Abnormal Blood and Urine Chemistries In An Alcohol and Drug Population: Dramatic Reversals Obtained Quickly From Potentially Serious Diseases

Alfred F. Libby, Ph.D.[1]. Charles R. Starling, M.D.[1], David K. MacMurray, Ph.D.[1], Robert E. Kline II, R.T.[1]

Background of the Study

This article, just as the two previous papers, concerns itself with the same addictive population and treatment facility. When we arranged to do this Study, we took eight months to carefully arrange the protocol as there were several hypotheses we wanted to test. This was a realistic, even uncooperative, yet rigidly controlled patient setting. Several metabolic abnormalities had been observed in drug and alcohol patients over the past four years in their blood chemistries, hematology and urine. These areas were of vital interest to the investigators; therefore, an exacting protocol for close scrutiny was designed.

Discussion of Serum Sodium Levels

One of the primary and burning issues by antagonists has always been the unanswered question regarding the wisdom of using sodium ascorbate in mega dosages. There have always been questions raised concerning the safety of using sodium ascorbate at what would be considered by orthomolecular physicians as therapeutic levels (30 grams or more per day).

Critics of this usage have stated that these amounts will upset the electrolyte balance, elevate the blood pressure, cause cardiac stress and possibly poison a patient by excesses of elemental sodium.

Drs. Klenner and Libby have each stated separately that sodium does not appear to freely dissociate from the ascorbate as it does from chloride. We drew pre-treatment and post-treatment blood samples to see what effect, if any, the ingestion of 30 plus grams of sodium ascorbate for three days and eight to ten grams daily for the remainder of the test period would have on the electrolyte system. All pre-treatment, post-treatment chemistries were drawn at the same time in the AM and were fasting specimens.

1. The Libby Institute
For Orthomolecular Medicine and Research
1402 N. El Camino Real
San Clemente, California 92672

ORTHOMOLECULAR PSYCHIATRY, VOLUME 11, NUMBER 3, 1982, Pp. 156-181

SERUM SODIUM LEVELS

Normals - 135 - 150 mEg/L

Patient	Pre-Treatment	Post-Treatment	%
#1	142	139	2.1% ↓
#2	140	143	2.1% ↑
#3	146	138	5.5% ↓
#5	143	145	1.4% ↑
#8	144	144	0
#10	140	141	0.7% ↑
#13	144	139	3.5% ↓
#14	139	145	4.3% ↑
#15	139	147	5.8% ↑
#17	144	142	1.4% ↓
#18	139	141	1.4% ↑
#19	141	143	1.4% ↑
#20	138	140	1.4% ↑
#21	138	143	3.6% ↑
#24	138	144	4.3% ↑
#25	139	143	2.9% ↑
#26	140	144	2.9% ↑
#27	146	145	0.7% ↓
#28	139	143	2.9% ↑
#29	144	142	1.4% ↓

Discussion of Serum Sodium Test Results

It is obvious to the reader that all serum sodium levels were well within normal limits, both pre-treatment and post-treatment! Examining the data reveals an average increase of 1.3 mEg/L, which is statistically insignificant.

Discussion of Potassium Levels

Potassium is the major intracellular cation, with only two percent of the total body potassium being extracellular. We would naturally be concerned with deficiencies or excesses of this most important electrolyte. Hypokalemia occurs with gastrointestinal fluid losses, in renal disease, with diuretic administration, in mineralocorticoid excess and in alkalemia. Hyperkalemia is seen in acute and sometimes in chronic renal diseases, in renal tubular acidosis in which the exchange of sodium for potassium and/or hydrogen is impaired, and in acidotic states.

Elevated and depressed serum potassium concentrations may have profound adverse effects on the neuromuscular system, and especially on the myocardium. Serious arrhythmias can develop, even death.

POTASSIUM LEVELS

Normals - 3.5 - 5.3 mEq/L

Patient	Pre-Treatment	Post-Treatment	Δ%
#1	4.0	4.8	20.0% ↑
#2	3.9	3.7	5.1% ↓
#3	4.0	4.0	0
#5	4.0	4.0	0
#8	4.5	4.5	0
#10	4.1	4.6	12.2% ↑
#13	5.3	6.1	15.1% ↑
#14	4.1	4.1	0
#15	4.6	5.1	10.9% ↑
#17	5.1	5.1	0
#18	4.2	4.4	4.8% ↑
#19	4.7	4.6	2.1% ↓
#20	4.5	7.7	71.1% ↑
#21	3.7	5.4	45.9% ↑
#24	4.5	3.7	17.8% ↓
#25	4.5	6.2	37.8% ↑
#26	4.6	4.7	2.2% ↑
#27	4.6	4.3	6.5% ↓
#28	4.8	5.5	14.6% ↑
#29	5.0	5.8	16.0% ↑

Discussion of Test Results

Patients 13, 20, 21, 25, 28 and 29 had elevated potassium levels. While these elevations are not extremely high, they are elevated and therefore ostensibly present a potential threat to the patients involved in creating an imbalance to the electrolytes.

Closer investigation into these elevated potassium levels indicate they are false high levels. Our rationale for this statement is the following: their sodium, chloride and C02 levels are normal, in each case their LDH levels were elevated. S.L. Skinner reported his findings in Lancet 1, 478, 1961, "Cause of Erroneous Potassium Levels." Dr. Skinner states: "False high potassium levels are also seen if a tourniquet is left on too long with juxtavenular cellular injury and leakage of potassium into the plasma. This effect is markedly enhanced if the fist is repeatedly clenched prior to and during drawing. Any hemolysis will yield an elevated value owing to the high concentration of potassium within erythrocytes."

Patients 20, 21 and 29 had the longest histories in drug abuse and their veins were very difficult to penetrate, thus necessitating the longer use of the tourniquet and fist clenching. Their agitation can be told in the LDH levels. Patient 29 finally had to draw the blood sample on himself by using a vein in his ankle. In addition, none of these patients had signs or symptoms of hypo- or hyper-kal-emia. In fact, they looked and felt "terrific."

Discussion of Chloride

Chloride is the major extracellular anion. Most of the ingested chloride is absorbed and the excess is excreted along with the cations into the urine. Low serum chloride levels are observed in prolonged vomiting with loss of HCL; in metabolic acidotic states in which there is an increased accumulation of organic anions.

Elevated chloride values are seen in metabolic acidosis associated with prolonged diarrhea with loss of NaHCC»3, and in renal tubular disease in which there is a decreased excretion of H+ and therefore a decreased reabsorption of HCO3-.

CHLORIDE LEVELS

Normals - 95-105 mEq/L

Patient	Pre-Treatment	Post-Treatment	Δ%
#1	104	103	1.0% ↓
#2	102	101	1.0% ↓
#3	103	102	1.0% ↓
#5	104	102	1.9% ↓
#8	103	101	1.9% ↓
#10	104	102	1.9% ↓
#13	103	102	1.0% ↓
#14	103	102	1.0% ↓
#15	103	103	0
#17	102	101	1.0% ↓
#18	103	102	1.0% ↓
#19	102	101	1.0% ↓
#20	102	101	1.0% ↓
#21	98	101	3.1% ↑
#24	103	102	1.0% ↓
#25	102	101	1.0% ↓
#26	103	101	1.9% ↓
#27	102	101	1.0% ↓
#28	101	101	0
#29	102	102	0

Discussion of Serum Chloride Results

What is of significance here is that the serum chloride levels were within normal limits, before and after treatment, but yet observe the downward adjustment. We feel it is absolutely essential to the successful detoxification of a patient to have and maintain a state of diarrhea for about four hours. One might assume, therefore, with the added sodium and the diarrhea, a shift in the chloride would occur; such was not the case.

Discussion of Total CO2

The potential uses of carbon dioxide as a pharmacological agent and its toxicity lie primarily in its marked effects on respiration, circulation, and the CNS. Several hundred milliliters per minute of carbon dioxide are produced by the body's metabolism. The gas diffuses easily from the cells that produce it into the blood stream, where it is carried partly as a bicarbonate ion, partly in chemical combination with hemoglobin and plasma proteins, also in physical solution at a partial pressure of about 46mm HG in mixed venous blood. It is transported to the lung, where it is normally exhaled at the same rate at which it is produced.

Acid-base disorders have traditionally been placed into four categories: respiratory acidosis, respiratory alkalosis, metabolic acidosis and metabolic alkalosis. This classification takes cognizance of respiratory versus nonrespiratory disorders. The following classification of acid-base disorders focuses primarily on changes in hydrogen concentration induced by a change in the ratio of the acid and base form of the buffer system.

(1) Acidosis (increased hydrogen ion con centration)
(a) Primary excess of acid.
(b) Primary reduction of base.

(2) Alkalosis (decreased hydrogen ion concentration)
(a) Primary excess of base.
(b) Primary reduction of acid.

(3) Mixed disturbances of acid-base balance.

CARBON DIOXIDE

Normals - 21-29 mEq/L

Patient	Pre-Treatment	Post-Treatment	(%)
#1	33	26	21.2% ↓
#2	37	35	5.4% ↓
#3	30	25	16.7% ↓
#5	34	31	8.8% ↓
#8	36	33	8.3% ↓
#10	36	29	19.4% ↓
#13	34	28	17.6% ↓
#14	32	31	3.1% ↓
#15	32	32	0
#17	35	31	11.4% ↓
#18	32	27	15.6% ↓
#19	34	34	0
#20	33	32	3.0% ↓
#21	37	34	8.1% ↓
#24	31	26	16.1% ↓
#25	35	33	5.7% ↓
#26	34	33	2.9% ↓
#27	37	34	8.1% ↓
#28	37	34	8.1% ↓
#29	36	32	11.1% ↓

Discussion of Total CO_2 Test Results

According to the test results, this patient population was suffering from chronic metabolic acidosis. Use of the word "metabolic" in this instance is unfortunate, because carbon dioxide is also a metabolic product. Yet, by convention, carbonic acid resulting from dissolved carbon dioxide is called a respiratory acid, while any other acid in the body, whether it be formed by metabolism or simply ingested by the person, is called a metabolic acid.

H. Valtin published in 1973 a book entitled, **Renal Function: Mechanisms Preserving Fluid and Solute Balance in Health.** Dr. Valtin stated, "A large quantity of acid is produced daily in the body as a result of metabolism. Between 13,000 and 20,000 mmol/1 of CO_2, which can be converted to carbonic acid (H2CO3), is produced as a result of the complete oxidation of carbohydrates, proteins, and fats. Another 40 to 60 mmol/1 of acid in the form of ketoacids, as a result of the incomplete oxidation of lipids, and sulfuric and phosphoric acid as a result of the oxidation of sulfur-containing compounds."

All metabolic reactions are catalyzed by enzymes which function at optimal hydrogen ion concentrations. It is therefore necessary that the body possess efficient mechanisms to maintain the pH of both the extracellular fluid and therefore the intracellular fluid within narrow limits (7.35 to 7.45). (The pH is the negative logarithm of the hydrogen ion concentration.) This is accomplished by the blood buffers, by renal tubular mechanisms, and by respiration.

The blood buffers in descending order of importance are:

(1) Bicarbonate/carbonic acid,
(2) Hemoglobin,
(3) Plasma proteins, and
(4) Erythrocyte and plasma phosphate.

Metabolic acidosis can result from (1) failure of the kidneys to excrete the metabolic acids normally formed in the body, (2) formation of excessive quantities of metabolic acids, (3) intravenous administration of metabolic acids, or (4) addition of metabolic acids by way of the gastrointestinal tract, or (5) loss of alkali from the body fluids. Some of the specific conditions that cause metabolic acidosis are the following: diarrhea, vomiting, uremia, diabetes mellitus, carbonic anhydrase inhibitors, high extracellular fluid potassium concentration.

Since none of these conditions existed, we looked to other areas to learn why 100 percent of the patient population had elevated CO_2 levels. It is our belief at this time that the reasons for this 100 percent elevation is threefold: (1) the population were heavy smokers, (2) there was no organized exercise program until we arrived, and (3) there was a lack of available plasma protein buffers as evidenced by the anion gap values. One who is familiar with laboratory procedures will quickly realize that the predictable CO_2 results might have been much higher than reported. When the oxalated blood is unstoppered for testing, the CO_2 begins to dissipate. In the post-testing 100 percent of the patient population revealed a decrease in their CO_2 levels, although the majority had not returned to normal at the time of post-treatment blood drawing.

The Anion Gap

The anion gap is a mathematical approximation of the difference between the unmeasured anions and cations in the serum. Routinely, electrolyte measurements include NA+, K+, CI-, HCO3- (as total CO2). The unmeasured cations (e.g., Ca++, MG++) average 7 mmEq/L, and the unmeasured anions (e.g., protein-, P04 =) average 24mEq/L. Therefore, normally, there is a net difference of 17mEq/L in unmeasured anions in the serum. If the CI- and total CO_2 concentrations are added together and subtracted from the Na+ and K+ concentrations, the difference should be equal to or less than 17mEq/L. If the anion gap exceeds 17mEq/L, it indicates that an increased amount of one or more of the unmeasured anions is present, with electrical neutrality of the serum being conserved. Etiologies for an increased anion gap are (1) uremia with retention of anions, (2) ketotic states (diabetes, starvation), (3) toxic ingestion (methanol, salicylate), ethylene glycol, and paraldehyde, (4) lactic acidosis (shock), and (5) increased plasma proteins (dehydration). A decreased anion gap (less than lOmEq/L) is seen only rarely, in some patients with multiple myeloma.

TEST RESULTS OF ANION GAP VALUES

$Na+ + K+$
$-Cl- + C0_2$
= Anion Gap Values

Group of nine (only one test result)

Patient		
#4	=	8.1 mEq/L
#6	=	8.2 mEq/L
#7	=	8.6 mEq/L
#9	=	5.5 mEq/L
#11	=	9.5 mEq/L
#12	=	6.1 mEq/L
#16	=	5.5 mEq/L
#22	=	10.3 mEq/L
#23	=	8.3 mEq/L

Group of Twenty with
Pre-Treatment and Post-Treatment Test Results

Patient	Pre-Treatment	Post-Treatment	
#1	9.0 mEq/L	14.8 mEq/L	↑
#2	4.9 mEq/L	10.7 mEq/L	↑
#3	17.9 mEq/L	15.0 mEq/L	↓
#5	9.3 mEq/L	16.7 mEq/L	↑
#8	9.5 mEq/L	14.5 mEq/L	↑
#10	4.1 mEq/L	14.6 mEq/L	↑
#13	12.3 mEq/L	16.1 mEq/L	↑
#14	8.1 mEq/L	16.1 mEq/L	↑
#15	8.6 mEq/L	17.1 mEq/L	↑
#17	12.1 mEq/L	15.1 mEq/L	↑
#18	8.2 mEq/L	14.1 mEq/L	↑
#19	9.7 mEq/L	12.6 mEq/L	↑
#20	7.5 mEq/L	14.7 mEq/L	↑
#21	6.7 mEq/L	13.4 mEq/L	↑
#24	8.5 mEq/L	14.7 mEq/L	↑
#25	6.5 mEq/L	15.2 mEq/L	↑
#26	9.6 mEq/L	14.7 mEq/L	↑
#27	11.6 mEq/L	14.3 mEq/L	↑
#28	5.8 mEq/L	13.5 mEq/L	↑
#29	11.0 mEq/L	13.8 mEq/L	↑

Discussion of Anion Gap Results

These test results appear to be one of the most dramatic and exciting of the entire Study. We feel these astounding results are a first reporting of a finding in alcohol and drug patients that is contrary to the information contained in the current literature. We have shown without question that none of the electrolytes is affected by the use of megadoses of sodium ascorbate, vitamins, minerals, and L-amino acids. Further, the anion gap is also a useful tool for quality control of laboratory results for Na+, K+, C1-, and total C02- We are quite convinced of the validity and accuracy of the electrolyte values by applying the anion gap mathematical technique. Because of the results obtained, there is extremely strong presumptive evidence that the hypotheses of Kwashiorkor, Libby and Stone put forth in their 1977 paper is correct. By mathematically measuring the unmeasured anions in the serum, we are able to show rather conclusively that protein is simply unavailable for use as a blood buffer; hence the metabolic acidosis. The patients were given daily dosages of 22.5 grams of free L-amino acids and the anion gap values reversed themselves in just 29 days. It is important to note how severely depleted these patients' unmeasured anions were (protein), in the pre-treatment testing.

Discussion of Factors That Affect The Plasma Cholesterol

1. An increase in the amount of cholesterol ingested each day increases the plasma concentration slightly. However, when cholesterol is ingested, an intrinsic feedback system for control of body cholesterol causes the liver to compensate to a great extent for this by synthesizing smaller quantities of endogenous cholesterol. As a result, plasma cholesterol concentration usually cannot be changed upward or downward more than ± 15 percent by altering the diet.

2. A saturated fat diet increases blood cholesterol concentration as much as 15 to 25 percent. This presumably results from increased fat deposition in the liver, which then provides increased quantities of acetyl-coenzyme-A in the liver cells for production of cholesterol. Therefore, to decrease the blood cholesterol concentration, it is equally important to maintain a diet low in saturated fat as it is to maintain a diet low in cholesterol concentration. There are other factors that affect the plasma cholesterol concentration, but they do not apply to this patient population.

CHOLESTEROL

Normals - 150-300 mg/dl

Patient	Pre-Treatment	Post-Treatment	Δ%
#1	344	275	20% ↓
#2	164	197	20% ↑
#3	239	220	8% ↓
#5	190	193	2% ↑
#8	184	203	10% ↑
#10	148	129	13% ↓
#13	136	112	18% ↓
#14	139	201	45% ↑
#15	198	238	20% ↑
#17	223	242	8% ↑
#18	185	198	7% ↑
#19	157	206	31% ↑
#20	219	252	15% ↑
#21	195	238	22% ↑
#24	101	160	58% ↑
#25	208	257	24% ↑
#26	262	253	3% ↓
#27	195	180	8% ↓
#28	252	238	6% ↑
#29	228	217	5% ↓

Discussion of Cholesterol Test Results

Most of the cholesterol in the body is used to form cholic acid in the liver. As much as 80 percent of the cholesterol is converted into cholic acid. This, in turn, is conjugated with other substances to form bile salts which promote digestion and absorption of fats.

It appears that contrary to the opinion of modern physiologists that plasma cholesterol concentration usually cannot be changed upward or downward more than ± 15 percent by altering the diet is in error, particularly if you add vitamin C to the altered diet. It appears statistically significant that 12 patients out of 20 showed variations in excess of ± 15 percent.

It is also of interest to note that only one patient (#1) had an elevated cholesterol. Observe the incredible drop of cholesterol in this patient. We feel the other changes in the cholesterol, upward and downward within normal limits, represent body chemistry adjustments toward normal for each patient.

Discussion of Triglycerides Levels

The principal functions of the liver in lipid metabolism are: (1) to degrade fatty acids into small compounds that can be used for energy, (2) to synthesize triglycerides mainly from carbohydrates and to a lesser extent, from proteins, and (3) to synthesize other lipids from fatty acids, especially cholesterol and phospholipids.

Large quantities of triglycerides appear in the liver, (a) during starvation, (b) in diabetes mellitus, or (c) in any other condition in which fat is being utilized rapidly for energy. In these conditions, the triglycerides are mobilized from the adipose tissue, transported as free fatty acids in the blood, and then redeposited as triglycerides in the liver, where the initial stages of much of the fat degradation begin. Thus, under physiological conditions, the total amount of triglycerides in the liver is controlled to a great extent by the overall rate at which lipids are being utilized for energy.

TRIGLYCERIDE TEST RESULTS

Normals - lO-igOmg/d-L

Patient	Pre-Treatment	Post-Treatment	%	
#1	188	37	80%	↓
#2	131	91	30%	↓
#3	481	36	93%	↓
#5	55	131	138%	↑
#8	51	58	14%	↑
#10	73	88	20%	↑
#13	45	21	53%	↓
#14	64	115	80%	↑
#15	100	160	60%	↑
#17	82	79	4%	↓
#18	57	32	44%	↓
#19	90	191	112%	↑
#20	162	286	77%	↑
#21	105	109	4%	↑
#24	127	108	15%	↓
#25	67	90	34%	↑
#26	99	206	108%	↑
#27	86	72	16%	↓
#28	77	71	8%	↓
#29	124	380	206%	↑

Approximately 40 to 50 percent of the calories in the "normal" American diet are derived from fats, which is about equal to the calories derived from carbohydrates. Therefore, the use of fats by the body for energy is equally as important as the use of carbohydrates. In addition, an average of 30 to 40 percent of the carbohydrates ingested with each meal is converted into triglycerides, then stored, and later used as triglycerides for energy. Therefore, as much as two-thirds to three-quarters of all energy derived directly by the cells is supplied by triglycerides rather than by carbohydrates.

Discussion of Triglyceride Test Results The results in this chemical area are not surprising when one considers the amount of sugar each patient ingested daily. Also, when we consider the "junk foods" eaten by these patients, we can quickly see what they used for energy. Increased values may result from a genetic predisposition and are often seen in association with diabetes mellitus. Triglycerides may also be increased by excess of carbohydrate in the diet, particularly free sugar, and in excess of alcohol. It is essential to exclude the presence of diabetes mellitus and alcoholism when hypertriglyceridemia has been detected.

3LOOD CHEMISTRIES THAT WERE CONSIDERED WITHIN ''NORMAL LIMITS'' BY MEDICAL LABORATORY

TOTAL PROTEIN 1.0-8.2 g/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	6.7	6.5	2.3% ↓
#2	7.5	7.5	0
#3	6.6	6.9	4.5% ↑
#5	7.3	7.0	4.1% ↓
#8	7.0	7.4	5.7% ↑
#10	7.5	7.4	1.3% ↓
#13	7.5	7.4	1.3% ↓
#14	6.7	7.9	17.9% ↑
#15	7.2	8.0	11.1% ↑
#17	7.3	7.5	2.7% ↑
#18	6.4	6.7	4.7% ↑
#19	7.3	7.6	4.1% ↑
#20	8.0	8.5	6.2% ↑
#21	7.9	7.5	5.1% ↓
#24	7.3	7.6	4.1% ↑
#25	7.8	7.5	3.8% ↓
#26	7.1	7.3	2.8% ↑
#27	8.2	7.5	8.5% ↓
#28	7.3	7.5	2.7% ↑
#29	8.6	7.7	10.5% ↓

ALBUMIN 3.5-5.0 g/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	3.9	3.7	5.1% ↓
#2	4.1	4.4	7.3% ↑
#3	4.3	4.9	13.9% ↑
#5	4.3	4.3	0
#8	4.3	4.7	9.3% ↑
#10	3.9	4.2	7.7% ↑
#13	4.3	4.3	0
#14	4.1	5.5	34.1% ↑
#15	4.4	5.8	31.8% ↑
#17	4.5	4.9	8.9% ↑
#18	4.1	4.6	12.2% ↑
#19	4.2	4.3	2.4% ↑
#20	3.4	4.5	32.4% ↑
#21	4.7	4.8	2.1% ↑
#24	4.4	4.7	6.8% ↑
#25	4.3	4.5	4.6% ↑
#26	4.1	4.5	9.8% ↑
#27	4.8	4.5	6.2% ↓
#28	4.4	4.7	6.8% ↑
#29	4.6	4.1	10.9% ↓

GLOBULIN 2-3 g/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	2.8	2.8	0
#2	3.4	3.1	8.8% ↓
#3	2.3	2.0	13.0% ↓
#5	3.0	2.7	10.0% ↓
#8	2.7	2.7	0
#10	3.6	3.2	11.1% ↓
#13	2.2	3.1	40.9% ↑
#14	2.6	2.4	7.7% ↓
#15	2.8	2.2	21.4% ↓
#17	2.8	2.6	7.1% ↓
#18	2.3	2.1	8.7% ↓
#19	3.1	3.3	6.4% ↑
#20	5.4	4.0	25.9% ↓
#21	3.2	2.7	15.6% ↓
#24	2.9	2.7	6.9% ↓
#25	3.5	3.0	14.3% ↓
#26	3.0	2.8	6.7% ↓
#27	3.4	3.0	11.8% ↓
#28	2.9	2.8	3.4% ↓
#29	4.0	3.6	10. % ↓

A/G RATIO 1.1-2.2

Patient	Pre-Treatment	Post-Treatment	%
#1	1.4	1.3	7.1% ↓
#2	1.2	1.2	0
#3	1.9	2.5	31.6% ↑
#5	1.4	1.6	14.3% ↑
#8	1.6	1.7	6.2% ↑
#10	1.1	1.3	18.1% ↑
#13	1.3	1.4	7.7% ↑
#14	1.6	2.3	43.8% ↑
#15	1.6	2.6	62.5% ↑
#17	1.6	1.9	18.8% ↑
#18	1.8	2.2	22.2% ↑
#19	1.4	1.3	7.1% ↓
#20	0.6	1.1	83.3% ↑
#21	1.5	1.8	20.0% ↑
#24	1.5	1.7	13.3 ↑
#25	1.2	1.5	25.0% ↑
#26	1.4	1.6	14.3% ↑
#27	1.4	1.5	7.1% ↑
#28	1.2	1.5	25.0% ↑
#29	1.2	1.1	8.3% ↓

GLUCOSE (65-110mg/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	90	92	2.2% ↑
#2	81	86	6.2% ↑
#3	120	132	10.0% ↑
#5	103	96	6.8% ↓
#8	110	99	10. % ↓
#10	73	82	12.3% ↑
#13	110	86	21.8% ↓
#14	85	74	12.9% ↓
#15	96	90	6.2% ↓
#17	90	84	6.7% ↓
#18	92	83	9.8% ↓
#19	82	92	12.2% ↑
#20	87	89	2.3% ↑
#21	109	87	20.2% ↓
#24	92	85	7.6% ↓
#25	94	97	3.2% ↑
#26	85	87	2.4% ↑
#27	100	90	10.0% ↓
#28	86	94	9.3% ↑
#29	99	90	9.1% ↓

URIC ACID (M 2.1-7.8mg/dl
(F 2.0-6.4mg/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	3.0	3.0	0
#2	5.3	4.7	11.3% ↓
#3	5.8	4.8	17.2% ↓
#5	4.5	3.8	15.6% ↓
#8	6.6	5.1	22.7% ↓
#10	4.1	4.6	12.2% ↑
#13	4.8	3.7	22.9% ↓
#14	5.8	8.2	41.4% ↑
#15	5.1	5.1	0
#17	4.1	4.4	7.3% ↑
#18	4.7	3.6	23.4% ↓
#19	7.1	6.3	11.3% ↓
#20	4.4	3.6	18.2% ↓
#21	6.7	6.9	3.0% ↑
#24	4.6	4.5	2.2% ↓
#25	5.5	4.7	14.5% ↓
#26	4.4	4.1	6.8% ↓
#27	6.8	4.4	35.3% ↓
#28	5.3	4.9	7.5% ↓
#29	6.0	5.3	11.7% ↓

BUN (8-22mg/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	14	13	7.1% ↓
#2	12	11	8.3% ↓
#3	13	13	0
#5	13	14	7.7% ↑
#8	14	16	14.3% ↑
#10	15	11	26.6% ↓
#13	9	10	11.1 ↑
#14	10	14	40.0% ↑
#15	12	12	0
#17	11	16	45.4% ↑
#18	14	12	14.3% ↓
#19	10	8	20.0% ↓
#20	10	12	20.0% ↑
#21	15	10	33.3% ↓
#24	12	11	8.3% ↓
#25	15	15	0
#26	15	13	13.3% ↓
#27	13	11	15.4% ↓
#28	14	13	7.1% ↓
#29	14	12	14.3% ↓

CALCIUM (8.5-10.5mg/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	9.4	8.7	7.4% ↓
#2	9.1	9.3	2.2% ↑
#3	9.6	9.7	1.0% ↑
#5	9.0	9.3	3.3% ↑
#8	9.2	9.3	1.1% ↑
#10	8.8	9.3	5.7% ↑
#13	9.7	8.9	8.2% ↑
#14	8.6	10.3	19.8% ↑
#15	8.9	10.5	18.0% ↑
#17	9.6	9.7	1.0% ↑
#18	8.9	9.9	11.2% ↑
#19	9.3	9.7	4.3% ↑
#20	9.3	9.9	6.4% ↑
#21	9.4	9.5	1.1% ↑
#24	8.8	9.0	2.3% ↑
#25	9.4	9.6	2.1% ↑
#26	9.2	9.5	3.3% ↑
#27	9.8	9.5	3.1% ↓
#28	9.4	9.5	1.1% ↑
#29	7.6	9.0	18.4% ↑

CREATININE (0.5-1.5mg/dl

Patient	Pre-Treatment	Post-Treatment	%
#1	1.0	0.9	10.0% ↓
#2	0.8	0.9	12.5% ↑
#3	1.2	1.0	16.7% ↓
#5	0.8	0.9	12.5% ↑
#8	1.1	1.0	9.1% ↓
#10	0.9	0.8	11.1% ↓
#13	0.9	0.8	11.1% ↓
#14	1.0	1.0	0
#15	0.8	1.2	50.0% ↑
#17	0.8	0.9	12.5% ↑
#18	1.0	0.9	10.0% ↓
#19	1.0	1.0	0
#20	0.8	0.8	0
#21	0.8	0.9	12.5% ↑
#24	0.8	0.9	12.5% ↑
#25	1.0	1.2	20.0% ↑
#26	1.0	1.0	0
#27	1.2	1.1	8.3% ↓
#28	0.9	0.9	0
#29	1.4	1.3	7.1% ↓

LACTATE DEHYDROGENASE
(100-225 IU/L)

Patient	Pre-Treatment	Post-Treatment	%
#1	167	159	4.8% ↓
#2	195	188	3.6% ↓
#3	183	198	8.2% ↑
#5	205	179	12.7% ↓
#8	194	190	2.1% ↓
#10	202	187	7.4% ↓
#13	147	138	6.1% ↓
#14	232	215	7.3% ↓
#15	134	90	32.8% ↓
#17	220	173	21.4% ↓
#18	130	173	33.1% ↑
#19	209	275	31.6% ↑
#20	234	325	38.9% ↑
#21	174	209	20.1% ↑
#24	96	190	97.9% ↑
#25	195	201	3.1% ↑
#26	194	185	4.6% ↓
#27	201	158	21.4% ↓
#28	159	189	18.9% ↑
#29	292	365	25.0% ↑

Discussion of Lactate Dehydrogenase Results

Anxiety and hemolysis of RBC's will account for abnormal levels of patients 19, 20, and 29. As stated elsewhere, numbers 20 and 29, ages 45 and 42 respectively, had the longest history of drug abuse and their veins reflected that abuse by the difficulty in penetrating their veins.

Discussion of "Normal" Blood Chemistry Results

It is of extraordinary interest to examine these within "normal" limits test results. It appears highly significant that even within "normal limit" chemistries, there are patient population trends and fine tune adjustments being made in each individual's chemical make-up.

It remains a constant source of amazement what decontamination process, a change of life style, a change of nutritional status, and a little supplementation can accomplish for a patient. **Truly there is better living through chemistry.**

Discussion of Serum Glutamate Oxaloacetic Transaminase (SGOT) and Serum

Glutamate Pyruvate Transaminase (SGPT)

Although a large number of substrate-specific transaminases have been demonstrated in various animal tissues, only two have been described in the serum, glutamate oxaloacetic transaminase (GOT) and glutamate pyruvate transaminase (GPT).

Abnormal levels of GOT are seen in patients with hepatic disease, myocardial and skeletal necrosis, and other diseases of the renal and cerebral tissues. Glutamate pyruvate transaminase (GPT) elevations are absent or slight in disease that does not involve the liver either primarily or secondarily.

PRE-TREATMENT AND POST-TREATMENT VALUES

SGOT 0-40 IU/L

Patient	Pre-Treatment	Post-Treatment	Δ%
#1	60	40	33.3% ↑
#2	130	154	18.4% ↑
#3	26	40	53.8% ↑
#5	39	103	164.1% ↑
#8	15	25	66.7% ↑
#10	58	70	20.7% ↑
#13	60	70	16.7% ↑
#14	300	400	33.3% ↑
#15	37	40	8.1% ↑
#17	55	69	25.4% ↑
#18	20	35	75.0% ↑
#19	134	126	6.0% ↓
#20	36	87	141.7% ↑
#21	20	31	55.0% ↑
#24	14	32	128.6% ↑
#25	28	29	3.6% ↑
#26	20	27	35.0% ↑
#27	30	18	40.0% ↓
#28	20	22	10.0% ↑
#29	600	66	89.0% ↓

SGPT 0-30 IU/L

Patient	Pre-Treatment	Post-Treatment	Δ%
#1	17	28	64.7% ↑
#2	59	59	0
#3	16	34	112.5% ↑
#5	29	59	103.4% ↑
#8	14	19	35.7% ↑
#10	41	91	121.9% ↑
#13	82	51	37.8% ↓
#14	112	380	239.3% ↑
#15	17	29	70.6% ↑
#17	92	40	56.5% ↓
#18	13	15	15.4% ↑
#19	55	49	10.9% ↓
#20	17	62	264.7% ↑
#21	13	20	53.8% ↑
#24	10	25	150.0% ↑
#25	29	14	51.7% ↓
#26	23	17	26.1% ↓
#27	85	12	85.9% ↓
#28	25	11	56.0% ↓
#29	QNS	33	0

Discussion of Glutamate Oxaloacetic Transaminase (GOT) and Glutamate Pyruvate Transaminase (GPT) Test Results

We are discussing these two transaminases together because of the highly dramatic results that have caused us to formulate the following hypothesis for your consideration: First we must restate that the anion gap offers strong evidence that the protein levels were extremely low (supporting the Kwashiorkor theory) and after treatment, they returned to the normal ranges.

The plasma concentration of each type of amino acid is maintained at a reasonably constant value. There is a general principle of reversible exchange of amino acids among the different proteins of the body. Even with starvation or during severe debilitating diseases, the ratio of total tissue proteins in the body to total plasma proteins remains relatively constant at about 33 to one. Except for the excess protein in the diet, or the 30 grams of obligatory protein degradation each day, the body uses almost entirely carbohydrates or fats for energy as long as these are available. However, after several weeks of starvation, when the quantity of stored fats begins to run out, the amino acids of the blood begin to be rapidly deaminated and oxidized for energy. From this point on, the proteins of the tissues degrade rapidly—as much as 100 grams daily—and the cellular functions deteriorate precipitously.

As is true for monosaccharide absorption, very little is known about the basic mechanisms of amino acid transport. However, it is known that four different carrier systems transport different amino acids. One transports neutral amino acids, a second transports basic amino acids, a third transports acidic amino acids, and a fourth has specificity for two amino acids, proline and hydroxyproline. Also, the transport mechanisms have far greater affinity for transporting L-stereoisomers of amino acids over D-stereoisomers. (In the treatment aspect, we used only L-amino acids.) Experiments have demonstrated that pyridoxal phosphate, a derivative of the vitamin pyridoxine, is required for transport of many amino acids.

Transamination is promoted by enzymes called transaminases, all of which are derivatives of pyridoxines. Without this vitamin, the non-essential amino acids cannot be synthesized and, therefore, protein formation cannot proceed normally.

Amino acid transport, like glucose transport, occurs only in the presence of simultaneous sodium transport. Furthermore, the carrier systems for amino acid transport, like those for glucose transport, are in the brush border of the epithelial cell. It is currently believed that amino acids are transported by the same sodium gradient mechanism as that of glucose transport.

In order to explain our position in the GOT and GPT serum values, we must digress a bit to carbohydrate absorption. Essentially all of the carbohydrates are absorbed in the form of monosaccharides, only a small fraction of a percent is being absorbed as disaccharides and almost none as larger carbohydrate compounds. The transport is selective, specifically transporting certain monosaccharides without transporting others. The order of preference for transporting different monosaccharides is: (1) galactose, (2) glucose, (3) fructose, (4) mannose, (5) xylose, and (6) arab-inose.

Galactose and glucose transport becomes blocked whenever sodium transport is blocked. Therefore, it is assumed that the energy required for most, if not all, monosaccharide transport, is actually provided by the sodium transport system. A theory that attempts to explain this is the following: it is known that the carrier for transport of glucose and some other monosaccharides, especially galactose, is present in the brush border of the epithelial cell. However, this carrier will not transport the glucose in the absence of sodium transport. Therefore, it is believed that the carrier has a receptor site for both a glucose molecule and a sodium ion, and that it will not transport the glucose to the inside of the cell if the receptor sites for both glucose and sodium are not simultaneously filled. The energy to cause movement of the carrier from the exterior of the membrane to the interior is derived from the difference in sodium concentration between the outside and inside. That is, as sodium diffuses to the inside of the

cell, it "drops" the carrier and therefore the glucose with it, thus providing the energy for transport of the glucose. For obvious reasons, this explanation is called the sodium gradient theory for glucose transport.

It is recognized that sodium transport is also required for transport of amino acids, suggesting a similar theory of "carrier-drag" mechanism for amino acid transport. This theory postulates that the carrier has a receptor site for both an amino acid molecule and a sodium ion. Only when both of the sites are filled, will the carrier move to the interior of the cell. Because of the sodium gradient across the brush border, the sodium diffusion to the cell interior pulls the carrier and its attached amino acid to the interior where the amino acid becomes trapped. Therefore, their concentrations increase within the cell, and they then diffuse through the sides or base of the cell into the portal blood.

We have clearly stated the foregoing to establish a foundation for what we believe has occurred to account for the GOT and GPT non-pathological serum elevations**.** We have already discussed the anion gap and its relationship to protein. Further evidence of the protein deficiencies and imbalances is reflected in observing the values of the total proteins, albumin, globulin and A/G ratios. Observe the dynamic activity that has occurred. Next, observe a similar phenomenon occurring in glucose metabolism. Third, as previously reported, the 24-hour urine Cortisol levels were balanced. Also, as previously reported, consider the amount of teaspoon equivalents of sugar intake each patient admitted to taking daily. Consider the impact this intake had on the sodium transport system. It is our opinion at this time that via means of orthomolecular applications of vitamins, minerals, L-amino acids and sodium ascorbate, we have eliminated the chronic deficiencies and placed the body chemistry in a state of dynamic equilibrium. More importantly, by using sodium ascorbate, it appears we balanced the critical sodium transport system, and by accomplishing that, carbohydrate and protein metabolism could then proceed to operate in a normal manner. Adding a total of 22.5 grams of free L-amino acids in three equally spaced dosages daily for 29 days, we corrected the chronic protein deficiencies, as evidenced by the adjustment of the anion gap. As explained earlier, if a deficiency of vitamin B6 is present, the protein formation could not proceed normally, so we took the precaution to correct this possible hazard. With each of the three equally spaced dosages of ten L-amino acids, we gave one 500mg B6 tablet. In addition to the l,500mg of vitamin B6 daily, there was included in the other formulas an extra 180mg of B6 added for a period of eight days, making a grand total of l,680mg of B6 daily. For the remainder of the treatment days, there was a daily total of l,560mg of vitamin B6 given. Since transamination is promoted by enzymes called transaminases, all derivatives of pyridoxine, we simply supplied too much of vitamin B6, causing this non-pathological rise in the SGOT and SGPT levels.

Pre-Treatment URINALYSIS TEST RESULTS

Patient #	Color	Sp.Gr.	PH	Protein (qual)	Glucose (qual)	Ketones	Occult
#1	clear yellow	1.026	6.5	neg.	neg.	neg.	neg.
#2	clear yellow	1.024	6.0	neg.	neg.	neg.	neg.
#3	clear yellow	1.27	6.5	neg.	neg.	neg.	neg.
#5	clear yellow	1.015	6.5	neg.	neg.	neg.	neg.
#8	clear yellow	1.026	6.5	neg.	neg.	neg.	neg.
#10	clear yellow	1.026	6.0	neg.	neg.	neg.	neg.
#13	clear yellow	1.028	6.0	neg.	neg.	neg.	neg.
#14	clear amber	1.027	6.0	neg.	neg.	neg.	neg.
#15	clear yellow	1.026	6.0	neg.	neg.	neg.	neg.
#17	clear yellow	1.028	6.0	neg.	neg.	neg.	neg.
#18	clear yellow	1.034	6.0	neg.	neg.	neg.	neg.
#19	clear yellow	1.015	6.0	neg.	neg.	neg.	neg.
#20	clear yellow	1.026	6.0	neg.	neg.	neg.	neg.
#21	clear yellow	1.010	7.0	neg.	neg.	neg.	neg.
#24	clear yellow	1.030	6.0	neg.	neg.	neg.	neg.
#25	hazy yellow	1.025	6.0	neg.	neg.	neg.	neg.
#26	clear yellow	1.030	6.0	neg.	neg.	neg.	neg.
#27	hazy yellow	1.027	6.0	neg.	neg.	neg.	neg.
#28	clear yellow	1.028	6.5	neg.	neg.	neg.	neg.
#29	clear yellow	1.023	6.0	neg.	neg.	neg.	neg.

Pre-Treatment URINALYSIS MICROSCOPIC

Patient	WBC	RBC	BACT.	CRYSTALS	CASTS	MUCUS	EP.CELLS
#1	1-3	0	0	1+Ca.Oxalate	0	1+	few
#2	1-3	0	0	0	0	1+	few
#3	0-4	0-2	0	2+Ca.Oxalate	0	1+	few
#5	0-2	0	0	rare Ca.Oxalate	0	1+	few
#8	0-3	0-2	0	1+Ca.Oxalate	0	1+	few
#10	0-2	0-2	0	0	0	2+	0-1
#13	0-3	0-1	0	1+Ca.Oxalate	0	1+	few
#14	0-2	0	0	0	0	2+	few
#15	0-1	0-1	0	0	0	2+	few
#17	0-2	0-1	0	1+Ca.Oxalate	0	2+	few
#18	0-3	0	0	1+ amorph.	0	4+	few
#19	few	0	0	0	0	0	0
#20	2-4	0	0	1+Ca.Oxalate	0	1+	6-8
#21	0-2	0	0	0	0	0	0-2
#24	0-2	0	0	1+Ca.Oxalate	0	1+	0
#25	0	0	0	few Ca.Oxalate	0	3+	few
#26	0	0	0	1+Ca.Oxalate	0	3+	0
#27	0-3	0-1	0	2+Ca.Oxalate	0	2+	few
#28	0-2	0-2	0	1+ amorph.	0	0	0
#29	0	0	0	rare Ca.Oxalate	0	1+	few

Post-Treatment URINALYSIS MICROSCOPIC

PATIENT	RBC	BACT.	CRYSTALS	CASTS	MUCUS	EP.CELLS
#1	0-1	0	0	0	0	few
#2	0	0	0	0	0	0
#3	0	0	0	0	0	0
#5	0	0	0	0	0	0
#8	0	0	0	0	0	0
#10	0	0	0	0	0	0
#13	0	0	0	0	occ.	few
#15	0	0	0	0	0	0
#17	0	0	0	0	0	0
#18	0	0	0	0	0	0
#19	0	0	0	0	0	0
#20	0	1-3	0	0	occ.	few
#21	0	0	0	0	0	0
#24	0	0	0	0	0	0
#25	0	0	0	0	0	0
#26	0	0	0	0	0	0
#27	0	0	0	0	0	0
#28	0	0	0	0	0	0
#29	0	0	0	0	0	0

Post-Treatment URINALYSIS

Patient	Color	Sp.Gr.	PH	Protein (qual)	Glucose (qual)	Ketones	Occult Blood
#1	clear yellow	1.010	5.5	0	0	0	0
#2	clear yellow	1.022	6.0	0	0	0	0
#3	clear yellow	1.012	5.5	0	0	0	0
#5	clear yellow	1.020	6.0	0	0	0	0
#8	clear lemon	1.018	6.0	0	0	0	0
#10	clear yellow	1.015	6.0	0	0	0	0
#13	clear yellow	1.017	6.5	0	0	0	0
#14	clear yellow	1.022	6.0	0	0	0	0
#15	clear yellow	1.012	6.0	0	0	0	0
#17	clear yellow	1.010	6.0	0	0	0	0
#18	clear yellow	1.010	5.5	0	0	0	0
#19	clear yellow	1.018	5.0	0	0	0	0
#20	clear yellow	1.020	6.0	0	0	0	0
#21	clear yellow	1.022	6.0	0	0	0	0
#24	clear lemon	1.018	5.5	0	0	0	0
#25	clear yellow	1.012	5.5	0	0	0	0
#26	clear yellow	1.016	5.5	0	0	0	0
#27	clear yellow	1.015	5.5	0	0	0	0
#28	clear yellow	1.010	6.0	0	0	0	0
#29	clear yellow	1.012	6.0	0	0	0	0

Discussion of Urinalysis Results

Another effective demonstration of the value of detoxifying and decontaminating the body. The pre-treatment and post-treatment microscopic examination tells it all. Notice there are no WBC, RBC, casts, crystals or mucus threads. Also notice the change in the specific gravity.

Pre-Treatment HEMATOLOGY

Patient	RBC	Hemo-globin	Hema-tocrit	MCV	MCHC	MCH	Platelet	Morph.
#1	3.86	13.1	3.9	98	34.5	33.6	App.Adq.	Norm.
#2	4.98	14.7	45.3	90	32.7	29.3	App.Adq.	Norm.
#3	5.10	14.8	41.1	81	34.7	27.8	App.Adq.	
#5	4.62	14.7	44.8	96	33.1	31.5	App.Adq.	Norm.
#8	5.37	15.5	45.8	83	34.9	28.5	App.Adq.	Norm.
#10	5.56	13.8	43.6	78	31.6	24.6	App.Adq.	Norm.
#13	4.92	14.1	42.9	85	33.8	28.5	App.Adq.	Norm.
#14	4.88	14.9	45.0	91	33.1	30.1	App.Adq.	Norm.
#15	5.47	15.2	46.2	84	33.0	27.5	App.Adq.	Norm.
#17	4.80	14.5	43.4	88	34.2	29.7	App.Adq.	Norm.
#18	4.53	13.0	39.0	85	33.3	28.3	App.Adq.	Norm.
#19	6.01	16.9	53.8	89	31.5	27.8	Adq.	Norm.
#20	4.16	11.0	35.2	84	31.4	26.2	Adq.	Norm.
#21	5.82	16.9	50.9	86	33.2	28.5	App.Adq.	Norm.
#24	4.29	13.0	39.2	90	33.2	30.1	App.Adq.	Norm.
#25	5.06	15.6	47.0	92	33.5	30.4	App.Adq.	Norm.
#26	5.18	15.5	47.7	91	32.8	29.6	App.Adq.	Norm.
#27	5.40	16.0	47.7	86	34.5	29.3	App.Adq.	Norm.
#28	5.18	14.6	45.4	87	32.3	27.8	App.Adq.	Norm.
#29	5.45	16.4	50.8	92	32.5	29.8	App.Adq.	Norm.

Pre-Treatment HEMATOLOGY

Patient	WBC	SEGS	BANDS	EOSIN	BASO	LYMPHS	MONO	MORPH.
#1	6100	54	2	2	0	33	9	Norm.
#2	6100	50	3	9	0	32	6	Norm.
#3	5300	50	2	6	0	34	9	Norm.
#5	5500	52	0	5	1	31	6	5ATYP.
#8	8700	38	3	4	0	38	9	6ATYP.
#10	7600	49	2	5	1	34	4	5ATYP.
#13	6800	50	1	4	0	39	6	Norm.
#14	6100	34	1	9	0	40	3	13ATYP.
#15	6200	69	0	4	0	27	0	7ATYP.
#17	4900	56	2	1	0	33	8	Norm.
#18	6200	58	2	0	0	36	2	2ATYP.
#19	9300	68	1	2	0	26	1	Norm.
#20	6500	54	4	1	0	34	3	4ATYP.
#21	12,200	78	0	1	0	19	2	Norm.
#24	6500	59	0	3	0	33	1	4ATYP.
#25	8400	66	1	4	0	25	4	Norm.
#26	8200	49	0	10	0	32	9	Norm.
#27	7900	40	2	3	0	45	5	5ATYP.
#28	8400	54	0	3	0	39	4	Norm.
#29	8900	54	4	3	0	26	3	10ATYP.

The nine patients who were released from the program had pre-treatment hematologies and we want to bring to your attention the atypical, granular lymphocytes. Patient #4 had 1 atypical, granular lymphocyte out of 32 counted. Patient #9 had 4 out of 37 counted. Patient #12 had 7 out of 33 counted. Patient #22 had 2 out of 33.

Post-Treatment HEMATOLOGY

Patient	RBC	Hemo-globin	Hema-tocrit	MCV	MCHC	MCH	Platelets	Morph.
#1	4.04	13.4	38.1	96	35.3	35.1	App.Adq.	Norm.
#2	4.92	15.3	43.1	89	35.7	32.8	App.Adq.	Norm.
#3	5.5	15.6	47.7	92	33.6	30.4	App.Adq.	Norm.
#5	4.53	15.1	41.4	93	36.6	35.3	App.Adq.	Norm.
#8	5.55	16.0	45.2	83	35.4	30.5	App.Adq.	Slt.anso
#10	5.45	14.1	42.1	79	33.7	27.5	App.Adq.	Norm.
#13	5.03	14.6	41.5	84	35.1	30.7	App.Adq.	Norm.
#14	5.35	16.8	47.7	91	35.2	33.1	App.Adq.	Norm.
#15	5.41	15.8	44.6	84	35.5	30.9	App.Adq.	Norm.
#17	4.96	15.1	42.3	87	37.7	32.2	App.Adq.	Norm.
#18	4.68	13.8	40.2	88	34.2	31.2	App.Adq.	Slt.anso Slt.Polk
#19	6.15	18.6	53.3	89	35.1	32.0	App.Adq.	Norm.
#20	5.10	14.8	42.7	86	34.9	30.7	App.Adq.	Slt.anso Slt.Polk
#21	5.39	16.3	45.1	86	36.1	32.0	App.Adq.	RBC Slt.anso
#24	4.69	13.9	40.9	88	34.3	31.3	App.Adq.	Norm.
#25	5.12	16.2	64.9	90	36.1	33.6	App.Adq.	Norm.
#26	5.40	16.9	47.1	89	36.0	33.0	App.Adq.	Norm.
#27	5.18	15.6	43.5	86	35.8	31.9	App.Adq.	Norm.
#28	5.19	15.8	43.3	85	36.6	32.2	App.Adq.	Norm.
#29	5.4	17.0	47.2	89	36.6	33.5	App.Adq.	Norm.

Post-Treatment HEMATOLOGY

Patient	WBC	Segs.	Bands	Eosin	Base	Lymphs	Mono	Morph.
#1	8600	62	9	2	1	22	4	Norm.
#2	7000	43	0	4	0	37	4	12ATYP.
#3	5600	61	4	1	0	32	2	Norm.
#5	4800	61	1	3	1	3	4	Norm.
#8	7900	56	1	1	0	27	9	6ATYP.
#10	7600	49	5	9	0	30	7	Norm.
#13	5900	54	3	1	7	27	0	8ATYP.
#14	6600	75	0	0	0	24	1	Norm.
#15	4900	52	0	1	0	42	5	Norm.
#17	6600	59	2	3	0	27	4	5ATYP.
#18	5800	58	2	1	0	35	4	N
#19	9300	57	1	0	0	37	5	Norm.
#20	6200	27	1	2	0	57	2	11ATYP.
#21	5800	51	0	0	0	34	9	6ATYP.
#24	6100	59	3	2	0	32	4	Norm.
#25	8900	58	3	4	0	22	6	7ATYP.
#26	7000	56	1	6	1	32	4	Norm.
#27	8200	54	0	5	0	32	4	Norm.
#28	7700	54	0	5	0	35	6	Norm.
#29	8100	54	1	4	0	30	4	7ATYP.

Discussion of Hematology Results

The values of the hematologies speak for themselves as a remarkable balancing of the hemapoietic system. Rather than elaborate on the several changes, we will concentrate instead on an extremely interesting phenomenon occurring with the lymphocytes. Due to our prior experience with the alcohol and drug patient, we had asked the medical laboratory who donated their services for this Study if they could provide a hematologist to study the lymphocytes for toxic granulations in the cytoplasm. This request could not be met. The reason it was asked prior to the Study was the fact this phenomenon had repeatedly been observed in the drug patient, but strangely enough, not in the alcoholic patient. These lymphocytes had been reported by medical laboratories we had used and they reported them as "toxic granulations", either 1+, 2+ or 3+. We felt it important to investigate these atypical cells more closely in a controlled Study.

As predicted by the senior investigator, 10 out of the original 20 patients had from a low of 2 to a high of 13 of these atypical "toxic granulation" lymphocytes. These "toxic granulations" were not understood, but were often observed over the past four years. Typing of these cells was not economically possible, so we are unable to state whether these lymphocytes were T cells or B cells.

Based on current literature on the subject, it appears that these lymphocytes are of the T cell variety for the following reasons: within the last seven years a cell has been discovered called "Natural Killer" (NK) cells. These NK cells have been linked with lymphocytes and recent evidence (1981) indicates that virtually all human and rat NK activity is mediated by large granular lymphocytes (LGL). This recent study with highly enriched populations of LGL, various tumor cell lines, viruses, mitogens, bacterial, and other adjuvants were shown to induce considerable production of interferon after culture overnight. It was noted that during

these short-term incubations, only the LGL and not T cells or monocytes produced interferon in response to most of the stimuli.

One bit of difficulty, however, is that none of the authors, or for that matter none of the medical laboratory staff we used in this Study, have knowingly observed an NK cell in the microscope. Therefore, it is not known with any certainty whether these "atypical" lymphocytes are LGL cells or NK cells.

As noted in the pre-treatment and post-treatment results, 10 of the 20 patients (excluding the 9 patients who had only one examination) demonstrated what appears *to* be LGL cells. What is interesting to note is that patients #5(5), #10(5), #14(13), #15(7), #18(2), #24(4), and #27(5) lymphocytes returned to normal in the post-treatment examination. However, what at this time is unexplainable, is that several patients who had normal lymphocytes on the first examination, developed LGL cells on the second examination. Patients #2(12), #13(8). #17(5), #21(6), and #25(7) demonstrated this number of atypical lymphocytes. What is even more perplexing to the investigators is patient #8(8) remained the same, patient #20 elevated from 4 to 11, and #29 decreased from 10 to 7.

It would be of interest to determine more clearly the lineage of NK cells and their relation to the T cell and myelomonocytic lineages; the nature of the recognition receptors on NK cells and of the antigens on the target cells; the detailed mechanisms for the regulation of their activity; and the biochemical sequence of events that lead to their lysis of target cells. The recent findings of the intimate association between NK cells and LGL, and the concomitant ability to purify these cells and to expand their members in culture, have provided an excellent basis for further studies on these questions. It would also be of interest to determine the nature of the granules in LGL and whether these granules play an important role in their functions, particularly in relation to drug patients and the production of interferon.

AREAS OF SPECIAL INTEREST: EOSINOPHIL COUNT

(normal 2-3)

Patient	Pre-	Post-	%	
#1	2	2	0	
#2	9	4	5	↓
#3	6	2	4	↓
#5	5	3	2	↓
#8	4	1	3	↓
#10	5	9	4	↑
#13	4	1	3	↓
#14	9	0	9	↓
#15	4	1	3	↓
#17	1	3	2	↑
#18	0	1	1	↑
#19	2	0	2	↓
#20	1	2	1	↑
#21	1	0	1	↓
#24	3	1	2	↓
#25	4	4	0	
#26	10	6	4	↓
#27	3	5	2	↑
#28	3	5	2	↑
#29	3	4	1	↑

Areas of Special Interest

Patients #2(9), #3(6), #14(9), and #26(10) had Eosinophil counts of twice normal or above. On three consecutive mornings a stool examination was collected for Ova and Parasite examination. All 4 patients proved to be negative for Ova and Parasite contamination. All 4 of the aforementioned patients demonstrated decreases in their Eosinophil count on post-treatment examination. Of the 20 patients, 2 showed no change, 7 demonstrated increases, and 11 decreased on the post-treatment examination. These changes are unaccounted for at the present time.

Evaluation of Voice Recordings of Patients

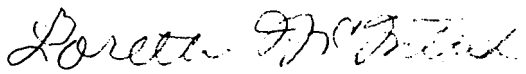
Throughout these papers we have singled out patient #20, largely because she had been in this facility for six months prior to our arrival and to test our hypothesis that the patients' voices improved, we hired a retired U.S. Treasury Department Agent who had been in charge of narcotic enforcement for California, Nevada and Arizona. He was declared a narcotic expert witness in Federal and State courts. He and his staff stated her voice showed "stress" when she spoke of the pain she was feeling. Later on, "no stress" when she stated she was "feeling great," and "no stress" regarding drugs. They also indicated her voice levels increased considerably over the first recordings—"more forceful and animated."

Examination of Patients' Handwriting

On each occasion when the patients received their supply of vitamins, minerals, and L-amino acids, they were required to sign their name for close patient control purposes. Once again, we used patient #20 for signature analysis changes.

Experience has shown us that as the mental, emotional and physical status improves, so do the voice and signatures. We again hired a retired U.S. Treasury Department Agent who was the Supervisor of Questioned Documents, Fingerprints and Photographs for all divisions of the Treasury Department in a 38 state area. He was also qualified as a Document Examiner in Federal, State, Military and Tax courts in 38 states. It was this man's expert opinion that patient #20's signature became clearer. Perhaps you should see for yourself and judge.

5-26-80 Signature



7-3-80 Signature

Conclusions

1. Looking carefully at the electrolyte values, it is extremely easy to see that the megadoses neither disrupt the electrolyte balance, nor does the use of sodium ascor-bate in mega dosages affect the serum sodium levels.

2. The Anion Gap appears to be an important finding in the drug and alcohol culture. Obviously the majority of the patients did not have multiple myeloma that was corrected in 29 days.

3. This paper demonstrates irrefutable evidence that all addictions represent serious physical illness. This illness affects behavior and/or pathological functioning of each system.

4. We collected pre-treatment and post-treatment 24-hour urine specimens for a quantitative amino acid assay to be done by column chromatography. Our purpose was to demonstrate not only the Kwashiorkor these patients were afflicted by, but the corrective therapy that would occur by the administration of the 22.5 grams of L-amino acids. An arrangement was made with Francis S. Markland, Ph.D., Associate Professor of Biochemistry, LAC-USC Cancer Center, to perform these assays. The urine specimens were submitted. No results were ever obtained.

5. The dramatic percentile changes that occurred within the "normal limits" blood chemistries, should establish very clearly that the use of vitamins, minerals and L-amino

acids in orthomolecular dosages ("The right molecules in the right amounts and delivered to the right place to assure optimal health") does not—I repeat does not—change a vitamin or mineral to a drug effect in the organ system. Putting ice cream on a piece of pie merely creates pie a la mode, and this act does not suddenly and mystically make this a medication!

6. From the beginning to the end of the Study took 40 days, ending during July, 1980. As a result of our on-going research, we are now able to accomplish all that we have reported on in the short space of just 15 days instead of the 40 days it previously took us, and even more efficiently than before. With predictable modifications, we obtain the same dramatic results in an acute detox setting with alcohol and drug addiction.

7. The most dramatic conclusions we must draw from the successes reported in this paper are the following:

The lack of proper food intake will lead to inadequate vitamin availability. More significantly, the lack of protein intake leads to a negative nitrogen balance which translates into the fact that these patients will have a diminished capacity to synthesize apoenzymes. So, despite the fact that they have adequate vitamins in their circulation, they will be unable to utilize these vitamins as coenzymes to form enzymes. The reverse is also true; they may have vitamin deficiencies, but sufficient apoenzymes, yet enzyme metabolism will still suffer. The key is to decontaminate the body first and then change the negative nitrogen balance to a positive nitrogen balance by the addition of L-Amino acids, given in sufficient therapeutic amounts. Damaged liver cells lose their ability to bind vitamins; the dire consequence of this fact is quite clear.

Summary

From this and the preceding two papers, it must now be obvious that the alcohol and drug patients heretofore have not been given an equal opportunity to get well as have other patients who are suffering from metabolic dysfunctions. The archaic and oftentimes barbaric techniques employed in treating these patients is reprehensible and must be changed.

We feel these papers will lead the way in encouraging other disciplines to explore the usage of orthomolecular techniques in their particular specialties.

We apologize that there were certain areas we could not tie down in this Study. The problem was one of finances and lack of equipment. We intend to pursue these areas vigorously and will report our progress just as soon as we are able.

Bibliography

BHAGAVAN, A.: (Nutrition Research Coordinator, Hoffman-LaRoche): Personal Communication Regarding Transaminases, 1981.

GOODALE, R.H.: Clinical Interpretation of Laboratory Tests, 1965.

GOODHART, R.S. and SHILLS, M.E.: Modern Nutrition in Health and Disease, 1975.

GUGTON, A.C.: Textbook of Medical Physiology, 1976.

HALSTEAD, J.A.: The Laboratory in Clinical Medicine, 1976.

HERBERMAN, R.B. and ORTALDO, J.R.: Natural Killer Cells: Their Role in Defenses Against Diseases. Science, pp. 24-29, October 2, 1981.

HOFFMAN, W.S.: The Biochemistry of Clinical Medicine, 1970.

KLENNER, F.: Personal Communication —Regarding Non-Dissociation of Sodium Ascorbate, 1978.

PAULING, L.: Personal Communication —Regarding Fate of Vitamin C in Body, 1978.

SCRIVER, C.R. and ROSENBERG, L.E.: Amino Acid Metabolism and Its Disorders, 1973.

SKINNER, S.L.: Cause of Erroneous Potassium Levels. Lancet 1, 478, 1961.

TODD, SANFORD and DAVIDSON: Clinical Diagnosis and Management by Laboratory Methods. Sixteenth Edition, 1979.

VALTIN, H.: Renal Function: Mechanisms Preserving Fluid and Salute Balance in Health. Bosto, Little, Brown and Co., p. 149, 1973.