific visual function. Night blindness is an early sign of vitamin A deficiency. It is also necessary for stabilization of cellular and intracellular membranes, synthesis of normal steroid metabolites and synthesis of mucopolysaccharides (19).

Two major functions of vitamin A which are retinol dependent are the visual cycle and spermatogenesis. The conversion of retinol to retinal occurs through an oxidative process requiring ADH (alcohol dehydrogenase) and NAD. Conversion of retinol to retinal can be impaired by competitive utilization of ADH. Ethanol has a high affinity for the enzyme alcohol dehydrogenase. Alcoholics may fail to convert retinol to retinal. This may provide a mechanism to explain a common cause of male sterility in chronic alcoholics. It also explains why alcoholics may become night blind. Other contributing factors to vitamin A deficiency in alcoholics may also include poor food intake of the vitamin and malabsorption of the vitamin (19).

Vitamin E is present in the diet in vegetable oils, nuts, eggs, liver, wheat germ oil and margarine. Tocopherols are considered as dietary vitamin E. Gamma-tocopherol is found in soybean oil. Alpha-tocopherol is also present in the diet (19).

When fat absorption is affected, the absorption of a-cocopherol is also affected. Vitamin E functions as a lipid antioxidant, it prevents the production of toxic lipid peroxides in the tissues. Vitamin E has a highly specific function in many tissues. Activity of the selenium-gluthathione peroxidase system is related to inhibition of lipid peroxides. Cell components are protected from the toxic effects of peroxide by selenium-gluthathione peroxidase which breaks lipid peroxides down to hydroxy fatty acids and stops a further breakdown of peroxides to free radicals which can then again initiate peroxidation. Vitamin E works with the selenium-gluthathione peroxidase system to provide a chain breaking effect against peroxide formation (19).

Patients studied who were given the drug clofibrate showed a reduction in serum lipids and serum vitamin E levels. When the drugs were withdrawn, vitamin E returned to pre-treatment levels. A coagulation defect has been reported with patients on warfarin who are also taking moderately high doses of vitamin E and clofibrate. It is suggested that vitamin E may interfere with vitamin K utilization. Studies with laboratory animals show a prolonged prothrombin time and a hemorrhagic condition with hypervitaminosis E. These effects can be reversed by administration of vitamin K (19).

Levels of total tocopherol in the body are related to total lipid content of the serum and to levels of certain lipid fractions. Serum tocopherol levels are high if total serum lipids are high (19).

Minerals

Iron is present in meats and other animal proteins as heme iron. Iron added to cereals is usually reduced iron, but in some instances

ferrous sulfate is used. Heme iron is absorbed better than other food sources. Ferrous iron sources are absorbed better than ferric salts or ferric complexes. Absorption of non-heme iron in the intestine is improved in the presence of ascorbic acid, fructose, sorbitol, meats and certain organic acids (lactic, citric, pyruvic and succinic). Some amino acids enhance iron absorption. Phosphate and phytates depress iron absorption (19).

Some drugs depress iron absorption in man and animals. Bicarbonate depresses absorption of iron in guinea pigs. This may be of importance to patients who take quantities of bicarbonate as an antacid. Roe (19) found that tetracycline caused an inhibition of protein synthesis in the intestine. It has been suggested that alterated protein synthesis is associated with impaired iron uptake. One investigator (19) showed that tetracycline depressed the intestinal mucosa uptake of radioiron in rats.

Inorganic iron preparations have been shown to depress blood levels of some antibiotics such as doxycycline, methacycline, tetracycline and oxytetracycline. Roe (19) showed that cholestyramine binds both heme iron and inorganic iron in vitro and in rats the drug impairs the absorption of inorganic iron. He also found that there were decreased non-heme iron stores in rats on long term treatment with cholestyramine.

Aspirin or other salicylates are considered to be a prominent cause of iron deficiency anemia. If the aspirin taken amounts to 1 to 3 gms per day, bleeding in the gastrointestinal tract occurs in about 70 per cent of normal subjects. In pre-existing gastrointestinal diseases such as alcoholic gastritis, esophageal varices or peptic ulcer, aspirin may cause gastric hemorrhage in some patients (19).

Once iron is absorbed from the gastrointestinal tract, it is transported bound to transferrin in the plasma. Estrogens or contraceptive steroids increase the levels of transferrin in the plasma. One study has shown that oral contraceptives increase iron absorption. It has been postulated that this may occur because of high iron transferrin concentrations (19).

Isoniazid (INH), a vitamin B_6 antagonist, may impair the uptake of iron and cause sideroblastic anemia (19).

Zinc is found in the diet in animal protein foods. Women receiving contraceptive steroids have been shown to have lower zinc plasma levels. There have not been studies to show women are zinc deficient when taking contraceptives (19).

Corticosteroids have an effect on zinc status by increasing zinc renal losses (19).

Malabsorption of zinc occurs with persons having a high level of phytate in the diet--a diet consisting primarily of cereal grains such as in countries like Iran and Egypt (19).

Zinc plays an important role in the body in several processes. It is important for certain enzymes necessary for cellular oxidative processes, for normal protein synthesis, in metabolism of vitamin A. Zinc deficiency creates some syndromes which are not well explained. Zinc deficiency is believed to be involved in gonodal immaturity, dwarfism, and anemia, and is found in Iranian and Egyptian boys and girls on a high phytate diet. One investigator (19) has suggested growth retardation in malabsorption conditions such as cystic fibrosis may be due to zinc deficiency.

Evidence of vitamin A deficiency in alcoholics may be related to an impaired activity of alcohol dehydrogenase due to zinc deficiency (19).

Magnesium excretion is increased with the use of some diuretics including ammonium chloride, mercurial diuretics, chlorothiazide and hydrochlorothiazide. Roe (19) found that magnesium depletion caused an increased sensitivity to digoxin induced arythmias.

Low blood magnesium levels have been associated with malabsorption which may be drug induced (19).

Magnesium is an important activator of ATPase, enzymes associated with ribosomal protein synthesis and coenzyme A (19).

Chronic alcoholics develop hypomagnesemia due to poor diet and hyperexcretion of magnesium in the urine. Magnesium deficiency is characterized by seizures, tetany, muscle weakness, ataxia, tremors and behavioral disturbances (19).

Appetite Suppression Due to Drugs

Regulation of appetite is a complex process involving peripheral, neural, and metabolic components. Neural mechanisms include a "feeding center" in the lateral hypothalamus and a "satiety center" in the ventromedial hypothalamus. Catecholamines in the brain seem to serve as neurotransmitters in the regulation of appetite (21).

Some drugs impair appetite due to their central nervous system effects. Digitalis, phenformin and amphetamines fall in this category. Some drugs have an anticholinesterase property. They cause dryness of the mouth and impair taste sensation. Drugs which fall in this group are anticholinergics, phenothiazine tranquilizers, tricyclic

antidepressants and all antihistamines. Some drugs have gastic irritative properties that cause anorexia and nausea. These include potassium salts and iron, salicylates, theophyllin and antibiotics such as tetracycline and erythromycin (22).

Some illnesses alter taste sensitivity. Carson and Formican (20) reviewed the literature concerning altered taste due to disease-medication relationships. They have compiled the following tables (Tables VII and VIII).

Drug Problems in the Geriatric Patient

Advancing age modifies the drug response in man because renal function is lessened and active and passive absorption may be impaired due to lessened acid production in the stomach (23) (25). Ward and Hannah (26) state that the aging process is only partially understood. However, certain conditions are commonly encountered in the elderly, such as cardiovascular disease, atherosclerosis, osteoporosis, and hormonal imbalances. When there is a decline in renal function, drug overdose is likely to occur.

Nine drugs which can cause special problems for the elderly are: analgesics, antibiotics, anticoagulants, atropine, digitalis, diuretics, laxatives, sedatives and tranquilizers (26). Many of these are routinely used for the geriatric patient.

Elderly patients with Parkinson's disease are often given the drug levodopa. Treatment of Parkinson's disease with levodopa was proposed when it was found the dopamine content in portions of the brain was lower than normal in patients with the disease (27). Levodopa rather than dopamine is given orally, since levodopa crosses the blood-brain

TABLE VII

MEDICAL PROBLEMS AFFECTING TASTE SENSITIVITY

Disorder	Effect	Reference
Endocrine disorders		
Adrenal cortical insufficiency	Increased detection sensitivity	22
Turner's syndrome (chromatin negative gonadal dysgenesis)	Decreased sour and bitter sensitivity	21
Pseudohypoparathyroidism	Decreased sour and bitter sensitivity	22
Diabetes	Decreased sweet sensitivity with high blood sugar	23,24
	Not correlated with high blood sugar	25
Other diseases		
Cancer	Decreased sweet and salt sensitivity	26-28
	Altered bitter sensitivity	26,27
Familial dysautonomia	Decreased sensitivity	29
Aglycogenesia	Inability to recognize sweet	30
Renal failure	Decreased sweet, salt, and sour sensitivity	31
Sjogren's syndrome	Decreased sensitivity, especially for bitter, salt and sour	32
Cystic fibrosis	Increased sensitivity	33
Direct nerve damage		
Tongue lesions	Decreased detection of salt and sweet	34
Facial paralysis	Decreased sensitivity	35
Trauma		
Gunshot wounds	Decreased sensitivity	36
Head injuries	Decreased sensitivity	37
Burns	Decreased sensitivity	37

Source: J. S. Carson and A. Gormicane, Disease-Medication Relationship in Altered Taste Sensitivity, J. Am. Dietet. A. (1976).

TABLE VIII

EFFECTS OF MEDICATIONS ON TASTE SENSITIVITY

Drug	Effect	Reference
Amphetamines	Decreased sweet sensitivity in some; differs with individuals	38
	Increased bitter sensitivity	39
Anesthetics		
Cocaine	Decreased sensitivity, especially sweet and bitter	38
Eucaine	Decreased bitter and sweet sensitivity	38
Amydricaine	Decreased bitter and sweet sensitivity	38
Amylocaine	With high intake, loss of salt detection, decreased bitter sensitivity	38
Isococaine and tropacocaine	Decreased sweet sensitivity	38
Benzocaine	Decreased sour sensitivity	38
Amethocaine	Increased bitter sensitivity; decreased sweet sensitivity	38
Lignocaine	Decreased salt and sweet sensitivity	38
Acetyl sulfosalicylic acid	Decreased sensitivity	40
Clofibrate	Decreased sensitivity	40
Dinitrophenol	Loss of salt taste; general hypogeusia	38
d-Penicillamine	General decrease in sensitivity	41
5-Fluorouracil	Some alterations in bitter and sour sensitivity; increased sweet sensitiivity	28
Griscofulvin	Decreased sensitivity	40
Insulin	With prolonged use, decreased sweet and salt sensitivity	40
Lithium carbonate	Strange, unpleasant taste	42
5-Mercaptopyridoxal	Altered taste	9
Phenindione	Decreased sensitivity	43
Phenytoin	Decreased sensitivity	44
Oxyfedrine	Decreased sensitivity	45

TABLE VIII (Continued)

Drug	Effect	Reference
Anti-thyroid agents		
Methimazole	Decreased sensitivity	46
Methylthiouracil	Decreased sensitivity	47

Source: Carson and Gormicane, J. Am. Dietet. A. (1976).

barrier more readily. Dopamine is formed in the presence of a decarboxylase enzyme by the decarboxylation of L-dopa.

Vitamin B_6, pridoxine, has been shown to interfere with levodopa. It is known that the decarboxylase enzyme is dependent upon pyridoxine for the conversion of L-dopa to dopamine (28). Pyridoxine in oral doses of 10 to 25 mg reverses the antiparkinson effects of levodopa, therefore, multivitamin preparations or foods fortified with pyridoxine should not be given to patients on L-dopa therapy (28) (29). Foods particularly high in pyridoxine such as beef liver, pork, beans, oatmeal, sweet potatoes, bacon, and pyridoxine fortified breakfast cereals should be avoided. Some investigators feel the pyridoxine available in the standard daily diet is not likely to interfere with L-dopa activity (29).

Some drugs used in the treatment of depression block the activity of the enzyme monoamine oxidase in the body. Monoamine oxidase is present in the liver and gastrointestinal tract of man and the enzyme is responsible for detoxifying tyramine and other pressor amines ingested in foods. Two drugs which are monoamine oxidase inhibitors are phenelzine and tranylcypromine. These drugs block the activity of the enzyme monoamine oxidase and allow tyramine to reach the circulation. The result may be a potentially fatal hypertensive crisis. The severity of the reaction is dependent upon the amount of tyramine or other pressor agents consumed. Six milligrams of tyramine may increase blood pressure and 25 mg may produce severe hypertension. Certain cheeses, chicken livers, herring and some wines are particularly high in tyramine (30) (31).

Hall (24) suggests the following simple rules to observe in prescribing drugs for the elderly:

1. Know the pharmacological action of the drugs being used and in particular how it is metabolized and excreted.
2. Use the lowest dose that is effective in the individual patient.
3. Use the fewest drugs the patient needs.
4. Do not use drugs to treat symptoms without first discovering the cause of the symptoms.
5. Do not withhold drugs on account of old age when drug therapy may improve the old person's quality of life.
6. Do not use a drug if the symptoms it causes are worse than those it is supposed to relieve.
7. Do not continue to use a drug if it is no longer necessary. It is wise to review repeat prescriptions quarterly in all elderly patients (p. 582).

CHAPTER III

PROCEDURE

This research was stimulated by an awareness of increasing reports in the literature of drug-nutrient interactions. The geriatric patient has been particularly susceptible to drug-nutrient interactions because of factors having to do with the total health care and long term institutionalization. Through dietary consultation for a number of years in long term care facilities, personal observations regarding the problems of the elderly patient has further stimulated this interest.

Selection of Subjects

Five administrators of nursing homes were contacted for permission to study charts and interview patients in each facility. Of the five facilities chosen, three were 100 bed facilities, one was a 50 bed facility, and one had a bed capacity of 60 patients. All facilities were located in Oklahoma City, Oklahoma. The particular facilities in this study were chosen because of a previous knowledge of the facility and acquaintance with the administrator, by which it was felt permission would be obtained to conduct this study among the patients.

After discussion with a statistician at Oklahoma State University, it was decided to do a striated random sample of each wing in the particular facility being studied. This was due to the fact that one wing might contain bed patients who needed heavy nursing care, another

might contain patients who were up walking around and would require lighter nursing care. It was, therefore, felt a better sampling would be obtained by a straited random sample of each wing since the medical problems could differ. Three of the facilities had four wings and two facilities had three wings.

The subjects were selected by placing all the numbers of the patient beds in one wing in a container. The numbers were then drawn out of the container at random. It was decided to study approximately half of the patients in each wing. In facilities A and B, the number of patients studied would be approximately 50 each, or a total of 100 patients for the two facilities. In facilities C, D and E, the number of patients studied would be approximately 35 each, or 100 bed patients for the three facilities. The goal of this study would be to study approximately 200 patients in the five nursing homes. The number of patients studied in each wing of a particular facility is shown in Table IX. A total of 206 patients was chosen for the study.

Data Collection

A form was developed to collect the data from the patients (Appendix A). It was pre-tested in one facility among approximately 15 patients and found to be suitable for collecting the desired information. In the first part of the form the following information was collected from the patient's chart: patient's name, admission date, sex, height, weight, physician's name, diagnosis, age, physical limitations, diet order, supplements, where meal taken, written records of food intake and lab work significant to study. The second part of the form included medications given, date ordered, dosage and times of administration. The last

part of the form was a questionnaire regarding the specific foods the patient ate. The data was collected by personal interview with the patient. Observation of the patient was also written during the interview.

TABLE IX

NUMBER OF PATIENTS SELECTED IN EACH FACILITY

Facility	Wing				Total
	I	II	III	IV	
Facility A (100 beds)	15	9	13	16	53
Facility B (100 beds)	12	11	14	14	51
Facility C (50 beds)	10	12	10	None	32
Facility D (60 beds)	14	7	13	None	34
Facility E (100 beds)	10	7	10	9	36

Analysis of Data

The first objective in analyzing the data was to categorize each drug listed on the patient's chart. The purpose of this table was to show all drugs which were prescribed by the physician for all patients in the study. The chart is shown in Appendix B. The American Hospital Formulary Service (32) and the Physician's Desk Reference (33) were used

to place the drugs in the correct drug categories.

The second objective was to look at each drug listed in the chart and prepare a second chart showing the drugs which are reported in the literature as having drug-nutrient interactions. This chart is shown in Appendix C. The reference used to prepare the chart was the Handbook of Interactions of Selected Drugs With Nutritional Status in Man (34). The handbook format is based on the American Hospital Formulary Service.

The third objective was to relate the interactions found for specific drugs to the individual patient. A chart was prepared showing each patient by number and the following information: age, time in facility, weight gain or loss in one year's time, diagnosis, diet, number of drugs prescribed and drugs prescribed which have a drug-nutrient interaction reported in the literature. Data was collected for the study in June and July of 1976. In late May and early June of 1977, the weight of each patient studied was rechecked to note whether there had been change. The chart showing the information discussed above is shown in Appendix D.

The final goal of the analysis of data was to compare related factors such as patient's age, length of time on the drug and patient's eating habits, and to make recommendations for improvement in methods used for recording information in the patient's chart. Better methods of recording would benefit the patient because someone might become aware of a drug-nutrient interaction which would otherwise be missed.

CHAPTER IV

RESULTS AND DISCUSSION

Data collected from approximately 200 patient charts in five nursing homes was studied. One of the major goals of the study was to determine if the medications prescribed for the nursing home patient included some of the medications that have been reported in the literature to have drug-nutrient interactions. The other major goal was to assess the nutritional status of the patient by observing weight change during one year and by checking records of food intake of the patient to see if there was an obvious relationship between drugs having a drug-nutrient interaction and patient's food intake.

Drugs Prescribed

Each medication prescribed on the chart by a physician was recorded as written on the chart and placed in the proper drug category. Drug categories were determined by following the listings in the American Hospital Formulary Service (32) and the Physician's Desk Reference (33). The drugs prescribed by categories are shown in Table X. The highest number of medications prescribed were narcotic and non-narcotic analgesics. A total of 213 prescriptions for analgesics was prescribed for 206 patients. The next highest group of drugs prescribed was laxatives, 173 prescriptions for 206 patients. Some patients had more than one prescription for a laxative. A small number of patients had more than

TABLE X

NUMBER OF DRUGS PRESCRIBED IN FIVE NURSING HOMES
BY DRUG CATEGORIES IN RANK ORDER

Drug Categories	Facilities					Total
	A	B	C	D	E	
Total number of all drugs prescribed:						1299
Analgesics						213
Non-narcotic	48	51	45	27	22	
Narcotic	5	1	9	3	2	
Laxatives	63	38	28	19	25	173
Vitamin and Mineral Supplements	60	30	24	23	22	159
Tranquilizers						109
Major	14	22	7	11	11	
Minor	9	9	8	11	7	
Vasodilators	36	19	14	13	15	97
Sedatives (hypnotic)	15	16	16	6	10	63
Cardiotonics	12	17	8	12	12	61
Diuretics	11	15	13	9	11	59
Anti-hypertensive	3	6	10	8	15	42
Antacids	8	7	8	3	4	30
Antitussive	3	8	13	6	0	30
Stool Softeners	7	6	10	2	1	26
Antinauseants	6	5	6	2	1	20
Antibacterials	5	6	2	4	1	19
Antihistamines	6	4	3	1	2	16
Potassium Replacements	3	4	2	4	2	15
Antibiotics	6	4	0	1	5	14
Hypoglycemia Agents	2	4	4	1	2	13
Antidiarrheals	0	7	1	1	4	13
Antiarthritics	3	1	5	1	2	12
Antidepressants	1	8	0	0	2	11
Thyroid	5	3	1	0	1	10
Antiasthmatics	4	2	0	1	2	9
Anticonvulsants	4	0	1	1	3	9
Insulin	0	2	1	4	2	9
Androcortical Steroid	2	4	1	2	0	9
Hormonal	2	2	1	1	2	8
Anti-Parkinsonism Drugs	1	3	2	0	1	7
Anti-psychotic Drugs	0	2	1	2	2	7
Analeptic	1	2	2	0	1	6
Anti-arrhythmics	0	2	0	2	1	5
Anticoagulants	1	2	2	0	0	5
Uricosuric	0	2	1	0	1	4
Anticholinergic	1	1	1	0	0	3
Antimalarials	1	1	0	0	0	2
Digestant Aid	2	0	0	0	0	2
Anti-hyperlipemics	1	1	0	0	0	2
Antipruritic	0	1	0	0	1	2
Anti-inflammatory	0	0	1	0	0	1
Oral Contraceptives	1	0	0	0	0	1
Appetite Suppressant	1	0	0	0	0	1
Antineoplastics	0	0	1	0	0	1
Spirits of Fermenti	0	0	0	0	1	1

one type of analgesic prescribed. Vitamin and mineral supplements accounted for 159 prescriptions. Major and minor tranquilizers totaled 109; vasodilators, 97 prescriptions; and sedatives, cardiotonics and diuretics, approximately 60 prescriptions in each category. The total number of drugs prescribed for 206 patients was 1,299. This was calculated to be an average of 6.4 prescriptions per patient in the study. The actual number of drugs prescribed per patient is shown in Table XI. Twenty-nine patients had four drug prescriptions, 20 patients had six drug prescriptions, 19 patients had seven drug prescriptions, and 18 patients had eight drug prescriptions. The highest number of drugs prescribed to one patient was 22. The next highest number of drugs prescribed to one patient was 17. The majority of patients were in the range of three to eight drug prescriptions. Eight patients were prescribed two drugs, six were prescribed one drug and only one patient did not have any drugs ordered. Table IX shows the average patient in the nursing home is taking a large number of drugs. A complete listing of all drugs prescribed is shown in Appendix B. The number of drugs prescribed to each patient in the study is shown in Appendix D.

Many of the drug orders in the nursing homes studied were PRN (see abbreviations) orders. PRN orders in the facilities may have been given only rarely or they may have been a regular daily medication. Studying the PRN orders in detail would be another whole study. The number of PRN orders per patient ranged from one to ten. The average number of PRN orders was between two and six drugs. PRN orders are listed in parentheses for each patient in Appendix D.

TABLE IX

RANGE IN THE NUMBER OF DRUGS PRESCRIBED PER PATIENT

Number of Drugs per Patient	Facility					Total
	A	B	C	D	E	
0	0	0	0	1	0	1
1	3	1	0	1	1	6
2	2	3	1	2	0	8
3	3	4	2	7	1	17
4	11	3	3	5	7	29
5	5	7	4	4	6	26
6	6	7	2	5	1	21
7	5	5	4	1	4	19
8	5	5	3	3	2	18
9	5	0	3	2	1	11
10	2	0	1	2	2	7
11	2	2	2	1	2	9
12	3	3	2	0	0	8
13	2	3	3	1	0	9
17	0	1	0	0	0	1
22	0	0	1	0	0	1

Drugs With Reported Drug-Nutrient Interactions

The next step in the study was to see if a drug-nutrient interaction had been reported for that particular drug in the Handbook of Interactions of Selected Drugs With Nutritional Status in Man (34). Since this particular handbook is categorized with the same format as the American Hospital Formulary Service (32), it is not difficult to find the generic name of the drug. Accuracy in evaluation is possible, even though the drug name may not be familiar. The drugs which have a reported drug-nutrient interaction are shown in Appendix C. Each drug on the chart found to have a drug-nutrient interaction in the study is listed by drug category, trade name or generic name as listed on the patient's chart, and by the generic name and the number as shown in the American Hospital Formulary Service (32) and the Handbook of Interactions of Selected Drugs With Nutritional Status in Man (34).

Two drug categories which were shown to have drug-nutrient interactions are analgesics and laxatives. Both of these drug categories are prescribed to a large degree to the elderly patient. Other categories of drugs prescribed which have interactions were antacids, tranquilizers, antipsychotic drugs, sedatives, antidepressant drugs, diuretics, potassium supplements, antihypertensive drugs, cardiotonics, anticonvulsant drugs, anticoagulant drugs, antineoplastic drugs, vasodilators, antihistaminics, antiasthmatic drugs, antilipemia agents, anti-arthritic, uricosuric drugs, hormonal drugs, andrenocortical steroids, anti-parkinsonism drugs, antibiotics, antibacterials, analeptic drugs and hypoglycemia agents.

Levodopa and drugs which are monoamine oxidase inhibitors were found to be prescribed for a small number of patients. Only one drug order was for a monoamine oxidase inhibitor and only three drug orders were for levodopa. In each instance, there was no indication in the diet order of possible interaction with certain foods.

Vitamin and mineral supplements were sometimes prescribed in large doses. Examples were vitamin C, 250 mg, 500 mg, 600 mg, or 750 mg per day; vitamin D, 50,000 I.U. per day, iron, 325 to 500 mg per day. In the review of literature, interactions with other nutrients when excessively large amounts of certain vitamins are ingested was discussed. The RDA (36) (see abbreviations) for vitamin C is 45 mg per day, a drug order for 600 mg per day is 13 times the RDA. The RDA for vitamin D is 400 I.U. (see abbreviations) and the RDA for iron is 10 mg for older men and women. Large amounts of iron medication may cause nausea and diarrhea in some patients.

Individual Patient Comparisons

A table (Appendix D) was compiled which showed each individual patient and the drugs each received reported to have a drug-nutrient interaction. In order to make comparisons, the table also shows the age of the patient, the number of years in that particular facility, weight gain or loss in one year, the diagnosis of the patient, the diet preparation, the number of drugs ordered and the number of drugs on each patient which were PRN orders. The PRN orders are shown on the table in parentheses following the total number of drugs ordered. Almost every patient evaluated received at least one drug which had a reported drug-nutrient interaction. Many of the patients were receiving

two, three or more drugs which were shown to have an interaction. Some patients showed as high as six or seven of the drugs they were taking had reported interactions.

Weight

Comparisons of weight were made on each patient between June, 1976 and June, 1977. The object was to note weight gain or loss within one year. It was not possible to obtain a weight for each patient in this study. Many of the charts were marked "unable to weight." In discussing this with the Directors of Nursing, each stated it was not possible to weigh bed patients or wheel chair patients. When weights were rechecked in June, 1977, two facilities had obtained methods to weigh bed and wheelchair patients. Facility C had purchased an instrument which looked like a flat piece of plywood to which two bathroom scales had been attached on the underneath side. One scale was attached to each end of the piece of plywood. The piece of plywood was large enough to roll a wheelchair upon it. A reading was taken on each scale and the two figures were added together for the total. The weight of the wheelchair was then subtracted. The Director of Nursing in this facility maintained all bed patients could be weighed in a wheelchair. Weights were actually being done once a month for these patients and recorded. For this facility, weight changes are shown for three months for patients who were originally "unable to weight." In facility B, scales had been purchased which could be attached to a patient lift. This, however, did not seem to be as convenient because it was still not being used on a regular basis. If a follow-up weight check was not possible because the patient had moved from the facility to another facility or had expired, it is

marked on the table with "M" or "Exp." Of the total number of patients studied, it was possible to obtain weights on 119 (or 58 per cent). Even though weight loss did appear on some patients (refer to Appendix D) who were taking drugs with a reported drug-nutrient interaction, it would be very difficult to make a statement as to cause and effect without lab work and without knowing something more about the food intake of the patient.

Food Intake Records

Food intake records were difficult to find. In an attempt to study food intake in relation to drug-nutrient interactions, a questionnaire was drawn up for a personal interview with the patient. In facility A, all 53 patients were visited. Much of the information obtained was questionable. The Director of Nursing was asked which patients could give reliable information. In each facility, when the list of patients was reviewed with the Director of Nursing, very few patients were considered able to give reliable information. Memory was a problem in trying to obtain reliable data even if the patient was oriented to the present. It was decided the only accurate food intake information would have to be obtained from the charts. This, too, proved to be disappointing. Food intakes were recorded as poor, fair, and good by the nursing staff, but recording on the chart was not done until sometime after the meal. Again, the information was questionable as to the memory of the aide or nurse charting on the nurses notes. Actual food intake records (by checking the patient's tray at mealtime) were kept in three facilities. These were, however, kept on a sporadic basis. The food service supervisor stated she recorded the information if she had the time. She

also may have recorded only the meals while she was on duty. The recording was usually by percentage of the total meal served. The patient ate 100 per cent, 75 per cent, 50 per cent, etc. of the total amount served. While this did not identify which foods were eaten, it did tell something as to the amount. One of the excellent factors was the fact that in each facility, the food service supervisor was a HIEFSS (see abbreviations) member. The importance of a proper menu being served and the patient eating the food was well understood by the food service supervisor. Another positive factor was that each facility studied had a dietary consultant who was a registered dietitian. The menu in each facility was adequately written and portions served were correct in size to meet nutritional needs. In each case, the food service supervisor was very interested in the patient. The best information about how a patient was eating was verbal from the trained food service supervisor. She did observe, even though it was not recorded, and seemed to know who was eating well and who was not. In all five facilities studied, the available data in writing was so poor that it was not possible to calculate the exact nutrients received by the patient.

Time of Administration of Drugs

The relationship of time of administration of drugs to meals seemed important in view of the many drugs which show interactions such as anorexia, nausea, and vomiting. Drugs were not passed at the same time in all facilities. Table XII shows times of administration of drugs.

Meal hours in facilities A, B, C and E were breakfast at 7:30 a.m., dinner, 12:00 noon, and supper at 5:30 p.m. In facility D, the five meal plan was used and the meal hours were 7:00 a.m., 10:00 a.m., 1:00

p.m., 4:00 p.m. and 7:00 p.m. In each facility, passing drugs involved a period of time--approximately one to one and one-half hours was necessary to pass all medications ordered. This meant that 8:00 a.m. medications were begun at 7:30 a.m. and finished at 8:30 or 9:00 a.m. The Directors of Nursing stated they sometimes received the medication before the meal, during the meal or after the meal. They stated they were not concerned with the meal hours in relation to medications unless it was stated on the label of the prescription by the pharmacist. In facility D, the meals were always before medication, no one received medication on an empty stomach unless so stated by the pharmacist.

TABLE XII

TIME OF ADMINISTRATION OF DRUGS

Facility	b.i.d.	t.i.d.	q.i.d.
A	8-8	8-12-8	8-12-4-8
B	8-8	8-12-8	8-12-4-8
C	8-8	8-12-5	8-12-5-8
D	8-8	8-12-4	8-12-4-8
E	8-8	9-1-5	9-1-5-9

Length of Time on Drug

The length of time a patient was on a drug could only be evaluated by the date of the drug order on the chart. In most cases, little

change occurred in the drugs ordered or in dosages given from the time a patient was admitted to the facility. The exception was for antibiotics which were always given for short terms. If a patient was having side effects from a drug, the prescription was reviewed by the pharmacist and the physician.

Other Related Factors

Of the 206 patients in the study, 65 were male (31.5 per cent) and 141 (68.5 per cent) were female. All patients were Caucasian with the exception of one Negro patient. Each facility had a house physician which saw one-third to one-half of the patients in the facility. In facility A, 16 different physicians saw the 53 patients studied (House Physician 29); in facility B, 27 different physicians saw the 51 patients studied (House Physician 10); in facility C, 10 different physicians saw the 32 patients studied (House Physician 23); in facility D, four different physicians saw 32 patients studied (House Physician 29) and in facility E, 14 physicians saw the 36 patients studied (House Phsyician 21).

Many of the charts did not show the height of the patient; 32 charts (or 16 per cent) showed a record of the patient's height, 174 (or 85 per cent) did not have a record of the patient's height. In discussing this with the Director of Nursing in each facility, it was stated they did not make an effort to get a patient's height upon admission. If the transfer sheet that came with the patient, or the physical examination which was completed by the physician, did not have a recorded height, it was not done.

The age of most patients in the study was between 75 and 100 years. Seventy per cent, or 145 patients, were in this age group. Table XIII shows the age of the patients in the study and the percentage in each age group.

TABLE XIII

NUMBER AND PER CENT OF PATIENTS IN STUDY BY AGE

Patient Age	Facility					Total	%
	A	B	C	D	E		
20-64	9	2	2	6	8	27	13.2
65-69	3	3	2	3	2	13	6.4
70-74	5	4	1	7	4	21	10.2
75-79	3	5	5	9	4	26	12.6
80-84	7	13	6	4	10	40	19.4
85-89	12	12	10	5	4	43	20.8
90+	14	12	6	0	4	36	17.4
Total						206	100.0

The length of stay in a facility ranged from one year to 13 years. The average length of stay was from five to six years. The time a patient was on a medication could be stuied only in relationship to the length of stay in the particular facility studied. The length of time each patient was in a particular facility is shown in Appendix D. It must be noted that facility E was a new facility which opened in June

of 1976 so it was not possible to record an accurate long-term length of stay in this facility.

Laboratory data was limited for the patients studied in relation to identifying a drug-nutrient interaction. Lab work is ordered by the physician for the patient. Because long-term care facilities are not intended for diagnostic centers but rather for care and treatment, lab work is usually ordered when a problem arises. Urine analysis may be ordered for a suspected bladder or kidney infection. Blood work may be ordered when it is important for treatment of the patient. It was often possible to find a hemoglobin and hematocrit on the chart included with blood work. But, very little was present in regard to nutrient levels in the blood.

Medically defined conditions in the patients studied are shown in Table XIV. It was interesting to note that 76 patients had a diagnosis of Arteriosclerotic Heart Disease and 61 patients had a diagnosis of Arteriosclerosis. Many of the other diagnoses reported were related to these two conditions. Other conditions such as Parkinson's disease, osteomyelitis and rheumatoid arthritis were a small percentage of the total number of conditions. Diagnosis as listed on the chart for each patient studied is shown in Appendix D.

The tables and discussion in this chapter present some of the factors which can play a part in a drug-nutrient interaction for a patient.

TABLE XIV

MEDICALLY DEFINED CONDITIONS IN FIVE NURSING HOMES IN RANK ORDER

Medically Defined Condition	Facility					Total
	A	B	C	D	E	
Arteriosclerotic heart disease	20	23	9	15	9	76
Arteriosclerosis	24	10	10	5	12	61
Organic brain syndrome	4	9	4	13	10	40
Fractures	9	12	5	2	2	30
Hypertension	4	3	4	8	9	28
Cerebrovascular accident	10	5	4	2	3	24
Arthritis	9	6	3	2	3	23
Diabetes mellitus	3	6	4	4	5	22
Osteoarthritis	9	3	0	4	4	20
Mental retardation	6	0	1	2	5	14
Congestive heart failure	1	1	2	4	5	13
Cerebral arteriosclerosis	1	6	5	0	1	13
Osteoporosis	7	1	1	4	0	13
Chronic obstructive pulmonary disease	1	0	0	7	0	8
Parkinson's disease	2	3	1	0	1	7
Cancer	3	0	2	2	0	7
Psychotic	3	1	1	0	1	6
Schizophrenia	3	1	0	1	1	6
Anemia	2	1	1	1	0	5
Rheumatoid arthritis	1	1	0	2	1	5
Cardiovasulcar insufficiency	3	0	0	1	0	4
Osteomyelitis	0	1	1	1	0	3
Total number of conditions						428

CHAPTER V

SUMMARY AND RECOMMENDATIONS

In this research an attempt was made to show some of the factors present with the older individual which may make him or her more at risk in the area of a drug-nutrient interaction. To identify a drug-nutrient interaction in the older individual is a challenge because the older individual generally already has many physical problems and the appearance of the older individual may make it hard to spot change. The emotional state is often poor if the individual is neglected by the family who may have placed him or her in the institution. To identify a drug-nutrient interaction, the nurse, dietitian, pharmacist and the physician must be aware of physical changes in the older person. Two changes that should always be questioned are a dramatic weight change from normal and a definite change in termperament from normal.

It is important that correct heights and weights be kept on the charts for each patient. This study shows this is a weak area in the institutions. The importance of a height and weight is to provide some objective physical data by which to evaluate the patient. Taking a height is a one-time task and is not time consuming. A tape measure can be used if the person is unable to stand. Sometimes it is not always obvious by looking at someone whether or not there has been change. Often it goes unnoticed in the patient in which the change is gradual and the personnel seeing the individual daily are not aware the change

is occurring.

This study shows that many institutionalized individuals are on a large number of drugs. While data is available concerning mode of action and safety of a single drug, comparable data is not available when a variety of drugs are given to the same patient. In a study conducted by the Department of Health, Education and Welfare on Physician's Drug Prescribing Patterns in Skilled Nursing Facilities (35) it was found an average of 6.1 drugs per patient were prescribed. The data for the study was drawn from a sample of 288 nursing homes and 3,458 patients across the United States. The range in number of prescriptions per patient was 0 to 23. This data closely parallels this study in which the average number of drugs prescribed was 6.4 per patient and the range in number of drugs was from 0 to 22 for the individual patient. The study completed by HEW and published in 1976 states:

> Patients in skilled nursing facilities tended to have more prescriptions for drugs that alleviate symptoms of illness than for drugs which might have a direct therapeutic action on the underlying causes of diseases (35, p. 7).

One of the points covered in the review of literature is that the drug metabolizing enzymes are affected by lack of the correct nutrients in the body. This in turn affects how the body handles a particular drug or a group of drugs. The danger that is pointed out in the review of literature is in the individual who has many drugs prescribed for a long period of time and is not eating well in order to obtain the correct nutrients for the body's needs. It is sometimes thought that this can easily be remedied by simply taking a vitamin pill. A vitamin pill ordinarily contains at the most 10 nutrients and there are now 52 known nutrients found in food. Vitamin and mineral supplements in large doses

must be classified as drugs. It is the correct combination of foods that provide all the needed nutrients for the body. It is not difficult to meet the RDA for a nutrient with foods and a patient receiving the needed nutrients in the body is more nearly protected from a drug-nutrient interaction. With the trained food service supervisor and the registered dietitian in the health facility, proper menu planning and portion size is present to provide the nutrients for the patient in the daily diet. It is, however, the amount of those nutrients that the patient actually takes into the body that is important rather than what is perfectly planned and served. When a meal is not eaten, no drastic change occurs to signal someone and only by keeping records at mealtime can it be known what is happening with the patient and his food intake.

Recommendations from this study are:

1. proper recording of heights and weights,
2. charting of food intake on all patients, and
3. through further in-service education, an awareness be created of possible drug-nutrient interactions among all health care personnel.

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APPENDIXES

APPENDIX A

QUESTIONNAIRE USED FOR COLLECTION OF DATA

INFORMATION FROM THE CHART

Patient's Name _____ Admission No. _____

Admission Date _____ Sex _____ Birthdate _____

Physician's Name _____ Height_____ Weight _____

Diagnosis from chart (physical and mental) _____

_____

_____

Physical limitations (amputee, paralysis, wheelchair or bed patient)____

_____

Diet order _____

Supplements _____

Where meals taken _____

Record of food intake:

Lab work significant to study:

Patient's Name _____

Date Ordered	Medication	Dosage	Time Administered

Description of Patient (physical signs and symptoms--activity of patient)

PATIENT INTERVIEW

Name _____ Room _____ Facility _____

Food Intake:

Milk: Do you drink milk? ____ How much? _____
Have you always drank milk? _____ Do you eat cheese, ice cream, etc.? _____

Meat: Do you eat all meat served on trays? _____
Do you have trouble chewing meat? _____
Do you have trouble swallowing? _____

Fruits and Vegetables: Do you like fruits and vegetables? _____
Dislike: _____
Do you drink orange juice daily? _____
Can you eat fresh vegetables and fruit raw? _____

Breads and Cereals: Do you eat cereal for breakfast? _____
Kind: _____
Do you eat bread? _____ White or whole wheat _____

Fluids: Do you drink water? _____ How much? _____

Snacks: Do you eat foods or fluids between meals? _____
Does your family bring you food? _____
What items? _____
Does your food taste good to you? _____
What is wrong with it? _____

What would be your chief complaint about the food served to you in this facility? _____
_____
_____

APPENDIX B

LISTING OF DRUGS IN USE

	Facility				
	A	B	C	D	E
Vitamin and Mineral Supplements					
Vitamin C, 250 mg	1	0	0	1	0
Vitamin C, 500 mg	3	1	1	0	1
Albee with C	2	2	1	0	1
Theragran M	4	3	0	4	4
Theragran Liq	0	2	0	0	0
Theragran Hematinic	2	0	0	0	1
Stuarts Hematinic	1	0	0	0	0
Ferrous Sulfate, 325 mg	4	1	1	3	2
Feosol Spansules	0	0	0	1	1
FeSoy	0	1	0	0	0
Ferro Grad 500	8	0	0	0	0
Ferrolip 50	0	0	1	0	0
Feosol Elixir	0	0	0	1	0
Ferrous Gluconate	0	1	0	0	0
Ferro Folic	0	1	0	0	0
Fergon	0	0	0	0	1
Inferon (Iron Inj.)	1	0	0	0	0
Multiple Vitamin	7	4	2	3	0
Deca Vi Sol	1	0	0	0	0
Abdec Vitamins	1	0	0	0	0
Surbex T	3	2	0	1	0
Surbex	0	1	0	0	0
Myadec	0	0	1	0	2
Stress Tabs	4	0	0	0	2
Riboflavin	1	0	0	0	0
LiVitamin Tabs	0	0	1	0	0
Trinsicon	3	0	2	0	1
Gevrabon	1	2	0	1	1
Vitamin E, 100 I.U.	0	0	0	1	0
Myade C	0	0	1	0	0
Geritol	0	0	1	0	0
Yeast Tabs	0	0	1	0	0
Liquid Calcium	1	0	0	0	0
Ca Gluconate	0	2	2	0	0
Vi-Daylin	0	0	2	0	0
Stress Caps 600	1	0	0	0	0
Folbison	1	0	0	0	0
Poly-vi-sol	1	0	0	0	0
Thera Combex	0	1	0	0	0
Mi-Cebrin T	0	1	1	1	0
Vitamin D, 50,000 I.U.	0	0	1	0	0
Vitamin (kind not specified)	3	1	0	0	0
One-a-Day Vitamin	0	1	0	0	0
ViCon Forte	0	0	0	1	0
Aquasol A	0	0	0	1	0
DiCal D	0	0	0	1	0
Geriflex	0	0	0	0	1
Oscal	0	0	0	0	1
Unicap	0	0	2	0	0
Nicotinic Acid	1	0	0	0	0
Pyridoxine	0	0	0	0	1

	Facility				
	A	B	C	D	E
Vitamin and Mineral Supplements (Continued)					
B-Complex	0	1	0	0	0
B_{12} IM	5	2	2	2	0
Nicobid	0	0	0	1	0
Sodium Chloride Tabs	0	0	0	0	1
Sodium Flouride	0	0	1	0	1
Laxatives					
Nature's Remedy	1	0	0	0	0
Solace Syrup	1	0	0	0	0
Colace	4	0	1	3	1
Pericolace Cap	0	4	0	0	0
Laxative of Choice	9	12	6	1	13
Ducolax Supp	4	2	1	1	1
Ducolax Tabs	5	0	0	0	0
Enema	7	0	1	0	0
Fleet's Enema	1	4	0	1	0
Dorbantyl Forte	0	0	0	2	1
Milk of Magnesia	21	10	16	8	3
Alophen Tab	1	0	0	0	0
Doxidan	2	0	1	0	1
Dorbane	0	1	0	1	1
Feen-a-Mint	0	0	0	0	1
Anavac	2	0	0	0	1
Cascara Sagrada	2	0	0	0	0
Mineral Oil	1	1	0	1	0
Modane	2	4	1	1	2
Anti-Diarrheals					
Lomotil	0	6	1	1	3
Kaopectate	0	1	0	0	1
Stool Softeners					
Doxidan	0	1	3	0	0
Surfax	3	0	1	2	0
Metamucil	2	0	2	0	1
Dioctyl Sodium Sulfoc	0	0	1	0	0
Dialose	2	3	1	0	0
Dialose Plus	0	1	1	0	0
Milkinol	0	0	1	0	0
Glysennid	0	1	0	0	0
Analgesics (Non-narcotic)					
Aspirin	0	1	0	1	1
Tylenol	22	16	3	10	7
Anacin	0	1	0	1	1
ASA	13	14	15	7	11
ASA Supp	0	1	0	0	0
Darvocet N 100	3	1	7	1	0
Darvocet N	1	2	2	0	0
Darvon and ASA	1	0	0	0	0
Darvon	2	5	1	1	0
Darvon Cmp 65	3	4	7	2	0
Bufferin	2	2	3	0	0

	Facility				
	A	B	C	D	E
Analgesics (Non-narcotic) (Continued)					
Pabalate	0	0	0	1	0
Ascriptin (ASA with Maalox)	1	2	1	0	1
Norgesic Tabs	0	0	2	0	0
Phenaphen	0	0	1	1	1
Equagesic	0	1	0	0	0
Fiorinal	0	0	0	1	0
Empirin	0	1	2	0	0
Acetamerophen Supp	0	0	1	0	0
Riopan	0	0	0	1	0
Analgesics (Narcotic)					
Mepergan Fortis	0	0	1	0	0
Percodan	2	0	0	0	0
Demerol	1	0	1	0	0
Sparine	0	0	1	0	2
Tylenol #2	2	0	0	3	0
Tylenol #3	0	1	0	0	0
Empirin #3	0	0	6	0	0
Anticholinergic Agents					
Robinul	1	0	0	0	0
Antacids					
Pepto-Bismol	0	1	0	0	0
Gaviscon (appetite suppressant)	1	1	0	1	0
Mylanta	0	1	1	0	0
Mylicon	0	0	0	0	1
Aldurox	0	0	1	0	0
Maalox	3	2	1	0	0
Gelusil	0	1	0	1	1
Digel Liq	1	0	0	1	1
Donnagel	0	0	1	0	0
Donnatal	2	1	3	0	0
Kolantal Gel	0	0	1	0	0
Tums	0	0	0	0	1
Respiratory and Cerebral Stimulants					
Metrazol	1	0	0	0	0
Ritalin	0	0	0	0	1
Tranquilizers (Minor)					
Librax	0	2	0	0	0
Valium	6	3	3	3	6
Equanil	0	1	0	1	0
Librium	0	2	1	2	1
Atarax	1	0	0	0	0
Meprobamate	0	0	2	1	0
Tranxene	1	0	0	1	0
Vistaril	1	1	1	2	0
Serentil	0	0	1	0	0
Dantrium	0	0	0	1	0

	Facility				
	A	B	C	D	E
Tranquilizers (Major)					
Mellaril	10	15	2	3	6
Compazine	0	4	2	2	0
Haldol	1	0	0	6	0
Thorazine	3	3	3	0	5
Anti-Psychotic					
Prolixin	0	0	0	0	1
Navane	0	0	0	2	0
Stelazine	0	0	1	0	1
Synalgos	0	1	0	0	0
Lithium Carbonate	0	1	0	0	0
Caffiene and Sodium Benzoate	0	1	0	0	0
Sedatives (Hypnotic)					
Sominex	0	1	0	0	0
Chloral Hydrate	3	5	2	1	2
Seconal	0	1	0	0	0
Dalmane	8	7	3	3	6
Carbital	1	0	2	0	1
Noctec	0	0	0	1	0
Placidyl	0	0	2	0	0
Phenobarbitol	1	1	6	1	1
Paraldehyde IM	1	0	0	0	0
Nembutal	0	0	1	0	0
Butisol Sodium	0	1	0	0	0
Doriden	1	0	0	0	0
Anti-Depressants					
Aventyl	0	1	0	0	0
Elavil	0	1	0	0	0
Triavil	0	1	0	0	1
Serax	0	1	0	0	0
Tofranil	1	3	0	0	1
Caffiene and Sodium Benzoate	0	1	0	0	0
Diuretics					
Thiomerin Inj.	0	1	0	0	0
Lasix	6	8	8	6	6
Hydrodiuril	0	2	0	3	1
Diuril	2	1	0	0	0
Dyazide	1	1	1	0	0
Hydroten	1	0	0	0	0
Aldactazide	0	2	1	0	2
Diamox	0	0	1	0	1
Esidrex	1	0	1	0	0
Hydrochlorthiazyide	0	0	1	0	0
Dyrenium (Triamterene)	0	1	0	0	1

	Facility				
	A	B	C	D	E
Potassium Replacements					
Koan	2	1	0	1	0
Kaoclon	0	0	1	0	0
K-Lyte	0	1	1	0	0
Klor-Vess	0	1	0	0	1
Kaochlor Tabs	0	0	0	0	1
Potassium Elixir	0	0	2	2	0
Kolyum	1	0	0	0	0
Potassium Chloride	0	1	0	1	0
Anti-Hypertensive					
Hydropres	0	0	0	0	1
Ser-ap-es	1	0	1	3	1
Rauzide	0	0	0	0	1
Aldomet	0	1	3	3	3
Metatensin	0	0	1	0	0
Ismelin	0	1	0	0	1
Salutensin	0	0	1	0	0
Zaraxolyn	0	0	2	0	0
Apresoline (diuretic)	0	0	0	1	1
Diupress (diuretic)	1	0	2	0	0
Aldoril	0	1	0	0	1
Reserpine	0	0	0	0	2
Naturetin	1	1	0	1	0
Catapres	0	1	0	0	1
Aldactone	0	1	0	0	1
Eutron (MAO Inhibitor)	0	0	0	0	1
Cardiotonics					
Digitoxin	1	1	1	0	1
Lanoxin	9	10	4	7	6
Crystodigin	1	1	1	1	2
Digoxin	0	4	2	4	2
Purodigin	0	1	0	0	1
Persantine	1	0	0	0	0
Antiarrhythmics					
Quinidine Sulfate	0	0	0	0	1
Inderol	0	2	0	2	0
Anticonvulsants					
Dilantin	3	0	1	1	2
Mysoline	1	0	0	0	0
Luminal Sodium	0	0	0	0	1
Anticoagulants					
Coumadin	1	2	2	0	0
Antineoplastics					
Cytoxan	0	0	1	0	0

	Facility				
	A	B	C	D	E
Vasodilators					
Cyclospasmol	1	1	0	0	3
Nitroglycerine	5	1	2	2	1
Nitrobid	4	0	1	3	3
Arlidin	0	1	1	0	0
Pavabid	12	7	5	3	3
Peritrate	0	1	0	0	0
Hydergine	10	3	1	0	4
Isordil	1	1	2	1	1
Ethaquin	1	0	0	0	0
Papaverine	0	0	0	1	0
Quinam (used for leg cramps)	0	1	1	2	0
Robaxin	0	0	0	1	0
Vasodilan	2	1	1	0	0
Cerespan	0	2	0	0	0
Antihistaminics					
Triton	1	0	0	0	0
Benadryl	1	1	2	1	0
Benylin	1	2	0	0	0
Contac	1	0	0	0	0
Ronical Timespan	0	0	1	0	0
Oranade Span	1	0	0	0	0
Dimetane	0	1	0	0	1
Drixonal	1	0	0	0	0
Disophrol	0	0	0	0	1
Anticholinergic					
Cogentin	1	0	1	0	0
Bentyl	0	1	0	0	0
Anti-Malarial					
Quinine	1	1	0	0	0
Antitussive (against cough)					
Tussiorganidan DM	0	1	0	0	0
Ulo Syrup	0	1	0	0	0
Vicks Formula 44	0	1	0	0	0
Tussend (mild CNS depressant)	0	1	6	0	0
Hycomine	0	0	1	0	0
Ambelyn Exp	0	0	1	0	0
Phenergan with Codeine	0	0	0	1	0
Tessalon Pearl	0	0	0	1	0
Robitussin	1	1	2	3	0
Actified C	1	1	1	0	0
Pherergan	1	2	1	1	0
Entex	0	0	1	0	0

	Facility				
	A	B	C	D	E
Anti-Asthmatic					
Elixophylin (diuretic, stimulant)	1	1	0	1	0
Quibron	1	0	0	0	0
Alupert	0	1	0	0	0
Chlortrimeton	1	0	0	0	1
Sudafed	0	0	0	0	1
Brethine	1	0	0	0	0
Anti-Inflammatory Anti-Arthritic					
Butazolidin	0	0	2	0	1
Indocin	0	1	0	1	0
Motrin	3	0	3	0	1
Uricosuric (Blocks for matron of uric acid-gouty arthritis)					
Zyloprin	0	2	1	0	1
Allopurinol	0	0	0	1	0
Thyroid Hormone					
Synthroid	4	2	0	0	1
Thyroid	1	1	1	0	0
Hormonal					
Delatestyrl	1	0	0	0	0
Depo-Provera	0	0	0	0	1
Depo-Estradial	0	1	0	0	0
Tace	1	0	0	0	0
Deladumone Inj.	0	0	0	1	0
Premarin	0	1	1	0	1
Adrenocortical Steroid					
Durabolin	0	0	0	1	0
Deca-Durabolin	1	1	0	1	0
Prednisone	1	1	1	0	0
Deltasone	0	1	0	0	0
ATCH Decadron	0	1	0	0	0
Anti-Parkinsonism					
L-Dopa	0	0	0	0	1
Sinumet (contains L-Dopa)	0	0	1	0	0
Dopar	1	0	0	0	0
Artane	0	2	1	0	0
Kemadrin	0	1	0	0	0
Antihyperlipemics					
Antromid-S	1	1	0	0	0

	Facility				
	A	B	C	D	E
Antibiotics					
Ampicillin	0	0	0	0	2
Tetracycline (Sumycin)	1	1	0	0	0
Vibramycin	1	0	0	1	0
Keflex	2	3	0	0	1
Emycin Supp.	2	0	0	0	0
Anti-Bacterial					
Gantanol	0	1	0	0	0
Gantrisin	1	1	0	0	0
Neg Gram	0	1	0	0	0
Mandelamine	1	2	0	3	0
Macrodantin	2	0	0	1	0
Bactrin (urinary)	0	0	1	0	0
Bacterium	0	1	1	0	0
Septra Tabs	0	0	0	1	1
Thiosulfil Forte	1	0	0	0	0
Anti-Nauseant					
Dramamine	4	2	4	0	0
Antivert	1	3	1	0	1
Tigan Supp	0	0	0	2	0
Bonine	1	0	0	0	0
Wans Supp	0	0	1	0	0
Appetite Suppressant					
Sanorex (non-amphetamine)	1	0	0	0	0
Digestant Aid					
Kuzyme	1	0	0	0	0
Kanulase	1	0	0	0	0
Analeptic					
Tofranil	1	2	1	0	1
Tegretol	0	0	1	0	0
Oral Contraceptives					
Ortho Novum	1	0	0	0	0
Hypoglycemic Agents					
Tolinase	0	0	1	0	0
Orinase	1	0	1	0	1
Diabinese	1	1	2	1	1
Dymelor	0	1	0	0	0
DBI	0	2	0	0	0
Insulin					
NPH	0	0	0	2	2
Regular	0	1	1	0	0
Lente	0	1	0	2	0

	Facility				
	A	B	C	D	E
Spirits of Fermenti					
Whiskey	0	0	0	0	1
Unclassified					
Derifil	2	0	0	0	0

APPENDIX C

LISTING OF DRUGS SHOWING DRUG-NUTRIENT INTERACTIONS

Drug Category	Drugs as Listed on Chart	Generic Name	Drug-Nutrient Interactions
Analgesics	Aspirin Anacin ASA Darvon & ASA Bufferin Pabalate Ascriptin Phenaphen Fiorinal Empirin	Salicylates 28:08*	Dyspepsia, decrease absorption of tryptophan, possibly other amino acids, glucose; decrease plasma, platelet, ascorbic acid levels; decrease serum folate in patients with rheumatoid arthritis; increase urinary loss of ascorbic acid, potassium, amino acids; antagonize Vit K heartburn in 7% patients on high doses
	Tylenol Darvocet N-100 Acetaminophen Supplement Phenaphen	Para-aminophenol 28:08	May cause anorexia, nausea, vomiting, dyspepsia, diarrhea or constipation
	Darvon	Propoxyphene 28:08	Can cause nausea, vomiting, constipation, abdominal pain
	Mepergan	Promethiazine 28:24	Anorexia, constipation, dry mouth, epigastric distress, nausea, vomiting
	Percodan	Oxycodone, Salicylate & Para-Aminophenol 28:24	
	Demerol	Meperidine Para-Aminophenol 28:24	
Laxatives and Stool Softeners	Colace Dialose Dorbantyl Pericolace Surfax Doxidan Dioctyl Sodium Sulfoc Milkinol	Diotyl Suffo-succinate Salts 56:12	Can decrease intestinal glucose absorption; causes intestinal hyper-peristalsis, rapid propulsion
	Ducolax	Bisacodyl 56:12	Decreases absorption of carotene, Vit A.D,E,D, calcium, phosphate; can cause indigestion, anorexia, flatulence; increases intestinal motility; dissolves fat-soluble vitamins
	Mineral Oil	Petrolatum, Liquid 56:12	Decreases absorption of carotene, Vit. A. D, E, K, calcium, phosphate; can cause indigestion, anorexia, flatulence; increases intestinal motility; dissolves fat-soluble vitamins
	Milk of Magnesia	Magnesia Magma 56:04	Inactivate thiamine; thiamine unstable in basic pH; decrease Vit A absorption
Antacids	Gelusil	Aluminum Hydroxide 56:04	Inactivate thiamine decrease Vit A absorption
Tranquilizer	Thorazine	Chlorpromazine 28:16.08	Can cause weight gain; can increase serum cholesterol

Drug Category	Drugs as Listed on Chart	Generic Name	Drug-Nutrient Information
Anti-Psychotic	Synalogos	Promethazine 28:24	Can cause anorexia, constipation, dry mouth, epigastric distress, nausea, vomiting
Sedatives	Chloral Hydrate	Chloral Hydrate 28:24	May cause gastric irritation
	Phenobarbitol	Phenobarbitol 28:12	Decreases serum levels of folate, B_{12}, pyridoxine, 25-hydroxy, Vit D, calcium; decreases absorption of xylose; decreases erthrocyte folate; increases turnover of Vit D, Vit K, especially in children; can decrease bone density, cause osteomalacia
	Doriden	Glutethimide 28:24	May increase Vit D turnover; may alter calcium need; increase bone resorption; can cause polyneuropathy
Anti-depressants	Aventyl	Nortriptyline 28:16.04	Can cause constipation, nausea, vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, abdominal cramps, black tongue, weight gain or loss
	Elavil Triavil	Amitriptyline 28:16.04	Can cause nausea, epigastric distress, vomiting, anorexia, stomatitis, diarrhea, swelling of parotid glands, black tongue, constipation, peculiar taste, dry mouth, weight gain or loss
	Tofranil	Nortriptyline	Can cause constipation, nausea, vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, abdominal cramps, black tongue, weight gain or loss
			The antidepressant drugs all affect the autonomic nervous system which are listed above.
Diuretics	Lasix	Furosemide 40:28	May cause nausea, vomiting, diarrhea; possibly cause peculiar sweet taste, oral and gastric burning sensation; increases excretion of calcium, magnesium and potassium; decreases serum calcium in hypercalcemia; decreases serum, muscle magnesium; increases serum zinc; decreases carbohydrate tolerance; may be diabetogenic; acts on medullary diluting segment of kidney tubule

Drug Category	Drugs as Listed on Chart	Generic Name	Drug-Nutrient Interactions
Diuretics (Continued)	Diuril Hydrodiuril Dyazide Esidrex	Thiazides 40:28	May cause anorexia, gastric irritation, nausea, vomiting, cramping, diarrhea; increase urinary excretion of potassium, magnesium, zinc, riboflavin; decrease carbohydrate tolerance; can cause magnesium, potassium depletion; single dose increases calcium excretion; long-term use tends to decrease calcium excretion and increase serum calcium; may increase intestinal calcium absorption or increase bone resorption
	Aldactazide Aldactone	Spironolactone 40:28	Increases urinary calcium, magnesium
	Diamox	Acetazolamide 40:28	Can cause anorexia; increases urinary calcium, potassium
Potassium Supplements	Koan Kaoclon K-Lyte Klor-vess Kaochlor tabs Potassium Elixir Kolyum Potassium Chloride	Potassium Supplement 40:12	Can decrease absorption of Vit B_{12}; slow release of potassium chloride; causes decrease of Ileal pH (acidification)
Anti-Hypertensive	Hydropres Diupress	Reserpine 24:08	Increases gastrointestinal motility and secretion; can cause nausea, vomiting, anorexia, diarrhea, weight gain
	Aldomet Aldoril	Methyldopa 24:08	Can cause dry mouth, gastrointestinal distention, constipation or diarrhea
	Ser-ap-es Apresoline	Hydralazine 24:08	Can cause anorexia, nausea, vomiting, diarrhea; can cause paresthesias, peripheral neuritis, pyridoxine depletion; forms hydrazone with pyridoxine; inactivates the vitamin and increases its urinary excretion; may chelate trace metals
	Eutron	Methylclothiazide (not listed)	MAO inhibitor--avoid tyramine containing foods (see review of literature)
Cardiotonics	Digitoxin Lanoxin Crystodigin Digoxin Purodigin	Digitalis Glycosides 24:04	Toxicity symptoms include anorexia, nausea, vomiting, diarrhea; may competitively inhibit glucose absorption; may increase urinary magnesium, calcium

Drug Category	Drugs as Listed on Chart	Generic Name	Drug-Nutrient Interactions
Anti-convulsants	Dilantin	Phenytoin 28:12	Decrease serum levels of folate, Vit B_{12}, pyridoxine, 25-hydroxy Vit D, calcium; decreases absorption of polyglutamic folic acid, calcium; decreases erythrocyte folate level; can decrease cerebrospinal fluid Vit B_{12} despite normal serum Vit B_{12}, in absence of hematologic abnormalities, increases turnover of Vit D, Vit K, especially in children; increases serum copper; can cause megoblastic anemia, polyneuropathy; can decrease bone density; cause osteomalacia, osteoporosis
	Mysoline	Primidone 28:12	Can cause anorexia, nausea, vomiting; can decrease serum levels of folic acid, Vit B_{12}, pyridoxine; can decrease absorption of calcium; can decrease erythrocyte folate; increases turnover of Vit D, Vit K, especially in children; can decrease bone density; can cause osteomalacia
	Luminal Sodium	Phenobarbitol Sodium	(check Phenobarbitol)
Anti-coagulants	Coumadin	Warfarin 20:12.04	Can cause nausea, vomiting, cramps, diarrhea, mouth ulcers, competitive antagonism of Vit K and vice versa; drug effect may be antagonized by high doses of Vit E
Anti-neoplastics	Cytoxan	Cyclophosphamide 10:00	Causes anorexia, nausea, vomiting, hemorrhagic colitis, oral mucosal ulceration
Vasodilators	Pavabid Papaverine Cerespan	Papaverine 86:00	Can cause nausea, abdominal distress, anorexia, constipation
	Robaxin	Methocarbamol 12:20	Can cause nausea
Anti-histaminics	Dimetane	Brompheniramine 4:00	Can cause nausea, vomiting, anorexia
Anti-asthmatic	Quibron	Theophylline 86:00	Can cause nausea, vomiting, epigastric or substernal pain
	Alupent	Methocarbamol 12:20	Can cause nausea

Drug Category	Drugs as Listed on Chart	Generic Names	Drug-Nutrient Interactions
Antilipemia Agents	Antromid-S	Clofibrate 24:06	May cause nausea, loose stools, dyspepsia, flatulence, vomiting, stomatitis, gastritis; causes decreased taste acuity, unpleasant aftertaste; decreases absorption of glucose, xylose, iron, carotene, Vit B_{12}, electrolytes, medium-chain triglycerides
Anti-inflammatory Anti-arthritic	Butazolidin	Oxyphenbutazone Phenylbutazone Pyrazolone Derivatives 28:08	Can cause ulcerative esophagitis, acute and reactivated gastric and duodenal ulcer, gastritis, epigastric pain, dyspepsia, nausea, vomiting, abdominal distention; can decrease absorption of tryptophan, possibly of other amino acids
	Indocin	Indomethacin 28:08	Decreases plasma and platelet ascorbic acid levels; causes dyspepsia in 8% of patients; may decrease absorption of amino acids, xylose
Uricosuric	Zyloprin	Allopurinol 92:00	Infrequently causes nausea, vomiting, diarrhea, intermittant abdominal pain; may increase liver storage of iron; decrease iron absorption; may inhibit conversion of mucosal ferritin to ferrous iron (transportable form)
Hormonal	Premarin Ortho-Novum	Estrogenic substances conjugated 68:12	Can cause nausea, vomiting, gastrointestinal cramps, bloating; alter plasma amino acid pattern; alter tryptophan metabolism; decrease serum, leukocyte, platelet ascorbic acid levels; may decrease serum Vit B_{12}, folate, pyridoxine, riboflavin, magnesium, zinc, albumin; decrease serum calcium; increase erythrocyte zinc; increase hemoglobin, hematocrit; can cause polyneuropathy, peripheral neuritis, megaloblastic anemia; enhanced effects occur in patients on marginal diets
Andrenocortical Steroid	Prednisone Deltasone ACTH Decadron	Corticosteroids 68:04	Decrease absorption of calcium, phosphorus; increase urinary ascorbic acid, calcium, potassium, zinc, nitrogen; decrease nitrogen balance; decrease serum zinc; increase blood glucose, (continued on next page)

Drug Category	Drugs as Listed on Chart	Generic Name	Drug-Nutrient Interactions
Adrenocortical Steroid (Continued)			(Continued) serum triglycerides, serum cholesterol; increase need for folate, Vit D, pyridoxine, Vit C; delay wound healing, promote gastrointestinal ulceration
Anti-Parkinson	L-Dopa Sinumet Dopar	Levodopa 92:00	Antagonizes pyridoxine, 10-25 mg pyridoxine decreases drug effectiveness; increased need for ascorbic acid, pyridoxine; decreases absorption of tryptophan and other amino acids; drug effect decreased by high protein diet; increases urinary sodium, potassium
Antibiotics	Ampicillin	Penicillins 8:12.16	Can cause nausea, vomiting, diarrhea, hypokalemia, renal potassium wasting
	Tetracycline	Tetracyclines 8:12.24	Can cause nausea, vomiting, diarrhea, anorexia, glossitis; decrease absorption of calcium, iron, magnesium, zinc, amino acids, fat, xylose; decrease synthesis of Vit K by intestinal bacteria
Anti-bacterial	Macrodantin	Nitrofurantoin 8:36	Frequently causes anorexia, nausea, vomiting, can cause megoblastic anemia; decreases serum folate
	Bactrim	Salicylazo-sulfapyridine 8:24	May cause gastric distress, nausea, vomiting, anorexia; decreases serum folate, serum iron; antagonizes response to folate supplement
Analeptic	Tofranil	Imipramine 28:16.04	Can cause nausea, vomiting, anorexia, diarrhea, epigastric distress, peculiar taste, stomatitis, abdominal cramps, black tongue
Hypoglycemia Agents	DBI	Phenformin 68:20	Can cause unpleasant metallic taste, anorexia, nausea; decreases rate of glucose absorption in human ileum; dose related effect decreases absorption of Vit B_{12}, fat, calcium, amino acids

*Referencing number following the system reported in Interactions of Selected Drugs with Nutritional Status in Man. Am. Dietet. A. October, 1976.

APPENDIX D

COMPILED PATIENT INFORMATION

Facility A

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
1	72	1	0	Cerebral AS, Fx rt leg, arterial occulusive disease, limited alertness	Reg	4(2)	Pavabid MOM Tylenol
2	96	6	+2	Gen. AS, arthritis, osteoarthritis, osteoporosis, senility	Reg	5(4)	ASA Lax of Choice
3	79	6½	+8	AS, osteoporosis, osteoarthritis senile agitation	Reg	6(2)	Pavabid MOM ASA
4	73	4	-9	ASHD, COPD, Fx left leg & shoulder, OBS, history of GI bleeding, some disorientation	Reg	9(6)	Lax of Choice Darvon 65 Quibron
5	86	2½	+4	Shingles, ASHD, osteoarthritis, CVI	Low Res	7(5)	Tofranil Ducolax Tylenol Mineral Oil
6	93	1	0	Senility	Reg	4(1)	Lax of Choice
7	88	2½	-3	ASHD, Fx third vertebra	Reg	11(3)	Pavabid Lasix Koan formula Dialose Lax of Choice Tylenol
8	77	1	-8	Parkinson's disease, hypertenson, rt hemiparesis, Fx rt leg, post CVA	Reg	6(1)	Pavabid Dilantin MOM
9	98	1½	+14	ASHD, Gen AS, post FX rt hip, osteoporosis, osteoarthritis, anemia	Reg	8(5)	Pavabid Lax of Choice Tylenol
10	71	1	M	AS, cerebrovascular accident, post open heart surgery	Reg	10(3)	Dilantin Surfax Darvocet N-100 Dopar Antromid S Macrodantin
11	63	7	-49	Diabetes mellitus, obesity, AS, OBS	1500 cal ADA	6(2)	ASA Ducolax Thorazine
12	87	4	-6	ASHD, blind, senile, arthritis	Reg	5(4)	ASA Lasix

Facility A (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
13	82	2	+15	Malnutrition, arthritis Rt. inguinal hernia, hiatus hernia, psoriasis	Reg	7(3)	ASA Lasix Lanoxin
14	71	2	-3	ASHD, diabetes, CVA, Fx rt hip	1800 cal ADA	8(1)	Pavabid Tylenol MOM Chloral hydrate
15	87	9	+4	ASHD, AS, confused deg. arthritis, anemia	Reg	5(4)	Ducolax Tylenol Lax of Choice
16	82	2½	Unable	Cerebral thrombosis, post CVA	Soft	6(4)	Ascriptin Darvon Comp 65 Chloral hydrate
17	32	4	-7	Psychotic, MR, pre-frontal lobotomy, schizophrenia	1200 cal ADA	9(1)	Colace MOM Phenobarbitol Thorazine
18	82	2	Exp.	Senile, AS, hypertension, arthritis	1500 cal ADA	7(4)	Lanoxin Bufferin Dulcolax Esidrix
19	80	1½	+2	Parkinsonism, OBD, CVI, ASHD, coronary and vascular disease	Reg	8(2)	Lax of Choice Chloral hydrate
20	87	4	Unable	Senility, AS, osteoarthritis	Mech soft	1(0)	ASA
21	38	1	-83 Exp.	Post-op astrocytoma, CA brain & extension to thoracic spinal cord	Reg	14(8)	Percodan Dulcolax Colace Tylenol Darvon
22	98	7	Unable	Mental deterioration, AS, CV disease, osteoporosis	Pur-eed	3(0)	Lax of Choice Crystodigin
23	90	3	Unable	ASHD, OBS, CVD	Reg	12(6)	ASA Maalox Tylenol
24	87	2½	Unable	ASHD, deg. arthritis, CVA	Reg	4(2)	MOM Tylenol
25	94		Unable	Senility, deg. arthritis, AS	Reg	7(2)	Pavabid MOM Lasix
26	69	1	Unable	ASHD, Ca prostate to dorsal spine, osteoarthritis, hypothyroidism	Reg	12(7)	Darvocet N 100 Demerol Doxidan MOM Percodan

Facility A (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
27	89	4	Unable	Confused, senile, Fx left hip, osteoarthritis, osteoporosis	Pur-eed	11(4)	Diuril Koan Liq MOM ASA Lanoxin Maalox Darvon Cmp. 65
28	87	2	Unable	Arthritis, AS, hypertension osteoarthritis	Pur-eed	5(1)	Diuril Lax of Choice Macrodantin
29	101	5	Unable	Confused, ca of colon, stroke affecting rt side	Pur-eed	6(2)	Surfax Pavabid Ser-ap-es Darvocet N
30	87	7	Unable Exp.	ASHD, Fx pelvis, hypertension	Pur-eed	6(2)	Lanoxin Diupress ASA
31	68	1	Unable	AS, stroke with left hemiplegia, osteoporosis, osteoarthritis	Lo Na 2 gm	6(2)	Colace Tylenol
32	92	5	Unable	AS, CVI, senile	Reg Gr.	7(3)	Lanoxin Tylenol Dulcolax Lax of Choice
33	78	9	Unable	Senile psychosis, gen AS, osteo-arthritis	Pur-eed	9(3)	Dialose Darvon Dulcolax ASA Tetracycline
34	74	1	Unable Exp.	ASHD, AS, senile psychosis	Pur-eed	3(2)	MOM Tylenol
35	58	1	Unable Exp.	AS, post cva with spastic paralysis and brain stem lesion, post MI, decubitus low back and hip	Pur-eed	3(2)	Lanoxin Doxidan Colace
36	87	7	Unable M	AS, ASHD, blind, confused and dis-oriented, osteoporosis	Pur-eed	6(2)	Tylenol Dyazide
37	82	4½	Unable Exp.	Gen. AS, ASHD, post cva, some confusion	Pur-eed	8(3)	Dilantin Lanoxin Tylenol MOM
38	91	2½	+2	Senile, agitated de-pression, Fx rt hip	Reg	7(2)	Lanoxin Lasix Tylenol

Facility A (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
39	90	1½	Unable	Confusion, ASHD, AS	Reg	12(3)	MOM Pavabid Lasix Prednisone Bufferin
40	32	6	-3	MR, congential hip deformity	1000 cal	4(2)	Tylenol
41	57	9	Unable	Severe deformed rheumatoid arthritis	Reg	4(2)	MOM Tylenol
42	29	9	0	MR	1200 cal	5(2)	MOM ASA Ortho Novum
43	63	9	+1	Reg. enteritis, chronic schizophrenia	Reg	10(2)	Ducolax Pavabid Thorazine
44	82	1½	-22	Post Fx rt hip, post CVA	Reg	14(5)	Darvon ASA Maalox Kolyun MOM Lanoxin
45	62	3	Unable	Confused and dis-oriented, AS, cerebral Thrombosis with hemiplegia	Reg	2(2)	MOM Tylenol
46	94	3	-9	ASHD, senility, confused	Reg	8(5)	ASA MOM Digitoxin
47	74	6	-4	Schizophrenia, paranoid, confused	Reg	1(0)	MOM
48	86	4½	-24	Diabetes mellitus, OBS, ASHD, CHF, prior MI	1400 cal	5(3)	Tylenol
49	93	4½	Unable Exp.	Fx Femur, hostile, confused, arthritis, acute leukemia	Reg	12(5)	MOM Darvocet N 100 Tylenol
50	89	9	-3	CVA, gen AS, alert, oriented, benign prostrate hypertrophy	Reg	1(1)	MOM
51	90	4	+5	Gen AS, ASHD, chr. cholecystitis, osteoarthritis	1200 cal	9(6)	Surfax Pavabid Tylenol
52	22	4	Unable	MR, CP, multiple birth defects, thrombophlebitis	Reg	4(1)	Ducolax Tylenol Coumadin
53	36	7	+7	MR	1200 cal	4(3)	Mysoline ASA MOM

Facility B

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
1	87	2½	+5	AS, blind	Reg	2(0)	None
2	82	3	+4	ASHD	1600 cal	5(2)	Lax of Choice Tylenol Chloral Hydrate
3	79	5	Unable Exp.	Cerebral As, CBS, diabetes mellitus, mod. confusion	Reg	5(2)	MOM DBI
4	78	3	+13	Senile dementia	Lo Res	11(7)	Dialose Darvocet N Aldactazide Digoxin MOM
5	87	2½	+17	AS, OBS, confused	Reg	3(2)	Tylenol
6	79	2	Unable Exp.	Post MI, post CVA	Reg	9(4)	Pavabid MOM Lanoxin ASA K-Lyte Lasix
7	93	2	Unable M	Cerebral AS, CBS, cerebral thrombosis, confused	Reg	10(2)	ASA Lanoxin Pericolace Ducolax
8	96	2½	-3½	ASHD, post Fx rt hip, osteoarthritis, depressed	Reg	9(3)	Thorazine Peri-Colace Ascriptin ASA Digoxin
9	73	1	Same	Rheumatoid arthritis, infected left hip with draining sinus	Reg	5(3)	Darvon Ascriptin
10	68	3	-28	Schizophrenic reaction	1000 cal	6(1)	Chloral Hydrate Thorazine Tetracycline
11	75	2	Same	AS, aortic aneurysm below renal vessels, vertigo	Lo Salt	3(1)	Pavabid Tylenol Coumadin
12	82	2½	Unable Exp.	ASHD, diabetes mellitus	Reg	8(4)	MOM ASA Lanoxin Lasix Koan
13	69	7	Same	Spinal cord deterioration, spastic movement of lower extremities	Reg	7(4)	ASA
14	90	3	-3	ASHD, arthritis, confused	Reg	5(3)	Peri-colace Lax of Choice Bufferin

Facility B (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
15	84	2½	+5	Deg. joint disease, obesity, ASCVD	Lo Na	11(5)	Prednisone Lasix Tylenol Lax of Choice
16	63	2	Unable M	CBS, psoriasis, chr. alcoholism	Reg	17(8)	Dyazide Peri-colace Digoxin Maalox Phenobarbitol
17	84	2	Unable M	ASCVD, diabetes mellitus, gouty, arthritis, hypertension	Reg	7(5)	Digitoxin Tylenol Darvocet DBI
18	87	2	+2	ASHD, hip Fx, post hip pinning	Lo Fat	6(5)	Empirin Lax of Choice Tylenol
19	84	10	+1	Diabetes mellitus, hypertension, osteomyelitis	Lib. Diab	8(3)	Crystodigin MOM ASA Hydrodiuril
20	88	4	+12	Gen. AS, post Fx rt. femur with jewett pinning	Reg	2(2)	Maalox Lax of Choice
21	84	1	Unable Exp.	Cerebral AS with possible Parkinsonism	Reg	4(2)	Pavabid ASA Lax of Choice Bactrium
22	94	3	+17	ASHD	Reg	8(3)	Lanoxin, Dimetane Lasix, Tylenol
23	98	3	+3	Arthritis, HCVD controlled, ASCVD anemia	Lo Chol	13(6)	Indocin Aldoril Lasix Tylenol
24	71	7	Unable Exp.	ASHD	Reg	4(1)	Tylenol Premarin
25	84	4	Unable	ASHD	Reg	11(5)	Lax of Choice Diuril Pavabid Anacin
26	91	6	+12	ASHD, Fx left hip with pinning, osteoporosis, confused and disoriented	Reg	5(3)	Lax of Choice Bufferin Darvon 65 Thorazine
27	83	3	+2	Arthritis, ASHD	Reg	5(3)	Doxidan Cerespan
28	85	3	+1½	confused, disoriented, hypertension, osteoarthritis, vascular senility	Reg	2(1)	ASA Darvon

Facility B (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
29	88	1	+1	Very confused, cerebral AS, CBS, Fx left hip, left mastectomy	Reg	3(1)	None
30	82	3½	-2	Senile, disoriented, AS, post colostomy	Soft	5(2)	ASA Darvon
31	80	4	Unable	Confused, disoriented, ASHD, post hip Fx, osteoarthritis	Lo Na	7(4)	Lanoxin Pavabid Darvon 65 Aspirin Lasix
32	68	3	-19	Psychotic, hypothyroidism	Reg	6(3)	Chloral Hydrate Tylenol MOM
33	86	9½	Same	Senile, disoriented, cerebral AS, arthritis, cortical atrophy	Reg	4(3)	ASA Lax of Choice
34	91	1	+14	Confused, ASHD, post Fx rt hip, left inguinal hernia	Reg	8(4)	Pavabid Darvon Cmp. 65 Tylenol
35	91	1½	+9	Senile, deterioration, confusion, post coronary, post hip Fx	Reg	7(4)	Digoxin Tylenol
36	97	2½	Unable Exp.	Aortic stenosis, AS aneurysm of the thoracic aorta, Fx rt hip with Jewett nailing	Reg	5(1)	Lanoxin Tylenol
37	87	1½	Unable	ASHD, CVD, recent stroke, memory impaired	Lo Na	13(2)	Tylenol Klor Vess Decadron Lanoxin Lax of Choice
38	62	6	+3	disoriented, AS, OBS	Reg	5(4)	MOM Ducolax
39	93	1	Same	ASCVD, CHF controlled, severe varicose veins	Lo Na	5(2)	Coumadin Lanoxin Lasix MOM Darvon
40	88	1	-1½	Fx rt. hip, alert	Reg	3(2)	ASA
41	85	3½	Same	Confused, post Fx hip & pelvis, AS, arthritis, colitis	Reg	6(6)	ASA
42	79	4½	Same	CBS, ASHD, post Fx hip--agitated easily	1000 cal	6(2)	Pavabid Chloral hydrate
43	86	7	+11	ASHD with coronary artery insufficiency, senile	Reg	8(4)	Tofranil Lanoxin Lax of Choice ASA

Facility B (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
44	84	2½	Unable	ASHD, OBS, mild diabetes mellitus, confused, agitated, disoriented	Reg	1(1)	None
45	84	1	Unable Exp.	Parkinsonism, CVA	Reg	7(2)	Atromid Dialose Zyloprin
46	97	4½	-4	Gen. AS, post CVA, oriented to time and place	Reg	6(4)	Cerespan MOM Darvon 65
47	91	1	+14	Deg. joint disease, mild diabetes	1500 cal	6(0)	MOM Mineral Oil Purodigin
48	86	2½	Unable	CVA, ASHD with aortic stenosis and insufficiency, pernicious anemia, recurrent urinary infection, confused and depressed	Soft	8(4)	Aldomet Lasix Lax of Choice Tylenol Darvocet N 100
49	73	1	Unable Exp.	CVA, rt side paralysis	Reg	7(2)	Lanoxin Elavil Tylenol
50	73	1	-7	AS, CBS, Fx left hip, Gout	Reg	8(2)	Zyloprin Aldactazide Dialose Chloral Hydrate Darvon Tofranil
51	83	1	Unable M	Cerebral AS, Parkinsonism, decubitus ulcer	Reg	6(2)	Aventyl ASA

Facility C

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
1	81	1	Unable Exp.	Confused, adv. AS, AS dementia	Soft	5(3)	Esidrex Darvon 65 Maalox
2	82	1	-3	AS, Osteoporosis, Senile	Bland	13(3)	Premarin Darvon 65 Chloral Hydrate Pavabid MOM
3	65	7	Unable	Psychosis, ca skin, L side face	Reg	6(5)	Thorazine MOM ASA Empirin #3
4	59	6	-5½	MR, osteomylitis of left hip, hernia	Reg	3(2)	MOM ASA
5	93	1	+15	Acute depression, aphakic rt eye, glaucoma rt eye	Soft	11(0)	Diamox Tofranil
6	86	13	+1	AS, senility	Reg	12(7)	Bufferin
7	93	5	Unable	Fx rt. femur	Pur-eed	8(5)	MOM ASA Darvon 65
8	85	2	+5	AS, arthritis	Lo Salt	9(5)	Lasix Butazolidin Kaochlor Lax of Choice
9	87	3	-12	Senile, diabetes mellitus, aortic stenosis, peripheral vascular disease	1500 cal	5(2)	Dialose ASA MOM
10	95	1½	Unable Exp.	Mild confusion, senility, mild nutritional anemia	Reg	10(6)	Pavabid Lanoxin MOM ASA Darvon Pl 65
11	78	5	Unable	AS, CBS, arthritis	Pur-eed	4(3)	Chloral hydrate Lax of Choice ASA
12	86	7	Unable Exp.	AS, ASHD with compensated CHF diverticulosis, Parkinson's disease	Bland	12(5)	Lanoxin MOM Bufferin
13	86	4½	Unable	Post CVA, left hemiplegia, CBS, diverticulosis	Pur-eed Lo Res	8(4)	Colace Ascriptin Bactrim Darvon N Tylenol Darvocet

Facility C (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
14	79	1	Same	ASHD, Fx rt hip, CVA rt hemiplegia, tracheotomy	1500 cal	9(5)	Diupress Pavabid Demerol Phenaphen Darvon 65 MOM
15	87	4	+5	ASHD, dementia, diabetes mellitus, epilepsy	1400 cal	5(3)	Phenobarbitol MOM Lasix ASA
16	66	1	+5	Mentally confused, ca of rectum with metases, cerebral AS, colostomy	Soft	5(2)	MOM ASA
17	81	1	-4	Fx L knee cap, post pneumonia, thrombosis L deep vein	Reg	2(1)	Coumadin Tylenol
18	82	3½	Unable	CVA with hemiplegia, diabetes, speech impaired due to stroke	1200 cal	13(9)	Ser-ap-es MOM Phenobarbitol Empirin ASA
19	88	12	Unable Exp.	AS, CHF, CVA, adv. OBS, comatose	1800 cal tube	7(2)	Lanoxin Pavabid Acetomenophen
20	86	9	Unable	ASHD, senility, dis-oriented, Glaucoma	Soft	4(2)	Lasix ASA
21	77	2½	Same	AS, AS dementia, hypertension	Reg	13(8)	Aldomet Lasix Lax of Choice ASA Empirin #3
22	81	6	-16	AS dementia, ASHD, disoriented	Reg	4(1)	Digoxin ASA Hydrochlorthiazide Dioctyl Sodium Sulfoccinate
23	89	7	+10	Hypertension, CBS, cerebral AS, depression	Soft Lo Salt	14(7)	Digoxin MOM Ducolax Darvocet
24	76	5	Unable Exp.	Reticular cell sarcoma, CHF, cerebral AS	Lo Na	22(10)	Digitoxin Prednisone Lasix Aldactiazide Coumadin Pavabid K-Lyte Lax of Choice Tylenol Empirin #3 Zyloprin

Facility C (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
25	49	12	+52	Epilepsy, brain surgery performed, withdrawn, mental deterioration	Reg	8(4)	Lasix Lax of Choice ASA Dilantin Phenobarbitol
26	90	3	Unable Exp.	Disoriented, Fx L hip, AS, adv. ASHD	Soft	7(4)	Empirin #3 Thorazine ASA Lanoxin Doxidan
27	83	2	Unable Exp.	Post TUR, senility	Reg	3(2)	Aldomet Thorazine
28	91	1	Unable	Diabetes mellitus, ASHD, hypertension	1400 cal	11(8)	Crystodigin Phenobarbitol Butazolidin Bufferin MOM Empirin #3 Diupress Lasix
29	79	2	-9	Fx rt hip, ASHD, hypertension	Reg	6(5)	Lax of Choice Aldomet Empirin Darvon Cmpd 65
30	101	11	Unable Exp.	AS, inguinal hernia rt, arthritis	Reg	9(3)	ASA Darvon 65 Dyazide MOM
31	72	6	-23	CBS, AS, very poor memory, chronic alcoholism pancreatitis	Reg	7(4)	MOM Phenobarbitol
32	89	1	Unable Exp.	ASHD with heart failure, old MI, pneumonia	Soft	7(5)	Lanoxin MOM Empirin #3 Lasix Phenobarbitol

Facility D

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
1	76	2½	Unable Exp.	ASHD, MR, hysterectomy (ca)	Reg	3(0)	None
2	86	1½	-6	ASHD, OBS, hypertension, senile	Reg	4(0)	Ser-ap-es Lanoxin
3	88	1	+25	ASHD, OBS, Fx L hip, disoriented, osteoporosis	Soft	4(1)	Crystodigin MOM Darvon 65
4	44	5	-2	Birth injury, dorsal scoliosis, mute	Soft	10(5)	ASA
5	46	6	Unable 3 mo. -8	Incomplete quadraplegia, Blind L eye, cellulitis perineum, iliostomy, chronic cystitis	Reg	5(1)	Dorbantyl Lasix ASA
6	87	1½	+7	Hypertension, osteoarthritis, lumbar spine, deg. cervical disc syndrome	Lo Na	13(6)	Ser-ap-es Tylenol MOM Fiorinal Anacin
7	86	1½	+13	Hypertension, ca L mastectomy, conjunctivitis	Lo Salt	8(4)	Gelusil
8	44	3	+2	Rheumatoid arthritis, osteoporosis, iron def. anemia, Fx L femur, Duodenitis	Reg	5(2)	Dilantin Phenabarbitol Surfax Darvocet N-100
9	74	1½	+6	Pneumonia, active gastric ulcer	Lo Ca Hi Fiber	3(0)	None
10	74	5	+6	ASHD, OBS, osteoporosis, osteoarthritis of spine, acute rhinitis, diabetes mellitus	1200 cal	8(3)	Lanoxin Darvon Tylenol
11	78	1	Unable 3 mo. +1½	Bilateral AK amputation, CVI, ASCVD, peripheral vas. disease	Reg	4(0)	Colace Hydrodiuril Kcl Elixir
12	71	2	Unable 3 mo. +4	Hypertension, CVA, cellulitis L leg, rt. inguinal hernia	Soft	4(2)	Papverine Ser-ap-es MOM ASA
13	78	7½	-4	AS, COPD, PVD, BPH, Pulmonary TB (inactive)	Soft Lo Salt	8(2)	Digoxin Hydrodiuril Kcl Elixir Dorbantyl Tylenol

Facility D (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
14	67	1	Unable Exp.	CHF, ASHD, osteoarthritis, diabetes, paranoid, schizophrenia	1500 cal 2 gm Na	6(0)	Digoxin Kcl Elixir Lasix Colace Aspirin
15	79	7	Unable Exp.	CHF, OBS marked senility, deg. osteoarthritis, L side paralysis	Lo Fat	9(3)	Indocin Pavabid Darvon 65
16	84	2	Unable 3 mo. +6	Hypertension, COPD, arthritis, edema, blind L eye	Reg	3(1)	Tylenol Lanoxin Pabalate
17	68	2	Unable Exp.	Nephrectomy, azotemia, CVA with L side paralysis, hypertension	1500 cal	10(4)	Digoxin ASA Surfax Phenaphen Tylenol
18	52	3	Unable 3 mo. +3	Deg. CNS, scites, multiple sclerosis, hiatus hernia	Soft	3(2)	Ducolax ASA
19	67	1	Unable M	ASHD, OBS, disoriented, 2nd and 3rd degree burns, rheumatoid arthritis	Reg	5(2)	Colace MOM Tylenol
20	75	2	Unable M	Diabetes mellitus, hypertension, ASHD, OBS, L vent hypertrophy, senile dementia	1600 cal	3(1)	None
21	73	2	Unable	COPD, OBS, ASHD, chr. cystitis, acute bronchitis	Soft	2(1)	MOM
22	77	1	+6	ASHD, COPD, OBS, dehydration, arrested TB	Reg	3(1)	Tylenol
23	79	4½	Unable Exp.	OBS, ASHD, AS, aphakia bilateral	Reg	4(3)	Mineral Oil MOM
24	82	2½	+1	Osteoporosis, ASHD, OBS, statis edema, dehydration, L mastectomy	Reg	6(3)	Lasix Chloral Hydrate ASA MOM
25	79	4	Unable 3 mo. +20	Acute gastroenteritis, acute CHF, diabetes, cystitis, bladder calculi, L renal tumor, cerebral apoplexy with L hemiparesis	1800 cal	11(1)	Lanoxin Hydrodiuril Koan Cl Aldomet

Facility D (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
26	85	1	Same	MI, AS, CHF, ASHD, angina pectoris, senility	Reg	6(2)	Lanoxin Lasix Lax of Choice
27	72	1½	+3	Disoriented, OBS, AS, chronic pylonephritis, acute cystitis, multiple trauma with multiple Fx ribs and pulmonary contusion	Pur-eed	1(0)	Tylenol
28	49	5	Unable Exp.	ASHD, hypertension, diabetes, chronic renal failure, hypo-glycemic reaction	1200 cal	7(1)	Aldomet Digoxin Lasix Apresoline
29	82	2½	Unable Exp.	ASHD, OBS, TIA, acute urinary tract infection	Pur-eed	2(0)	Aldomet Macrodantin
30	81	1½	-12	ASHD, COPD, chr. UTI, OBS, acute cystitis	2 gm Na Soft	6(4)	Pavabid Lanoxin Tylenol
31	50	7½	+3	MR, schizophrenia, paranoid type, Deg. arthritis	Reg Avoid Sweets	9(3)	Lasix MOM
32	72	2	-18	ASHD	Soft	6(2)	Pavabid Lanoxin, ASA
33	76	2½	+30	ASHD, COPD with acute bronchitis, osteomyelitis, acute cellulitis	Soft	0(0)	None
34	71	3	-8	ca throat and lung, COPD, OBS, AS, obstipation	Reg	5(3)	Tylenol

Facility E

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
1	83	New Facility Open June 1976	+½	CHF, CBS, old burn right hand	Reg	7(0)	Purodigin
2	64		Unable M	CVA, juvenile diabetes, CHD, hypertension	1800 cal	11(3)	MOM Aldomet Lanoxin Aldactazide Aprensoline Tylenol
3	81		Unable M	CBS, diabetes	1800 cal	4(1)	Eutron (MAO Inhibitor)
4	84		Unable M	ASHD, arthritis, L hip Fx, colitis, hypertension, hemmoroids	Lo Salt	5(2)	Digoxin MOM
5	73		Unable M	Post-op Fx rt hip, hysterectomy, thrombus neck 1962	Reg	8(1)	Phenaphen
6	86		Unable M	Gen AS, back injury, disorientation	Soft	7(11)	Lanoxin Tylenol
7	84		+4	ASHD, adv. rheumatoid arthritis, osteo-arthritis	Reg	1 (5)	Crystodigin Aldomet Pavabid ASA
8	51		Same	Post-op removal rt hip, prosthesis, osteoarthritis	Bland #3	4(1)	Tylenol Lax of Choice
9	73		Unable M	CBS	Reg	5(1)	None
10	40		Unable	Diabetes, MS	1800 cal	1(0)	Lasix
11	81		-11	ASCVD, senile	Reg	3(1)	Chloral Hydrate
12	84		Unable M	AS, CBS, CVA	Reg	3(0)	Crystodigin Lasix Dilantin
13	91		Unable Exp.	Mental confusion weakness	Pur-eed	4(0)	None
14	77		-15	AS, hypertension, arthritis, diabetes, diverticulitis	1500 cal	3(2)	Ser-ap-es Tylenol
15	89		Unable	ASHD, angina pectoris, mild confusion, diverticulosis, dizziness	Reg	6(0)	Digitoxin Pavabid
16	84		-1	Gen AS cerebral and coronary, hypertension, diabetes mellitus, confused	1200 cal	11(3)	Aldomet Lasix Dorbantyl

Facility E (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
17	76		Unable M	ASHD, arthritis	Reg	9(0)	Digoxin Kaochlor Lasix Premarin
18	35		Unable M	Severe MR, club feet bilateral, decubitus on coccyx area	Pur-eed	10(3)	Dilantin Klorvess Phenobarbitol Tylenol Lax of Choice
19	93		Unable M	CBS, AS, Fx pelvis	Reg	5(2)	Ampicillin Ducolax
20	71		+8	AS, CHF, anxiety, depression, osteoarthritis	Lo Salt	8(4)	Tofranil Lanoxin ASA Lax of Choice Tylenol
21	84		+3	CBS, AS, CHF, arthritis, hypertension	Lo Salt	7(2)	Hydrodiuril Lanoxin Aldactone ASA Lax of Choice
22	77		-4	ASHD, CBS, several small CVA's, mental confusion, hypertension	Reg	7(4)	Aldoril ASA Lax of Choice Chloral Hydrate
23	74		+6	Psychosis, AS, gross tumor, rt upper extremity	Reg	5(3)	Thorazine Lax of Choice ASA Gelusil
24	69		-3	MR, ASHD, AS	Reg	4(2)	Pavabid Thorazine Lax of Choice ASA
25	67		+7	Paranoid Schizo-phrenia, hypertension, pyorrhea, constipation	Reg	5(1)	Thorazine Colace ASA
26	45		+2	Severe MR	Reg	3(2)	ASA Lax of Choice
27	64		Unable M	MR, diabetes, hypertension, obesity	1500 cal	4(2)	Lax of Choice ASA
28	89		Unable Exp.	AS, CBS, CHF, Parkinsonism	Pur-eed	12(0)	L-Dopa Lanoxin Lasix Ampicillin
29	59		-18	Psychosis, surgery for hernia	Reg	4(2)	Thorazine Lax of Choice
30	99		Unable	AS, CP	Reg	2(2)	ASA Lax of Choice

Facility E (Continued)

Patient No.	Age	Years in Facility	Weight + or -	Diagnosis	Diet	No. of Drugs	Drugs with Reported Interaction
31	63		Unable M	Cirrhosis, emphysema, ascites, umbilical hernia	Salt Free	7(3)	Aldactone ASA
32	98		+7	CHF, ASHD	Soft	10(3)	MOM Lasix Lanoxin Aldactazide Zyloprin Aspirin
33	84		+7	CBS, dog bite with skin graft L leg	Reg	3(2)	Thorazine
34	85		Unable	AS, CBS, Fx L arm, mental illness, hypertension	Lo Salt	3(2)	Hydropres Lax of Choice Tylenol
35	75		Unable	Senile brain syndrome	Reg	4(1)	Butazolidin Ascriptin
36	84		+2	Gen. AS, alert, osteoarthritis	Reg	4(0)	Lax of Choice Doxidan

VITA 2

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Master of Science

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