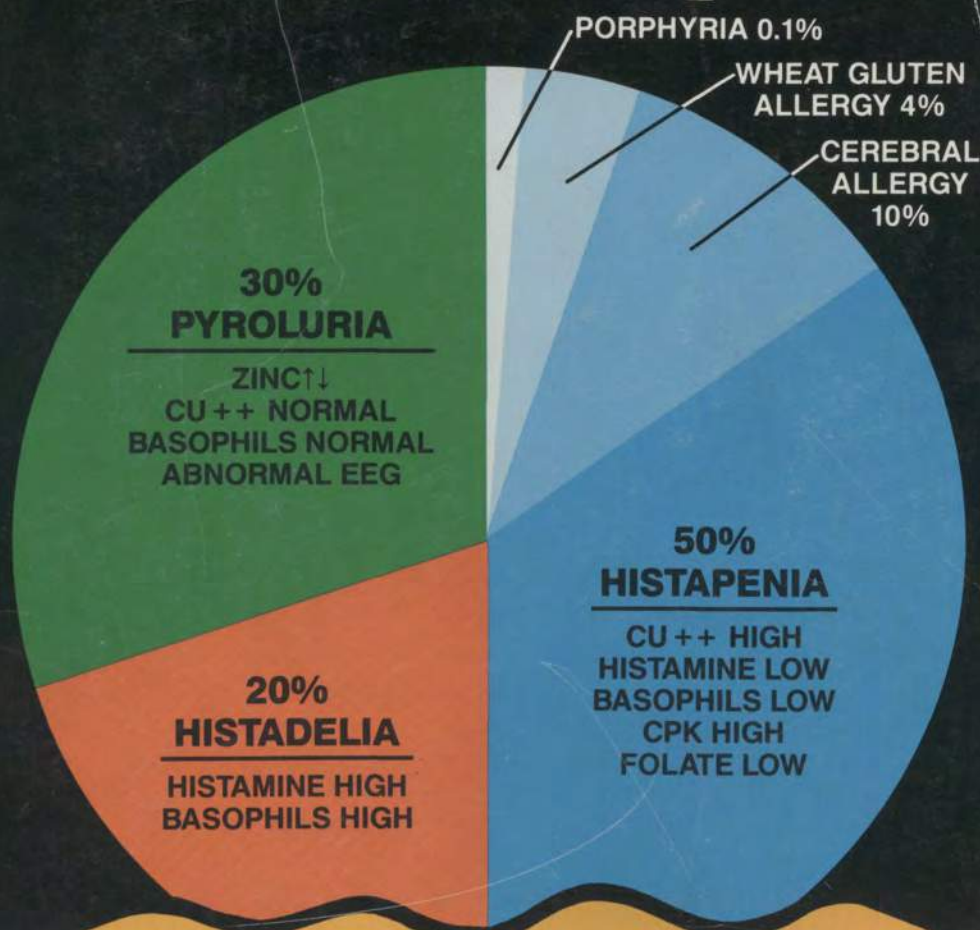


THE SCHIZOPHRENIAS

Ours to Conquer



HYPOGLYCEMIA

Carl C. Pfeiffer, Ph.D., M.D.,
Richard Mailloux, B.S. and Linda Forsythe, B.A.

THE SCHIZOPHRENIAS: OURS TO CONQUER

by

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Richard Mailloux, B.S.
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THE SCHIZOPHRENIAS: OURS TO CONQUER
Bio-Communications Press / January 1988

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For information address:

BIO-COMMUNICATIONS PRESS
3100 North Hillside Avenue
Wichita, Kansas 67219 USA

ISBN 0-942333-02-0
(previously published by Pyramid Books, ISBN 0-515-02178-4)
Library of Congress Catalog Card Number: 87-72306

Revised Edition

Published in The United States of America

BCP and Bio-Communications Press are service marks of
The Olive W. Garvey Center
for the Improvement of Human Functioning, Inc.

ACKNOWLEDGEMENTS

The authors wish to thank Sarah Rodgers, Phyllis Lofton and Marie Arcaro for excellent typing and secretarial assistance in the preparation of this book. They never exclaimed, "What—another revision?"

The authors also wish to thank Keith Jordan for The Easy Alpha Test.

This book is dedicated to the many patients who had sufficient faith in a new approach to the schizophrenias to tolerate the several ups and downs in their mental health as we slowly determined biochemical causes. We also appreciate the devotion of our underpaid professional staff who are motivated to continue because the patients' health and even their personal health is improved by the nutritional approach.

Special thanks must be given to Professor Roger Williams and Donald R. Davis, Ph.D., who allowed Lianne Audette and Norma Rahn to make the colored charts of their cartwheel graphs depicting nutrients in food and diets (see back cover).

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PREFACE

by A. Hoffer, M.D., Ph.D.

Schizophrenia is a syndrome, not a single disease. It is a collection of diseases characterized by two main sets of symptoms and signs: perceptual and thinking disorders. Carl Pfeiffer and his colleagues properly emphasize the plurality by use of The Schizophrenias. This conclusion is accepted by orthomolecular psychiatrists who base treatment using this concept, and by most research psychiatrists even when they insist on treating them all with similar tranquilizers. For a new approach to the schizophrenias we must credit the senior author of this book.

The term schizophrenia was first used by Eugene Bleuler about eighty years ago. He classified the group of schizophrenias behaviorally into clinical or psychological types like paranoid, catatonia, etc. This classification has little interest today for it does not matter whether they are called paranoid or simple—the drug treatment is always the same. A second classification has been one by default. If the schizophrenia was cured by specific treatment it was reclassified as a neurological disease. In 1900, the four main schizophrenias were general paresis of the insane (GPI), scurvy, pellagra and dementia praecox. Penicillin cured GPI and paretics were rediagnosed as a neurological syndrome. Diet and vitamin B-3 cured pellagra and these schizophrenics were rediagnosed then as pellagrins. Scorbatic psychoses vanished as did scurvy and dementia praecox was replaced by schizophrenia. The only schizophrenics that psychiatrists continued to treat were the ones with no cause and no specific treatment.

Psychiatrists should have realized that if so many schizophrenics were caused by biochemical abnormalities, more would be found. But attempts to discover other

PREFACE

causes of schizophrenia failed because so much effort was wasted trying to relate simple biochemical findings to the Bleulerian sub-types. Additional effective specific treatments were needed. This was given a great impetus when Dr. Humphrey Osmond and I showed that acute schizophrenic patients were much better off when treated with optimum doses of Vitamin B-3. This demonstrated for the first time that we had another group—those responsive to Vitamin B-3.

Orthodox psychiatrists continued in vain to search for a single causative factor (and still do) which might show a connection to the real schizophrenia. Orthomolecular psychiatrists began to use biochemical abnormalities to identify schizophrenic biotypes. Instead of using factor A to characterize all schizophrenics, we began to ask how do all patients with factor A differ from patients without factor A. This would then point to a specific biochemical treatment.

Our leading pioneer in this scientific way of diagnosing and treating the schizophrenias is Carl C. Pfeiffer, assisted by his excellent team of Brain Bio Center associates and his colleagues. Carl Pfeiffer and associates divided the schizophrenias into a number of subtypes based upon biochemical findings and accurate clinical observations. These diagnostic groups have a reality never before attainable. They are described in this book. Not only are they diagnostically homogeneous, but they can be treated rationally, i.e. treatment is determined by the biochemical diagnosis.

The word schizophrenia is bad. It frightens patients and their families. It gives others the wrong impression about the disease, and it is misused by writers and politicians who believe it means being changeable or even two-faced and they use the word schizophrenia to insult each other. A rational biochemical diagnostic system will help us get rid of the schizophrenias. It will be replaced by

PREFACE

rational terms such as pyroluria, histadelia, histapenia, cerebral allergy, and so on. All these syndromes which cause bizarre mental quirks are described in this book.

This impressive volume contains the observations and theories about the schizophrenias gathered by Carl Pfeiffer and colleagues over more than twenty years. I have heard Dr. Pfeiffer lecture dozens of times and have always enjoyed listening to his clear exposition of his work. More important, I always learn something. Unfortunately, too few psychiatrists have been wise enough to attend his lectures. They can remedy this deficiency by studying this book, and then coming to his lectures to learn more!

Today, with orthomolecular treatment, schizophrenic patients have the best prognosis since 1955 when nutrient treatment first expressed itself. For this they should always be grateful to the orthomolecular scientists. In my opinion, patients denied the benefits of this fine body of scientific work will one day be able to sue their therapists for egregious ignorance and even for malpractice.

EXPLANATION OF CARTWHEEL DIAGRAMS

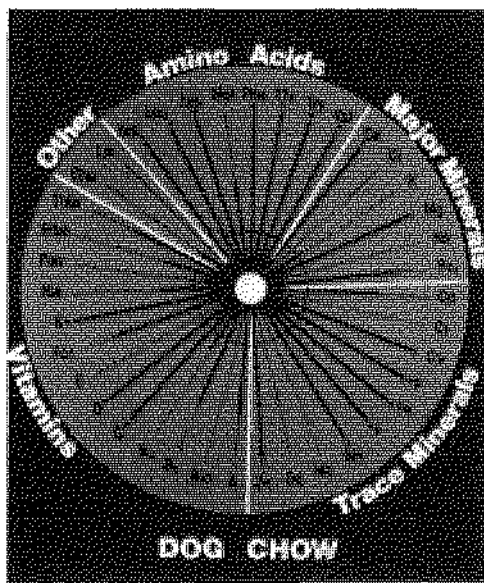
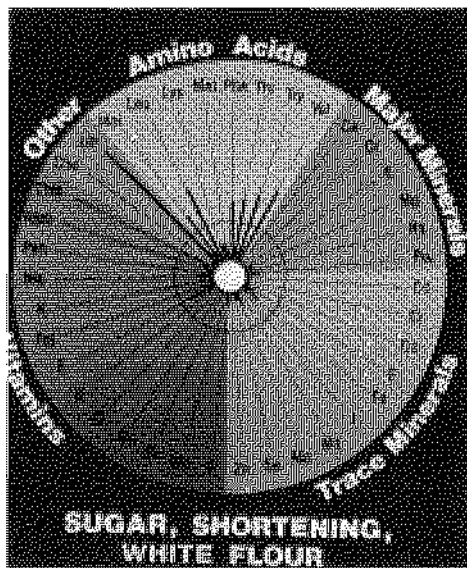
THESE DIAGRAMS TELL HOW WELL CERTAIN FOODS SUPPLY THE GROWTH AND MAINTENANCE CHEMICALS WHICH ARE ESSENTIAL TO HEALTHY HUMAN BEINGS.

THE DIAGRAMS SHOW THE RELATIONSHIP OF NUTRIENTS TO THE CALORIC OR ENERGY CONTENT OF AN ACTUAL FOOD.

STARTING AT THE CENTER OF THE CARTWHEEL AND RADIATING OUTWARD, THE 39 LINES REPRESENT THE ESSENTIAL NUTRIENTS.

- SEE KEY -

THE INTERMEDIATE CIRCLE REPRESENTS THE RECOMMENDED DAILY ALLOWANCES (RDA) OF ALL 39 NUTRIENTS. THE HEAVY BLACK LINES INDICATE THE AMOUNT OF EACH NUTRIENT PRESENT IN 2,500 CALORIES OF A SPECIFIC FOOD. IF THE HEAVY BLACK LINE EXTENDS AS FAR AS THE INTERMEDIATE CIRCLE, THEN THE SPECIFIC NUTRIENT NEEDS OF MAN ARE SATISFIED. IF THE HEAVY LINE EXTENDS PAST THE INTERMEDIATE CIRCLE, IT MEANS THAT MORE THAN ENOUGH OF A PARTICULAR NUTRIENT IS PRESENT AND AVAILABLE TO MEET HUMAN NEEDS. DOTTED LINE REPRESENTS FORTIFIED OR "ADDED" NUTRIENTS.



**INTRODUCTION/CHARACTERISTICS
OF THE SCHIZOPHRENIAS**

INTRODUCTION: TESTING THE SPOOR OF SCHIZOPHRENICS

As a biologist in 1929 at the University of Wisconsin, I first became interested in the schizophrenic when I saw several catatonic patients temporarily recover when given carbon dioxide inhalations by Dr. Ralph Waters. In this instance, Ralph Waters and Chauncey Leake were the biologists, while Bill Bleckwenn (the psychiatrist) supplied the schizophrenic patients. This group later extended their findings to intravenous sodium amytal as an agent for producing a lucid interval. When given carbon dioxide, or the depressant sodium amytal, a catatonic patient may "come to" and relate episodes of his past life and his present ward life with a clarity of detail that astounds the listener. Later at Manteno State Hospital we predicted that arecoline (the active ingredient of betel nut) would produce a lucid interval in catatonic schizophrenia. Our predictions were justified, and arecoline also produced, in a reliable fashion, lucid intervals in the catatonic schizophrenic.

These demonstrations convinced me, and should convince others, that the severe schizophrenic who is frozen into a waxlike statue still has a normal brain anatomically—only the physiology, or function, of that brain is in biochemical imbalance.

Later, as an intern at the Wisconsin General Hospital, I wanted as much anesthesiology as possible. Since we were allowed to trade off monthly services, I got three months of anesthesiology and four months of neuropsychiatry, a service that nobody wanted. This simple numerical balance toward neuropsychiatry helped to shape my future.

I recall one of my patients who was continuously psychotic with status epilepticus. I gave him sodium

phenobarbital, 100 mg intramuscularly every hour all night long. He was restrained because of his violence. At 5:00 A.M. he appeared to be better and asked for a cigarette. I hesitated, and he boasted, "If you'll give me a cigarette, Doc, I'll make smoke come out of my ears." The restraints were loosened, and he got his lighted cigarette, took a big puff, held his nose, and smoke came out of both of his ears! Chronic holes in ear drums on both sides! As an unemployed epileptic, he made a living in local bars demonstrating his trick.

But Enough of These Tricks—What Really Are the Schizophrenias?

As a child you may have watched the public circus parade which always featured the elephants lined up, trunk hooked to tail, following one another with a slow, waddling gait. These gray behemoths with their gold and red trappings were the apogee of all that was impressive and formidable. The lead elephant would have a small turbaned mahout perched on its broad and massive head. For sanitary reasons and comic relief, the last in the parade was the white-garbed dyschondritic dwarf who pushed a two-wheeled cart. With his wide shovel he ceremoniously picked up the elephant droppings, placed them in his cart, and closed the lid. Everybody laughed.

Since the turn of the century, research in the schizophrenias has been like the circus parade. In my biological fantasies the parade of gray behemoths represents the schizophrenias while the dyschondritic dwarf represents the biological scientist collecting the spoor of the schizophrenias. The mahout riding majestically on the head of the lead elephant might be the psychiatrist.

The biologist soon discovers that all elephants are not alike. The spoor collected at the end of the line of gray behemoths can come from any one of the six or seven

schizophrenias (elephants) in the parade. The biologist needs to keep constantly in mind which schizophrenia supplied the spoor.

For example, the pragmatic biologist has determined in a safe manner the body temperature of the elephant by the prompt insertion of a rectal thermometer into the fecal ball. In real elephant hunting the elephant boy sticks his finger into the spoor to determine the degree of heat at the center. This will tell his educated finger exactly the distance to the game, and it is the solution to the problem of elephant hunting. Also in the forest are the dung beetles which make smaller balls of the large ball and roll them off to nurture more dung beetles. In my fantasies these are the non-biologically trained therapists who study grantmanship in order to employ technicians to negate and neutralize the findings of the biological scientist—the dwarf without the grant.

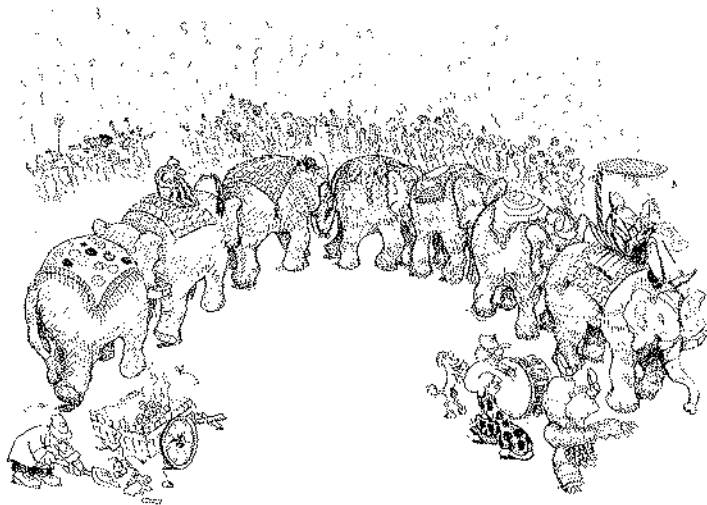


Figure 1: From the biologist's viewpoint, research into the schizophrenias has been like a circus parade with the therapist riding the lead elephant, while the humble biologist gathers the droppings for analysis.

In research the urine, blood, cerebrospinal fluid, and even the brain are analyzed. These studies enable biologists to separate seven different entities from the hodgepodge of disease entities that constitute the schizophrenias. The motivated biologist finds the spoor more difficult to acquire now that the psychiatrists have learned the bare fundamentals of statistics, which they apply freely to their heterogeneous schizophrenic populations as they develop the clinical data on me-too drugs.

I learned early that therapists are not to be trusted in the study of biochemicals. A nose-drop preparation produced a drop in body temperature and some anti-schizophrenic effect. A psychiatrist friend wanted to use this on a problem patient. He left it with the ward nurse with written directions: "Give 10 drops and take body temperature every hour." The nurse gave 10 drops every hour and took body temperature likewise. After six hours the patient was uncomfortable, cold with goosebumps, pallor, and a heart rate of 30. When the medical team arrived the patient, who was just schizophrenic and not stupid, said weakly, "I think electroshock is better than this new therapy." The patient recovered uneventfully, and the nose drops are no longer marketed. As one enlightened government official once said, "For research in schizophrenia, the psychiatrist should be on tap but not on top." A psychiatrist trying to do biological research is like an aborigine who is handed a new boomerang. The poor fellow spends the rest of his life trying to throw the old one away!

From 1948 to 1957 we ran a research ward for the chronic unresponsive schizophrenic at Manteno State Hospital. We examined the spoor of the schizophrenic, and we continuously gave the patient many naturally occurring biochemicals which we thought might mimic the action of carbon dioxide.

The human ethics committee consisted of myself,

the biological scientist, and the interested graduate students who applied the good Golden Rule by trying on themselves and their peers the biochemicals that we planned to use on the patient. We were not trying new drugs, but only pure biochemicals. These biochemicals were used to produce an overbalance in the human body—ours or the patient's.

These smart biochemicals, which we hoped would know where to go and what to do in the brain, included methyl guanidine, pyridoxine, multivitamins, choline, methionine, glutamine, glutamic acid, aspartic acid, l-tryptophan, calcium, magnesium, potassium, guanidine, ACTH, and cortisone—to name a few. Hydrazides produced effective thalamic epilepsy for convulsive therapy which started in the thalamus, but this was no more effective than ECT (electroshock) which originated in the cortex, and ECT was much more convenient.

At Illinois the period of 1945 to 1954 was one of intense learning under the tutelage of Warren McCullough, Fred and Erna Gibbs, Percival Bailey, Paul Bucy, Lasslo Meduna, Adolf Rostenberg, and Max Samter. The graduate students under Klaus Unna, Ted Sherrod, and James A. Bain contributed as they prepared and defended their theses. We were all interested in the brain and how it worked.

In 1950 Martin Pilot perfected the absolute eosinophil count under our direction. The schizophrenic patients had such low counts, 11 to 22 per cu mm (stress), that we couldn't use the eosinophil-lowering effect of ACTH to compare the patients with normals who have an eosinophil count of 150 to 250 cells/cu mm. This showed us that the physiological state of the schizophrenic was one of constant stress—a finding to be proven years later by our colleague, Leonide Goldstein.

We were also innocent bystanders in 1951 when Morris Lipton and Nat Apter had Charles Huggins remove

the adrenal glands from six schizophrenic patients. The adrenal glands, which were tested in vitro by the Hudson Hoagland group, were normal, and none of the schizophrenics got any worse or any better. The patients might have improved if schizophrenia responded to the tender loving care that was bestowed on these patients. Neither happened! No improvement at all! The patients were maintained thereafter on DOCA, cortisone, and extra salt in their diet.

Since schizophrenia was not modified by the removal of the main source of catecholamines—the adrenal—we developed a distinct prejudice against the adrenochrome-adrenolutin theory and the whole catecholamine (dopamine) theory of schizophrenia. As a clincher, one weekend in 1956 we got pure adrenochrome, a natural biochemical, from a nearby pharmaceutical firm and injected recovered alcoholics who volunteered for the study. We told the men that some form of the “DTs” (delirium tremens) might occur with the injection. The individual dose was increased gradually from 0.5 mg to 7 mg intravenously. There was no effect on the blood pressure, electrocardiogram, or pulse rate, but with the larger doses the purple adrenochrome came through the kidneys into the urine. No DTs, no adverse effects, and we concluded that if natural adrenochrome could not be destroyed it would be excreted harmlessly in the urine. This convinced us even more that catecholamines had little to do with the schizophrenias.

In 1954 we showed that arecoline with its acetylcholine-like effect would also produce a lucid interval in the severe schizophrenic patient protected by methyl atropine. Arecoline, the active ingredient of the betel nut, is a mood-elevating chew for many Asians.

The choline and arecoline research did lead us to deanol which is the tertiary amine precursor of both choline and acetylcholine, a known neurotransmitter in

the brain. In our open studies, deanol had as great an effect in the schizophrenias as did Vesprin; one of the me-too tranquilizers then available. But, as usual, only a small but definite proportion of these patients responded to our new smart biochemical. Should we fight for our new smart biochemical, or should we continue the search for the main cause of "schizophrenia"? We chose to follow the elephant's spoor to see what else might be hiding in the medical forest and masquerading as the true schizophrenia. Some scientists may make their reputation by learning more and more about less and less. If we chased the disease, schizophrenia, we might learn more and more about elephant spoor, but not much else! Furthermore, by chasing the spoor of a disease we were labeled scientific dilettante—hardly deserving of grant support.

In the Fall of 1959 (October 17), I had a massive heart attack, and as I lay in the oxygen tent breathing rapidly, as my lungs filled with fluid, I decided to give up my teaching and my administrative duties and study schizophrenia for the rest of my scientific days. Accordingly, I resigned my directorship at Emory University Medical School and sought a post in psychiatric research—Head of Neuropharmacology at the New Jersey Neuro-Psychiatric Institute which planned to develop a research ward for male schizophrenics. Joseph M. Tobin was the Director, A. Arthur Sugerman was Head of Psychiatry, and we all shared medical duties. The years 1960 to 1965 were partly convalescent and mainly staff- and equipment-building years. Dr. Leonide Goldstein showed for the first time by quantitative EEG methods that the chronic male schizophrenic was constantly overaroused even when stiff with catatonia. No wonder carbon dioxide or amytal worked! Some sort of natural depressant was thus needed. Contrariwise, what would cause the brain to be continuously overaroused? Could it be neurohumors, hormones, or an ion? If so, which, how, and where in the

brain?

In August and September of 1966 we made a discovery—the chronic male schizophrenic was much lower in blood histamine (27 ng/ml) than comparable normal males (47 ng/ml) and, furthermore, the histamine rose to normal as the patient improved with an effective antipsychotic drug. We requested financial help from the National Institutes of Health to study this finding. The grant was refused because histamine had no effect (at the dosage used) on the transcallosal pathway of the cat brain, a special but enigmatic laboratory preparation used interminably by one of the scientists of the reviewing panel. Histamine is certainly one of the most important neurotransmitters in the hippocampal region of the brain. This action is overlooked by the dopamine and serotonin workers who multiply like rabbits when nourished by their tax-supported grants. At a recent international meeting a young scientist flippantly discussed the neurohumors of the brain, but failed to mention histamine and many others.

Left to our own financial and our own aging neuronal resources, we did what we could with our new knowledge. Outpatients—72 of them—volunteered to be tested for their blood histamine levels. The new test, the Experiential World Inventory, devised by Drs. Osmond and El-Meligi, was used to assay the dysperceptions, paranoia, depression, and impulsivity of our outpatients. A normal score was 15, and a highly dysperceptive individual outpatient might have a score of 220. The study was open. Would the blood histamine rise as the numerical score of psychopathology went down? In our 22 successes, the negative correlation coefficient was a modest -0.28. The significance was such that this would only occur once in a hundred times by chance! We were on our way; a blood sample might now tell us the degree of improvement in a given low-histamine patient. We also found low-histamine (histapenic) patients to be high in

copper. Copper is contained in histaminase, and excess copper is found in the drinking water of many suburban houses where well water is pumped through copper piping.

We analyzed our outpatient group of schizophrenics and found some patients with very high (above 100 ng/ml) blood histamine levels. With these patients the blood histamine slowly came down with therapy. Their individual correlation coefficients between degree of psychopathology and level of blood histamine might be as high as +0.66 compared to an equally significant—0.88 for a histapenic or low-histamine patient. We discovered many more of the high-histamine patients, so we coined the word “histadelic” for their type of illness. Thus histapenic and histadelic patients are equal in their degree of thought disorder and overarousal, but the histapenic patient has hallucinations and paranoia while the histadelic patient is severely depressed and usually compulsively involved with one type of suicide. The latter also have other rituals and abnormal fears. We found that the changes in histamine (low or very high) only occurred in about two-thirds of the schizophrenics—so we continued to study the spoor of many more patients.

In 1968 we decided that the only biochemical event which could provide histapenia and histadelia would be the mechanism of the storage of histamine in the basophils and nerve endings, which in turn would depend in part on some trace elements such as copper, manganese, calcium, or zinc, any of which could be in excess or in undersupply in the body. The inner core of the elephant spoor was getting warmer to the tip of the index finger!

In 1970, when we found that most or all of the blood histamine occurred in the basophils of the blood, we sent our findings to *Nature* magazine and got our paper back within three days with the comment that it was too specialized. We then sent it to *Science* magazine, which turned

it down with the comment that high and low histamine occurred in only two-thirds of the schizophrenics. When were we going to tell the world about the other third? This unfair criticism spurred us on to learn more about the other third of the schizophrenias.

To make a long story short, histapenic schizophrenic patients were found to be high in serum or tissue copper. They excreted less copper than the normal controls. Excess copper could be removed by giving zinc and manganese by mouth. With zinc and manganese, some patients improved in their extremes of histamine, and also their extremes of behavior. We were most happy when zinc gluconate and manganese gluconate appeared in the health-food stores. Now the druggist would no longer label our Ziman drops (zinc, 10%, manganese, 0.5%) for external use only. Now, the FDA could no longer breathe down our very vulnerable necks.

In 1970 we found that patients excreting the mauve factor (a lavender-colored spot when tested with Ehrlich's reagent) had significantly more zinc in their urine than did a group of schizophrenic patients who did not have the mauve factor. Dr. Arthur Sohler, at my suggestion, found that the kryptopyrrole urinary excretion would take with it pyridoxal and zinc to produce a double deficiency: namely, deficiency of a vitamin and an essential element. Zinc is a trace element needed by more than 20 enzymes in the human body. For instance, zinc is needed by the brain to produce RNA and DNA, those nuclear substances which deal with cell reproduction and perhaps storage of memory.

These mauve-factor patients have stress-induced mental difficulties frequently starting at age 17 because of zinc and B-6 deficiency. Without zinc they grow numerous metabolic white spots in their fingernails, and because of their B-6 deficiency, they fail to recall their dreams. With enough B-6 they again remember their dreams.

When they ask why we want them to recall their dreams, we answer, "Dream recall is normal and we just want you to be normal." One pyroluric patient called excitedly to relate that his first dream was about the terrible time he had had in the mental hospital (a real Id catharsis). He asked if I had planned that for him. Because of my usual busy day I said, "Yes!" and went on to the next dreamless patient.

Another 13-year-old young lady who had had only nightmares in the past two years found that now she had pleasant dreams. Her only comment was that "some of my dreams are awfully sexy." I responded by saying pragmatically that at least she wouldn't get pregnant from dreams. She replied. "Oh, I wouldn't go that far even in my dreams!"

Yet another 17-year-old patient had her nightmares changed to pleasant dreams. Her psychoanalyst protested that nightmares were useful for the working out of aggressions and that the change to pleasant dreams was a step backward!

In our book Mental and Elemental Nutrients (Pfeiffer, 1975b), we summarize the three common types of schizophrenia, namely, 1) histapenia, 2) histadelia, and 3) pyroluria. If we now add the disorders that masquerade as schizophrenia—namely, 4) hypoglycemia, 5) wheat gluten and cerebral allergy—we will probably include 95% of the present disorders that can easily be separated by the competent clinical laboratory connected with an outpatient clinic. With these five main variables we can for the first time calculate and list the combinations of disorders that have been labeled schizophrenia.

SCOPE OF THE SCHIZOPHRENIAS

In the past, the schizophrenias led both general disease and mental disease as a cause of hospitalization. Since only one-third of the patients were hospitalized it is, indeed, the world's greatest disease.

In 1962, 712,174 or 51 percent of the 1,406,818 patients who make up the daily hospital census were patients in psychiatric hospitals. This is slightly more than one out of every two hospital beds. That is for 1962, when schizophrenic patients had a roof over their heads. Now they are on the street or being exploited in boarding homes for their monthly social security disability income. From the street to the boarding homes they periodically appear at emergency clinics to get a quick intra-muscular fix of depot tranquilizers or a prescription for the cheapest drug which will calm their hallucinations. As street people the schizophrenics run afoul of the law, so some jails have a population which sociologists estimate to be 50% schizophrenic.

Diagnoses on patients with mental illness who were in a hospital in the United States for the year 1960 are as follows:

Schizophrenia	23.0%	One-fourth are basically schizophrenic
Alcoholism	14.6%	
Personality disorders	7.3%	Diagnosis?
Psychoneurotic reactions	6.9%	Diagnosis?
Mental deficiency	3.0%	
Epilepsy, Parkinsonism	13.9%	
Senility, brain damage	23.0%	
Other disorders	8.3%	
	100 %	

Because of the relative youth of schizophrenic patients and their low death rate, the schizophrenics tend to

accumulate in society as chronic disease patients.

It is often repeated that heart disease is the number one killer. However, the nation's main cause of hospitalization is schizophrenia, which for the forgotten patient may mean a living death! These living deaths far exceed the actual deaths from any other disease. Yet the research effort in schizophrenia is minimal when compared to its massive morbidity. Voluntary contributions to heart diseases total four to five times the contribution to the whole field of mental health. The disproportion of these two donations is even more striking since more than one-third of the heart funds went into research efforts while only one-sixteenth of the mental health funds was spent in research.

Though limited as it is by the funds available, research into the causes of the schizophrenias has made important breakthroughs in recent years, and many thousands of schizophrenics have already benefited greatly from these results. Beyond this, however, these pioneer studies have disclosed many other avenues which show much promise and should be promptly explored. Facilities and skilled personnel in most instances are available. Only financial support is lacking.

Various suggestions have been made for a schizophrenia research institute but few are funded. Now that half of the schizophrenias are in jail, one might combine the goals and establish at each large prison a biological research institute to study violence, aggression, psychopathic behavior, and the schizophrenias. The need for research in these fields is great.

For the sake of the hundreds of thousands of people whose lives are being blighted by this affliction and for the millions now living who otherwise will follow them, means must be found to advance this vital research. It is difficult to imagine any more worthwhile and promising utilization of the talents of our nation's skilled personnel.

INTRODUCTION/CHARACTERISTICS OF SCHIZOPHRENIAS

Cancer usually hits life as it ebbs, while the schizophrenias cut down genius as it flows to fruition.

PSYCHOMETRIC TESTS FOR THE DIAGNOSIS OF SCHIZOPHRENIA

Psychological tests can be helpful, first in making the diagnosis as well as in following the progress of the patient. Tests can be roughly separated into three kinds: intelligence tests, personality tests, and behavioral diagnostic tests. Some tests combine the two functions of personality assessment and the diagnosis of psychological abnormalities.

Some of the more objective of these tests are designed to measure more or less specific types of mental activity such as: intelligence, achievement, vocational interests, or aptitude. Except for intelligence tests and the HOD or EWI test, these tests have little relevance to the schizophrenias. Intelligence tests can be used not only to gauge overall intelligence in comparison with other people's but also to determine whether a person is making the fullest use of his capabilities. If the person shows some impairment in intellectual functioning, the intelligence test can help pinpoint the specific areas of difficulty. Patients with specific kinds of mental illness suffer from disabilities in different sorts of mental activity. For instance, persons with brain damage may be unable to deal with abstract or symbolic problems, while schizophrenics can manipulate these symbols, but often do so in unusual ways. Both schizophrenics and brain-damaged patients have trouble with some kinds of visual-motor tasks, but the troubles they have are so different as to be diagnostic. The intelligence test can also pinpoint family difficulties. Thus, a child with a subnormal I.Q. of 80 will have trouble when the rest of the family are up in the genius range of I.Q., or 140.

The most widely used behavioral diagnostic tests are either projective or self-rating objective inventories.

Projective tests consist of a series of more or less structured stimuli such as inkblots or pictures. There are no definitely right or wrong solutions, and the results consist of the patient's own perceptions, thoughts, and feelings that he has put into (projected into) the test material. There are many kinds of such tests with many ingenious means of drawing out expressions of the patient's inner mental machinery. Some are more or less explicit and have the advantage that scoring is on a reliable numerical scale, and then a statistically reliable comparable basis is possible. The disadvantage is that the leeway afforded to the patient to put things in his own way is limited. Tests in which the problem material is less explicit give the patient more room for diverse responses, but become correspondingly harder to score on a quantitative or numerical basis. The choice of tests, as well as the complex sifting and weighing of results, is the work of highly trained personnel, usually clinical psychologists who are specially trained in projective techniques.

To a trained observer, there is seldom much difficulty in deciding from a well-selected group of tests whether a patient has one of the schizophrenias. Indeed, even to an experienced clinician the change in the records of these patients from the original tests are sometimes surprising. This occurs when the patient is in a state of spontaneous remission or, for instance, when he is well-managed with modern medication. The tests also show surprising differences—sometimes between actual behavior and test results. This type of patient is sometimes hanging on to reality by sheer willpower.

A noteworthy development is the availability of computer scoring of the Minnesota Multiphasic Personality Inventory (MMPI). This is very complete and scores all possible factors. The evaluation of the report is still up to the physician, since even this test is not infallible.

Precise detailing of the test findings in the

schizophrenias would be lengthy, complicated, and outside the scope of this book. In general, though, the disease causes a loss of adaptive ability. The patient cannot deal effectively with various kinds of mental tasks except insofar as they happen to coincide with one or another of the vagaries produced by the disease. Otherwise, his functioning is haphazard, maladaptive, and often completely out of step with reality. When the patient is better, this disperception diminishes; as he gets worse, the variation grows, and normal thought, feeling, and perception slip away. Only a chaotic shambles of disperception remains, and the patient is depressed about the situation.

Recently, two simple questionnaire tests have been used to test improvement in the patient's state of disperception and schizophrenia. One is the HOD card-sort test of Drs. Abram Hoffer and Humphrey Osmond, and the other is the EWI test. The HOD card-sort test is the Hoffer-Osmond Diagnostic Test. The EWI is the Experimental World Inventory test as developed by Dr. Moneim El-Meligi and Dr. Osmond while working at the New Jersey Neuropsychiatric Institute, Princeton.

Both tests give numerical ratings for nine different abnormalities of behavior which are most helpful in judging the patient's degree of illness and progress with therapy. The tests also disclose suicidal or homicidal tendencies. Several enterprising high school counselors have used these tests to ascertain the type and degree of illness of teenagers.

The use of the HOD and EWI tests will make it possible to correlate by a scientific method the biochemical imbalance and psychological disorders of the schizophrenias and eliminate to a great extent the usual behavioral diagnostic guesswork.

THE EASY ALPHA TEST FOR DEPRESSION AND SUICIDE PREVENTION

Suicide in young people is one of the most tragic problems of our day. Statistics reveal that suicide is the third leading cause of death among American adolescents. This harsh fact makes the need for early detection of suicide proneness in youth both urgent and pressing.

Suicide in both adolescents and adults is frequently the consequence of depression and other mental disorders of biological origin. Based on the research recounted in this book, and that of other workers in the fields of psychiatry and nutrition, we now understand the nutritional and biochemical bases of many kinds of mental illness and can provide nutritional treatment to correct the underlying imbalances. Hence, this questionnaire, the Easy ALPHA Test. This test has been designed, based on research encompassing both the work of BBC staff and other investigators, to measure symptoms of depression and other mental syndromes and to check for other symptoms of the nutritional disorders which often produce mental illness. Thus, the Easy ALPHA Test checks for depression, impulsivity, paranoia and disperceptions on the one hand, and on the other hand also measures the somatic symptoms and traits of pyroluria, histadelia, copper excess and several vitamin deficiencies. To our knowledge it is the first test designed to evaluate depression in young people that takes nutritional factors into account.

We are in the process of testing this scale in order to validate it, and would appreciate your assistance in this endeavor. We shall always need patients and normal controls between the ages of thirteen and thirty-two to see if significant differences between scores on the Easy ALPHA Test exist between these groups. Our preliminary results suggest that the Easy ALPHA Test may well be

useful in making this distinction, but much more testing needs to be done. Readers are thus encouraged to replicate and fill out this form themselves and those readers of this volume who are school counselors, psychologists, clinicians or public servants of any sort with the ability to test this questionnaire on populations of the indicated age level are encouraged to do so if possible. Please return the form to Keith Jordan, c/o Princeton Brain-Bio Center, with any information that may be useful in evaluating the results.

Our hope is that one day this test will be used to screen populations of young people in school systems, clinics, prisons and other agencies so that those suffering from dangerous mental disorders can be isolated, investigated and administered adequate treatment as indicated by the nature of their difficulties. Every year thousands of young people take their own lives. We need a tool to help us prevent such unnecessary deaths. We hope that the Easy ALPHA Test will aid us in this endeavor. We would greatly appreciate your help. A key and a guide to the scales has been provided to evaluate responses.

Princeton Brain Bio Center of the S. F. of N. J.
862 Route 518, Skillman, New Jersey 08558
(609) 924-8607

THE EASY ALPHA TEST
A CORRELATION OF DEPRESSION WITH
NUTRIENT DEFICIENCIES
copyright 1987

Directions: Circle the answer that best fits your situation.
Exclude experiences while using street drugs.

- | | | |
|---|-----|----|
| 1. Do you often feel very down in the morning? | Yes | No |
| 2. Do you always look for excitement? | Yes | No |
| 3. Do you feel the need to travel or wander? | Yes | No |
| 4. Do you think you can read people's minds or tell the future? | Yes | No |

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- | | | |
|---|-----|----|
| 5. Have you suffered much from constipation recently? | Yes | No |
| 6. Are your fingernails clear of white spots? | Yes | No |
| 7. Do you have any known allergies? | Yes | No |
| 8. Do you hear ringing in your ears? | Yes | No |
| 9. Do your gums bleed frequently? | Yes | No |
| 10. Do you frequently have cold sores? | Yes | No |
| 11. Do you never feel down or sad without a reason? | Yes | No |
| 12. Do you usually stop and think before acting? | Yes | No |
| 13. Do people often seem to be watching you? | Yes | No |
| 14. Do you ever feel as if parts of your body were dead? | Yes | No |
| 15. Do you ever have pains in your knees? | Yes | No |
| 16. Do you rarely have headaches? | Yes | No |
| 17. Do you frequently have a metallic taste in your mouth? | Yes | No |
| 18. Do you bruise easily? | Yes | No |
| 19. Are you usually free from guilty feelings? | Yes | No |
| 20. Do you blurt things out often without thinking first? | Yes | No |
| 21. Do you feel safe with your friends? | Yes | No |
| 22. Do you hear voices (unnaturally) talking to or about you? | Yes | No |
| 23. Does your mouth seem dry all the time recently? | Yes | No |
| 24. Do you remember your nightly dreams? | Yes | No |
| 25. Do you feel driven to achieve without deadlines? | Yes | No |
| 26. Is it easy for you to control your anger? | Yes | No |
| 27. Are you often irritable? | Yes | No |
| 28. Would you do almost anything on a dare? | Yes | No |
| 29. Do you often think about your enemies? | Yes | No |
| 30. Do your thoughts appear like a voice outside your head? | Yes | No |
| 31. Are you usually free of nausea in the morning? | Yes | No |
| 32. Do you have many head colds? | Yes | No |
| 33. Do you find it easy falling asleep at night? | Yes | No |
| 34. Do you often feel restless? | Yes | No |
| 35. Do you find it easy to forgive people? | Yes | No |
| 36. Can other people hear what you are thinking? | Yes | No |
| 37. Do you have no difficulty tasting flavors? | Yes | No |
| 38. Do you have no trouble putting on weight? | Yes | No |
| 39. Does life seem hopeless to you? | Yes | No |
| 40. Do you mind having to wait for something? | Yes | No |

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41. Are you not suspicious of most people?	Yes	No
42. Do you get pains in your belly?	Yes	No
43. Do you have many abnormal fears?	Yes	No
44. Do you have early morning insomnia?	Yes	No
45. Are you free of violent urges and intentions?	Yes	No
46. Do people often seem to be laughing at you?	Yes	No
47. With cuts and burns, do you heal rapidly?	Yes	No
48. Are you inwardly driven to have sex?	Yes	No
49. Do you usually feel you can cope with life?	Yes	No
50. Do you frequently laugh at the jokes of others?	Yes	No

THE EASY ALPHA TEST—A CORRELATION OF DEPRESSION WITH NUTRIENT DEFICIENCIES (ages 13-32)

This test discloses symptoms of depression and related psychiatric syndromes as well as symptoms of nutrient deficiencies which have been documented to cause depression and other mental disorders. Ten categories of moods or experiences and/or physical symptoms can be derived from the Easy ALPHA Test.

Scale 1 Depression

This scale checks for symptoms of clinical depression, including disturbance of mood, depressive cognition and biological symptoms.

Scale 2 Impulsivity

This scale measures your degree of self-control and how well you make judgements before following your impulses and entering or initiating a situation.

Scale 3 Paranoia

This scale measures abnormal reactions to others such as undue suspicion and ideas of reference.

Scale 4 Disperceptions

This scale measures your senses such as vision and hearing; normally these are in good balance and free from aberrations such as hallucinations. This scale also measures delusions or bizarre interpretations of life experiences.

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Scale 5 Pyroluria

This scale measures symptoms of pyroluria, the illness associated with the mauve or pink factor in urine. Pyroluria is a hereditary tendency to zinc and B-6 deficiency. It is a major cause of depression and schizophrenia in young people.

Scale 6 Histadelia

This scale measures traits of the histadelic or high histamine biotype. Histadelics are driven, high-energy people who get things done in the world. They frequently suffer from allergies, headaches and depression.

Scale 7 Copper Excess

This scale measures symptoms of having too much copper—from plumbing, containers, commercial vitamins, birth-control pills and other sources—in the body. This metal poisoning can give rise to mental symptoms including depression.

Scale 8 Vitamin C Deficiency

This scale measures symptoms of a deficiency of this important nutrient. During the course of evolution, man lost his ability to manufacture his own Vitamin C, and is now dependent on external sources.

Scale 9 B-6 Deficiency

This scale measures symptoms of deficiency in this vitamin, which is part of the biological deficit in Pyroluria.

Scale 10 B-2, B-3 Deficiencies

This scale assesses for symptoms of a lack of an adequate amount of these nutrients in one's diet.

Key—Easy Alpha Test

Depression—Ques. 1(y), 5(y), 11(n), 16(n), 19(n), 23(y), 27(y), 33(n), 39(y), 44(y), 49(n)

Impulsivity—Ques. 2(y), 12(n), 20(y), 28(y), 34(y), 40(y), 45(n)

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Paranoia—Ques. 3(y), 13(y), 21(n), 26(n), 29(y), 35(n),
41(n), 46(y), 50(n)

Disperception/Psychotic Symptoms—Ques. 4(y), 14(y),
22(y), 30(y), 36(y)

Pyroluria—Ques. 6(n), 10(y), 15(y), 24(n), 31(n), 37(n),
42(y), 47(n)

Histadelia—Ques. 7(y), 16(n), 25(y), 32(y), 38(n), 43(y),
48(y)

Copper Toxicity—Ques. 8(y), 16(n), 17(y), 26(n), 33(n)

Vitamin C Deficiency—Ques. 9(y), 18(y), 47(n)

Vitamin B-6 Deficiency—Ques. 10(y), 24(n)

Vitamin B-2, B-3 Deficiency—Ques. 10(y)

MODELS OF MADNESS, MODELS OF MEDICINE

We all know that inside the skull is the brain, not the "mind," but since it does not have palpitations, or cramps, since it does not hurt, itch, or sting, we have no direct experience of it and we do not know when it is "sick."

— Models of Madness, Models of Medicine
p. 189

There comes a fateful moment in the lives of those suffering from schizophrenia when they first present themselves for help, either to a private physician or psychiatrist, or to a hospital admitting staff. Upon giving an account of the symptoms and current state, the person seeking help learns from the practitioner that either a physical disorder exists, or does not. The course of action in the two cases is almost entirely different. In the former case, the doctor will elect to postpone discussion of other problems until the physical one is under control. In the later case, the problems in happy, fruitful and useful living will remain the prime concern.

The clinically oriented "medical model" which basically treats schizophrenia as a symptom of some underlying disease or disorder, and the "psychoanalytical model" which means to correct some basic misunderstandings in the minds of schizophrenics, are only two of the eight models presented by authors Miriam Siegler and Humphrey Osmond in Models of Madness, Models of Medicine and used to better understand the schizophrenias.

The other six models are all derived from the basic "psychiatric" model and include: "The impaired model" wherein schizophrenics are permanently considered to be of lowered human status; "the moral model" which

classifies schizophrenics with other behavior problems; "the social model" which blames society and views schizophrenia as yet another symptom of our "sick" society; "the family interactional model" which blames the family for its "sick" form of communication which has surfaced as schizophrenia in at least one member; "the conspiratorial model" which basically argues that although schizophrenics are not significantly different from the rest of us, they have been labeled as if they were different; and finally "the psychedelic model" which romanticizes schizophrenics as people with gifts of exceptional insight to whom we simply cannot relate.

As a result of all these models, the use of the word "sick" has become corrupt. To say that someone is "very sick" no longer implies a physical ailment. The speaker's intention may just as well have been to say "the fellow has an extremely warped and unpleasant personality," or that he is "too impaired to undertake responsibilities." Some "sick" people are merely thought to be symptoms of a "sick society" or of a "sick family" and not truly "sick" themselves. Such fundamentally different beliefs can be seen in how various people (including the professionals) treat "sick" patients and their families. Clearly, different models are used by different therapists.

The Clinically Oriented Medical Model of Schizophrenia

We have all frequently experienced the affects of illnesses on our mental or emotional state—making us groggy, or irritable, or depressed, or forgetful. Even the common cold has a profound effect upon our mental abilities and on our mood. Many illnesses, including among many others, organic brain tumors, cerebral allergies, infections such as brain syphilis, and, most commonly, biochemical imbalances such as simple nutritional deficits and environmental intoxications, are known to

have their most deleterious affects on brain functioning and should clearly be treated within the medical model. In this model, schizophrenic patients are considered to be physically ill in a way that not only affects their brains, but also their behavior.

Whenever we are in fact ill, most of us would like the advantages of competent medical advice preferably given in the clear, familiar doctor/patient relationship. A clinically oriented medical model of schizophrenia is designed for this purpose. As a clinical medical model, it focuses on biologically relevant information about patients, which leads to diagnosing the appropriate illness, and planning the best possible treatment approach available.

Because the true function of the clinical medical model is not only to treat the ill, but also to ensure that, within the realm of medicine, no one is blamed for the illness, all schizophrenics and their families are granted freedom from illness-related blame and guilt. Only the clinical medical model can perform both treatment and absolution of blame functions and it can do so only through the proper use of its two main components: "Aesculapian authority" and the "the sick role."

What Is "Aesculapian Authority?"

One of the most eloquent accounts of the power and quality of pure Aesculapian authority is found in the following story recounting Dr. Schweninger's treatment of Chancellor Bismarck:

...Schweninger at last imposed moderation on the genius who had imposed it on others, but never on himself. At their first meeting, Bismarck said roughly: "I don't like being asked questions." Schweninger replied: "Then get a vet. He doesn't question his patients." The battle was won in a single round. Bismarck ate and drank less, kept more regular hours. When

Schweninger was present, he even kept his temper. He underwent a slimming diet, which consisted exclusively of herrings...it did the trick. Bismark's weight went down from eighteen to fourteen stones; he slept long and peacefully; his eyes became clear, his skin fresh and almost youthful.

(one stone = 14 lbs.)

— A. J. P. Taylor, 1967 BISMARCK: The Man and the Statesman

Aesculapian authority was first named and defined by T. T. Patterson in 1957 to be a unique combination of wise, moral, and charismatic authority, through which the doctor, having agreed to do all he can to help the patient get well, proceeds to give his orders. According to Patterson, the doctor with Aesculapian authority has been granted the right to be heard by reason of knowledge and expertise, and the right to control and direct stemming from the goodness of his goal to better the health of the patient (as expressed in the Hippocratic Oath) and by means of his powerful and mysterious abilities in the face of illness and death. Without such authority few, including Bismarck, would be willing to submit to most medical treatments since they may be too frightening, disgusting, painful, expensive, life-threatening, or simply unpleasant.

What Is Meant By "The Sick Role?"

Dr. Schweninger's authority carried no weight until Bismarck properly accepted "the sick role." Doctor's so-called orders are really only advice, for the doctor (as doctor) has no structural authority over the patient and cannot truly order him to do anything. Therefore, once it is clear to the doctor that the patient at hand is in fact sick, he or she must persuade the patient, through evidence and personality, to accept the sick role. As did Bismarck at

first, the patient often resists. Illness brings with it the fear of death or disablement, the miseries of medical treatment, the loss of personal privacy, the expending of money, and many other undesirable consequences.

In accepting "the sick role," the patient agrees to do everything in his own power to get well, which usually requires curtailing many normal, habitual, and instinctual habits, submitting to treatments which allow people to do things to us that we normally resist, and following the doctor's "orders." In a sense, the doctor uses the patient's drive to survive to enforce acceptance of treatment. And what does the patient get in return?

In addition to the best treatment possible, which comes with the doctor's knowledge, expertise, and professional commitment to the patient's health, the conference of the sick role absolves all blame. By the authority invested in the doctor, within the confines of the medical model, neither the patient, nor anyone else can be blamed for the illness at hand. This use of Aesculapian authority enforces an attitude toward unavoidable misfortune through which usual fears and moral judgments must be temporarily suspended. Such an authority is much to our advantage, for without such authority we are unlikely to suppress the natural tendency to blame ourselves or others for the many appalling misfortunes that attend serious illness. Aside from other troubles, a doctor who fails to absolve a patient and family from blame for the illness, may find it difficult to gain the cooperation that is so crucial to success within the medical model.

Scientific, Public Health, and Psychiatric Medicine Are Aberrations

From this definition of the medical model with its Aesculapian authority used to confer the sick role, it follows that there are two categories of aberrations which

may occur: the sick role may be adopted without the use of Aesculapian authority, or Aesculapian authority may be used without conferral of the sick role. In the first instance, the patient believes the doctor is employing the Hippocratic Oath and putting the patient's health as the goal of all actions when, in fact, the doctor is not working from within the clinical medical model at all.

Many doctors today employ the scientific medical model instead and proceed to use patients as guinea pigs without the patient's understanding or consent. Another equally sinister set of circumstances occurs when the patient in the sick role is being treated by a doctor who is instead acting according to the rules of public health medicine, treating the patient in such a way that is perhaps best for the masses yet potentially lethal to the patient. Patients who are aware of the alternative roles that motivate doctors are better equipped to handle such circumstances should they arise.

The seven non-medical models of madness, when used by physicians, are instances of Aesculapian authority without the conferral of the sick role. Instead of the sick role, a variety of other roles are conferred: the "impaired" role, the "bad" role, the "psych" role, etc. None of these carry freedom from blame, or the promise of the best treatment possible, or any of the other benefits accrued to the sick role. The most important aspect about Aesculapian authority may be its power, but the oddest thing is that we fail to notice when it is being abused. Many non-medical models employ physicians who then abuse Aesculapian authority by using it to enforce non-medical orders. And in spite of intermittent grumbling about these "doctors" and their arbitrary ways, most meekly accept outrages that would not be tolerated for a moment in other instances with lesser authorities.

Although all people would agree that sick people should be granted the sick role, it is not always crystal clear

who is the sick one.

Impaired Model of Madness

Sick is different from impaired or disabled in that the sick person can be treated while the impaired cannot. The problems of the impaired are thought to be outside the realm of medical treatment—presumably they cannot be medically treated. The impaired model is used to spare the patient and the family “unreasonable hope.” Because medical doctors can be of no help, services come by way of support staff. Impairing disabilities are considered to be permanent. In a sense, people in the impaired role are behavior problems who do not “carry their own weight,” to which no expectations of improvement are added. They do not fear getting worse because they have been counseled to accept their fate. They have given up hope for treatment and appear to be resigned to living as second-class citizens, in the community but not of it.

Those who are truly impaired are unable to manage in the outside world and, therefore, deserve protective environments. Because there is no medical treatment appropriate to those who are impaired (clinicians of the medical model have given up hope), the Aesculapian authority of medical doctors has no right to claim or feign power over the impaired in the area of their disability. Schizophrenics who need help in areas such as social graces, or occupational therapy, can be rightfully granted such aid alongside medical treatment, but not in lieu of medical treatment and not under medical authority.

Administrators of large “asylums” who also have medical degrees and attempt to misuse their Aesculapian authority poorly mix the impaired model with the medical model. The result is often an inhospitable, non-protective, human warehouse, falsely run under medical auspices. Doctors who administrate these large asylums need to be

reminded that they are not treating, they are not putting each patient's best health interests ahead as the primary goal, and therefore are not adhering to a medical model. In such a setting they are unfairly misusing their authority as a medical doctor.

A schizophrenic patient gets caught in the impaired role whenever treatment is abandoned. For the schizophrenic patient, hope is still reasonable and the impaired model is still inappropriate. A doctor who gives up hope for a schizophrenic patient has most likely not tried all treatment approaches.

As long as patients follow their treatment plan, or at least have faith in treatments yet to come, they are rightfully granted the sick role and rightfully exempt from those responsibilities they cannot presently handle. However, once a patient gives up hope and abandons treatment, he is no longer exempt from responsibilities. He immediately falls into the impaired role and is granted second-class status.

Unfortunately, many schizophrenics are inadvertently placed in the impaired role by members of the community who simply believe that the schizophrenias cannot be cured. They do so simply by treating schizophrenics as if they were impaired, as if there were no treatment, and as if they deserved lower societal status. The impaired role is usually conferred by default.

The Moral Model of Madness

In this model, schizophrenics are again viewed as behavior problems, as people with irritating, troublesome, disgusting, frightening, or eerie behavior which should and presumably can be corrected or at least brought within acceptable limits. People who work within the moral model frequently believe that these "ill-mannered" schizophrenics are intentionally and illegitimately trying

to get the privileges naturally awarded to the sick. As a result many schizophrenics get classified, along with malingerers, as people who enjoy benefits of the sick role, like being exempt from social and societal responsibilities, while having nothing organically wrong. A favorite, though innocent, example can be found in actions of a young child who intends to get out of school by fooling his mother and placing the thermometer on the heating pad.

A schizophrenic patient can easily place himself in the "irresponsible" or "immoral" role simply by ignoring a clinically oriented medical doctor's treatment orders for relieving his schizophrenic symptoms. In this sense, the patient is remaining in the privileged sick state without accepting the responsibilities of the sick, which include doing everything in their power to get well.

The greatest abuse which results from the use of this model is seen when other people place a schizophrenic in this category without agreement from the patient. Many people who do not understand how illnesses affect the brain and behavior do not see schizophrenics as sick. According to these people, many schizophrenics are simply lazy and poorly behaved. "What they need is an environment that won't allow them to get away with such behavior!" or "They'll teach you how to behave in that state hospital!"

Moral models are often inappropriately used in hospital settings. When isolation is used as a means of punishment, then the moral model prevails. Medical models reserve isolation for patients with contagious infections. If the practitioner, the family, or others who know the supposedly sick person are treating the patient with condescension or contempt, or are moralizing about his erring ways, the patient is not in the sick role.

Unlike the impaired, it is assumed that the immoral could hold their responsibilities if properly trained to do so. No societal expectations are lifted. Behavior

modification is often used and is frequently successful in altering a patient's behavior and can rightfully be used in conjunction with medical treatment, but again, not instead of medical treatment. The one fear which must remain in the forefront is the possibility that behavior modification may hide warning signs of suicidal intentions. Many suicides have been attempted in patients whose behavior "seemed fine."

Ideally, one would appropriately relate the impaired and the moral models to the medical model. Once the schizophrenic was safely installed in the sick role under the protection of the medical model, then techniques derived from the moral and the impaired model can be brought to bear on the secondary effects of the illness. From these other two, a person long ill can get help in sustaining, repairing, and learning the social and psychological skills which are so often damaged by the complex illness.

The Psychoanalytical Model and Its Derivatives: the Social, the Family Interactional, the Conspiratorial, and the Psychedelic

When we are physically ill we all know enough to go to the family medical doctor, but the task before us seems much more complicated when faced with something like schizophrenia that first appears as a mental disturbance. Although we often limit our thinking of brain diseases to such classics as brain tumors, the fact is that most illnesses affect our brains. Unfortunately, many clinically oriented medical doctors are equally limited in their thinking about brain disorders. Many still examine a patient system by system—the digestive system, the respiratory system, the skeletal system, the nervous system, etc.—and, finding nothing with these particular systems, ask: "Do you think your emotional disturbances

might be psychological?" Or, finding something wrong, how seldom these less informed doctors ask: "Do you think this illness is affecting your brain and thereby leading to your disturbances of thought?" Very often the answer would be a relief-filled "yes."

The problem with the medical doctors in the example given above is really quite surprising. These doctors have simply forgotten to include the brain as part of the body. By doing so, they mistreat many clearly medically related illnesses. Having then removed these cases from the realm of medicine, and referred them on to psychologically oriented professionals, many medical doctors today do us all a serious disservice.

Blame is a sure sign that the sick role has not been conferred. Most likely, when the patient is viewed as a victim, the practitioner is not working from within the medical model. Perhaps he is a psychoanalyst, psychologist, social worker, or family therapist and the model used is the "psychiatric," the "social" or the "family interactional." Because these models are often muddled within the professions, they necessarily offer confusing roles to both the practitioner and the patient. Basically, they each see the patient as somehow the victim, and limit the treatment approach to talk therapy. Holding head strong to their particular treatment approach, few practitioners offer new avenues when their own treatments fail. Instead, many insist that their treatment approach is a life-long process (if the patient can pay!).

Some of the greatest damage is done by practitioners using the psychoanalytical model. Often they are also trained as medical doctors and make improper use of their Aesculapian authority as they blame the mother or the father for corrupting the mind of the patient. The patient is often told that a complete cure is possible and complete if only the psychoanalysis works. Only through psychoanalysis can the underlying trauma be revealed and

overcome. If it doesn't work, the psychoanalyst offers no other alternative.

Similarly, the social model maintains that a cure is possible and can be complete—if only society would substantially reform. Schizophrenic families are believed to be the victims of a “sick” society, told that it is their right to live in a more just and equitable world, and promised that once society's problems are ironed out, they will no longer be plagued with mental illness. This news must come as cold comfort indeed to a poor family trying to find help for their schizophrenic child.

The family interactional model offers the family the right to be treated as “sick” along with the index patient. Often this comes with a thick coating of blame and ridicule. Some family systems models do not confer blame and can prove to be very helpful alongside a medical model which treats the underlying illness before wasting time on hours or years of talk therapy. Whatever problems in living remain after the illness is controlled, can well be handled by the few family systems models that avoid blame.

The basic problem with all three of the above models, the psychoanalytical, the social, and the family interactional, is that they ignore the underlying illness and attempt to treat physical ailments through talk therapy alone. They each attempt to completely explain schizophrenia within the confines of what Siegler and Osmond call “continuous” models. Their considerations are cosmic; their views are global. Their strength derives from their capacity to satisfy our need for a cosmology, a way of putting it all together. But the effect of using these models without first treating underlying medical disorders is usually serious and often risky, and even gloomy. The general principles used by these models do not offer a set of instructions or procedures for distressing circumstances.

The two remaining continuous models, the conspirational model and the psychedelic model are even more egregious and grandiose. According to each of these, in their own way, schizophrenics do not have the problem. Instead, proponents of each of these models believe that the problems lie in those who interact with the schizophrenic.

The psychedelic model holds that although schizophrenics are different from "the rest of us," there is nothing actually wrong with them. They are simply living within another frame of reference that we simply do not understand and to which we simply cannot relate. It is our limited capacity which makes us unable to comprehend their transcended state.

The conspirational model as formulated is a logically derived alternative to all of the others. It states that so called "schizophrenics" are, by all standards, the same as all the rest of us. It was simply they and not us who got caught in the threads of the labeling system. Their few "out of the range of normal" behaviors were noticed while our "out out of the range of normal" behaviors were simply overlooked.

Some proponents of a related model believe that "mental illness" per se is merely a metaphor for brain illness. They claim there can be only physical illness. To them, if the brain or some other part of the body is not ill, then the person is in no way ill. Because the mind itself cannot be sick, one cannot rightfully speak of a "mental" illness. This implies, of course, that if the brain is in fact ill in schizophrenia, then the disorder is real and not a myth at all.

Schizophrenia is a tough and persistent disorder. No one imagines for a moment that this great and grave illness can be totally conquered by taking a pill. That is magic, not medicine. Great illnesses require great exertions by doctors, patients, families, and often the whole community.

They have psychological, social, moral, spiritual, financial, vocational, and other aspects which can, at times, make us of a variety of professions, and they cause major catastrophes.

In contrast, the medical model, a discontinuous model, offers immediate but limited dealings: symptoms are treated as best they can be and no attempt is made to totally explain schizophrenia. Although a lack of comprehensiveness exists, comfort and protection are offered at particular spots along the way as the medical model takes simple, practical and verifiable steps to alleviate the suffering. All in all, the medical model is best able to handle the complexities while providing the patient with the most humane and effective treatment.

People do hold the beliefs as defined in the above models and they will continue to hold them. Although no one model can be proven to be more correct than any others, some do seem to make life more miserable for the schizophrenic and their family. From the professional point of view, it can prove helpful to not only see where you fit, but to see whether, in all senses, you are as pure as allowed by your role, with its duties and responsibilities.

Finally, you can rightfully exclaim, "The only Aesculapian authority among doctors was my pediatrician or my old family doctor." This is true, the pediatrician knows more about growing up and the family practice doctor should know more about all aspects of medicine. The developing counterpart for adults is the ecological-nutritionally oriented doctor who can give advice in many fields and is the obvious choice for applying the medical model in the schizophrenias, for second opinions on operations, and other risky therapies such as cancer chemotherapy.

THE BEWARES OF SCHIZOPHRENICS

Psychiatry and medicine in general are services which are supplied to the patient by the therapist. Although by nature medicine differs with many of the services to which we are accustomed, it has one important consistency with which all should be aware: the quality of treatment will vary based on the knowledge, expertise, and motivation of your practitioner. Additionally, one must keep in mind that drugs usually require only days to have a therapeutic effect, while nutrients and biochemicals may require weeks. With this in mind,

The schizophrenic, and his loved ones who pay the medical bills, should beware of:

1. The therapist who says nutrients (vitamins & minerals) are unimportant in the treatment of mental disease.
2. The psychiatrist who states that he doesn't need to use the potent new mental drugs or nutrients in his practice.
3. The therapist who says the major tranquilizers are habit forming. Not true!
4. The therapist who, after his talking therapy has failed, states that brain damage must exist.
5. The therapist who says prolonged talking therapy should be tried first and nutrients should be tried later, or as a last resort.
6. The therapist who has hidden meanings for the stumbling of the accident-prone, disperceptive schizophrenic. After a simple accident he may say, "You injured yourself because of unresolved subconscious guilt!" or "You attempted self-castration!" (Wild bias indeed!) Any such statements should warn the patient that progress will be slow, indeed, with this therapist.
7. The therapist who never makes a diagnosis of

schizophrenia, or who refuses to give the patient and his family any recognized psychiatric or medical diagnosis.

8. Any therapist who states that the patient is only expressing the sickness of the entire family, implying that family members are well only because the patient is sick.

9. Any therapist who attempts to treat schizophrenia only by means of "conditioning therapy." In operant conditioning the patient gets a reward, such as candy, for a nonsensical mechanical response. In aversive conditioning he gets a mild electric shock for not cooperating. These are both research tools and should be kept in research programs. Antischizophrenic nutrient therapy must come first.

10. Any therapist who asks for a lump-sum payment in advance before undertaking to treat the patient. After all, you didn't contract to pay your orthodontist or pediatrician in advance.

11. A therapist who states that hypnotism or dream analysis may provide solid answers in the schizophrenias.

12. The therapist who raises the fee and states that you will get more benefit by paying a higher price. An exception occurs when the therapist asks that the patient work and pay the fee rather than the parents. In this case, the patient who earns his way will be more cooperative.

Midway in the Bewares

We should perhaps apologize not to, but for, the professions of psychology and psychiatry. Too many therapists unfortunately believe that they can perform miracles with some new (?) technique. If every therapist were required to spend a day in the medical library before starting a new technique, his optimism would be sobered. The "beware" above and below are based on years of observation of fads in the treatment of the schizophrenias. These fads would no longer be proposed if the proposer

would only study the documented reports of their inadequacy in the schizophrenias.

13. The therapist who says that nutrients will only make the patient more aware of his intellectual deficits and thus make the patient more unhappy.

14. The therapist who says, "We never put labels on patients with such actions." They put on the patient a mysterious hex mark instead of a label. This allows self-blame and self-pity to accumulate because no diagnosis means the patient cannot compare his biochemical anomaly with others with the same condition. The medical model of illness is not fulfilled.

15. The therapist who says taking a blood sample for study may give the patient an excuse to discontinue psychotherapy.

16. Beware of the therapist whose only feeble resource is to shuffle the deck of siblings and family. This therapist may say that in order to improve, John or Mary must live alone in an apartment away from the family. The patient is usually too sick with his biochemical imbalance to live alone and to take care of himself. Being alone can enhance the dis perceptions of schizophrenia. These patients need a carefully structured daily program of activity which may be provided in a home or in a halfway house.

17. Beware of the over-critical paramedical worker who says to the relatives, "What will happen to Linda (the patient) later on when the nutrient therapy fails to continue working." The nutrient therapy does not stop working, and furthermore, when the patient improves, careful supportive psychotherapy should be used rather than negative psychology.

Finally, a specific caveat (with a wild waving of red flags) should be given to the suggestion of any therapist who wants you (the patient) to stop all drugs (including vitamins) "in order to see (or disclose to him) the real you or the undoctored personality." This therapist obviously

does not believe in the beneficial effects of vitamins or drugs or he would not make this suggestion. Frequently, relatives may in ignorance reinforce the suggestion by saying "Well, the patient must learn to live without drugs someday." The vitamins and the trace elements are nutrients needed every day by the body. These are needed by the schizophrenic patients as much as you or I need our eyeglasses or warm clothing in winter, or the diabetic patient needs his insulin. Vitamins and trace metal elements are not drugs and may, in effective doses, reduce the amount of antischizophrenic drug which the patient may need each day.

A further fallacy in the therapist's desire to see the real you may be his failure to obtain in detail the careful psychiatric picture of you before you started psychotherapy or the meganutrient treatment. The HOD or MMPI profile will give the picture of the real you. Many of our patients who have stopped all drugs at the suggestion of a talking therapist have relapsed and become hospitalized. Unfortunately, a few have committed suicide. Obviously, this bold suggestion that all therapy be stopped is the invitation of the devil, and can only be done, with safety, in the locked ward of a psychiatric hospital. A Machiavellian variation of this (no drug) gambit can be the substitution of placebo (dummy capsules) for the active medication. The know-it-all therapist may secretly believe that dummy capsules will suffice and that talking therapy is supreme. This led one therapist to give a patient a big build-up about a new miracle drug from Europe (10,000 trials and no failures!) When the dummy capsules were substituted for the regular antipsychotic medication, the patient became so severely psychotic that he required hospitalization. The suggestion is also stupid in that only a single item of therapy should ever be stopped, and then at weekly or monthly intervals. In this way the patient can determine what really helps him and what he really needs.

DANGER SIGNS OF THE SCHIZOPHRENIAS

Most of the public associates peculiar behaviors and thoughts with the disorder of schizophrenia. However, those who have had no contact with a schizophrenic usually cannot elaborate further. Below is a list of common schizophrenic symptoms which may aid family and friends in the identification of the thought disorder. Early diagnosis and entry into the treatment system is paramount, as in the acute stages schizophrenia is disabling. Be aware that single symptoms are not diagnostic; schizophrenia is a syndrome with many manifestations appearing together. Also note that the symptoms listed are not exclusive for schizophrenia, as many will also appear in those suffering from mania and organic brain disease.

1. Stereotyped or repetitive behavior (grimacing, smiling, etc.).
2. Continued feelings of physical discomfort without cause.
3. Unnatural fears, timidity, or grandiosity.
4. Failure to make friends and unnatural ability to alienate loved ones; withdrawal from society.
5. Unfounded suspicions or threats.
6. Profound insomnia, i.e., the ability of a teenager to work or carouse all night.
7. Profound daytime fatigue or stuporlike sleep (he may go to bed when he comes home from school).
8. Continued angry excitement or temper tantrums in a teenager.
9. Complaints that voices are too loud or all lights are too bright.
10. Hallucinatory activity.
 - a. Hearing voices unnaturally.

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- b. Seeing things unnaturally.
 - c. Smelling bad odors without cause.
 - d. Unnatural abilities: "I read others' thoughts," "They read my mind." "Road signs have hidden meanings."
11. Talk of suicide.
12. Announced sudden belief in the great truth phenomena, such as:
- a. "God is Love."
 - b. "Love is the only thing that matters."
 - c. "I didn't ask to be born."
 - d. "My basic problems must be solved."
 - e. "Vegetarianism is the only way to eat."
 - f. "I'm taking off to find the real me."

IS THE SCHIZOPHRENIC DANGEROUS?

When the young student from Texas climbed the campus bell tower, with rifle in hand, and shot down innocent bystanders, the press and even therapists cried: "Beware, Schizophrenia!" Although the autopsy ultimately showed a mid-brain tumor as the probable cause of this antisocial act, the damage had been done. The public cringes at the thought or sight of a poor unfortunate who doesn't talk sense. Of all the schizophrenics, only the suspicious or paranoid patient is dangerous. And he represents only a small and usually stable fraction of the schizophrenias. Unlike a patient with a growing brain tumor, who may explode into violence unforeseeably, a paranoid develops his psychosis slowly and usually gives many signs of his illness before acting on his suspicions. Of course, these signs should not be ignored. This points up once again the importance of early diagnosis and treatment. Once classified, the paranoid usually stays that way—just overly suspicious, vocal, and extremely slow to back his words with action. If he can live an isolated life, with minimal social contacts, he can at most times be productive and respected. When pushed or ridiculed he may break under the stress, so he must be watched carefully!

In addition to patients with brain tumors, the patient in the manic stage of manic-depressive disorder can be destructive and assaultive. By far the greatest danger of assault arises from the individual with a character disorder who deliberately plans a violent career to meet his needs in life.

One psychiatrist has repeatedly said that if he were looking for a safe neighborhood to live in, he would choose one that was entirely schizophrenic, because the possibilities of violence in such a neighborhood would be

a great deal less than that in the average neighborhood.

Most untreated schizophrenics are meek, withdrawn, and occupied with their own thoughts or delusions. If they hear voices, the voices may even be heavenly or exalting. Only rarely do the voices suggest violence, and then many patients will tell their friends or doctor what the voices are suggesting. Again we are forewarned, and in the overwhelming majority of cases the schizophrenic does successfully battle with the negative voices if any. Under antipsychotic medication the patients come to doubt the voices or other hallucinations, so that while they may hear them, they don't believe them.

With appropriate therapy, the patient can be taught to recognize hallucinatory voices as opposed to real ones. For instance, such a simple thing as the question: "Can you talk to those voices without opening your mouth?" will help the individual differentiate between the real and false voices.

Finally, although remembered, the hallucinations disappear, and the patient may refer to them only as those silly voices that he used to hear; or preferably, the individual may be able to realize that the "silly voices" were or are hallucinations which are an expression of schizophrenia and, as such, are to be disregarded as a basis for action in life.

Since we are dealing with the schizophrenias, ours to conquer, we should not close this topic without some mention of the hyperideational paranoid. He may be the executive in high places who has many ideas—in fact, too many so that he himself gets little or nothing done. If he has understanding and controlling colleagues he may be of value to the organization. If, however, he has absolute control of the business he can soon send it into bankruptcy. Such an individual was Adolf Hitler, whose havoc and infamy will live forever in history. With this horrible example of the hyperideational paranoid in mind, many

organizations get psychometric tests on candidates for executive positions. Obviously these individuals can be used for their free flow of ideas but should not be depended on for logical administration.

THE VERY REAL RISK OF SUICIDE

In general, homicide is grossly overrated and suicide among schizophrenics underestimated. The rising suicide rate among students has alarmed us for a whole generation. Suicide by students now ranks third as a cause of death among college students. No greater tragedy can befall a family or the family doctor than to learn that a brilliant student has decided he's "had it" and takes final, successful action. The high incidence of suicide at this age period coincides with that period of life when the onset of the schizophrenias are at their highest incidence. Suicide in schizophrenics is 20 times more prevalent than in the normal population. Additionally, it is unclear how many schizophrenics commit suicide before their diagnosis is intact.

Suicide is more commonly taken to be a hazard of a temporarily depressed mind rather than that of a serious mental illness, because the relationship of obvious sadness and the wish to "end it all" is readily understandable. Yet self-destructive impulses and actions are common in schizophrenia. For example, young persons with so-called "emotional instability reactions," for all their obvious aggressiveness and destructiveness toward other people, are often intensely self-destructive as well. The manifestations of destruction vary in form and intensity, ranging from minor self-mutilation to more or less serious attempts at suicide, sometimes in bizarre and grisly ways. Much of this emotional illness may be early schizophrenia. In the days before the advent of modern drug therapy it was not at all uncommon to see schizophrenic patients who had burned themselves with cigarettes or had cut themselves with shards of broken glass. These acts may have a symbolic quality. The patient may burn himself on the hands and feet so that the scars are an imitation of the

wounds of Jesus. He may leap from a window, thinking that he has been transfigured and can fly. Thus these acts often arise from the patient's delusional thinking. In addition, however, schizophrenics may be severely depressed, and their self-injury may spring from this just as with other people who are depressed. There is a further hazard. In some patients, certain drugs may intensify depression, so judicious tailoring of drug therapy for each patient is imperative. This takes time and patience on the part of both doctor and patient.

As stated above, suicide as a result of schizophrenia may occur much earlier in life than that from other causes. Suicides caused by depression occur late in life, in association with the menopause, midlife crisis, or hardening of the arteries of the brain. Thus, in still another way schizophrenia cuts down the young, and the total years of useful life that are lost are greater. Unfortunately, suicidal actions are sometimes among the very first evidences of the onset of the disease. Many a patient is already dead by the time the shocked and grieving family realizes in hindsight that something was, indeed, dreadfully amiss. This makes the need for early diagnosis (such as thorough psychometric screening in schools) and prompt, effective therapy all the more pressing.

The high suicide rate among young schizophrenics may seem surprising, and many people have viewed these suicides as impulsive, "crazy" incidents. However, in talking with young schizophrenics, one soon learns of their despair about their distorted world and their lack of hope of ever getting well. They are often told that they have nothing wrong with them except emotional instability, and the "authoritative therapist," who says "get hold of yourself and behave," brings no relief. Given an experience such as this, it really does not seem so strange that they commit suicide.

Most discussions of suicide rapidly turn into a

detailing of statistics. However, statistics can be dehumanizing and to the family, friends, and physician, not to mention the patient, are irrelevant to the individual case. On this level, the statistics don't matter. The important thing is to save a human life whenever possible. If all present-day treatment has failed, at least the patient can be preserved until the advent of better therapeutic measures. With these new treatments perhaps the suicidal cases may respond the best.

Talk or threats of suicide must always be taken seriously by both relatives and therapists. Patients will frequently give their real intent more openly in their answers to the psychometric tests, such as the EWI, HOD, or MMPI tests. In these tests a high dysphoria score, a low euphoria score, and a high impulsivity score show that the patient is actively suicidal.

THE TYPES OF SCHIZOPHRENIAS

The types of schizophrenias are continually decreasing as researchers illuminate the causative factors in disorders which mimic schizophrenia in many ways. In 1900 the schizophrenias were $X + 14$ in number, yet now they are only X in number, where X is unknown. The fourteen separations are dementia paralytica (brain syphilis), pellagra (niacin deficiency), porphyria (abnormal form of chemical blood pigment), homocysteinuria (excretion of homocysteine in urine), thyroid deficiency, amphetamine psychosis, vitamin B-12-folic avitaminosis, hypoglycemia (low blood sugar), psychomotor epilepsy, cerebral allergy, wheat gluten sensitivity, histapenia (low blood histamine and high blood copper), histadelia (high blood histamine), and pyroluria (excretion of kryptopyrroles in urine). Undoubtedly, other specific entities will be separated from the hodgepodge we call the schizophrenias in the future. Hence, we refer to the disorder in the plural as we do the epilepsies.

Yet, another reason for speaking of the schizophrenias in the plural is the variation in their severity, duration, and symptoms. In the past, the serious schizophrenias have been labeled paranoid, simple, hebephrenic, catatonic, and mixed. These terms are of little help, since, except for the paranoids, the untreated patient may vary from month to month through all the diagnostic categories. The paranoid (unduly suspicious) usually stays true to his class and is the closest to normality in his quantitative brain waves, thoughts, and ideation. Simple therapy, such as adequate rest or quiet seclusion, can sometimes dispel the paranoia. Stress can aggravate paranoia. The aging process decreases paranoia.

Some females have schizophrenia only at their menstrual or premenstrual period. Others suffer from

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postpartum psychosis after the birth of a child. We now understand these episodes as the result of elevated estrogen and copper levels. Estrogen elevation causes a subsequent rise in serum copper levels and in this heavy metal intoxicant is the causative factor for these psychotic occurrences. One prolific housewife had postpartum psychosis after the birth of her fifth, sixth, seventh, and eighth babies! Her only complaint was that her present doctor was trying to cure her with talking therapy instead of electro-shock therapy which had been previously effective.

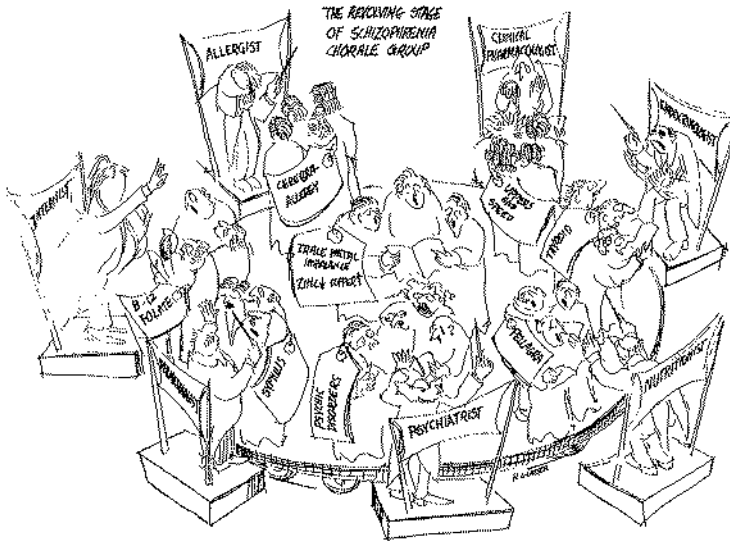


Figure 2: The schizophrenias are a hodgepodge of many biochemical imbalances which may send the patient to many perplexed specialists.

Schizophrenic patients usually should be told, when they ask about their illness: "Yes, you do have one of the schizophrenias, but this diagnosis, while real, covers disorders of extremely varying severity." Some patients have only mild disperception or thought disorder and go

through life without treatment, handicapped, but not seriously impaired. Others have only feelings of unreality which may come in waves when they are overtired, and the condition never gets any worse and may decrease with the aging process. A few may have serious symptoms which can be completely controlled by thyroid therapy. Some have a complete remission with specific drug therapy, and finally, some patients need every type of therapy that is now available. New research is constantly being done to improve antischizophrenic drugs, and our biochemical knowledge of the disorder is such that no one need any longer be afraid of the honest diagnosis of schizophrenia. As the late Dr. Nathan Kline remarked, "We have better treatment for the schizophrenias than for the neuroses." Only by facing up to the extent of the disease will we control it, and research progress is such that another decade should disclose the cause and provide the cure.

An analogy to the schizophrenias can be drawn in the case of rheumatism (the arthritic disorders). Every adult over the age of 30 has some type of arthritis—usually osteoarthritis of a single joint, such as a knee or the sacroiliac. Very few of us have rheumatoid arthritis in which multiple joints are hot and inflamed. In spite of the fact that arthritis can be a mild or a severe disease, the physician does not hesitate "to call a spade a spade" and tell the patient and close relatives that some type of arthritis exists. Effective treatments exist today for both arthritis and schizophrenia.

The recognition and treatment of schizophrenia has been held back by the failure of many physicians to realize its extent and by an unwarranted fear of schizophrenia. A patient recently remarked, "I am glad that I only have schizophrenia instead of a personality disorder. I know that schizophrenia responds to drugs, whereas any personality disorder would be hard to change."

Since the schizophrenias are not alike in outcome,

but only in behavioral symptoms, the recognition of a schizophrenia is important so that early treatment and regular counseling can be started. Some schizophrenias are mild and will be controlled by a reduction in everyday stress. Some are more severe and may need megavitamin therapy. For serious symptoms, one uses the effective antischizophrenic drugs, plus vitamins; and for the most severe, the patient may need electroshock therapy (EST), plus drug and talking therapy. Remember the rule of Dr. James Blake (circa 1800): "One-third of the psychotics recover spontaneously." Remember, too, that the hospital discharge rate for even the severest form of the disease is a whopping 75 per cent. Drugs and treatment yield a 90 per cent improvement.

Differential Diagnosis and Treatment of the Schizophrenias

We have already said that the schizophrenias were at one time much more numerous, namely X plus 14 similar disorders. Those fourteen syndromes, which were facsimiles of schizophrenia, are now separated from the schizophrenias. One may well ask if these disorders are still apt to be confused with schizophrenia. In regard to two, the answer is no; in regard to the last twelve, the answer is yes. In other words, the diagnostic tests for number one, namely brain syphilis, are now routine, and a blood serological test rules out the possibility of brain syphilis which might be confused with schizophrenia. The same is more or less true in regard to brain tumor. Brain tumors of the frontal lobe or of the mid-brain can be sometimes confused with schizophrenia; but the characteristic history, the presence of increased pressure of the spinal fluid and the frequent occurrence of convulsions, will usually eliminate brain tumor as a possible alternate diagnosis.

We then come to (number two) pellagra, which has almost disappeared, because many staple foods, such as corn meal and wheat flour, have niacin or niacinamide added in order to prevent pellagra when these foods are used as a main source of diet. Only in rare instances can an individual now get a dietary deficiency of niacin with a resultant pellagran psychosis. In other words, grave undernutrition is needed in order to produce this disease with present-day foods.

The diagnosis of porphyria (number three) is more difficult to eliminate, and, undoubtedly, some patients now labeled schizophrenic have had porphyria off and on during their lifetime. Fortunately, it is a rare disease, so the chance of a misdiagnosis is uncommon. Porphyria as a mental disease has been publicized by two British psychiatrists in the July, 1969, issue of the *Scientific American*. Drs. Ida MacAlpern and Richard Hunter point out that King George III of England, who was king of England at the time of the American Revolution, undoubtedly suffered from porphyria. At that time the cause of the disease was unknown, but he had the typical and classical symptoms of porphyria which are as follows: acute abdominal pain, which is followed by some constipation; darkening of the urine to a port wine color; weakness or even paralysis of the lower extremities; hoarseness; fast pulse, and convulsions. As the convulsions subside, the patient usually has a period of delirium, and the whole sequence of symptoms may come and go at yearly or ten-yearly intervals. In the case of King George III, these attacks were at ten-yearly intervals.

Thyroid deficiency (number four) has been called myxedematous madness, because many patients who have inadequate secretion of the thyroid hormone will show the symptoms of schizophrenia. A slight degree of hypothyroidism may go unnoticed, and a few patients who seem to have normal thyroid function and schizophrenia

will have their mental symptoms clear when thyroid or thyroid hormones are added to their therapeutic regime.

As for number five, amphetamine psychosis, we will discuss this in other parts of this primer. The use of weight-reducing tablets and the overuse of the amphetamine-type stimulant drugs can produce a syndrome which is indistinguishable from so-called schizophrenia. The patient may have paranoid schizophrenia or other classical types. This may happen to the unwary teenager or middle-aged woman who uses weight-reducing pills enthusiastically or even takes them exactly as prescribed by an enthusiastic doctor. Thus, weight-reduction medications may precipitate schizoid symptoms, and the juvenile who has access to "speed," methedrine, or "Bennie pills" can induce abnormal ideation and behavior as a result of overstimulating his brain. A careful history is usually adequate to eliminate this type of drug-induced schizophrenia.

Yet, another disease (number six) which may be confused with schizophrenia is homocysteinuria, which is one of the metabolic disorders in which the patient excretes the abnormal amino acid, homocysteine. This occurs in children and has been successfully treated by a low methionine diet plus additional pyridoxine, or vitamin B-6, in the diet. (These patients need 150 to 300 mg. pyridoxine daily.) Under these circumstances the patient can usually grow normally without symptoms of mental disorder.

One of the most difficult diagnosis at present (number seven) is the differentiation of schizophrenia from vitamin B-12 and folic acid deficiency. The onset of vitamin B-12 deficiency can be insidious in the adult and will produce all of the symptoms of the paranoid or other types of schizophrenia.

The doctor must always keep this deficiency in mind when an adult patient develops symptoms of

schizophrenia. Schizophrenia when it occurs for the first time in older adults is sometimes called "paraphrenia." Thus, the target organ for the vitamin deficiency may be the brain rather than the blood, and a patient may have a specific B-12-folate deficiency of the brain with only minimal anemia and, certainly, no signs of pernicious anemia. The importance of the early diagnosis of this deficiency is evident because we know that this type of disorder responds dramatically to B-12 and folate therapy and does not respond to the antipsychotic drugs. Thus, the differential diagnosis of schizophrenia late in life presents many more subtleties and nuances than does the diagnosis of schizophrenia in the teenage years when such a vitamin deficiency is not apt to occur.

Dr. Edward H. Reynolds of the National Hospitals for Nervous Diseases, London, has been a pioneer in studying folate and vitamin B-12 levels of the blood of mental patients and epileptics. He has been reported and others have confirmed that prolonged Dilantin use for antiepilepsy therapy will produce a specific folic acid deficiency. Thus a typical patient, who has had only grand mal seizures (epilepsy) during the early years of life, may develop by the age of 15 to 20 a folic acid deficiency and the full-blown symptoms of paranoid schizophrenia. This psychosis responds to the use of some other anticonvulsant, such as Valium, to control the seizures and the gentle use of small doses of folic acid and vitamin B-12 to raise the blood levels of these two vitamins and remove the deficiency.

Several patients at the Princeton Brain Bio Center who had a history of epilepsy of many years' duration and who had been treated vigorously with Dilantin to prevent grand mal seizures developed schizophrenia, and their blood samples showed their vitamin B-12 to be in the mid-range of normal and their folic acid levels to be one-half that of the minimal or lowest level ever encountered in

normal people.

Obviously, the old rule applies: "The doctor must think of the disorder in order to diagnose it." The difficulty in the accurate diagnosis of the schizophrenias, and mental disease in general, led Dr. Derek Richter of London to point out that, with present psychiatric diagnoses, one may be as accurate as children labeling dolls. One "Helen" is not another "Helen."

THE BORDERLINE PATIENT

There are a group of patients who, much like the fabled Richard Corey in Simon and Garfunkle's song, look normal to all the world and one day go up to their room and kill themselves. Impulsive behavior is common in this group and often presents with crises such as suicidal or homicidal ideation, gestures or attempts, drug abuse, fleeting psychotic episodes or major psychotic decompensations. This phenomenon has been variously referred to as latent schizophrenia, pseudoneurotic schizophrenia (Hoch and Polatin, 1949), the "as if" personality (Deutsch, 1942), the borderline personality (Kernberg, 1967), the borderline state (Knight, 1953), and the borderline syndrome (Grinker et al, 1968).

Although the use of the term "borderline personality" seems to imply uncertainty on the part of the diagnosing psychiatrist, it is behaviorally used to separate a fuzzy mish-mash of patients. Borderline patients are "merely at the edge of schizophrenia," but are clearly psychotic at times. Their behavior is not limited to classically neurotic patterns; they are not psychopaths, yet they are not normal. Some say they are best located in a "borderland" somewhere between an integrated personality and a disintegrated personality.

The four most prominent, behavioral, clinical features of the borderline personality include: 1) an intense anger that is not integrated into life's experiences, 2) intense and unstable relationships reflecting an inability to integrate positive and negative feelings about other people; 3) a self-identity dependent on role playing which, when present, covers over signs of illness and when absent leads an "overwhelming panic of non-being," and 4) periodic perceptions of profound isolation and consequent depression often expressed as an intolerance of being

alone. Some 20% of all patients in hospitals or in psychotherapy are diagnosed as borderline. Borderline patients as a category cry for definite diagnostic biological keys to their actual disorders. We have found them to be histadelic and poisoned with copper.

A careful history is usually needed to reveal the severity of the illness because borderline patients often look quite good even in a crisis. As Richard Corey's neighbor reported after his suicide, there is often no indication whatsoever that anything is wrong. This characteristic of play-acting a "healthy person" role is quite common and often the most dangerous aspect of the disorder. Everything seems black and white, all good or all bad. When they are good they can be very, very good. Many patients report spans of excellent achievement in school or at work. Their manners and appearance may even seem particularly appropriate in certain situations. A careful history, however, will reveal that these very appropriate features reflect the absence of any firm identity. The behaviors are generated by a force of mimicry or a form of rapid, superficial identification with certain people whereby they take on the predominant features of friends around them. The history may tell of many such episodes, often similar in form and rarely similar in content.

The problem is that there are often as many instances when these patients have been "bad." When they are troubled it can be scary, mostly because it is so unpredictable. The moment they seem to be in fine spirits may be directly followed by another moment, when they just as easily completely absorb themselves with dark feelings and complete a suicide quickly and silently; when they feel good, it seems as if nothing could ever go wrong, but when they are down suicide seems the only recourse available. Due to their impulsive behavior, these patients cannot be easily trusted; their decisions are most arbitrary. Clinically, such impulsive behavior may surface in an

emergency room as self-mutilation, an auto accident, or a drug overdose. Counseling rarely helps because a commitment to avoid such behavior does not last if a moment or two later they feel like doing anything!

Feelings directed towards the physician are known to change just as rapidly. Even if initially the doctor is viewed to be a savior with all the answers, as soon as one unanswerable question is unanswered, a drastic shift may follow and the patient may view the doctor as uncaring and alien. Urgent appeals for help are often mixed with innumerable attempts to undermine or devalue the care they receive. A great example of this is seen in the person who calls a suicide hotline, pleading to be stopped and then refuses to give an address or phone number. They may call the suicide and drug hotlines "for kicks" and to criticize.

As with the therapeutic relationship, all interactions with others are viewed as either all good or all bad with no ability to freely integrate the two opposing feelings. The unrecognized fact is that all of us are neither saints nor sinners, but rather somewhere in between. Not realizing this, these patients cling dependently then disappear completely, all the while oscillating between love and hate. A good history may also reveal vacillations between transient, superficial relationships lacking any real sense of commitment and intense liaisons marred by manipulation, insistent demands, and criticism.

Given the right opportunity, these patients may express just how angry, depressed and lonely they really are. Many cannot remember a time when they were not depressed and recognize their sudden bursts of intense anger. Although they hate to be alone, they quickly alienate their most patient friends through manipulation and abuse. Intimacy is scary because it threatens an already fragile sense of autonomy and self-identity. No amount of closeness or distance ever seems just right.

Borderline patients frequently decompensate into a

mini or major psychosis at times of extreme pressure, and then recompensate when the pressure is removed or released. Such transient psychoses are common and are the main feature which distinguishes borderline patients from psychotic patients. Psychotic patients do not bounce out of their state quickly and get right back in touch with reality as borderline patients are known to do. These brief dissociative experiences can prove dangerous, prompting patients to further exercise poor judgment which often leaves them in life-threatening situations.

Because their whole life centers around adaptation by defense mechanisms, and they do have a full array of such mechanisms, most successful therapies focus the attention on encouraging more mature, more stable defense choices. Medication is to be administered with extreme care to avoid any opportunity for patients to use the medications for their inordinate highs, lows, or suicides. If a patient is clearly dangerous, then restrictive measures may need to be taken; but in general, a consistent, limited, and steady approach is adequate to maintain a relatively stable relationship with someone of borderline personality. Such a steady state can be difficult to maintain given the inevitable emotional turmoil and obvious manipulation that often enrages those who try to help. Perhaps it will seem easier to come to the aid of a person whose illness is better understood. Surely early recognition and appropriate referral can at least lessen associated frustrations.

Certainly, borderline personality is a syndrome crying for adequate biological diagnosis. With treatment to eliminate heavy metals such as copper and aluminum, and nutrients to repair any deficiencies, these patients do well with orthomolecular care.

PARANOIA: THE DELUSION OF PERSECUTION WITH GRANDIOSITY AND NAIVETE

I include in paranoia only those patients in which a system of delusions is psychologically elaborated, slowly and continuously over a number of decades without deterioration, without mood swings, and without changes in external appearance. In dementia praecox hallucinations are frequent, whereas they are rare in paranoia.

— Emil Kraepelin, from his 1898 speech at Heidelberg wherein he differentiated Dementia Praecox from Manic Depressive Disorder. *Zeit. for Neurol. und Psych.* 1899

Introduction

Emil Kraepelin, the German psychiatrist, divided the schizophrenias into several types according to behavior. Paranoia was the name he selected for a state of extreme suspiciousness in which the individual may have delusions of persecution. Since Kraepelin's time, the name paranoia acquired its modern meaning, namely, undue suspicion. Of the Kraepelinian types of schizophrenia, the paranoid state is the most constant and also the closest to normal so that with proper and continued therapy a complete remission can result.

In a system run by pseudo-scientific clinicians whose main claim to fame is reclassifying and renaming of their patient disorders, the term paranoia and the paranoid state are remarkably constant. For instance, schizophrenics may vary from catatonia to confusion, but the paranoid patient usually remains remarkably paranoid. Paranoia is, therefore, a real diagnosis which may have a constant biochemical imbalance as its cause.

Personal Space in Paranoia

Psychologists have done studies on the distance between the heads of patients in normal dialogue. This is termed "personal space". The severe paranoid is apt to have a greatly decreased personal space. I remember a graduate student with paranoia who complained frequently to me as her professor. I always arranged to have a soft drink which I could raise to my lips and, thus, force her face back an extra six inches since her fashion was to complain nose to nose. The personal space varies in many regions of the World with the Asians having the smallest personal space. I also recall talking to a paranoid medical doctor in Germany who got closer and closer as he told me his symptoms. I shifted my briefcase to the floor in front of my feet to fend him off. After we had established good rapport, I pointed to the briefcase as my front line trench against his paranoia.

Lack of Voice Volume Control in Mental Disease

Many compensated schizophrenics will not only have a narrowed personal space but will also speak inordinately loud for the circumstances. Sensible people will modulate the volume of their voice to compensate for the distance of the listener—soft voice at 2 feet and louder voice at 10 feet. The compensated schizophrenic will use a single loud volume of voice output regardless of the distance to the listener. As the patient improves and the paranoia lessens, the normal personal space and volume control takes effect. I recall a scientist's wife with simple schizophrenia who always embarrassed us with her loud unmodulated voice. When in Stockholm at the N-K department store we heard an American say "But I can get it much cheaper in America." As predicted the loud voice was that of Henrietta from Madison, Wisconsin, still

embarrassing us in Stockholm.

Part of the difficulty of the paranoid is the stress produced by the hustle and bustle of present, everyday life. Historically, many paranoids became hermits, recluses, or gurus, who lived apart and were only consulted on important occasions. With urbanization, this secluded life is hard to find. In my early days, I knew a bachelor farmer who made a fair living, but we children were always warned to keep off his property because of his bad temper and difficult personality. When the interstate highway dissected his farm, the farmer went crazy and had to be hospitalized.

Diagnosis Can Be Difficult

The paranoid patient is probably the most difficult of all the schizophrenics to diagnose and adequately treat. In many instances, the patient may appear normal and be paranoid in only one particular area such as marriage, religion, or politics. Thus, a doctor may very well assume a patient to be normal if he fails to get his reaction on some specific subject. The doctor must question a suspected paranoid patient about many aspects of life and interests in order to make a correct diagnosis. The paranoid, aberrant at home, may simulate most effectively a normal personality when questioned by a therapist or judge.

One male patient who was serving in the army in Viet Nam when his paranoia began had the fixed notion that Central Intelligence had substituted puppets for his mother and father. Neither of the parents were his real parents—only imposters! This delusion had continued for several years and finally they persuaded the patient to visit the Brain Bio Center. In this setting, I, the doctor, was General LeMay placed behind the desk to extract information from him. The agents were everywhere and the only safe place for him was Russia or Red China

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because the American secret agents couldn't penetrate the iron or the bamboo curtains! He consistently refused hospitalization or treatment until he was arrested because he spanked a small boy for throwing a snowball at his car. He over responded to this minor insult and the judge wisely insisted on hospitalization and treatment.

Undue Suspicion with Naivete is the Paranoid Personality

Unwarranted suspicion is the basic paranoid trait. Paradoxically, this often co-exists with naivete. The paranoid is unduly suspicious of those who are trustworthy, yet he is trusting when he should be on his guard. For example, in his naive state, the paranoid may invite robbery by leaving his money, wallet or other valuables in plain view of strangers, assuming they won't be stolen, just because at that particular moment his mind is occupied with his paranoia. As yet another example, one paranoid patient left his valuable camera as security in the taxi while he went into a drug store to get change for a twenty dollar bill to pay the taxi fare. When he came out with the change, the taxi and his valuable camera were gone!

No Close Friends

A candid statement by one young adult paranoid patient was, "I can rap perfectly well with children and older people but I can't get along with people my own age." At 21 years of age he had never dated or had a girlfriend and he had no close friends because of his difficult personality.

Wants to Travel

One of the significant questions on a popular personality inventory states, "I have a wanderlust and wish to

travel continuously.” This applies specifically to the paranoid patient who cannot find friends his own age and hopes that humans in some other part of the country (or world) will be more understanding and friendly. Thus, he runs away and the constant youthful traveler may be an unduly suspicious psychiatric patient.

Rigid Personality

The paranoid is rigid and will not admit to minor faults which most people have. The paranoid wishes to be seen by others as always virtuous, which results in rigid, defensive and uncompromising behavior. This rigidity makes the paranoid insecure and frustrated so that hostility may break into the open in verbal tirades and childish temper tantrums which are ordinarily abnormal in the adult. The paranoid tolerates risk, but cannot tolerate surprise of any kind. Knowing this, his co-workers may make him the victim of practical jokes—which doesn’t help!

Grandiosity—the Eagle Ignores the Mosquito

A paranoid frequently believes that others are experiencing similar feelings. He also believes that minor external events are directed toward him which cause him to think that no one can be trusted, no one should be loved, and everyone doubted. He methodically builds a grandiose wall around himself and becomes ever more isolated in his lonely confusion. Unfortunately, he is rarely happy behind this self-imposed wall.

Often the paranoid may show an intense preoccupation with social issues. His chief interest may be “the principle involved.” This can lead into long drawn-out lawsuits or one-man reform campaigns. And yet, if the principle is actually upheld, the paranoid still remains

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unhappy. Because of this wish to carry on the fight for the sake of the principle involved, paranoid personalities are always poor losers in business or in social encounters.

The paranoid's basic unhappiness is further revealed by his lack of humor. If he is told a joke, he may react in anger or perhaps ask, "What do you mean by that?" When the paranoid is asked a question, he invariably answers with a question. For example, the simple greeting, "How are you?", is often answered with, "In what way did you mean?" Other times he might reply, "You're the doctor, you should know," or "Why do you want to know that?"

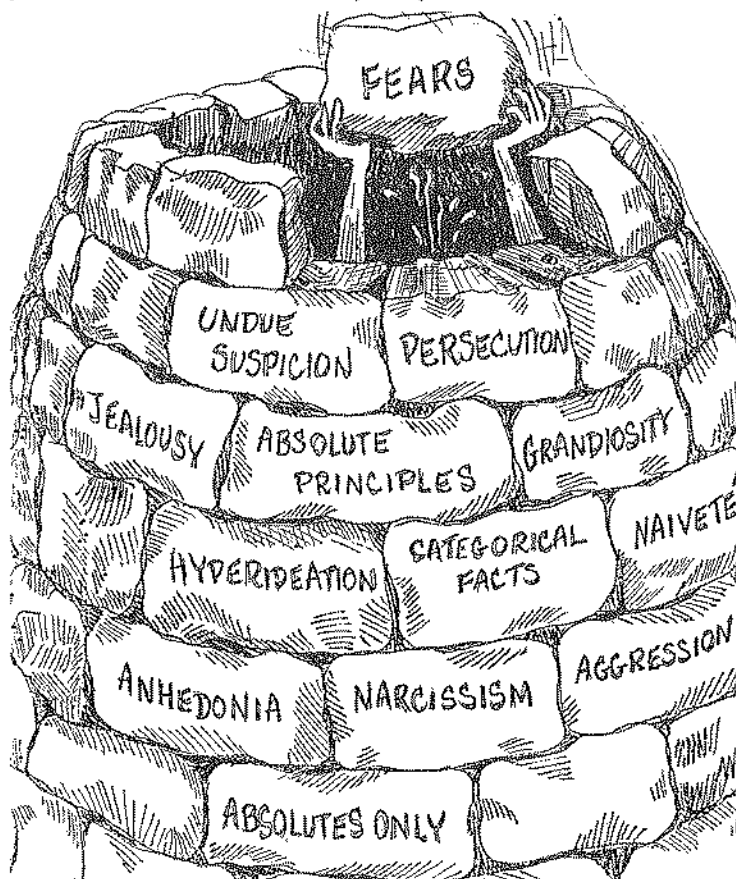


Figure 3: Paranoid patients build a wall around themselves by answering a question with a question.

The notion that everything is connected or explained by a Unitary Theorem: "My findings explain everything," the paranoid religious scholar stated. The seventh law of Moses states, "Thou shalt not commit adultery." The licentious activity of present day society is therefore responsible for drug addiction and all other ills of society according to this paranoid scholar. All is connected and easily explained by a single theory. "Allergy is the cause of most mental disease." "America should be reserved for blue-eyed blondes."

Limited Love Life

Generally speaking, the paranoid is his own and only true love. The female paranoid usually refrains from contact with the opposite sex. And while some paranoid males may have a harem of girls, as in the bizarre case of Charles Manson, this is an egotistical type of involvement rather than real affection. The paranoid's real life remains that of an unhappy and unfulfilled narcissist. Narcissism is falling in love with your own reflection or way of life. The term derives from Greek mythology where a beautiful youth spurned the love of Echo, so Nemesis punished his indifference by causing him to fall in love with his own face reflected in the calm water. He died of unrequited love and was changed into the flower Narcissus or Daffodil.

Grandiosity Promotes Autobiographies

The paranoid's self-interest is evident in his studies and writings. The grandiose personality of the paranoid leads him to believe that his life is unique and worth reporting in an autobiography. In many instances, his experiences have been harrowing. The paranoid state is very close to the hallucinatory state, and such proximity can indeed contribute colorful material to his autobiography.

If the patient is not already engaged in writing, the doctor can suggest this as a useful form of occupational therapy. The idea of a "best seller," such as Catcher in the Rye, is sufficient bait to keep the patient occupied.

The Grandiose, Hyperideational Paranoid

The idea man in business who is a hyperideational paranoid does not make a good leader. He may be the executive in a top job who has too many ideas; in fact, so many ideas that he gets little or nothing accomplished. If he has understanding colleagues who can restrain him when necessary, he may be of value to the organization.

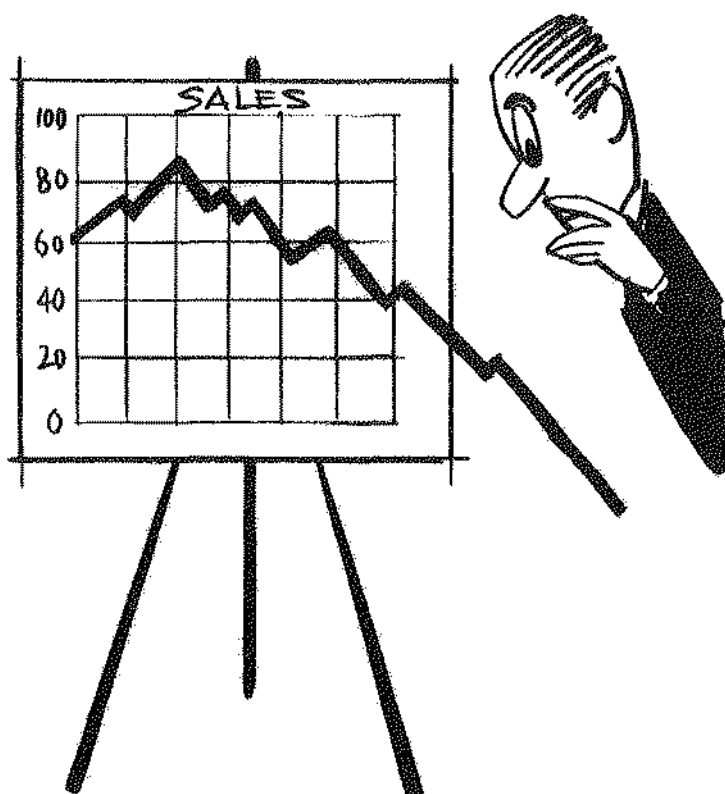


Figure 4: Hyperideational Paranoid—adequate title.

But if he has absolute control of the business, he can rapidly precipitate bankruptcy. These hyperideational individuals will not listen to words of caution, or other peoples' ideas, so many enemies are made in business encounters. Hitler is a perfect, but extreme, example. The havoc of his paranoia could have destroyed civilization if the German scientists had developed the hydrogen bomb instead of the "Buzz Bomb."

Diagnostic Interview

During the psychiatric interview, one paranoid has told me, "Doctor, you smile when you talk; you are laughing at me." I tried talking dead-pan thereafter! Harry Stack Sullivan, the famous psychiatrist, always kept his face turned ninety degrees or more away from the paranoid, realizing his usual smile could be misinterpreted. Experience had taught him full well the impossibility for a normal person to talk without smiling. Perhaps the couch in psychiatry was introduced for the same reason.

In terms of diagnostic categories, the paranoid's behavior usually remains within the limits of his classification. Compared with other mental patients, he is the closest to normality in his quantitative brain waves, thoughts, and ideation.

The Night People

Paranoids may be night owls who turn night into day, partly to avoid social contacts, but also because they realize that stress and noise are less at night. The geomagnetic forces of the sun, which may exert an influence on the schizophrenic, are also lessened at night. One paranoid patient did well in his first nighttime job as a short-order cook in an all-night restaurant, thus solving his previous difficulty in holding a daytime job.

Insomnia Causes Paranoia

If any patient comes to a doctor after several nights of insomnia, he will be abnormally paranoid. Insomnia or forced lack of sleep will produce paranoia in the strongest of individuals. This has been shown many times in the armies throughout the world and in carefully controlled laboratory experiments. These psychotic breaks are called battle fatigue in the army, although the predominant symptom may be paranoia. A definite diagnosis of paranoia should always be reserved until the patient has been adequately rested. The patient usually does not disclose his paranoia on either the HOD or the EWI tests. The MMPI test may disclose paranoia in young, naive patients.

Drug Use Can Cause Paranoia

Paranoia is frequently caused by amphetamines, as in diet pills, since light sleep or insomnia robs the brain of its rest. An acute episode of paranoia can be caused by cocaine usage, or "acid" (LSD-25) both of which are profound stimulants to the brain and will cause paranoia in susceptible individuals. Insomnia may contribute to this paranoia.

Downers such as alcohol, barbiturates, or other sleeping pills can unmask latent paranoia. The belligerent drunk, for example, will interpret normal advice as an insult and try to start a fight. On continued daytime and nighttime use of downers of any kind, the normal individual may get into a constant paranoid state like one of our doctors (on Nembutal) who always turned in a fire alarm when the setting sun was reflected in the hospital windows! Phenytoin (Dilantin) is an anti-folic acid drug which will, in high dosage over a period of several years, cause a severe paranoia which may be difficult to treat if other drugs fail to control the epileptic seizures.

Does Hearing Loss Cause Paranoia?

In older people, a syndrome of hearing loss and paranoia may build up together. This rarely happens with approaching blindness. Some writers have made much of the absence of sound causing paranoia, whereas really the absence of sight would be more frustrating and stressful. Perhaps accumulation of copper in the brain with age can cause both the loss of hearing and the paranoia. We have found in young patients that paranoia slowly decreases with age. Under some circumstances such as slowly developing deafness, paranoia may increase with the degree of deafness. The British call this paraphrenia, a term reserved for middle and old age schizophrenia. Doctor D. W. K. Kay and his colleagues in Newcastle, England, have studied paranoids whose onset of illness started at ages 50 through 79 (mean age 61.1 years). The prevalence of deafness was as high as 30% among the younger patients in the group. Environmental poisons such as lead and copper are known to produce ringing in the ears and overstimulation of the brain, so the late onset of paranoia may be a toxic effect from the slow accumulation of heavy metal poisons. Doctor Isabel Tipton of Knoxville, Tennessee, has shown that copper accumulates in the brain with age.

Treating the Paranoid-Homicidal Patient

An otherwise competent psychiatrist related how he had avoided treatment of a paranoid young man.

"On the initial interview the lad had a red aura above his head which clearly conveyed the message to me that he was dangerously homicidal. Sure enough! Within the next week he killed both his father and mother. I was lucky that I had not tried to treat him." Obviously, the psychiatrist had saved his own skin but what about the two lives

that were lost because of his inaction or incompetency?

As a biologist I would probably have difficulty in seeing a halo on Christ if he came to town, let alone seeing an aura of any hue above the head of a homicidal patient. We do see and feel bad vibes and we do pick up "paranoia" on the Experiential World Inventory as well as "impulsivity." When "depression" and "impulsivity" scores are high, the patient is suicidal. When all three are elevated, the patient is homicidal and we start effective treatment as soon as possible.

Fred, a man in a midwestern town wrote us a letter, the gist of which was "I am paranoid and homicidal. If you don't see me I shall kill myself with my 0.357 magnum but before I go I'm going to take my enemies with me." My secretary read the letter first and promptly referred the patient to a clinic in the middle west. I reviewed the correspondence and decided that our Brain Bio Center was probably the only place in the world where such a patient could get confidential, effective and careful treatment which would not activate his trigger finger. Fred had mentioned in his letter that his vacation was available in several weeks, so we wrote him that because of the urgency of his need we would see him for a two-day period to do our regular tests and, in addition, to test him for allergies which might be a factor in his paranoia. I suggested politely that he leave his weapon at home. Rumors flew as more of our help read his letter of threatened violence. I held a staff meeting to explain that homicidal acts occurred only in a patient who was dissatisfied with treatment and the professional's attitude toward the patient. Fred needed immediate help and only our BBC could give such help. The other professionals remained skeptical and unconvinced. As we planned for his arrival, some of the skepticism and fear diminished. We treated Fred successfully without any untoward incident.

We know that the paranoid patient is high in serum

and tissue copper. If zinc alone is used to treat these patients, they may become more paranoid because the copper is mobilized from the muscles and liver so that serum copper may increase the paranoia with fatal results. If a chelating agent such as Cuprimine (d-penicillamine) is given at the onset of treatment, the serum copper will not rise dangerously and the patient has the possibility of immediate improvement. Fortunately, this is exactly what happened to Fred; he got well and didn't become a mass killer.

Paranoia Decreases with Time

In our treatment of paranoid patients over a twenty-year period, we know now that even without treatment the degree of paranoia naturally decreases with age while the degree of depression naturally increases. A paranoid son who successfully sued his parents ten years ago is now happily visiting them and has gotten over his parental intolerance.

The Biochemical Imbalance of Paranoia

We have found that serum copper is high in the paranoid (130 to 200 mcg percent) and blood histamine is low (0 to 40 ng/ml). The high serum copper level has been confirmed by other workers. Hair copper is also high. Also, the blood folic acid level is low. These patients benefit from zinc, manganese, niacin, folic acid and vitamin B-12 therapy. However, progress is slow and the paranoid is impatient, so slow progress may not be enough! The paranoid is notoriously noncompliant often fearing that the therapist or the treatment prescribed have somehow become integrated into the immense plot against him.

Better Treatment of the Paranoid

The patient is reluctant and mistrustful to say the least. The paranoid requires results without side effects and fortunately Cuprimine (a copper chelator) morning and night may occasionally provide the answer. The paranoid is high in serum copper which can be removed slowly by the use of zinc and manganese. However, the patient may discontinue medication at the first sign of any side effect so we frequently use Cuprimine to chelate the excess copper out of the body via the urinary pathway. Cuprimine comes from penicillin so one must first ask if the patient is sensitive to penicillin. If not, Cuprimine can be used safely as long as zinc, manganese and vitamin B-6 are also given. The B-6 is given at noon and the zinc and manganese are given morning and night. Loss of taste occurs with zinc deficiency which Cuprimine may produce since the drug removes both zinc and copper from the body, and the precious zinc must be replaced.

Anti-schizophrenic drugs must be started at small dosage so that muscle effects are minimal. The cautious use of bedtime dosage is helpful and the patient must have easy access to the physician to provide quick reassurance. As the copper level in the blood and tissues decreases, the patient will sleep better and longer. Occasionally, the paranoid can be coaxed into accepting an intramuscular injection of Prolixin enanthate or decanoate which will provide effective medication for a one-week period. If Prolixin is used, the patient should have Benadryl at bedtime and Cogentin available for the muscular side effects.

Often, however, the paranoid has no insight into his illness and refuses the good advice from family and friends to seek medical help. If he refuses to take proper medication, Haldol concentrate may be put in his food or drink surreptitiously. This can be done because Haldol in liquid

form is tasteless and colorless. In many cases, this extreme action must be taken in order to get the patient on the path to recovery. However, caution must be used. If the patient discovers that he's "tricked," even though it is for his own good, he could become even more suspicious of those around him.

Self-Treatment with Niacin

Robert, a 23 year-old paranoid, came to us with the observation that he needed 30 grams of niacin per day in order to relieve paranoia and to do his work as a garage mechanic. We suggested the use of our top dose of niacin, namely, 3.0 grams per day to be supplemented with folic acid and vitamin B-12. Folate and B-12 helped in that he gradually reduced his dose to 15 grams of niacin, and on that dose he enlisted in the air force for training as an airplane mechanic. In the service, he ran out of niacin and folic acid. He relapsed and was given a medical discharge as paranoid with service-connected disability. This large dose is not advisable since on 30 grams of niacin his uric acid was 9.23 mgm percent—a level which will produce symptoms of gout. On 15 grams of niacin, the uric acid was 6.00 mgm percent, a much safer level. Niacin inhibits the urinary excretion of uric acid. Robert is now happily married and doing well in his own business.

In summary, the paranoid displays a remarkable genius for detecting in ordinary life situations those tiny inconsistencies, adversities or criticisms which others overlook but which the paranoid snowballs into "root causes," "crucial issues," and "the principle of the thing," all of which are seldom imagined but always over-emphasized. Once the paranoid is willing to assume the role of patient in a medical model, the paranoid can be successfully treated and the paranoia dispelled. Walt Kelly has Pogo say, "We have met the enemy and it is us." This

can be paraphrased for the paranoid into "I have met the enemy and it is me." When the paranoid has enough introspection to believe this, he is on the road to good mental health. The biochemical imbalance to be corrected is the high tissue copper level. With the patient's cooperation this imbalance can be changed towards normal.

Case 1

Gladys H., aged 30, was a long-term unresponsive paranoid patient when on our state research ward in 1950. Each day she would make a long list of charges (as she termed them) against the professional staff and the state. This list contained upward to ten to fifty items depending on her mood. Touching her arm was the beginning charge and the final charge against the staff was "thinking the way I do charges." The therapist, no matter how skillful, could not win even if he always agreed with this paranoid patient!

Drug Induced Paranoia

Case 2

Jean R. was aged twenty-six and severely paranoid since age eighteen. At age eight, she had fallen while skating and had a concussion. Epilepsy with major seizures developed at age 12. The seizures could only be controlled by large doses of phenytoin (Dilantin), an anti-folic acid compound. Six capsules (600 mgm per day) abolished all seizures but at this high dosage her paranoid mental state was unmanageable. On 400 mgm of phenytoin she had only one seizure per month. This monthly seizure was as therapeutic as EST, and for one week after the seizure she was completely manageable and much less paranoid. She was folic acid deficient from the prolonged phenytoin therapy. The use of folic acid, folinic

acid or citrovorum factor produced an epileptic seizure after each trial. Drugs, other than phenytoin, such as Valium would not control her seizures. Megavitamin therapy with Trilafon did not control her mental state, and she frequently was completely uncooperative in the taking of these vitamins. She died of a self-inflicted knife wound in the summer of 1970 before we knew about pyroluria and could use zinc and pyridoxine to control her phenytoin-induced paranoia. Phenytoin paranoia due to folic acid deficiency is a common result of long-term anti-epilepsy therapy.

AUTISM AND CHILDHOOD SCHIZOPHRENIA

Autism and childhood schizophrenia are two of the many schizophrenias. Both are found in childhood and are known to affect three or four children per 10,000 among any given peer age group. Until more is understood about the causal differences between the two illnesses, they will remain distinguished, both from each other and from the norm, with regard to behavioral symptoms. The fact that each is classified as "one of the many schizophrenias" is attributable to the great variety of symptoms and behaviors included in the schizophrenias.

Common Misconceptions

The term "autism" seems to tacitly imply an analogy between the autistic child's behavior and the "daydreaming" indulged in by the imaginative adult. That some inaccessible adults apparently preoccupy themselves with pleasant fantasies provides no warrant for believing that a child who has never been accessible is similarly preoccupied (Rimland). Autistic children are not simply daydreaming.

Childhood schizophrenics are also quite distinct from the normal child. Some few students of the mind (who do not understand the schizophrenias) have confused parents by claiming that most normal children live in a partial schizophrenic state or pass through schizophrenia as they mature. While it is true that normal children do play with fantasy, the experiences of the normal child are nothing like the experiences of the schizophrenic child who actually experiences fearful hallucinations. As one child said, "I have nightmares and daymares." A normal child's fantasy-land of imagination and play-acting

occurs naturally. It results from the simple fact that their minds are not yet disciplined by disappointment; they are not yet inhibited by their elders. The following verse by A. A. Milne is evidence of such natural childhood freedom. Contrary to some misconceptions, the many sorts "of funny thoughts" running 'round the head of this child are nothing like the hallucinatory state of the childhood schizophrenic.

Halfway Down

*Halfway down the stairs
Is a stair
Where I sit.
There isn't any
Other stair
Quite like
It.
I'm not at the bottom,
I'm not at the top;
So this is the stair
Where
I always
Stop.*

*Halfway up the stairs
Isn't up,
And it isn't down.
It isn't in the nursery,
It isn't in the town.*

*And all sorts of funny thoughts
Run round my head:
"It isn't really
Anywhere!
It's somewhere else
Instead!"*

The Differences between Autism and Childhood Schizophrenia

Basically, the autistic child remains unoriented,

having never been in touch with reality, while the child schizophrenic, like the adult schizophrenic, usually becomes disoriented and hallucinating following an initial period of normal development.

While both often seem withdrawn, their associated behaviors are quite distinct. The confused, anxious, concerned schizophrenic child frantically rejects prior to withdrawing. In contrast, the detached autistic child is usually aloof and alone, appearing disinterested, independent, and self-sufficient. The absence of the words "yes" and "I" in the autistic child language and the presence of hallucinations in the schizophrenic child are two of the most striking and unique symptoms which aid the often difficult task of differential diagnosis.

With regard to such attributes as overall health, appearance, responsiveness, and motor ability, autistic children are often quite characteristic. Physically, they are described almost invariably as beautiful, in excellent health, of handsome build, and light complexion. Their histories rarely include serious illness, allergies, asthma or skin problems. Although they show no explicit signs of intention, they are generally stiff and unresponsive, rarely accomodating either to the body of those holding them or to the emotional and social status of the environment surrounding them. According to Rimland, "the totality of an experience that comes to the child from the outside must be reiterated, often with all its constituent details, in complete photographic and phonographic identity."

It is even more remarkable to see an autistic child's incredibly coordinated motor and musical "skills" surface in their typical idiot savant performances. Such unusual memory, musical, motor and mechanical "talent" is not reported in schizophrenic populations. Schizophrenic children are more often poorly coordinated with regard to locomotion and balance. While schizophrenic motor symptoms include stereotyped repetitive actions, the

quality of these actions is likely to be hyperactive, gross, full-body whirling, clumsy toe-walking, or silly grimacing. Only occasionally a schizophrenic child is found who is catatonic or has waxlike rigidity. Overall, these children are noted for their strong tendency to physically "mold" to people when held and for their often intense need for attention and care. Also unlike the autistic counterparts, schizophrenic children are generally remembered to have been more sickly from birth, their poor health frequently complicated by serious respiratory, circulatory, metabolic or digestive difficulties.

The Autistic Child

While we admire, attempt to preserve, and even long for the active imagination and free-wheeling thought of the energetic normal child, the autistic child's pathetically overregulated thought elicits worry and apprehension. Dr. Bernard Rimland of San Diego has likened the autistic child's experience of life to the experience of a traveler whose dark path is only narrowly lit by the narrow beam of a flashlight. Both the traveler and the autistic child only focus on that which appears directly before them. Such a strong tendency to concentrate on minute details may be responsible for the idiot savant-like (typically a child able to solve large arithmetic problems) performances as described by Bettelheim. Such an absorbing focus on minute detail may also explain their complete inability to assimilate everyday speech and social interaction.

Dr. Leo Kanner, one of the foremost authorities on the subject of infantile autism, describes such infants as children who have been unusual from almost the beginning of life with autistic personalities, which cause them always to appear to be living in a private, inaccessible dream world, isolated, seemingly by choice, from contact with others. By two months of age, a normal child

consistently responds to friendly overtures, seems interested in his surroundings, and recognizes his parents as somehow more important than other people. A two-month-old autistic child, on the other hand, does not respond to friendly overtures. Although the first explanation for the dull, seemingly uninterested affect is simply that the child is "slow," after a long wait and no sign of recognition, parents relabel the problem as severe.

From a distance, the typical autistic appears only handsome and healthy. A closer look, however, distinguishes him from his more normal friends. (We refer to the autistic child as "he" because the disorder is four times more frequent in males.) The normal sparkle of childhood, so colorful and enthusiastic, is absent in the eyes of the autistic. Signs of colorful and enthusiastic life is absent in the eyes of the autistic. Signs of smiles and laughter are also missing. Even a hearty tickle or a boisterous game of "peek-a-boo" only occasionally elicits a giggle. His parents find all attempts to be friendly or interested are disappointingly one-sided.

As the child grows to the age of two and on, the problem becomes more perplexing. Although many individual things seem right, overall his parents know something is clearly wrong. They are naturally pleased that he can learn, yet also, quite naturally, expect more enthusiasm. He has clearly mastered too many skills to be retarded, so why doesn't he care about the things which usually hold a two-year-old's attention?

When requested to, their own child can readily learn to point to the letters or numbers in a children's book. This seems satisfying until parents see other children his age taking initiative and energetically seeking the names for all the pretty, brightly colored objects. Instead of playing with the other children in the room, the autistic child sits on the floor for hours, spinning around or simply rocking back and forth. Given a box of blocks, the other children

build things. The autistic child, given the same blocks will likely be found throwing them around the room or setting them in neat piles, one on top of another, again and again.

Some autistic children hammer. They sit on the floor, for long periods at a time, repeatedly hammering whatever is available. Other autistic ones "prefer" to bang their heads against the walls. Perhaps they enjoy seeing the phosphorus (stars) which appear as a result of the banging. A variation of this occurred in a seven-year-old girl who repeatedly bopped her chin with the back of her right hand. She not only chipped her teeth, but also developed quite prominent callouses in both impact zones. (With adequate nutrient therapy the chin bopping stopped within three weeks).

The autistic child who does develop speech uses words primarily to muse himself and rarely to communicate his needs. His speech typically has a high-pitched, sing-songy quality and content consisting of readily-learned nursery rhymes and short TV commercial jingles. These children are unable to warn of an emergency, or even to report one after the fact. One three-year-old autistic boy, having climbed up on a high shelf, found himself unable to get out of the precarious position. His crying brought mother to the rescue. Upon arrival, his mother attempted to capitalize on his predicament and finally got him to verbalize his needs. With this in mind, she repeated, "Say 'help me Mom! I can't get down!'" In a completely expressionless and rote manner, her autistic son repeated her whole sentence word for word! Another child, after hurting his hand, came to his mother saying, "Did you hurt yourself?" when he might have more properly said "I hurt myself." Stories such as these are often told about autistic children because they characteristically have great difficulty learning to actually use the words "yes" and "no" and to refer to himself as "I."

Dr. Rimland has noticed that the child with infantile

autism “is grossly impaired in a function basic to all cognition—the ability to relate new stimuli to remembered experience.” This crippling learning disability manifests itself in every area of the child’s experience. In order to learn a word, he must also “see” it. For example, if someone wanted to teach him about a red ball, they would get best results if they were to actually say “the red ball is rolling off the table,” while they proceed to roll the ball as described.

Without such persistent and formalized attention, the autistic child tends to “tune out.” Words alone have little effect and rarely get the intended meaning across. In the beginning, the autistic child may only listen to four word phrases before tuning out. Although with time he may seem to listen to short simple sentences, chances are he only catches key words and even then offers his own interpretation. In hearing, as with all learning related processes, the autistic child seems able to grasp only the very basic or concrete experiences of life. To these basics, he is unable to add the abstract concepts of experience required for sophisticated learning.

Clear examples of such learning difficulties will be apparent as the child goes through school. First grade reading will likely be normal owing to the concrete and basic nature of first grade primers. Through second and third grade, as reading progressively contains more and more abstract concepts, the child will learn to read the words, but not to understand their meaning. As he grows older, his inability to build on primary social experiences will become increasingly apparent. Socially and emotionally he may always function at a much lower level than he does intellectually.

What Causes Autism?

Not very long ago, many authorities thought

infantile autism was caused by an impaired mother-child relationship. According to their theories, the child became cold and aloof in response to his mother's failure to be physically or emotionally warm with affection. In line with this view, some professionals describe the parents of autistic children as "loners," as quite unsociable, rather withdrawn people. To our knowledge, this generalization is completely false. The parents of autistic children known to the staff at the Brain Bio Center are warm, outgoing, and responsive people. Most of them also have several normal children.

Genetic research expects to show that autism is a rare, recessively inherited trait involving biological disturbances. Along these lines, some authorities have reason to believe autism results from an imbalance in body chemistry, others are searching for some as of yet undetermined brain damage. All we know now is what we learned from the effects of various treatment programs.

Talk Therapy for the Autistic Child

To date, there is not one known instance of talk-related therapy actually curing a child of his autism. But we reserve curing for tobacco and sausages and know that many therapists have greatly helped both the child and the child's parents. Working as a friend through play therapy, for instance, a good therapist can bring an autistic child closer to his full, albeit limited, potential. As a result of such play therapy, the child may find it easier to relate to someone of authority other than his parents. One autistic child had an excellent relationship with his therapist and, as a result, was able to carry the positive experience over to camp counselors, teachers, and doctors.

Only the emotionally well-adjusted parents, who feel completely at ease with themselves and each other as well as comfortable in all other aspects, have a good

chance of successfully adjusting to life with an autistic child. All others are likely to have a difficult time accepting and learning to cope with the added tension brought on by an autistic child. The most troubled parents will be those already burdened with feelings of inadequacy and guilt and those already frustrated by or dissatisfied with the ways they have been adjusting to life's other problems. An invaluable therapist is one who, by working sympathetically, can help anxious parents sift through feelings of guilt, sorrow, and inadequacy. They know that faced with autism we are all inadequate.

Heavy Metals Burden Increased in Autism

We know that up to six months of age the blood brain barrier is inadequately developed so that heavy metals pass more freely to the brain in infants than in adults. A typical example is convulsions from lead poisoning which occurs only in infants and young children. Lead Poisoning may be diagnosed by chance when a wrist joint appears in the head X ray with a strong lead line apparent in the wrist. Any heavy metal adversely affects the brains of infants. We have found high levels of copper and lead in the blood of autistic children. We have also found high levels of lead, copper, cadmium and aluminum in the hair of autistic infants. Even the silver cup which is passed down in the family is suspect. Orange juice is sufficiently acid to dissolve silver and produce silver poisoning in the infants. Being born with a silver spoon in your mouth can perhaps lead to autism. Furthermore, low levels of these heavy metals are synergistic with each other so that 1/3 poisoning with copper and 1/3 poisoning with lead can lead to a full blown behavioral lead poisoning. We have found exactly this and many of the children have high uric acid levels indicating that the excess lead has also poisoned their kidneys. Many workers have found high lead levels in

autistic children but we have found the combination of heavy metals to be significantly poisonous. Any level of lead in children will result in learning disability.

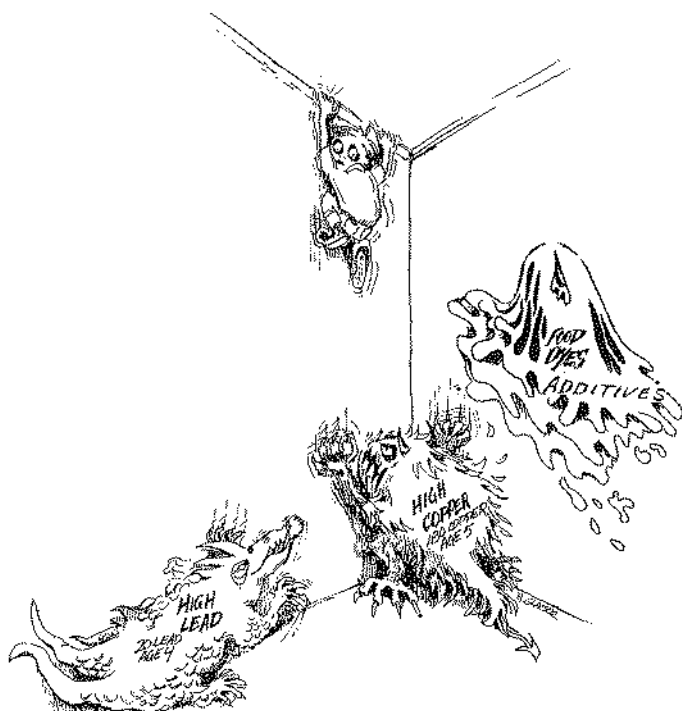


Figure 5: In juvenile autism and hyperactivity the ghosts which send "Johnny up the wall" are excess lead, aluminum, copper, cadmium, and the food additives.

Nutritional Therapy for Autism

We have had 20 years experience in the treatment of autism and have had good success with vitamin B-6, zinc, manganese and magnesium. About 10 years ago, Rimland and his associates did a double-blind trial of vitamin B-6 and magnesium in the treatment of autism. Their positive results have now been confirmed by at least six groups still using only vitamin B-6 and magnesium. The young

patients are poisoned with heavy metal (mainly copper and lead) and deficient in zinc, manganese, molybdenum, and vitamin B-6. Better results are obtained when the deficient trace elements are given with vitamin B-6 and vitamin C is used to rid the body of heavy metals.

EVIDENCE FOR THE THEORY OF OVERSTIMULATION IN SCHIZOPHRENIAS

Dr. Leonide Goldstein of Rutgers Medical School has shown by statistical analysis that the brain waves of the chronic male schizophrenics are relatively nonvariant or hyperregulated. This and other abnormalities are characteristic of a continued over-alertness or state of overstimulation and distinguish the schizophrenic from the normal subject, who has hyperregulated brain waves only under the influence of LSD, amphetamines, or other profound stimulants.

Dr. A. Arthur Sugarman, of the Carrier Clinic, and his colleagues have shown that treatment of schizophrenic patients with Thorazine or Trilafon is accompanied by a return of the brain waves to the normal range of variation and that the degree of schizophrenic behavior and overstimulation is decreased.

Dr. Goldstein has also shown that doses of one ug/kg of LSD, given to normal volunteers, causes hyperregulation of the brain waves, which then resemble the brain waves of the schizophrenic. This is because LSD is a profound, and probably specific, brain stimulant. All the hallucinogens have been shown to be brain stimulants in rabbits.

The use of antidepressants has given rise to a new toxic thought disorder as a result of the brain stimulation which accompanies overdose. Thus, a schizophrenic psychosis may develop upon treatment of a depressed patient who previously had frequent episodes of depression, but no episodes of schizophrenia. Similarly, a recent study of such stimulants in schizophrenics under blind test conditions has shown that these medications will make schizophrenics significantly worse. A study of an

antidepressant by the Princeton group showed that as the quantitative brain waves became more hyperregulated, the behavior of the schizophrenic patients worsened. The use of Iproniazid as a stimulant was in part the direct outgrowth of the euphoria and stimulation seen in tuberculosis patients treated with this, which in overdosage produced a psychotic state.

Dr. Carl Pfeiffer has shown by both behavioral and objective EEG (electroencephalogram) data that schizophrenics can tolerate a cerebral depressant, such as an oral dose of 200 mg. of pentobarbital, better than can the normal volunteer. In contrast, as judged by emotional outbursts, the schizophrenic tolerated one ug/kg of LSD less well than the normal volunteer.

In February, 1968, at a meeting in New York, three different groups of investigators essentially agreed with Dr. Goldstein that the schizophrenic is overstimulated both from behavioral and brain wave evidence.

1. Dr. P. H. Venables, of England, has presented evidence for overstimulation, which has been confirmed by Dr. Conan Kornetsky of Boston University School of Medicine.

2. Dr. J. H. Marjerrison, of Saskatchewan, also finds the brain waves of the schizophrenic to be hyperregulated, and furthermore, the degree of arousal anxiety increases when the patients have periods of hallucinations.

3. Dr. Enoch Calloway, of San Francisco, states the quantitative brain wave is a useful index of arousal anxiety which is present in the schizophrenic (even when sitting with eyes open), and he agrees that the so-called withdrawn, unresponsive chronic schizophrenic is extremely aroused. He is unresponsive only because he is pushed to the limit.

The schizophrenic patient has long been known to be an insomniac. This insomnia may precede the psychosis, extend through catatonic states, and be evident in the

hospital wards at night if the patients are not treated with tranquilizers. Preliminary brilliance or prepsychotic stimulation may be one of the first signs of a psychotic break. The psychosis of prolonged wakefulness may also provide a similar example of the psychogenic effect of forced overstimulation which is physically induced. While no quantitative data are available, the brain waves of prolonged wakefulness are, by inspection, similar to that of the schizophrenic. Thus, these various observations would indicate that the schizophrenic is, indeed, in a chronic overstimulated state.

This overstimulation does not respond to the usual doses of barbiturates, alcohol, or other antianxiety drugs. The patient can be sedated by means of adequate dosage of the truly antischizophrenic drugs, such as Thorazine, Reserpine or Haldol. These drugs oversedate the normal individual. We suggest that excess brain copper is involved in the overstimulation.

EVERYDAY DRUGS AND THE SCHIZOPHRENIC

The overstimulation of schizophrenics can often be exacerbated when they knowingly or unknowingly take brain stimulants. Some of the common stimulants which may worsen the degree of schizophrenia are coffee or tea (because of caffeine), antihistamines (as in cold capsules), cocaine (as in "crack"), and amphetamines (Bennie pills or Methedrine, as in weight-reducing drugs). All stimulants should be avoided, and the patient should take decaf rather than coffee, milk rather than tea, and orange juice rather than cola or similar beverages.

Some of the prescription drugs which may markedly increase brain stimulation are antidepressant drugs, the MAO inhibitors, and some local anesthetics. These must be avoided except when the use is closely supervised by the physician.

Alcohol, a central nervous system depressant, is associated with four of the identified types of schizophrenia. Depending on the underlying biochemical abnormality, alcohol may be an easily attainable avenue for self-medication or an agent which exacerbates the symptomatology. The high histamine patient is a compulsive, chronically depressed individual. Both of these factors may lead to a tendency for alcohol abuse, and histadelics are commonly chronic, hard-core alcoholics. Additionally, alcohol has a slight histamine releasing effect and thus, may result in lower body histamine and some mediating effect. The low histamine or histapenic patient occasionally suffers from periods of overstimulation and therefore, may turn to the depressant, alcohol, for relief. Such episodes are usually transient, resulting in binge drinking. The cerebral allergy patient is often allergic to the malt and yeast in beer, leading to a pleasant stimulation

immediately following ingestion. However, a severe let-down or withdrawal follows several hours later. The hypoglycemic individual may also succumb to alcohol, as it may provide relief from the symptoms of fatigue, irritability, and depression. Alcohol will depress the hypothalamus in the brain, decreasing the body's demand for glucose. Additionally, alcohol may substitute for glucose as a cellular fuel.

Drugs such as simple aspirin, Motrin, or Tylenol are sedative to the brain, and these may be used freely for pains and mild sedation. The compounded aspirins, such as Anacin and APC tablets, contain caffeine and should be avoided. The amino acid phenylalanine has some pain-killing effect and can be used as well. Please note that due to the ubiquitous nature of many of the drugs discussed, use or abuse cannot be considered diagnostic. However, they may reinforce a diagnosis already in place.

HOPE IN THE SCHIZOPHRENIAS

SCHIZOPHRENICS HAVE MADE AND CAN MAKE NOTABLE ACHIEVEMENTS

Alexander Pope's observation that "great wits near madness lie" was not original. Men have speculated since the earliest days on the dividing line between genius and madness. The results of these speculations have usually been confused and confusing. Some claim that genius, itself, implies the presence of schizophrenia, and others with equal dogmatism allege that all those who have any degree of schizophrenia become more stupid. The truth must be more complicated and dependent on the individual personality and profession. In all probability, the artist can get by with more schizophrenia than the minister or teacher.

Many medical men, over the centuries, have commented upon the brilliance which is not infrequently, but by no means always, associated with early schizophrenia. Indeed, at one time it was popularly supposed that the overstrain of this brilliance had "induced brain fever" resulting in collapse of the mind. The late Dr. Alan Gregg pointed out that the relatives were wrong when they said, "John overworked and had a nervous breakdown." One of the first signs of the disease is the ability to work day and night without adequate sleep.

Some years ago, Dr. Joan Fitzherbert in England found, while treating children at a child guidance clinic, that in the period immediately preceding a psychotic "break" their intelligence quotient (I.Q.) when measurable was high; but as they became well, it returned to a lower level some 20 or more points below that shown in the period of prepsychotic brilliance. It seems not unreasonable to infer that here for a period, at any rate, the schizophrenia was enhancing the children's abilities. How might this occur? Modern biochemical theory

suggests that these young schizophrenics add methyl groups to the brain amines, and this results in a continued druglike stimulation of the brain, and of course insomnia.



Figure 6: John overworked and had a nervous breakdown. The first sign of a schizophrenic process may be the ability to turn night into day and to go without sleep.

The great seventeenth-century neuroanatomist Thomas Willis described the illness which we now call schizophrenia in these terms: "For these kinds of brains, like distorted looking glasses, do not rightly collect images of things nor truly display them to the rational soul." Since we now know that changes in perception of the world are an essential aspect of the schizophrenias, then it might seem probable that from time to time such

changes would enhance certain abilities and that in some circumstances this enhancement, when coupled with an already intelligent mind, would result in something remarkably different. James Joyce, in the writing of his Ulysses, may possibly be an example of disperceptual brilliance. Joyce was probably histadelic.

From about the years 1700 to 1900, our view of the universe was governed and largely limited by the formulations of the great Sir Isaac Newton, who combined the most extraordinary intuitiveness in physics with superb qualities as a mathematical technician. Sir Isaac was a remarkable man; he did much of his greatest work alone; he spent two years during the Black Death or plague in rather mysterious seclusion on the family farm in Woolstenhough in Norfolkshire doing, it seems, nothing. He had no books; there were no journals at the time; he did not correspond with anyone. At the age of 30 he became increasingly unwilling to undertake "natural philosophy," which we now call science, and his energies in subsequent years were devoted to an occult philosophy, Biblical interpretations, and governing of the mint to influence the financial policy of the country. Newton was abnormally suspicious, cantankerous, and in many ways very difficult. In his mid-fifties he had a clear-cut psychotic break that lasted for two years, possibly brought on by heavy metal poisoning. The diarist Samuel Pepys, a fellow member of the Royal Society, notes with satisfaction that "Sir Isaac is improving and no longer believes that we Fellows of the Royal Society wish to introduce royal duchesses to his bed." Sir Isaac's delusion seems to have been that plans were afoot to breed a race of supermen for whom he would be the involuntary father. He objected to this on the grounds that he had not been consulted. However, Sir Isaac did recover and lived to a very ripe old age. He also was probably histadelic.

Much more recently we have the great philosopher,

Ludwig Josef Johann Wittgenstein. He was a student of engineering and a philosopher and played a large part in the development of the branch of philosophy known as logical positivism, although he himself later abandoned it. Wittgenstein published very little indeed, and most of his writings have been collected by his students. Although his philosophical statements are like strange and obscure riddles, there is no question that his contribution to philosophical thinking is brilliant. His behavior, however, was very erratic. He disappeared for some years and was finally found working as a schoolteacher in the Austrian Alps. He was then coaxed back to Cambridge. During World War II he became an orderly at Guys Hospital in London. He had a curious habit of sitting for hours in the front row of a theater, looking at any film that happened to be running; he claimed that this would help to clear his thinking! Yet another histadelic!

There is no doubt at all that Nijinsky, probably the greatest dancer who ever lived, was a victim of schizophrenia. We have a long account of his illness by his wife, Romela. He eventually died in a hospital suffering from schizophrenia. Nijinsky's dancing, it is said, had an eerie quality. He, himself, explained this by saying that he got outside himself, watched himself dancing, and so controlled his movements. This was thought by many to be a figurative statement. The evidence is, however, that that was how Nijinsky experienced it. Again, we would suspect histadelia.

Franz Kafka, the great Austrian author, was, according to Rudolf Altschule, undoubtedly schizophrenic. He also suffered from tuberculosis. His novels foreshadowed, in an extraordinary and horrible preview, the Nazi regime and ultimately an authoritarian world. There are also vivid and horrible accounts of some of his schizophrenic experiences. Perhaps his most appalling story is "Metamorphosis," in which the writer turns into some

kind of bug. This is described with such complete conviction that one feels the author is giving an account of something that happened to him. This is by no means impossible, for many schizophrenic people have extraordinary changes in the perceptions of their body which cause them the greatest distress. There is an interesting story from Janoush, Kafka's friend, about walking down a street when a little dog ran out of a doorway. Fanz shied away and pushed Janoush back as if a lion were coming at them. Janoush said, "Don't worry, Franz, it's only a little dog." Kafka replied, "Some people would say it was only a little dog." Many schizophrenic people suffer from the experience that people and objects coming toward them get larger instead of seeming to come closer. This is called the breakdown of size constancy, and this little story of Janoush's illustrates it perfectly. As the dog moved closer towards him, it appeared to get much larger and so less like a dog and more like a lion! No wonder Kafka shied away. Yet another histadelic.

William Blake is generally held by critics to be among those few who made the apex of English poetry. He is also regarded as one of the finest artists of all times. To his contemporaries, however, he was regarded as completely mad. He spoke of God the Father and talked to Him personally; he claimed he had seen his brother's ghost mounting to Heaven. He described one of his paintings as the ghost of a flea, which he had seen. He catalogued all of his paintings and announced that he was among the greatest of poets and painters. This was considered patently absurd at the time and merely the raving of a madman. That history has vindicated his self-estimate does not alter the fact that he may well have suffered from schizophrenia, probably histadelia.

There are many others who have had or been suspected of having this grave illness. Some have made great contributions for good, while others have been less

beneficial. There are, at least, strong suggestions that Adolf Hitler's extraordinary visions of "The Thousand Year Reich" may have been derived from a schizophrenic illness as a young man, and this may have been the basis of his paranoia, which caused his rabid and appallingly destructive anti-Semitism. Hitler probably had pyroluria and copper intoxication.

Van Gogh's pictures and life strongly suggest that he had schizophrenia, although there is considerable disagreement and discussion about this. The breakdown of the perceptual field is extremely suggestive of histadelia.

In Arthur Miller's After the Fall there is an upsetting and beautiful account of Marilyn Monroe's steadily developing illness. It is, of course, known that her mother was ill for many years in a mental hospital. Although Miller never mentions schizophrenia, it seems at least possible that Marilyn's elusive charm and innocence sprang from those misperceptions which we know do occur with schizophrenia. She had no cavities in her teeth, which is a sign of histadelia.

These examples of great schizophrenics should not be interpreted to mean, of course, that all those with schizophrenia are necessarily geniuses or artists. They do suggest that talented people have been able to make use of their illness in a remarkable, and sometimes highly constructive, way. Thus, the illness should not be seen as being purely negative. It should be recognized for what it is, part of the human condition, and therefore, worthy of our sympathetic understanding, study, and encouragement.

Most of the productive people in society are histadelic, so we find that these great people are probably high in histamine.

REQUISITE FAITHS FOR RECOVERY

Patients suffering from any type of chronic illness have probably already learned about the need for a generous supply of faith. They know the very large role faith plays in all recovery.

Patients must have faith in themselves and in their own ability to combat an offending disease. Such crucial faith need not come from an evaluation of present assets. Honor and respect for the quality of life and the power to heal can stem from a focus on the sanctity of life itself, from a spiritual foundation, or simply from an awareness of inner essence.

Because the therapist not only selects, but also directs treatment, much of the future lies in his or her hands. Faith in the therapist will lend support as they encourage and advise. Who else but the therapist searches the literature each night hoping to discover a new, improved treatment approach which is uniquely designed for the individual problems of each particular patient? Remember to have faith in the therapist, but do not allow blind faith. Be wary of catch-all diagnoses. A multiple sclerosis label is often placed on a zinc and vitamin B-6 deficiency. Alzheimer's disease labels frequently disguise simple aluminum poisoning and B-12 deficiency. Because these distinctions suggest different therapies, an accurate diagnosis can be the key to a speedy recovery.

Have faith in the nutrients and medications given by the therapist. Do not lose confidence when weeks or months of therapy bring only minimal improvements or annoying side effects. Instead, focus on the fact that it can take a long time to correct the serious biochemical and physiological imbalances which lie at the base of chronic disorders. Remember and be assured that one does not live this year with last year's body! All body tissues permeated

THE SCHIZOPHRENIAS: OURS TO CONQUER

by the circulation of blood and lymph can and will be influenced by better nutrition. Bones, muscles, and even the mind are constantly being reworked and made more efficient as proper nutrients and energy are supplied.

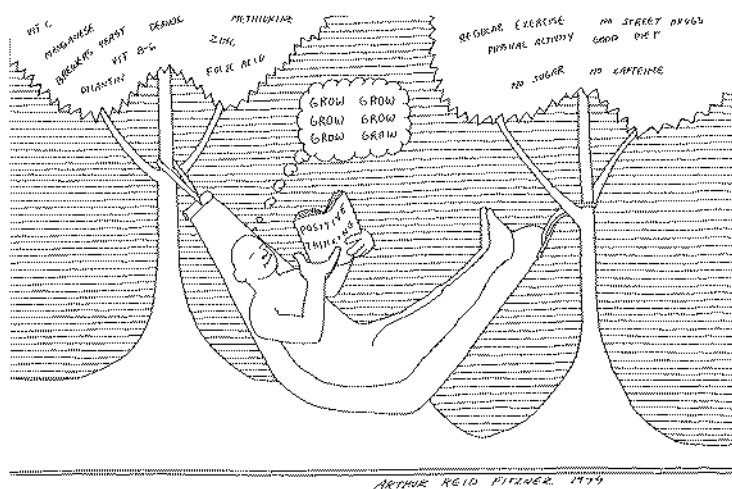


Figure 7: The beneficial effect of nutrients is slow, so that weeks and months of supplements may be needed to attain maximal benefits.

Medical and social science constantly progresses. By the time books are printed, they are often well behind the therapeutic knowledge published in journals. Faith in these journals and the doctors and scientists who read and write the articles, has supported new knowledge, and as a result, many disorders that were unmanageable ten years ago are now easily cured. As long as the future remains so thickly veiled, no man can predict what tomorrow will bring. Until such predictions are possible, faith in the continued progress of medical and social science will stand as a powerful personal resource. Believe in the future and the therapeutic rewards that come from

research and study.

If presently, a patient's symptoms cannot be allayed, remind them of the many people before who have gotten well while combining faith in themselves with faith in the therapist, the treatment process, and the constant progress of scientific research.

BEHAVIORAL VS. BIOLOGICAL DIAGNOSIS OF THE SCHIZOPHRENIAS

Degrees of Scientific Naivete!

1. Study fever as a single disease
2. Study anemia as a single disease
3. Study arthritis as a single disease
4. Study hypertension as a single disease
5. Report on the schizophrenias as a single disease as diagnosed behaviorally

In 1959, Kurt Schneider, a German psychiatrist, provided the first set of detailed symptoms on which to make a cross-sectional behavioral diagnosis of schizophrenia. He described 11 "first rank symptoms" (FRS), any one of which was sufficient for diagnosing a patient as schizophrenic in the absence of neurological disorders, such as retardation or brain disease. The first three deal with auditory hallucinations: voices repeat or anticipate the patient's private thoughts; they discuss or argue about the patient; or they keep up a running commentary on the patient's actions. Three more behavioral symptoms involve bizarre thinking: the belief that one's thoughts are removed by some outside force; that one's thoughts are magically broadcast for others to hear; and that thoughts are forcibly inserted into the patient's mind. Another three symptoms relate to the patient's experience of feelings, acts, and impulses being under the control of some external force, as if the patient were a robot or hypnotized. The patient also feels a passive reception of various sensations—heat, touch, or movement—imposed by outside forces. Finally, the patient's perceptions are deluded—what we would perceive as commonplace occurrences, the schizophrenic interprets as having a very special or profound personal significance.

Schneider's FRS are still regarded by many clinicians as the definitive behavioral criteria for making a diagnosis of schizophrenia. They are commonly used in Europe, and had a significant impact on British researchers and clinicians in particular. But studies show that FRS occur in about 50 percent of clinically diagnosed schizophrenics. That is, half of the patients labeled schizophrenic do not show any FRS. Also, FRS occur in other psychiatric disorders, especially mania, so they are not exclusive to schizophrenia.

Other cross-sectional diagnostic behavioral systems are more or less based on Schneider's FRS, but they include other symptoms as well. For example, the New Haven Schizophrenia Index (NHSI), developed in 1972 by B. M. Astrachan and colleagues, Yale University, New Haven, Connecticut, lists more than 20 symptoms, including visual hallucinations, confusion, catatonia, suspiciousness, and other paranoid signs. The flexible system, designed in 1973 by W. T. Carpenter and J. S. Strauss, National Institute of Mental Health (NIMH), Bethesda, Maryland, itemizes 12 relevant behavioral symptoms of schizophrenia, including poor rapport and insight, incoherent speech, and absence of early waking, elation, and depression. The presence of either five or six symptoms are necessary for a diagnosis of schizophrenia with the flexible system. The CATEGO system, a computer program used to process data from the Present State Examination, was developed by J. K. Wing and colleagues, Medical Research Council, London, in 1974. It allows for six separate diagnoses: definite or uncertain schizophrenia; definite or uncertain paranoia; and definite or uncertain "other" psychoses, such as simple and catatonic schizophrenia.

At the same time that these cross-sectional behavioral diagnostic systems were evolving, other researchers and clinicians developed a longitudinal approach still

using behavioral systems. In addition to considering present symptoms, they examined the patient's past history—whether or not other relatives were schizophrenic, the patient's work or school performance before the onset of the disease, the age at onset and duration of the illness.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-III) is the most current example of a longitudinal diagnostic system. DSM-III is a standard reference work in the U.S., and was published in 1980 by the American Psychiatric Association. It evolved from two earlier longitudinal diagnostic criteria: the Feighner criteria, defined in 1972 by J. P. Feighner and colleagues, Washington University, St. Louis, Missouri; and the research diagnostic criteria (RDC), developed in 1975 by R. L. Spitzer and colleagues, NIMH.

DSM-III draws a distinction between three classes of symptoms. "Prodromal" symptoms may develop before the onset of schizophrenia. They indicate that the individual's previous level of functioning is deteriorating—social withdrawal, personality changes, eccentric behavior, neglected hygiene and grooming, and diminished performance at work or school are listed as prodromal symptoms in DSM-III.

Active behavioral symptoms indicate the psychotic phase of schizophrenia. Again, they are derived from Schneider's FRS—delusions, hallucinations, and thought disorders. Any one of these behavioral symptoms is necessary for a diagnosis, but thought disorders alone are not sufficient. Thought disorders—incoherence, loose associations, and poverty of speech, for example—must occur with delusions or hallucinations, catatonic behavior, or flat or inappropriate emotions for a DSM-III diagnosis of schizophrenia.

"Residual" symptoms may persist after the active phase of schizophrenia. They are similar to the prodromal symptoms, but emotional flattening and impaired role

functioning are more common in the residual phase.

In addition to these symptoms, DSM-III requires several longitudinal factors: onset before age 45 and continuous illness for at least six months, for example. Also, a diagnosis of schizophrenia is not allowed if the patient has a history of manic or depressive disorders, or brain disease and retardation.

In summary, the DSM-III concept of "schizophrenia" emphasizes chronicity, hallucinations, paranoia, delusions of persecution, thought disorder, illogical thinking and other disturbances of feeling and behavior. To establish "schizophrenia" these symptoms must be continuous for six months and result in deterioration in functioning in work, social relations, and personal care at ages below 45 years.

We have found the many behavioral diagnostic systems to be ineffective in the classification of the schizophrenias, as they tend to view schizophrenia as a single entity rather than a collection of biological abnormalities manifesting similar neurologic impairments. A biological approach is logically more efficacious utilizing the underlying abnormality as a classification criteria rather than symptomatology and thus, implying a directed treatment program. In fact, a biological system is the manner in which medicine views all other ailments, and yet it has been curiously neglected with regard to schizophrenia. Certainly, we do not proclaim that all schizophrenics can be accommodated by our system at present. However, as research discovers new etiologies, new categories will be added.

The DSM-III definition behaviorally describes what can be chemically described as high copper, low histamine biotype of schizophrenia which we separated from the rest of the schizophrenias in 1971. Other definite biotypes are: histadelia (high histamine), pyroluria (zinc and B-6 deficiency), cerebral allergy and wheat gluten

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enteropathy. With adequate biochemical tests, the exact biochemical imbalances needing correction are evident. When these imbalances are corrected, the patient starts a slow but steady road to biochemical and behavioral recovery.

IMPORTANCE OF VARIOUS BIOCHEMICAL IMBALANCES

The body imbalances can be one of many types, ranging from simple unitary deficiencies of a nutrient to complex over or under production of a key metabolite. The exact measurement of the level of trace elements, vitamins, nutrients or metabolites provide the objective keys to disease processes both in the body and the brain.

- I. Deficiency of a single hormone
 Examples: a) Lack of thyroid produces Myxedematus Madness
 b) Lack of insulin produces Diabetic Coma
- II. Deficiency of a single vitamin
 a) Lack of niacin (B-3) produces Pelagrin psychosis
 b) Lack of vitamin B-12 produces Anemia and psychotic behavior
 c) Lack of pyridoxine (B-6) produces convulsions and psychosis
- III. Intolerance to an amino acid
 a) Phenylketonuria is caused by the inability of the body to use phenylalanine; hence, this neurological disease responds to diets devoid of phenylalanine
- IV. Need for optimal supplements for amino acids
 a) L-lysine controls herpes infections
 b) L-tryptophane allows normal sleep
 c) Methionine lowers elevated blood histamine and decreases depression
- V. Need for optimal supplements of vitamins
 a) Vitamin C helps eliminate heavy

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metals which produce learning disabilities

b) Pyridoxine (B-6) produces normal dream recall

c) Niacin lowers both triglycerides and cholesterol

d) Folic acid/B-12 raise blood histamine

e) Vitamin E prevents polycystic disease and cancer

f) Riboflavin prevents senile cataracts

g) Inositol produces natural sleep

h) Biotin prevents convulsions

i) Vitamin A prevents acne

j) Deanol increases acetylcholine and alertness

k) GTF (the glucose tolerance factor) helps insulin to burn sugar

These examples are as simple to understand as "No gas, No go" since there is a correlation between two single factors. We know, however, that trace elements (TE) are involved in enzyme activity, and each TE may be present in one to 100 enzymes. At present, cobalt is only known

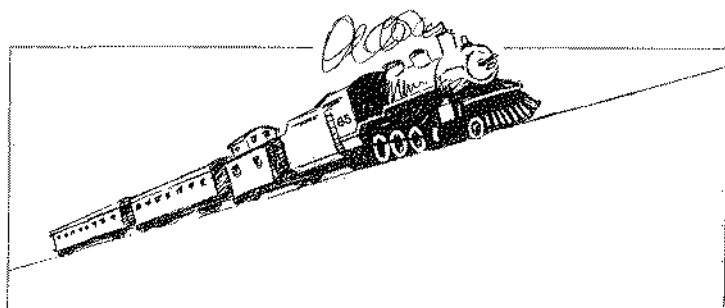


Figure 8: Nutrients are like the "little engine that could." Nutrients know where to go and how to do it.

to be needed in vitamin B-12, a vitamin most effectively made by bacteria. Thus, we can list above No B-12 = anemia and psychotic behavior, again a cause and effect relationship as simple as "No work, No pay!" But nutrients do pay off handsomely in that they know exactly where to go in the body and what to do! Drugs do not!

VI. Selenium is a trace element needed by one enzyme. Selenium is an essential element in glutathione peroxidase which promotes the destruction of tissue hydrogen peroxide. This is its only proven enzyme function in the human body. However, if deficiency of selenium is spread among the organs of the body, this small but vital role is sufficient to produce fatal heart disease, fatal cancer, and many less lethal lesions of tissues.

VII. Molybdenum is needed by four known enzymes in the body. These deal with the use of the amino acid, methionine, the detoxification of sulfite, and the formation of uric acid. If intravenous amino acids are given to the molybdenum-deficient patient, coma results.

VIII. Manganese is known to be needed by four important enzymes in the body. These deal with the formation of red blood cells, the making of an important neurotransmitter, cyclic AMP, and the formation of cartilage. Manganese prevents auto-immune disease, allergy and psychiatric depression. Manganese occurs in tropical fruits and is difficult to absorb orally.

IX. Copper deficiency (while almost unknown in the human adult) can effect eight enzymes in the body. Amazingly, the only two systems that are practically affected by deficiency are the formation of red cells and the conducting system of the heart.

X. Zinc is involved in twenty to one-hundred enzymes in the body and a deficiency of zinc produces a myriad of diseases from acne and total loss of hair to one of the schizophrenias. When combined with pyridoxine (B-6), zinc is a sure cure for one-third of the patients presently

labeled schizophrenic (pyroluria). Since zinc is involved in such a multitude of enzymes, we count 65 diseases wherein zinc deficiency plays a prominent role.

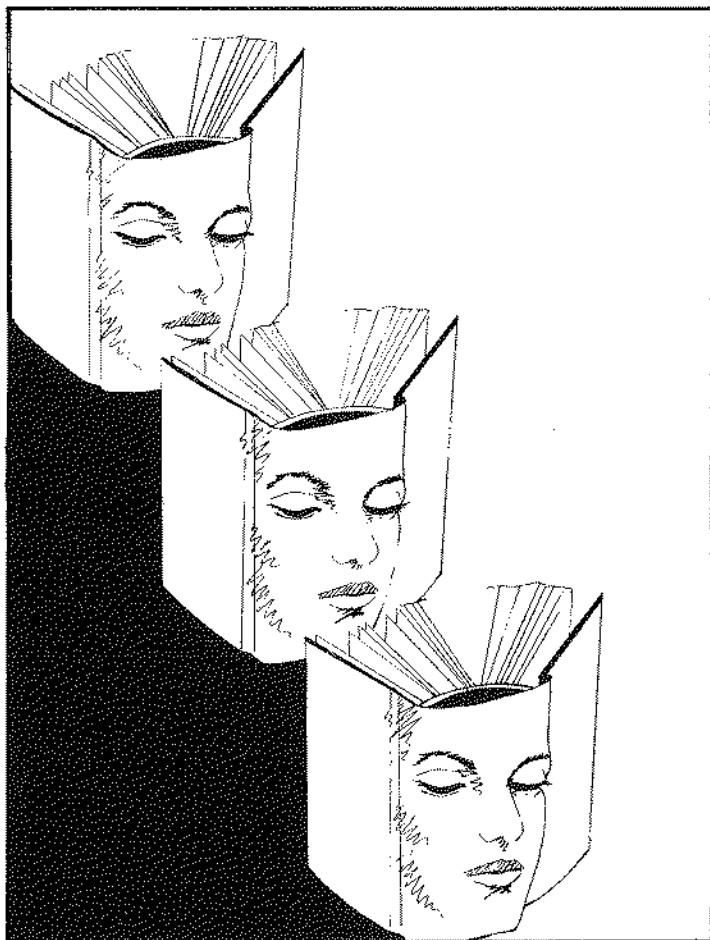


Figure 9: The three faces of Eve dissolve into the many facets of probable biochemical imbalances.

Thus, a biochemical imbalance can be a simple vitamin, hormone, amino acid, or trace element deficiency. A biochemical deficiency can involve a vitamin

and a trace element such as selenium - vitamin E, zinc - pyridoxine, or iron - folic acid. With malnutrition by jiffy foods and soft drinks, the human biochemical imbalances can be excessive with the patient truly a physical and mental basket case. Often such imbalances can only be diagnosed by a therapeutic trial of the needed nutrients. From the standpoint of disease, TE deficiency is the most basic of tissue needs and provides the most dramatic cure when the deficiency is relieved.

HEAVY METAL POISONING— HISTAPENIA—HYPERCUPREMIC PATIENTS

Man, the industrial genius, is continually redistributing and modifying the resources of this earth. Areas are being contaminated as the undesirable products of technology are abandoned, while other areas are stripped of top soil and trees and lie weathering so that the water pollution process continues. Copper is perhaps the most common of the heavy metal intoxicants with approximately double the tonnage mined compared to the closest competitors, lead and chromium (Shroeder, 1974).

Copper Deficiency is Rare

The National Academy of Sciences (1977) reports clinically apparent copper deficiency is extremely rare and difficult to achieve by dietary means. At the Princeton Brain Bio Center, where serum heavy metal leads have been routinely assayed since 1965, only three cases of low blood copper have been documented from over 25,000 patients treated. These deficiencies were precipitated by excessive zinc ingestion. Sloane (1985) describes three additional clinical situations where low blood copper may be expected, in premature infants, in patients receiving total parental nutrition, and in severely malnourished children. On the other hand, 51% of all female patients and 43% of all male patients at the Brain Bio Center have exhibited toxic levels of copper, accenting the pervasiveness of this oral poison in modern society.

Although commonly found in excess as a poison, copper is required by the body in small amounts as it is an essential component of several basic oxidative enzymes. Absorbed in the small intestine, copper binds to albumin,

transcuprein, and low molecular weight ligands which transport the heavy metal to the liver. Liver mechanisms incorporate copper in ceruloplasmin, a serum protein. Eighty percent or more of all serum copper is in the form of ceruloplasmin (Weiss and Linder, 1985) and as such is responsible for one of the most important enzymatic activities of copper, which involves the mobilization of iron and the regulation of hemoglobin. Other cupric enzymes include cytochrome C, superoxide dismutase, lysyl oxidase, tyrosinase, histaminase, and dopamine hydroxylase. Copper also has a role in blood clotting, namely as a constituent of factor V (Nutr. Rev., 43, 1985).

TABLE 1
Copper in Pellagra

	N	Serum Copper
Controls	6	92 mcg%
Pellagrins	18	167 mcg%
Treated Pellagrins	18	104 mcg%

Pellagrin Psychosis = Paranoia and Hallucinations

Data of K.A.V.R. Krishnamachari

Am. J. Clin. Nutr. 27: 108, 1974.

Copper Toxicity is Common!

The toxic effects of copper stem from its properties as a nervous stimulant. Thus, profound effects may be visible not only within the central nervous system but amongst the majority of the organ systems. The stimulative effects of copper were first described by Ussing in 1949 as he measured an increased electrical potential of frog skins placed in a copper-containing nutrient solution. Tonnie and Ferreira (1970) verified Ussing's findings with copper concentrations as low as 10 μ M and postulated the increased electrical potential may be due to unrestricted sodium movement across the membrane.

Pfeiffer and Goldstein (1984) using EEG (brain waves) demonstrated that 5 mgm of copper has in man the same central nervous system stimulative effects as 5 mgm of Dexedrine, a common amphetamine.

Copper Destroys Histamine

Pfeiffer and his colleagues (1975) were the first to detail a definite role of copper in mental illness as they defined histapenia, a schizophrenia-like disorder. Histamine, a neurotransmitter and chemical modulator of the body, is regulated by the copper-containing proteins, histaminase and ceruloplasmin. Abnormally high levels of free copper increase the activity of these two enzymes resulting in excessive degradation of histamine. This histamine deficit is responsible for some of the specific psychiatric deviations seen in the schizophrenias.

The neurotoxicity of copper may also arise from an energy deficiency resulting from the inhibition of the body's basic metabolic pathway, glycolysis. Lai and Blass (1984) have found high copper concentrations in rat brains inhibit hexokinase, pyruvate kinase and lactate dehydrogenase, essential enzymes in the production of energy. Not only may neuronal pathways be disrupted due to a neurotransmitter imbalance, but the neurons may actually be starved for energy.

TABLE 2
Inverse Relationship Between Copper and Vitamin C

Days	Body Wt. (Gms)	Adrenal C	Plasma Cu++ (mcg/ml)
0	416	145	0.65
8	372	25	0.68
12	371	7	1.37
20	258	Tr.	2.17

Hitier, Y., Int. J. Vit. & Nutr. Res., 46: p. 48, 1976.

Copper and the Brain

Behavioral manifestations of copper intoxication include disperceptions, thought disorders, hallucinations, paranoia, insomnia, violent behavior, withdrawal from reality, catatonic symptoms, and loss of drive. Depression and an obsession with suicide are significant clues that high copper and low histamine are responsible for the manifest psychiatric difficulties.

Physiological imbalances are visible outside the confines of the nervous system and provide additional diagnostic clues to histapenia. The body's histamine levels are severely depressed due to excessive degradation as previously described. As a chemical modulator, histamine is responsible peripherally for anaphylaxis, the body's natural response to an invading foreign particle. Histamine is responsible for the sneezing, runny nose, and swelling commonly seen in allergies, asthma and respiratory or peripheral infections. Histamine is carried in cellular elements, mainly the blood components basophils and platelets and the tissue components, the mast cells. Consequently, fewer basophils and mast cells are characteristic of histapenics. Allergies such as hay fever, asthma, or hives are rare; head colds are usually asymptomatic. Additionally, the non-secreting nature of such individuals leads to predisposition to poor dental health and difficulty in achieving sexual orgasm.

At the Brain Bio Center over a period of more than 20 years we have found the blood histamine of the histapenic-high copper patient to average (male) 20.2 and (female) 27.6 mg/ml, where normal is 42 and 46 mg/ml for male and females. We find 1% of these patients to have no detectable blood histamine and no basophils either in the blood cells which contain most of the histamine. The serum copper is usually above 120 mcg%. We have reported that these histapenic patients have only 6 mg/ml

of folic acid per ml of blood. This is one-half the level found in normals and pyrolurics. Their B-12 blood levels were normal at 649 pg/ml. Vitamin C in a 2.0 gram oral dose in schizophrenics will raise the urinary excretion of copper from 1.7 mcg/6 hrs. to 7.4 mcg/6 hrs. Similarly, niacin (vitamin B-3) will increase the urinary excretion to 8.3 mcg/6 hrs (Pfeiffer and Bachi, 1975). The combination as used in the treatment of these patients would be even greater.

Blood Type A: Sequesters Copper

Additionally, it has been revealed that certain blood types have an inherent propensity for accumulating copper in the body. Interest began in 1973 when Wiener et al. demonstrated significant differences among the three blood types of sheep with respect to copper levels in whole blood and plasma. Assuming that this suggested a biochemical difference between blood types, Bonnet, Pfeiffer, and Aston (1980) undertook an investigative study of humans. Chronically hospitalized schizophrenic patients were revealed to have an unusually high incidence of type A blood. It appeared that these individuals have great difficulty excreting copper and require extensive therapy to ameliorate their psychosis.

Drinking Water Has Excess Copper

A large portion of the copper we ingest is dissolved in our drinking water. Plumbing systems are frequently constructed with copper piping, rather than the older galvanized steel. Unusually acid or soft water dissolves the soft metal in some cases forming holes in the piping in as little as three years. Hard water, on the other hand, lines the pipe as its minerals precipitate out, protecting against the copper menace. Sharrett (1979) documented a positive

correlation (0.85) between whole blood copper and drinking water concentrations in five U.S. towns. In a subsequent study, Sharrett (1982) estimated the daily consumption of copper from drinking water in Seattle to be 2.2 mg, 0.2 mg greater than the U.S.R.D.A. The following states were reported to have soft water by the U.S. Geological Service (1974) and should be considered risk areas: Maine, Vermont, New Hampshire, Massachusetts, Rhode Island, Connecticut, New York, Maryland, Delaware, Virginia, North Carolina, South Carolina, Georgia, Mississippi, Arkansas, Oregon, and Washington.

Additionally, many sufferers add to their woes by taking multivitamin supplements, many of which contain 2-3 mg of copper. Such multivitamins should be avoided. Smoking also seems to contribute to the copper intake in modern society. The U.S. Public Health Service (1979) reports the average cigarette contains 0.19 ug of copper and this is accumulated in the body as the smoke is inhaled. Copper is just another component of the well publicized health hazards associated with smoking. Copper intoxication can occur at any age, so this may be the cause of late onset schizophrenia (paraphrenia).

TABLE 3
Elevated Copper in the Schizophrenic Population.
Note the lack of effect of drug treatment upon the
copper indices.

	No.	Copper	Cerul.
Normal controls	(98)	121	0.32
Untreated schiz.	(27)	202	0.36
Drug treated schiz.	(23)	208	0.26

cerul. = ceruloplasmin

Data of T. D. Chugh

Ind. J. Med. Research 61, 1147 (1973)

How to Remove Excess Copper

Therapy for copper intoxication involves the restriction of copper intake combined with copper antagonists which inhibit intestinal absorption of copper while promoting its excretion in the bile. Zinc, manganese and molybdenum along with vitamin C have been shown to decrease the body's copper burden. Additionally, selenium and vitamin E protect against some of the toxic peroxidative actions of elevated copper. In severe cases a pharmacological copper chelator, penicillamine (trade name Cuprimine), may be required to quickly reduce circulating copper levels; however, adverse side effects warrant the use of penicillamine only in extreme instances. Penicillamine should always be accompanied by zinc, manganese, and molybdenum since these useful metals are also removed by the chelating effect of penicillamine.

TABLE 4
The Time Course for the Relief of Symptoms
in Histapenia

Drippy palms		
Apthous ulcers		
Mind racing		
Insomnia		
Hypomania		
Hallucinations		
Obesity		
Paranoia		
One Week	One Month	One Year

A suggested treatment regime for histapenia is as follows:

Nutritional Treatment of Histapenia & Hypercupremia

1. Niacin, niacinamide 100 - 3000 mg

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2. Folic acid 1 to 2 mg
3. Zinc and manganese as gluconates
4. High protein diet
5. Lithium carbonate 300 to 900 mg
6. Prolixin 1 to 5 mg at bedtime
7. L-tryptophane, 500 mgm x 2 for sleep
8. B-12 injection weekly
9. 2000 mgm vitamin C daily
10. Molybdenum 500 mcg AM & PM

PYROLURIA—ZINC AND B-6 DEFICIENCIES

Thirty to forty percent of the schizophrenic patients fall within the normal range of blood histamine levels and, thus, cannot be diagnosed and treated according to this factor. Many of these patients can be identified by the excretion of urinary pyrroles (mauve factor), and this condition has been termed pyroluria. The first correlation between urinary pyrroles and episodes was noted in 1958 by Payza. Studying experimental LSD psychosis, Payza noted the occurrence of this new factor in the urine of some subjects. Irvine (1961) and Humphrey Osmond (1963) later found that abnormal pyrroles are excreted in greater frequency by schizophrenics. The mauve factor was structurally identified by Irvine in 1969 and confirmed by Sohler in 1970 to be 2,4 dimethyl-3-ethylpyrrole or kryptopyrrole—an aberrant product of hemoglobin synthesis. As this substance circulates in the body, it forms a stable Schiff's base with pyridoxal (the aldehyde form of pyridoxine or vitamin B-6) and subsequently complexes with zinc, stripping the body of these two essential substances as it is excreted. The psychopathology thus results directly from this double deficiency of zinc and vitamin B-6 and not merely by the presence of excessive pyrroles.

The presence of kryptopyrroles in the urine is not an absolute sign of schizophrenia or psychiatric disturbances, but simply signifies a susceptibility for such difficulties. O'Reilly and Hughes (1965) documented the presence of the mauve factor in 11% of normals, 24% of disturbed children, 42% of psychiatric patients, and 52% of schizophrenic patients. It is also simplistic to suggest that pyroluria is the only precipitating factor in schizophrenics excreting pyrroles in their urine. Frequently, clinical disease is complicated by a multitude of

biochemical imbalances. For example, the patient may be both pyroluric and histadelic. Although pyroluria can be easily distinguished through a urine test, the other biochemical factors must be probed to identify all biochemical imbalances to insure recovery.

As with many of the schizophrenias, pyroluria usually surfaces between the ages of 15 and 20 when numerous teenage stresses impact upon the turmoil of adolescence. Pressures of new jobs, living away at college, initial sexual experiences, and leaving home may combine with the biochemical imbalance inherent in the pyroluric and result in a psychotic break. Additionally, it is common, as with histadelia, for this disorder to be familial; thus, a family history may provide important clues.

TABLE 5
Kryptopyrroles in the Population

"Normals"	11%
Disturbed Children	24%
Psychiatric Patients	42%
Schizophrenics	52%

Data of O'Reilly, Hughes, Russell & Ernest
Dis. Nerv. Syst., 26: (1962)

Signs of Pyroluria

Physically, a number of striking features may be identified in the average pyroluric. Fingernails are commonly white spotted and fragile due to the absence of much needed zinc. Zinc and manganese are essential for the proper development of the musculoskeletal system; therefore, painful or poorly developed joints and crowded upper incisors frequently requiring extensive dental correction are present. Tooth formation will be impaired resulting in problems with dental caries. Both zinc and B-6 are required for the formation of body pigments. Skin

tends to be depigmented, tending to a “China Doll appearance” and individuals are intolerant of sunlight. In blacks, the pyroluric patient will frequently have the lightest skin of the family. Scalp hair is lightly colored, although natural color is frequently several shades darker. Pigment deficiencies may lead to premature graying in the young adult. Normal pigment returns with the implementation of proper therapy.

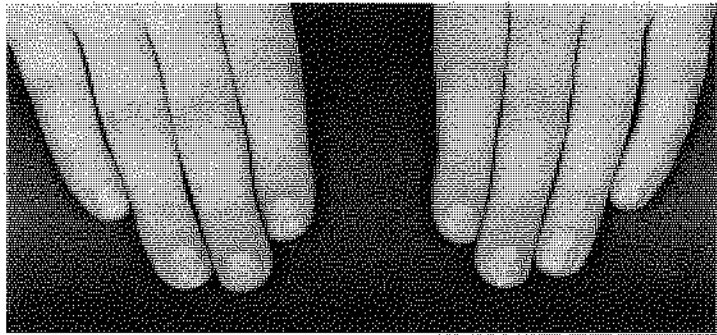


Figure 10: White Spotted Fingernails in the Zinc Deficient

Zinc is essential for the bacterial barrier of the normal skin, thus the pyroluric is plagued with frequent skin infections. Acne is the most common problem, often recurring far beyond the teenage years. Eczema and herpes (cold sores) are other common ailments, responding when proper therapy is implemented. Painful knees indicate poor cartilage formation.

Other factors which may surface with a more re-tailed examination include anemia resulting from the lack of B-6. This B-6 deficient anemia is resistant to the commonly implemented iron therapy. Vitamin B-6 is involved in the synthesis of oxygen binding hemoglobin of the red blood cells. Enlargement and tenderness of the spleen (upper left abdomen) develops due to recurrent hemolytic crises. Amenorrhea results in most young

females, and impotence may occur in males.

Patients often complain of morning nausea which is similar both in characteristics and etiology to the "morning sickness" experienced by pregnant women. The recurrent nausea upon waking stems directly from the B-6 deficiency caused by the long night without nourishment and the restoration processes of sleep. In the pregnant female, it is the fetus which strips the mother of B-6; in the pyroluric, it is the urinary kryptopyrroles.

Dream recall is rare or absent in the pyroluric. Vitamin B-6 is important for the functioning of short-term memory which allows one to remember dreams upon wakening. Dream recall is normal for a healthy human being; however, many falsely believe it is merely a phenomena of the young. Dreaming is an essential part of the human productivity and revitalization process as the mind may use dreams as a safe outlet for one's creative and sexual energies.

The pyroluric parent may ultimately affect the health and well-being of subsequent offspring. The mother, herself starved for zinc and B-6 certainly cannot maintain adequate nutrition for the developing fetus. Male children usually result in miscarriages or stillbirths probably due to the small but definite zinc demands of the developing testes and prostate. The female children whose nutritional burden is within the limitations of the mother and actually get born usually develop difficulties or impairments including delayed puberty, amnesia, "schizophrenia," teenage depression or delinquency, allergic symptoms, and even cancer in later life. The miscarriage of males results in all girl families.

The neurological symptoms of the adult are much more generalized due to the complex interactions of the brain and, thus, should not be considered diagnostic by themselves. Depression is the most pervasive of the psychiatric symptoms and may lead to suicide in the more

despondent individual. Teenage amnesia and delinquency is caused by pyroluria. Convulsive seizures and disperceptions may also occur. Pyrolurics often suffer from chronic insomnia, rarely sleeping for extended periods of time. Hallucinations of a visual and auditory nature have also been documented. B-6 deficiency may show slow waves distinguishable on brain wave studies.

We have determined both the pyridoxal phosphate levels and the E-GOT levels of pyroluric patients and find as expected that they are severely B-6 deficient. This deficiency is relieved by zinc and B-6 supplementation. When the patient is severely B-6 deficient, the serum zinc may be normal or elevated but usually the serum zinc level is below 100 mcg% and may be as low as 50 or 60 mcg%. Urinary kryptopyrroles are high, well above 10 mcg% and sometimes as high as 500 mcg%. All of these biochemical abnormalities are corrected by zinc, manganese, and vitamin B-6 therapy.

TABLE 6
The Effect of B-6 Therapy in the Pyroluric

	Kryptopyrrole	Coproporphyrin
Before Control	1046	225
Day 1	45	75
Day 2	127	48
Day 3	0	76
Day 4	32	52
After Control	141	370

Conventional psychiatric therapies are relatively ineffective in pyrolurics since they do nothing to improve the nutritional deficiencies which are at the root of the disorder. Electroconvulsive therapy and insulin coma yield no positive effects. Pyrolurics are characteristically intolerant to tranquilizers and barbiturates; therefore, attempts at sedation will only exacerbate symptoms.

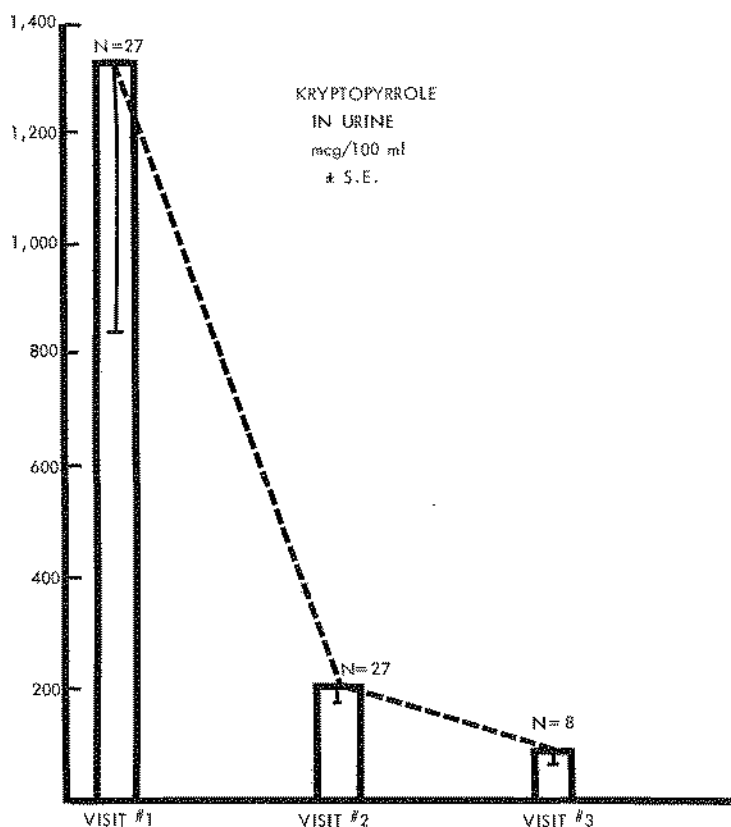


Figure 11: Decrease in Kryptopyrrole with B6 and Zinc Therapy

Nutritional therapy and the replacement of the missing zinc and vitamin B-6 offer the only avenue for complete recovery. Enough B-6 or pyridoxine should be given for nightly dream recall as this provides a convenient biological standard for normality. Zinc, approximately 30 mgm AM and PM, can be given as the gluconate or in the form of a multivitamin in conjunction with manganese (Ziman Fortified, Willner Chemists, Inc.). If the high B-6 dose produces numbness of the fingers or toes, the B-6 should

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be stopped and pyridoxal phosphate can be used instead at 1/5 to 1/10 the B-6 dose.

PORPHYRIA OCCASIONALLY MASQUERADES AS SCHIZOPHRENIA

In most countries and family trees porphyria is very rare indeed, but inbreeding is apt to bring porphyria to the forefront. This inbreeding has occurred in times past in France, Germany, and England, causing the craziness of King George III of England and both Charles VI and VII of France. Porphyria is more common in Sweden again because of consanguineous relationships, i.e. marriage between cousins.

Perhaps one of the most illustrative examples of porphyria exists among the white population of South Africa, where only 40 original settlers started the colony. In the early days when sailing ships were the main cargo carriers around the world, the Cape Horn of Africa needed a watering and green vegetable station for the ship's crews. Accordingly, forty prisoners were placed as immigrants near the Cape to start farming. These men complained of the lack of women so 40 Dutch orphan girls were transported to Africa to provide wives for the farmers. One of these orphan girls apparently carried the blood defect of porphyria. The result now is that an estimated 10% of the whites of South Africa have porphyria tendencies and life insurance policies state plainly that the policy is worthless if the patient dies of porphyria!

Porphyria is an inherited disorder of the body chemistry that rarely expresses itself until after puberty and is especially manifested during the reproductive years. George III experienced mild spells of porphyria in 1765, at the age of 26. These early attacks did not indicate insanity to his immediate court, simply stupidity. Later, however, it was popularly believed that George III must have been insane to sign the enactment of the Stamp Act of 1765, which sowed the seeds of the American

Revolution. His first severe attack came in 1788 when the king was 50 years old, and initiated the belief that the king was insane. The attacks began with abdominal cramps, constipation, and nausea, and a disabling weakness of the limbs. As the disease progressed, he suffered severe sweating, hoarseness, visual distortion, difficulty in swallowing, insomnia, mounting excitement, non-stop rambling, dizziness, stupor, and ultimately, convulsions. Most significant in detecting porphyria was the port-colored urine.

Because of the lack of medical knowledge and biochemistry, the king was never thoroughly examined. The covey of attending physicians would investigate the tongue and pulse, inquire about his excretory functions, and listen to his complaints. They would never dare to question the king unless he first addressed them, which of course the deranged king did rarely. Therefore, the mental ailment of George III covered up many of the physical ailments.

Dr. Francis Willis, called "Dr. Duplicate" because he was a doctor of medicine and theology, was summoned to Kew Palace, where he treated George III with rather unusual inhumane techniques: coercion and restraint. When the king would sweat and become restless and walk the halls, Dr. Willis would diagnose these actions as mania and slap a straight-jacket on the stricken king. Dr. Willis was later praised as the doctor who cured the king, when the disease ran its course and naturally subsided.

King George III's illness spurned the "Regency Crisis" debate in the power-hungry Parliament. The debate waxed and waned in Parliament for four months until, at the end of February, 1789, just as Parliament was about to pass the Regency Act, the doctors declared the mind of George III completely clear. Meanwhile, the American colonists had elected George Washington as their first president.

His ill-timed illness struck again in 1801 and 1804, which encompassed a war with France and the eventual peace treaty of Amiens in 1802. In 1810, the king was so severely stricken with porphyria that the Prince of Wales assumed the throne. By this time, the ailing monarch was past 70 years of age, blind and reduced physically and mentally by his repeated illness. His next few years were spent in relative tranquility, interspersed with the by now painful paroxysms. He died quietly on January 29, 1820, at the age of 81; a time when the Americans, who had desperately fought to free themselves from this so-called tyrant, were again beginning to prosper.

George III's hereditary illness has been traced to his 16th century ancestor, Mary Queen of Scots, includes Frederick the Great of Prussia, and has persisted to the present royal family.

As we celebrate our American Independence, let us think kindly of King George III. His image has been smeared with misconception for as long as we have been free. His stupidity was the confused rambling of an intoxicated brain. His putative "madness" was simply the porphyria-induced delirium and convulsions. King George III was neither a madman nor a tyrant, but a suffering individual inflicted not only with a crippling illness, but the backwardness of the medical profession and the ambition of the Parliament. It is interesting to ponder what might have happened if the king was rationally treated and better fed nutritionally rather than pushed into straight-jackets and inflicted with new, and often irritating, medical procedures and nostrums.

Porphyria or in actuality the porphyrias are a group of hereditary disorders resulting from an overabundance of porphyrias or porphyrin precursors. The porphyrin imbalance results in both neurologic manifestations, including psychoses, seizures, altered consciousness, paresis, and skin manifestations as a result of acute

photosensitivity. Porphyrins are excreted by the body in the urine and feces, darkening upon exposure to light producing the characteristic port-colored urine. Porphyrin is used by the body primarily for the biosynthesis of heme, a metalloporphyrin. Heme is an essential constituent of red blood cells as hemoglobin and is also a component of the detoxifying cytochrome P450 system; thus, porphyrin production occurs mainly in the bone marrow and the liver.

The porphyrias are classified according to the site of pathology into erythropoietic (bone marrow) and hepatic types. Attacks in the hepatic forms of porphyria are often precipitated by drugs or toxic chemicals which stimulate the cytochrome P450 system and, thus, such agents should be avoided. Three hepatic types commonly elicit neurological disturbances similar to those described in King George III, and our discussion will be limited to these disorders. Acute intermittent porphyria is due to a partial deficiency of porphobilinogen (PBG) deaminase and is characterized by excess PBG and δ aminolevulinic acid (ALA) in the urine. Acute neurologic attacks are common, although no cutaneous manifestations are noted with this type. Hereditary coproporphyria involves a partial deficiency of coproporphyrinogen oxidase leading to excess excretion of ALA, PBG, coproporphyrin and uroporphyrin in the urine and coproporphyrin in the feces. One-third of these patients will exhibit cutaneous manifestations as well as neurologic abnormalities. Variegate or South African porphyria is due to a partial deficiency of protoporphyrinogen oxidase resulting in excess ALA, PBG, coproporphyrin, uroporphyrin, and protoporphyrin in the urine as well as excess protoporphyrin in the feces. Cutaneous photosensitivity accompanies neurologic symptoms in the majority of these patients. Additionally, with all the porphyrias, although carriers may not exhibit all the symptoms previously mentioned, they often suffer from

mood swings and body pains. Management of an acute attack involves removal of an offending agent if present plus carbohydrate administration, correction of electrolyte abnormalities, and general supportive measures. Additionally, porphyria patients may have additional biochemical or nutritional abnormalities which may exacerbate their symptoms.

We have had two patients referred to the Brain Bio Center with a diagnosis of porphyria. We have found that they are severe pyrolurics, and both patients responded to a daily supplement of zinc and sufficient vitamin B-6 for normal dream recall.

DO SOME SCHIZOPHRENICS HAVE A CHARACTERISTIC ODOR?

The use of the sense of smell as a diagnostic tool is basic in diagnosis and may provide important clues to many biochemical disorders. Phenylketonuria in newborns is often detected by the "horsey," "musty" odor caused by the presence of phenylacetic acid. The observation of a maple syrup-like odor of urine can avert coma and death in sufferers from maple syrup urine disease. Other metabolic defects manifesting a characteristic odor include Oasthouse Urine disease, sweaty feet syndrome, cat's urine syndrome, fish odor syndrome, and rancid butter syndrome. The use of large oral doses of choline imparts a dead fish odor to the body, because bacteria in the intestine liberate the trimethyl amine from the choline. All basic amines not only stink, but they cling to hair and wool to give a persistent odor to the body.

Many of the older psychiatrists who treated schizophrenics noted that a distinctive odor accompanied the acute disease and that this odor disappeared with effective treatment. Most of the research work has been confined to the study of sweat, although the characteristic odor may also be present on the breath. Doctors and mothers are accustomed to noting acetone on the breath of a child with high fever or with diabetes, and therefore, the fruity odor of the breath of some younger schizophrenics should not go unnoticed. The odor is sweet and similar to that of an aldehyde such as acetaldehyde or a chemical ester.

First documented by Clark in 1917, the "backward" odor of many mental hospitals went relatively unnoted until a study by Smith and Sines in 1960. Sweat samples were taken from the clean skin of schizophrenics. Rats were trained using water as a reward to detect the presence of schizophrenics' sweat as opposed to the sweat of a

normal. The nose of humans was also able to detect a difference; however, their efficiency was not as great. Nine years later, Smith and her coworkers identified the malodorous component of the sweat to be trans-3-methyl-2-hexenoic acid using the methods of gas chromatography, infrared spectroscopy, mass spectroscopy, and nuclear magnetic resonance spectroscopy.

Subsequent studies have found trans-3-methyl-2-hexenoic acid to be present in the sweat of normal individuals and absent in some schizophrenics. Serious questions have subsequently arisen concerning methodology and the causative nature of trans-3-methyl-2-hexenoic acid; however, the presence of the characteristic odor remains well documented. The subjectiveness of the schizophrenic diagnosis, as well as the multitude of disease entities encompassed by the label certainly will complicate further research; however, more work is required to determine the cause of the odor. We have subsequently determined that only the pyroluric patient has this characteristic odor, so only 1/3 of the schizophrenics and 10% "normals" should be expected to be positive for trans-3-methyl-2-hexenoic acid in their sweat, breath and urine.

HISTADELIA— HIGH BLOOD HISTAMINE

Blood histamine is one of the important diagnostic keys against which many schizophrenics can be monitored and evaluated. Those characterized by inherently high blood histamine are referred to as histadelics or suffering from histadelia and compose approximately 20% of the patients presently labeled schizophrenic. The histadelic is one of the more difficult of the schizophrenic disorders to treat, not only due to the characteristics of the disease itself, but its poor understanding of the disorder by those in the psychiatric community. The histadelic responds poorly to conventional antipsychotic drug therapy and electro-shock therapy does little to improve the state. Frequently patients have received the entire gamut of the conventional therapies and are institutionalized or relinquished to the family or street. The hopelessness of this situation combined with these patients' propensity for suicide often results in the ultimate tragedy—the patient cut down in the prime of life.

Histamine is peripherally stored in basophils of the blood and the tissue mast cells. A strong correlation ($r = 0.699$) exists between these cellular components and the actual histamine levels and as such, they may be important diagnostic aids. However, absolute basophil counts performed in the average blood test may be in error. Cellular deterioration with time necessitates the immediate treatment of blood samples with Alcian Blue dye to preserve the accuracy of the count. This practice is uncommon in most offices and clinics. Basophil counts greater than 50 cells/cumm and histamine levels greater than 70 ng/ml (10 mcg%) are considered diagnostic for histadelia. The average histamine for males was 111 mg/ml and females 107 mg/ml in a study reported in 1975. This is

significantly different from normals, 42 and 46 mg/ml for males and females respectively.

The symptoms of histadelia and the unique characteristics of its sufferers often allow for the tentative diagnosis of the patient prior to any actual laboratory testing. Histamine is the chemical modulator of the body involved in many of the normal secretory processes and in anaphylaxis, the body's response to irritation or invasion. Multiple allergies are usually present and often severe. The familial nature of this disorder make allergies common in relatives as well as suicide (previously discussed). Their immediate family is frequently composed of many male children. The thin copious nature of the female histadelic's vaginal secretions provide an advantageous environment for the movement of the smaller, faster male sperm. So the race to fertilize the ova is won by the male sperm.

When sexually aroused, lubrication is heavy in the histadelic female. Orgasm is easily achieved in both sexes with males often complaining of premature ejaculation. Digestive enzymes are stimulated by the presence of histamine, thus salivary flow is usually heavy. The generous supply of saliva continually cleanses the teeth so dental caries are reduced. Gastric secretions are often heavy leading to a propensity for ulcers and heartburn in histadelics. The rapid digestion of foodstuffs is combined with an elevated metabolic rate. Therefore, these individuals have little subcutaneous fat and rarely gain weight despite often voracious appetites. Histamine is known to stimulate oxidation in the body. The accelerated metabolism is responsible for a slightly elevated body temperature and the body develops anatomically to aid in the dissipation of this extra heat. Ears and nose are often enlarged in attempts to increase surface area. The most common adaptation, however, is enlarged hands and feet. The fingers and toes are characteristically long with the

second toe frequently being longer than the first toe. Body hair is usually light and is especially noticeable in males with respect to facial and chest hair. Again, this allows for the more rapid loss of body heat. This patient is the hot one in a double bed and the doer in the family.

Psychiatric symptoms stem from histamine's central role as a neurotransmitter in the brain and the subsequent imbalance of histadelia. Most prominent is the compulsive nature of most histadelics; they are the perfectionists, the doers of the world and are often successful in the endeavors they deem important. Sociologists would label these individuals as Type A personality characterized by aggressive, productive behavior. The histadelic is obsessive, often living each day in accordance with rigid

TABLE 7

Distribution of the two psychiatric symptoms, paranoia or suicidal depression, among schizophrenic patients divided according to their blood histamine levels. All numbers are percentage of patients showing either paranoia or suicidal depression. The distribution of paranoia is greatest in low histamine patients, while suicidal depression is much more frequent in the high blood histamine patient. This skewed distribution would occur by chance only 1 in 200 times.

<u>Paranoid</u>				
	Low	Med.	High	
Male	36.7	16.7	27.3	%
Female	33.3	26.3	6.7	%
Total	35.0	20.0	18.9	%

<u>Suicidal Depressed</u>				
	Low	Med.	High	
Male	0	19.4	50.0	%
Female	2.1	21.0	60.0	%
Total	1.0	20.0	54.0	%

$$X^2 = 98.2 \quad p < 0.005$$

routine. Disperceptions, abnormal paranoia, and thought disorders provide the antagonistic elements in this otherwise productive syndrome. The obsessive-compulsive nature of the individuals may complicate matters if the patient manifests chronic depression. Chronic depression clouds the prognosis of this illness, impairing patient compliance to any treatment regime. Compulsive suicidal tendencies surface during these depressive bouts with a single method predominating (overdosing, cutting, etc.)

The compulsive nature of this disorder as well as the histamine releasing activity of many recreational drugs lead to a propensity for drug abuse and addiction among histadelics. Among their other neurological actions, the opioids are strong histamine releasers and include some of the more common street drugs and pain killers such as heroin, morphine, opium, and codeine. Alcohol has a small histamine releasing action; thus, the chronic alcoholic may, in fact, be histadelic. Stimulants may be continually pumped into the body to combat depression such as crack, cocaine, amphetamines, caffeine, or even sugar. A portion of the road to recovery involves the teaching of moderate use of these agents as the patient improves. Often this is difficult for no therapeutic regime can emulate the high, powerful feeling of many stimulant drugs.

With a few exceptions, conventional pharmacological agents and therapies are useless in the histadelic patient. Antipsychotics, even in large doses, do little to improve the prognosis of the patient. The antidepressant drugs such as amitriptyline or MAO inhibitors are ineffective. Lithium in a low dose of 600 to 900 mgm per day is somewhat effective; larger doses offer little in additional effectiveness.

Classical meganutrient therapy, vitamin B-3 and vitamin C offers only slight improvement. Folic acid leads to a rapid exacerbation of the symptoms. B-12 injections

may offer transient aid for depressive bouts.

More substantial and lasting gains are achieved through the reduction of the large histamine pool which accumulates in the histadelic. Calcium, taken in the form of calcium salts, induces the release of the body's store of histamine. Methionine detoxifies histamine by methylating the ring structure forming N-methylhistamine. We have shown that methionine significantly lowers blood histamine in their patients. Zinc and manganese should be supplemented to aid the calcium-methionine program and frequently provide sufficient relief. If symptoms persist, anti-folate agents such as the antiepilepsy drug, phenytoin (Dilantin) may be added. Phenytoin in a dose of 100 mgm, AM and PM may provide relief for the compulsion and depression. The amino acid L-histidine from which histamine is synthesized should be avoided in the diet if at all possible. Since histidine is found in most protein rich substances, a low protein, high fruit and vegetable diet is preferable.

Opioids provide relief but are addicting when used in these compulsive, depressed individuals. At one time in the early seventies, we had 23 histadelic patients on methadone maintenance therapy. We haven't used methadone or any opiod in the past ten years.

TABLE 8
The Time Course for the Relief of Symptoms
in Histadelia

Depression		
Blank Mind		
Obsessions		
Compulsions		
Rituals		
Phobias		
One Week	One Month	One Year

R_x 1) Methionine, 2) Calcium, 3) Phenytoin

THE SCHIZOPHRENIAS: OURS TO CONQUER

In summary, the treatment program for histadelia is:

1. Vitamin C 1.0 gm AM & PM
2. Vitamin B-6 to normal dream recall
3. DL-Methionine 500 to 1000 mg AM & PM
4. Calcium salt 500 mg AM & PM
5. Phenytoin (Dilantin) 100 mg AM & PM
6. Low protein diet
7. Avoid multivitamins and folic acid

CEREBRAL ALLERGY

Just as the allergic patient may manifest respiratory difficulties, skin ailments, or gastrointestinal problems, the exposure to specific allergens may irritate the brain and, thus, produce profound and distinct behavioral dysfunctions.

The allergic patient whose mental symptoms are so severe as to merit the label of "schizophrenia" has been known clinically for many years, comprising an estimated ten percent of the schizophrenic population. Like many other biochemical types of the schizophrenias, the psychiatrist may be the last to learn and accept cerebral allergy as a possible cause of some types of "schizophrenia."

The allergic child may suffer from the so-called "allergic-tension-fatigue syndrome," described by Dr. Frederic Speer in 1954, which results in irritability, "hyperactivity," and impaired concentration; thus, adversely affecting school performance. A similar syndrome in adults is called simply cerebral allergy. The offending allergen may be inhalants such as pollen, foods and preservatives, or even chemicals with which physical contact or proximity may be sufficient to precipitate an allergic attack. Commonly implicated foods are milk, wheat, beef, eggs, corn, sugar, and chocolate. Often food additives may be the insulting agent, greatly complicating the process of identification. Allergens have specific target organs in the body which are sensitive to their presence, and it is at these sites where the symptoms of the allergic attack are expressed. The skin may become spotted with hives, a blistery rash, or even open lesions. Nausea, vomiting, cramps, or diarrhea may plague the gastrointestinal tract. The respiratory system may show difficulty in breathing, excessive mucous production, post nasal drip, etc. If the allergen reaches the brain, a schizoid

episode may result. Such symptoms are not exclusive so that frequently more than one system is affected, thus, greatly complicating the diagnosis of the syndrome.

Allergic to a Favorite Food

The allergy may appear in a disguised form, where the initial contact with the allergen may produce favorable feelings, such as an uplift of mood or energetic behavior. However, within a variable number of hours a severe let-down occurs similar to the withdrawal syndrome of a drug addict. The patient may experience diffuse non-specific feelings of malaise, fatigue, depression, and paranoia. The uplifting nature of the initial exposure to the allergen may induce a daily routine of eating a particular food or walking through a particular area to get one's daily fix of allergic substances. This leads to the extreme mood swings which may typify the day of the cerebral allergic patient.

Again—A Familial Disorder

All allergies are familial by nature. A careful review of the clinical history of close relatives provides significant insight into the etiology of this type of schizophrenia. The allergic diseases have many presenting symptoms and common names so that the infant who cannot tolerate cow or goat milk may be starting a lifelong fight against allergies called colic or eczema or croup. Lack of breast feeding in an infant may predispose to allergies because the infant does not get the needed immune bodies from the mother. Colic in infancy, childhood eczema or allergic croup may be the first stellar symptom. The colic may progress into celiac disease wherein the food goes through the intestinal tract unchanged. If a sample of the intestinal wall is studied, the fingerlike villi that absorb food are

missing and the intestinal wall is found to be smooth and scarred. Asthma may occur and alternate with the other allergic diseases. Children eating food dyes or foods naturally high in salicylates may develop hyperactivity—a special form of cerebral allergy.

Various Treatments

Patients often have a history of many types of treatment, mostly futile and possibly even toxic or mentally retarding. Tranquilizers may be used to alter the extreme highs and anti-depressants may attenuate the lows but if these mood swings occur in a single day, how can both types of drugs be used effectively! Antihistamines may provide sleep at night and subdue some florid symptoms during the day. The availability of alcohol often leads to its abuse as the individual attempts to combat the daily mood shifts.

Several vitamins are noted for their effectiveness in reducing allergic symptoms. Vitamin C and B-6 (pyridoxine) are probably the most effective. Dr. William Philpott has used both of these vitamins intravenously to turn off allergic symptoms provoked by testing for allergies. The patient on adequate vitamin C will have fewer allergic symptoms. Vitamin B-6 should be given to the point of nightly dream recall and the minerals, calcium and potassium, should be in plentiful supply in the diet. Zinc and manganese are also needed by the allergic patient. Elimination of an offending food may be tried for several months or longer. For multiple food allergies in which this approach would severely limit the diet, a four-day rotation diet in which each food is eaten only once every four days should be tried. If this approach is unsuccessful, intradermal allergy testing to determine the degree of allergy and neutralizing dose of each allergen is recommended. In such procedures, the body is neutralized frequently

(usually twice per week) with injections of specific allergens and thus desensitized to their presence. Injections should be continued for at least one year to discourage reappearance of allergic reactions. Often, desensitization therapy is required for the more permeating allergens in modern society due to the difficulty of their elimination (pollen is a good example).

A number of different methods exist for determining the presence of allergies to specific substances. The strength and weakness of the available methods follows:

1. Eliminations

The oldest and simplest is the elimination of the allergen for a four-day period and then the challenge of the patient with the suspected allergen whether it be food, inhalant, or pollutant. If the patient is overweight, a four-day fast can be substituted for the elimination of a specific food. Foods can then be added one at a time to determine the patient's reaction to individual foods. The pulse rate increases when an allergic food is eaten. This procedure can be easily accomplished by the patient at home; however, it is qualitative and not quantitative, thus, neutralizing doses cannot be determined.

2. RAST Testing

When frozen serum from the patient is sent to a central laboratory, radio-antigens are used to determine what antibodies are present in the serum. This test seems to work better for inhalants and pollens than for foods. The tests are constantly being improved and perfected. However, the result is only useful if a patient has a few inhalant allergies which then can be avoided. Again, neutralizing doses cannot be determined.

3. Leukocyte Cytotoxic Testing

If the white blood cells are quickly separated from the patient's blood at the doctor's laboratory, then the white cells can be tested against common antigens such as solutions of the offending foods. This test can be used on

the umbilical cord blood at birth in order to find out what items the newborn baby is born sensitive to! With luck, eight or more antigens may be tested in one day from one sample of blood. Under these circumstances no more than three patients can be tested by one capable technician in one day. All of the studies are done with unstained white cells so the technician needs careful training. When a sensitive white cell comes in contact with a specific antigen, the cell may burst instantly and release all its granules. This is an extreme four-plus reaction. Lesser degrees of reaction are graded less and one-plus is the simple loss of movement of the white cells. Unfortunately, this test leads to an inordinate number of false positives due to the subjectivity of the tester. The absence of quantitative measures precludes the determination of neutralizing doses.

4. Provocative Testing

Some clinicians use diluted antigens given under the tongue of the patient to provoke allergic symptoms. The quantitative nature of provocative testing, however, is lost by this method.

5. Many clinics use the time-honored, but modernized, skin testing of the patient. This method usually will disclose the neutralizing dose of the allergen. When this neutralizing dose for each allergen or food is given twice weekly, the patient may be relieved of the annoying symptoms and can eat more foods.

Once allergy testing has been completed, the neutralization dose should be checked after six months, and the injections must be continued for at least a year. The individual needs to continue using the nutritional supplements listed previously, as well as utilizing food rotation and environmental control in order to limit future allergic responses.

Treatment of inhalant allergies includes the same supplemental support, as well as avoidance of the

offending agents. Again, if this is unsuccessful, testing for a neutralizing dose and injection therapy may be recommended.

Immunoglobulin A and E

A low serum immunoglobulin A (IgA) indicates pyroluria (see separate chapter) which is frequently accompanied by food allergies, while a high serum immunoglobulin E (IgE) indicates probable inhalant allergies. Most patients with food allergies also tend to have pyroluria, a stress phenomenon associated with excess pyrroles in the urine which bind vitamin B-6 (pyridoxine) and zinc. Some allergies such as wheat are accompanied by damage to the intestinal mucosa (celiac disease), thus resulting in the malabsorption of zinc and/or pyridoxine, as well as other nutrients. Blood histamine levels of the allergic patient tend to be depressed. Histamine is the chemical modulator of the anaphylactic reaction and is stored in the basophils of the blood. In the allergic patient who is continually being challenged with allergens, the frequent release of histamine prevents any storage of histamine in basophils.

Allergic patients may react adversely when exposed to food dyes, aspirin, foods with salicylates, food additives, food preservatives, and the insecticides used to reduce spoilage of food. Organic food eating is, therefore, recommended and carefully chosen vendors become most important. Was insecticide used? Were the crops sprayed? Was a preservative added?

Air deodorants and perfumes may also be offenders. In air travel, one can smell the surge of deodorant wafting through the cabin at regular intervals to the dismay and discomfort of those allergic to petrochemicals!

What Part of the Nation is Pollution Free?

One family was literally driven from the state of Connecticut when the government officials decided to spray the landscape for gypsy moths. Ultimately, the family broke up and we came to know the mother and her very allergic daughter named Chris. Chased from Connecticut, they moved to outside of Santa Fe, New Mexico. When Santa Fe got too polluted, they moved to a more rural area in New Mexico, but this was no better because it was too dry and too dusty! On the advice of an allergist, they tried Florida and as they looked for an ecologically clean homesite they would call the sheriff's office in each county and ask whether this county sprayed for mosquitoes. The hearty (but unwelcome!) answer was "of course we spray for those pests!" On the advice of Theron Randolph, M.D., they bought 80 acres in the Ozark Mountains at Seligman, Arkansas. Only hardwood trees existed and no evergreens with their heavy pollen. A ten-acre lake would provide fresh fish. They had the old musty farmhouse torn down. The well water tested free of metals and nitrates. A local trailer factory had a non-smoking crew of workmen build a trailer home of aluminum and fiberglass with no plastic or vinyl trim. This trailer home would be placed at the top of a hill facing their ten-acre lake. But then their hopes were dashed. The lake waters were hopelessly polluted with the pesticides that all the local farmers used to get a bigger but polluted crop.

Again, they sold the land and hit the highway to the only spot in the USA which is free of pollutants — Sequim, Washington, which is on the Pacific Ocean just south of Vancouver Island. Annual rainfall is only 15 inches per year. The Pacific winds are free of pollen and man-made pollutants. The soil is irrigated by mountain water and there are no mosquitoes, so no sprays. Seattle is only 70 miles away so supplies of nutrients are easily available.

The Sequin homestead came too late to save Chris, because she died in her mother's arms of chemical allergies and general malnutrition on 2/27/84 at the age of 32. One of her doctors, C. Orion Truss, M.D., wrote "The chemical intolerance problem is hopeless when it reaches the stage of violent reactions to every food." We all knew, loved and hoped to get Chris well but we failed.

The ultimate outcome of careful diagnosis and treatment of the allergic patient with cerebral symptoms may be excellent. The patient must watch for new allergies and follow the carefully prescribed diets and routines of avoidance.

WHEAT GLUTEN INTOLERANCE

“There’s a worm in the wheat...in stupidity street.”

— Ralph Hodgson (1871-1962)

Perhaps one of the most ignored of the schizophrenias is wheat gluten intolerance which can cause prolonged psychosis. This syndrome is the manifestation of the deleterious effects of gluten and its breakdown products on the cerebral functioning in genetically susceptible human population. The gluten found in wheat, oats, rye, and other cereal grains, is a protein which serves as a source of nitrogen for the developing endosperm. Although similar to an allergy, in many other ways wheat gluten sufferers cannot be desensitized in the way allergic patients can. Although wheat gluten intolerance comprises only a small portion of the schizophrenias, approximately 4%, it is indeed a tragedy if one endures a lifetime of mental illness when a simple change in the diet will result in a dissolution of the madness.

Celiac Disease in Childhood

The wheat gluten-intolerant patient is characterized by compulsive, ritualistic behavior. Mood and behavior swings occur and are often most noticeable right after the offending grain is eaten, subsiding if gluten-containing foods are avoided. Impaired speech development is common in children. Patients have a history of celiac disease, the name of the malabsorption syndrome caused by sensitivity to the gliadin function of the gluten protein. Symptoms of celiac disease usually arise during the first three years of life as the child is exposed to cereal grains. Typically, a second bout with celiac disease occurs in the third decade of life. The most characteristic symptom of

celiac disease is frequent, explosive bowel movements. Stools are foul smelling, loose and fatty, having a custard-like appearance due to the presence of unabsorbed fat. The child's abdomen is rotund and distended. Slowed growth and lethargy are common. Bloating, gas, and frequent stomach aches add to the gastrointestinal problems of the syndrome. In addition to the behavioral symptoms previously described, actual neuronal destruction has been described in conjunction with celiac disease. Kinney (1982) noted dementia, cerebellar ataxia, myoclonus, myelopathy, and peripheral neuropathy in celiac patients. Post mortem studies have uncovered neuronal loss in the thalamus, basal ganglia, amygdala, hypothalamus, periaqueductal grey, substantia nigra, red nuclei, cranial nerve nuclei, and the posterior spinal columns.

The physiological changes in the gastrointestinal tract are responsible for the malabsorption of nutrients and diagnosis is often made on the basis of intestinal biopsies. The irritation of the gluten causes a flattening of the villi, the thin, finger-like projections necessary for absorption. The damage is transient and the villi return as soon as gluten is removed from the diet.

Dermatitis Herpetiformis

Researchers have also noted a definite correlation between celiac disease and the skin ailment dermatitis herpetiformis. Dermatitis herpetiformis is the appearance of sometimes itchy, chronic lesions of the skin, frequently erupting on the back of the hand. Itching results in burning and stinging sensations. Physiologically, the lesions are the result of granular IgA, C3 protein and fibrin binding to the basement membrane of the skin. Chronic disruption of the skin can result in hyper or hypopigmentation. Renuala (1984) documented the presence of atrophied villi in all patients with dermatitis herpetiformis, with or without

gastrointestinal symptoms suggesting that all sufferers of the skin ailment have celiac disease whether symptomatic or asymptomatic.

Childhood Schizophrenia and Celiac Disease

One of the earliest observations of the relationship between cereal grains and "schizophrenia" was reported by Dr. Lauretta Bender in 1953 when she noted that the schizophrenic child was extraordinarily subject to celiac disease. By 1966, she had recorded 20 such cases from among over 2,000 schizophrenic children. In 1961, Graff and Handford published data stating that during one year, four of the 37 adult male schizophrenics admitted to the Institute of the Pennsylvania Hospital, Philadelphia, had a history of celiac disease in childhood. These early observations greatly interested Dr. F. C. Dohan of the Hospital of the University of Pennsylvania. He noted that these data indicate that "schizophrenia" occurs far more frequently than chance would predict in children and also in adults with celiac disease. Dohan believes that a polygenic inherited susceptibility to both celiac disease and "schizophrenia" may indeed exist and that one may contribute to the development of the other.

This belief is strengthened by the recent discovery of genetic markers for celiac disease and dermatitis herpetiformis. Cole and Kagnoff (1985) reported the presence of the class I antigen HLA-B8 and the class II antigen HLA-DR3 in 60-90% of the celiacs, depending on the geographic region.

Grain Eating Causes Schizophrenia!

Dohan, studying the general consumption of wheat, has noticed a curious correlation between ingestion of grain with frequency of schizophrenia. Investigating the

number of schizophrenic admissions to mental hospitals during World War II, Dohan found marked decreases in admission in countries with wheat shortages—Canada, Norway, Sweden, and Finland. In the United States, however, where no such shortage existed, admissions for schizophrenia increased as one would expect due to the numerous stresses of the war. Studying populations where grains are rare in New Guinea, the Solomon Islands and Yap, Dohan found the incidence of schizophrenia to be extremely rare. Subsequent Americanization of the peoples with increased consumption of wheat, and barley was followed by a rise in the incidence of schizophrenia to near expected levels.

Dohan (1985) hypothesizes that the behavioral abnormalities and schizoid episodes associated with wheat gluten intolerance result from neuroactive opioid peptides produced by the digestion of gluten (endorphins and schizophrenia is discussed in another chapter). In 1979, Klee and colleagues noted the production of opioid peptide *in vitro* through the treatment of wheat proteins with pepsins. Substantial evidence supporting Dohan's hypothesis is lacking at this date; however, it does warrant further investigation.

Treatment of wheat gluten intolerance simply involves the elimination of gluten from the diet. The pervasiveness of gluten in foods requires radical changes from the typical American diet. Thus, one might develop a strong nutritional awareness. Since the malabsorption associated with celiac disease often means the patient is nutritionally deficient, high potency supplements are dictated. Drug therapies are useless for obvious reasons; however, the symptoms of dermatitis herpetiformis may be relieved by sulfones and sulfapyridines. Once a gluten-free diet has been implemented, these drugs may be eliminated or reduced without exacerbation of the skin lesions. Since wheat gluten intolerance is not a true

allergy, desensitization cannot be performed. Consumption of gluten will result in the reappearance of any or all of the psychological, gastrointestinal, and dermatological symptoms.

In place of wheat flour, several substitutes can be used to retain nutritional value, taste, and the aesthetic qualities of prepared foods. Ground flour of brown rice, yellow corn, whole millet, potatoes, and soybeans are suggested. Many "gluten free" wheat flours contain residual gluten, as does oats, barley and rye and, thus, may not be tolerated. Toasted potato slabs can often take the place of bread in the diet and contain no gluten. One should remember that sensitivity is genetically controlled; therefore, some may tolerate foods, while others cannot. Experimentation will be required, consuming small amounts of a food or drink to see if it precipitates a reaction to determine what may be permitted in the diet.

NUTRITIONAL HYPOGLYCEMIA CAN GIVE SCHIZOPHRENIC SYMPTOMS

Many people suffer those “late afternoon blues,” a syndrome which often prompts a short nap or a quick snack to revive waning energy and sinking spirits several hours before dinner. What is one cause of these unpleasant symptoms?

Just as gasoline provides a source of chemical energy to run an engine, sugar (glucose) manufactured from various foods and transported in the blood, is the fuel from which body cells obtain the energy for all cellular activities. When the supply of gasoline diminishes, the engine begins to sputter erratically until replenished with fuel. Similarly, body cells can no longer produce adequate energy when blood glucose and mineral stores become depleted.

Of all the organs and tissues in the body, the brain is most dependent on the minute-by-minute supply of glucose from the blood. When the blood sugar level drops, the brain immediately suffers—resulting in fatigue and emotional chaos.

Hypoglycemia

Low blood sugar, technically termed “hypoglycemia” (hypo-low, glyc-sugar, emia-blood), which is usually responsible for those “late afternoon blues,” represents a chemical change in the body due to a decrease in immediately available glucose. This chemical change occurs in every person several times a day. Although hypoglycemia per se is not a disease, in recent times the term has become synonymous with chronic low blood sugar, a disease state resulting from an error in the body’s regulation of blood glucose levels because of inadequate

minerals. The lacking minerals are potassium, magnesium, calcium, phosphate and the trace elements manganese, zinc and chromium.

Specific Symptoms

- 1) Weakness, fatigue, faintness and dizziness
- 2) Nervousness, irritability, trembling and anxiety
- 3) Depression, forgetfulness, confusion and inability to concentrate
- 4) Palpitations of the heart and "blackouts"

The Blood Sugar Balance

Blood glucose levels are normally maintained within narrow limits despite intermittent food ingestion by a number of hormonal and neural pathways. Glucose can be manufactured from protein, fats, or carbohydrates, but carbohydrates are most rapidly and preferentially converted to glucose. Cells within the hypothalamus of the brain monitor blood sugar concentrations and initiate a series of biochemical readjustments in response to sugar surges.

Signals from the hypothalamus trigger the beta islet cells of the pancreas to release insulin, the hormone which promotes rapid absorption of glucose from the blood by the various tissues of the body. Insulin greatly enhances the transport of glucose into the liver where it is converted to glycogen for storage. Each molecule of insulin released from the pancreas aids in the removal of thousands of glucose molecules from the blood.

When blood glucose concentrations decrease, hypothalamic cells signal for the release of the insulin antagonists, namely adrenalin, glucorticoid hormones, and glucagon. Adrenalin antagonizes the action of insulin by promoting enzymatic activity in cells throughout the

body blocking glucose uptake. Glucagon release by the alpha cells of the pancreas promotes the conversion of glycogen to glucose in the liver. This newly formed glucose is then released into the blood stream to maintain normal circulating levels.

Sugar Cannot Be Stored or Used Without Minerals

Many vitamins and trace elements, including vitamin C, the B complex vitamins, calcium, potassium, magnesium, zinc, chromium, manganese and phosphorus are involved in glucose metabolism and the activities of the endocrine glands. The recently discovered Glucose Tolerance Factor (GTF) which contains chromium, nicotinic acid and three amino acids, is essential for the proper functioning of insulin, facilitating its action at specific insulin receptors, and is necessary for proper carbohydrate metabolism. Brewer's yeast is the best known source of GTF.

When any one of the mechanisms involved in blood glucose regulation becomes affected by disease or functions poorly, the result is a lack of balance between glucose, insulin and insulin antagonists. If too much insulin and/or too few insulin antagonists are produced, the result is chronic low blood sugar.

Certain Methods for Establishing Nutritional Hypoglycemia

For about thirty dollars, the hair analysis may be the most versatile of the diagnostic tests. On junk food, as the body runs low on zinc, manganese, chromium and other minerals such as calcium and magnesium are mobilized to use and store the excess glucose. The hair of the purely nutritionally hypoglycemic patient will, therefore, show a high calcium and magnesium with a low zinc, manganese

and chromium. Other diet indiscretions such as high copper or low potassium may obscure this finding.

For seventy-five dollars, one can obtain the oral 5-hour glucose tolerance test which will usually disclose nutritional hypoglycemia, diabetes, and prediabetic states.

For forty dollars at the Brain Bio Center, one can get a blood level of histamine, spermidine and spermine, three useful markers of possible biochemical imbalance. Histamine provides the answer to histapenia or histadelia. Spermidine is an indicator of rapid cell growth; and spermine, when low, indicates nutritional hypoglycemia.

Each of these tests requires a trained orthomolecular therapist for the interpretation.

Foods for Hypoglycemia Patients

Nutritionally oriented physicians have traditionally prescribed a high protein, low carbohydrate diet for their hypoglycemia patients, with an emphasis on frequent meals and snacks. Such a diet often includes large quantities of animal protein, while excluding carbohydrate-containing foods such as whole grains and fruits. Today, knowledgeable physicians agree that a diet low in animal protein, but high in complex carbohydrates gives consistently better results with hypoglycemic patients. The key is the emphasis on complex carbohydrates—not the pure white sugar so many Americans find addicting, but the type of carbohydrate found in vegetables, nuts, seeds, whole grains (such as oatmeal), and potatoes. When used as the core of the hypoglycemic diet, these naturally occurring carbohydrates help regulate blood sugar levels, thus, preventing the rapid blood sugar swings responsible for hypoglycemic symptoms. These foods also contain the trace minerals necessary for the transport and utilization of these carbohydrates.

Causes of Hypoglycemia

Impaired glucose metabolism engendered by disease is classified as organic or fasting hypoglycemia, since symptoms become more pronounced when food is withheld. An insulin secreting tumour of the pancreatic islet cells (the cells which make insulin) produces severe fasting hypoglycemia. Congenital liver enzyme defects; damage to the liver produced by alcohol, tobacco, or infection; encephalitis; brain tumours; hypopituitarism and Addison's Disease (an exhaustion of the adrenal gland) also cause hypoglycemia.

Numerous drugs have been implicated as the cause for hypoglycemia including sulfonylureas, salicylates (aspirin), prepanolol, pentamidine, and quinine. Additionally, disopyramide (Norpace), sulfamethoxazole, and trimethoprim (Bactrim, Spectrum) have been reported as causes when accompanied by renal problems. Despite the seemingly long list, all of these are rare causes accounting for only a few of the hypoglycemic disorders.

Defects in glucose metabolism resulting from secondary factors occur with far greater frequency. Such disorders are classified as nutritional, functional, reactive or fed hypoglycemia, because symptoms develop in response to food intake. Alimentary hypoglycemia, one type of nutritional glycemia, often develops in patients who undergo subtotal gastrectomy for peptic ulcers, as foods pass more rapidly into the small intestine when part of the stomach has been removed. Most often, however, prolonged stress, particularly the internal disturbance provoked by poor eating habits, precipitates hypoglycemia.

Stress Releases Blood Sugar and Minerals

Any physical or emotional trauma (i.e. pain,

overexertion, childbearing, anxiety, grief, fear) causes the adrenal gland to release adrenalin, prompting an increase in blood glucose to supply the extra energy needed to deal with the stress. When a person suffers continual stress, the adrenal gland must constantly supply adrenalin. Eventually, this persistent demand exhausts the adrenal gland. When challenged, it can no longer produce enough adrenalin and hypoglycemia results.

Sugar Provides Only Empty Calories Without Minerals

Nutritionally inadequate foods, without trace elements, exert a subtle but complex and damaging stress on the body's regulation of glucose metabolism. Refined carbohydrates are the worst offenders.

Sucrose, the refined sugar in baked goods, sweets and the sugar bowl, consists of a molecule of glucose and a molecule of fructose. When sugar is eaten, enzymes in the small intestine readily break the bond between the two simple sugars and glucose with fructose surges into the blood stream, signaling the pancreas to release battalions of insulin molecules. Insulin rapidly admits glucose to the cells and the level of glucose in the blood quickly decreases. This action is responsible for the quick, but temporary, energy provided by a bar of candy.

Empty Calories Stress the Pancrease and Adrenal Glands

When repeatedly forced to handle large amounts of glucose (derived from a diet rich in refined sugars), the pancreas becomes sensitized and hypoglycemia develops. Every time glucose enters the blood, the pancreas overreacts, releasing too much insulin which causes the cells to absorb and utilize glucose at top speed. The adrenal gland, striving to maintain the proper glucose level, becomes exhausted. Soon after a meal, blood sugar falls below the

MAJOR BIOCHEMICAL TYPES OF THE SCHIZOPHRENIAS

fasting levels and the body craves sugar; producing hypoglycemic symptoms. Another dose of sugar relieves symptoms for a short time, so many hypoglycemics snack continually on sweets, without minerals, a pattern which only aggravates the underlying metabolic disorder.

Too Much Sugar

Increased consumption of refined carbohydrates during the past 50 years probably accounts for the rising incidence of diabetes and hypoglycemia in recent times. In the 19th century, the per capita intake of sugar in England was only seven pounds per person a year. Today, people in the western countries consume as much as 128 pounds of sugar per year, and the human body cannot adapt to this drastic change.

Mark Twain once advised that the "secret to success in life is to eat what you like and let the food fight it out inside," but this statement came sometime before the present avalanche of sugary foods reached the market. Today, most people eat to satisfy their sweet tooth with refined carbohydrates and the food is indeed "fighting it out inside," and in many cases wholly defeating the glucose regulatory mechanisms. Doctors specializing in metabolic disorders estimate that at least one in twenty people suffer from hypoglycemia.

Governments Advise Dramatic Reduction in Sugar Intake

Both the US Senate Select Committee and the government sponsored NACNE Report advise a halving of average sugar consumption. But since sugar is cheap, mildly addictive (and there are many sugar addicts about) and extends the shelf life of many foods, it is little wonder that the food industry puts 30% sugar into ketchup and 23% into sauces and salad dressings. There is no doubt that

sugar-rich, convenience foods are making more and more people hypoglycemic.

What Are the Symptoms?

When cells utilize available glucose so rapidly that the blood cannot readily meet the constant demand for more fuel, the cells actually become starved. Glucose deficiency drastically alters the function of the brain, since the brain cells cannot store glucose and, thus, require a continuous supply to generate energy. In a state of glucose starvation, the brain suffers reduced efficiency and can no longer completely direct vital processes, thus disrupting physical and emotional behavior.

Physical and emotional disturbances in the hypoglycemic disorders vary according to the severity of the disorder and the individual affected. Mental symptoms frequently resulting from hypoglycemia include fatigue, irritability, nervousness, insomnia, mental confusion or forgetfulness, inability to concentrate, anxiety, phobias and fears, disperceptions, disruptive outbursts, and headaches. Such symptoms are non-specific and present in many disorders, but strongly indicate hypoglycemia when they occur from time to time, after fasting, late at night, first thing in the morning, or in direct relation to the time or content of a meal.

Low Blood Pressure and Low Body Temperature Is a Clue

A distinctive characteristic of hypoglycemia is low blood pressure and lowered body temperature. Hypoglycemics often complain of cold hands and feet and many experience cold sweats. Dr. Freinkel (1972) and Dr. Molar (1974) studied hypothermia in laboratory-induced hypoglycemia and found significant decreases in body temperature associated with the onset of other

hypoglycemic symptoms. Both doctors attribute this phenomenon to the effect of glucose deficiency on brain cells, since the hypothalamus controls body temperature. Many doctors note the low blood pressure but shrug it off with, "Well, you'll never die of high blood pressure." A normal blood pressure is needed to keep the hands warm and the mind alert. Manganese raises blood pressure and all hypoglycemic patients are deficient in manganese.

Hypoglycemia is Easy to Treat

For many "diseases of lifestyle" the outlook is grim but not so for hypoglycemia. All that is needed for the disease to go away is a change in lifestyle. Change to a "caveman's diet," high in complex carbohydrates, with daily exercise will not only do away with symptoms but make all the tests return to negative!

Specific Treatment

- 1) Avoidance of the junk foods sugar, alcohol and white bread
- 2) Exercise daily
- 3) Take Manganese, 10 mgm, AM & PM
- 4) Take Zinc, 15 mgm, AM & PM
- 5) Take Glucose Tolerance Factor (GTF), AM & PM
- 6) Take a low dose Multivitamin tablet (No Copper)

POSTPARTUM OR PUERPERAL PSYCHOSIS

Postpartum or puerperal psychosis compose two to eight percent of all female admissions to mental hospitals. These patients, either nearing completion of a pregnancy or having just given birth, experience disturbances as a result of their rapidly changing biochemical state. The majority of them are between 22 and 28 years of age, and their psychotic breaks generally occur within two weeks following a first birth. Statistics show more psychosis after male babies than after females.

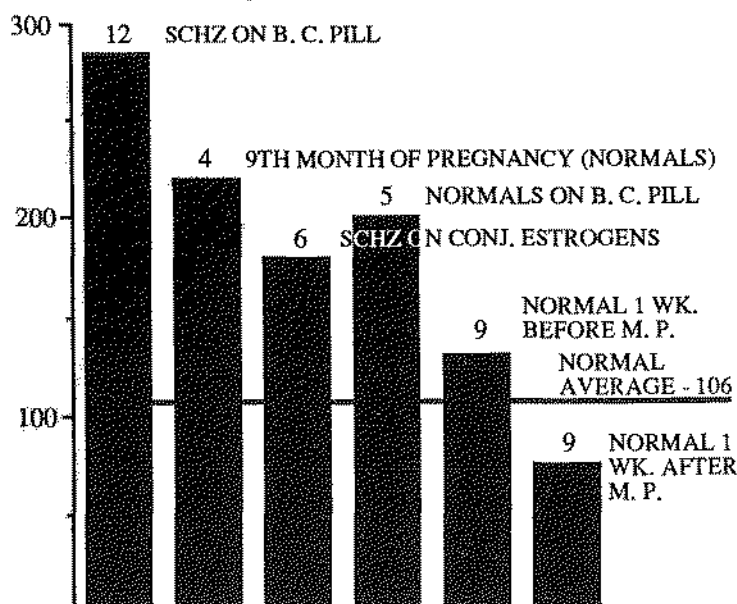
Such episodes of biochemical imbalance may be indistinguishable from schizophrenia. Symptoms may include a clouding of the conscious, disorientation, paranoia, euphoria or depression, aggression, unusual religious interests, self hostility, sleep disturbances, headache, nausea, vomiting and anorexia. The mother is often led to reject her newborn.

R. Gooch (1820) commenting on his experience with the malady states, "Those accustomed to the patient will notice her conduct and language become wild and incoherent, and at length she becomes decidedly maniacal; it is fortunate if she does not attempt her life before the nature of the malady is discovered." Hippocrates speculated the abnormal behavior was due to the effect of the mother's milk on her own brain. Nurses on the obstetrical ward call the psychosis the "milk let-down blues." Modern psychoanalysts cite psychological conflicts about the mothering of the infant as the major cause. The true determinant of this schizophrenia-like disturbance is not an individual or familial abnormality, but rather a biochemical imbalance due to the rapidly changing hormonal status of the body.

Since high copper levels can lead to psychotic

breaks, it is interesting to note that copper, and particularly ceruloplasmin (a copper-containing serum protein) are elevated by the female estrogen hormones. As estrogen levels fluctuate throughout the menstrual cycle, copper and ceruloplasmin also fluctuate. As the level of hormones rises progressively during pregnancy, the levels of copper double from approximately 115 mcg percent at conception to a mean value of 260 mcg percent at term. In some cases the rise in copper is so great the blood plasma actually takes on a green tinge. Malleeson (1953) was the first to link premenstrual tension and postpartum psychosis to this common cause. Rising copper levels are also responsible for the steady decline of blood histamine throughout pregnancy. Schizoid breaks are possible until the copper levels return to normal, and untreated, this can take months.

FIGURE 12
Women and Copper—"The Estrogen Link"
Blood Serum Levels of Copper—
Mcg Percent—in Women



Of perhaps greater concern to the sexually active female is the synthetic estrogen present in all oral contraceptives. Pfeiffer and Iliev have found some of these potent estrogens raise the body's copper burden to levels beyond those found in the ninth month of pregnancy, thus posing a threat to a woman's physical and mental health. Psychosis may last for several weeks after the discontinuation of the pill.

Therapy for estrogen-precipitated psychosis should be directed at decreasing the body's estrogen and copper levels and is similar to that discussed in conjunction with histapenia. Remission corresponds to the slow decline of accumulated copper in the blood and tissues; therefore, improvement may take many weeks after the implementation of effective treatment.

SCHIZOPHRENIA OR PSYCHOMOTOR EPILEPSY

An alternative diagnosis in patients with episodic schizophrenic behavior is psychomotor epilepsy. Epilepsy is characterized by uncontrolled, excessive electrical activity of all or part of the central nervous system. Epileptic attacks are produced when the activity of the nervous system rises above an inherent basal level. Precipitating factors include stress of a physical, mental, or emotional basis especially those which are unforeseen. Psychomotor epilepsy is a subdivision in which the seizures are localized to a specific region of the brain. Characteristically, psychomotor seizures involve part of the limbic system of the brain, such as the hippocampus, the amygdala, the septum, or the temporal cortex. The limbic system is that portion of our brain which integrates behavioral patterns, especially those involved with emotions, subconscious motor and sensory impulses, and the intrinsic feelings of punishment and pleasure. One half of all epilepsy is without apparent cause, thus suggesting a genetic predisposition; however, trauma, oxygen deprivation, tumors, bacterial or viral infections, and biochemical imbalances have been known to cause epilepsy in previously normal individuals.

Diagnosis of epilepsy is usually made on the basis of electroencephalogram (EEG) results which measure the summed electrical potentials of areas of the brain. Such measurements must be recorded during a seizure to be of great value to the therapist. Brain waves measured during an attack exhibit low frequency rectangular waves with an average frequency of four per second and a magnitude of approximately 100 microvolts. If EEG's prove to be inconclusive, diagnosis may be made on the periodicity of the attacks and response to certain drugs.

The sensory manifestations of psychomotor attacks include visual and auditory hallucinations. Patients may claim to see bright lights or objects and hear voices or sounds. Often seizures may be so organized that entire scenes with both visual and auditory stimuli may be experienced. Of course, the nature of the overt symptoms depends upon the areas of the brain involved in the seizure, which will vary from person to person and even with different attacks. Tactile disturbances in attacks may disrupt limb position sense; some may even claim limbs enlarge and change shape. Often the patient cannot recall the seizure once it has passed and brain functioning has returned to normal.

Psychiatric manifestations may consist of recurrent episodes of abnormal ideas, with or without some sensory involvement. Disruptions of one's perception of time may occur with patients claiming time rushes by or stands still. Delusions of depersonalization in which one feels separated from the environment or body are common. Emotional content in such attacks have been documented.

Treatment of psychomotor epilepsy involves the use of antiepileptic drugs, most commonly phenytoin (Dilantin). Dosage should be in sufficient quantities to control seizures and reduction may be feasible if on a nutrient supplement regime. Responsiveness to antiepileptic agents may be used as a diagnostic tool if a battery of clinical tests have revealed no clues as to the basis of the nervous disturbance.

Unlike drugs, nutritional therapy of epilepsy will affect and control all types of seizures, so one does not need to design nutrients for each type of seizure. You should know that lead-intoxicated children convulse, and other metals such as cobalt and aluminum produce seizures when applied to the brain of animals. Some natural dietary trace elements combat this effect and rid the body of convulsants such as lead and aluminum. In

patients with seizures we use zinc, magnesium, and vitamin C to reduce the body burden of lead and aluminum.

Adelle Davis taught, "give them all liberal doses of magnesium and their seizures will go away." This dictum is only, however, a small part of the nutritional needs of the epileptic. We do recommend milk of magnesia tablets (magnesia) 300 mgm 2 tablets AM and PM.

If any single trace element corrects the biochemical imbalance of epilepsy, it is manganese. Although often ignored by nutrition-conscious individuals, manganese is an essential trace metal frequently deficient in our diet. A component of at least six known enzymes, manganese is required for efficient sugar metabolism and for the production of cartilage, a vital structural component of our bodies.

Unfortunately, most diets, even the best planned, tend to be deficient in this important trace metal. Our manganese-deficient farmlands often produce fruits and vegetables lacking adequate levels of the element. And, many of our frequently-eaten foods fail to concentrate manganese even under the best conditions; for example, meat, even liver, provides little manganese. Foods rich in manganese include nuts, whole grains, spices, legumes, and tea leaves. Tropical fruits such as banana, papaya, and mango are particularly good sources; these fruits, growing in a sun-rich climate, have a high capacity of photosynthesis, and photosynthesis is a manganese-dependent process. Adult man needs at least 5 mgm per day, but manganese is poorly absorbed. This process is blocked by excess zinc or copper in the daily diet. We recommend, and successfully use, 50 to 100 mgm of manganese per day taken orally at bedtime.

If any single vitamin is needed in epilepsy, B-6 (pyridoxine) heads the list. Many children and teenagers have the mauve factor (kryptopyrrole) in their urine which robs them of zinc and B-6. They will have abdominal pain,

white-spotted fingernails, and will not remember their nightly dreams. They may need as much as 1000 mgm of B-6 in order to restore dream recall. We start with 50 mgm taken orally each morning and double the dose until dreams or a total of 1000 mgm is reached. Manganese is also needed for dream recall. B-6 doses in excess of 1000 mgm may produce numbness of the fingers or toes, an indication for shifting to pyridoxal phosphate at 1/5 to 1/10 the oral dose.

If any single amino acid is needed to control epileptic seizures it is taurine, the simplest of the amino acids. Taurine is not considered essential for man but cats need it for the normal development and function of the retina of the eye. The late Andre Barbeau, M.D., of Montreal, found taurine to be effective in the control of seizures. Oral doses of 500 mgm AM and PM are usually well tolerated. In histadelic patients, this dose may produce peptic ulcer distress which disappears when the taurine is discontinued.

The pyroluric patient with severe B-6 deficiency may show spikes and slow waves in the EEG which is corrected by adequate B-6, manganese, zinc and taurine. The anticonvulsant drugs must be discontinued slowly. In answer to "how long should the nutrients be continued in the epileptic patient?" We can only answer, as long as the patient eats—these are nutrient supplements.

PROLACTIN HORMONE AND THE SCHIZOPHRENIAS

Lack of sexual desire together with impotency in the male and lack of a menstrual period in the female may signify a stress-induced micro-tumor of the anterior pituitary gland. This micro-tumor secretes prolactin which can cause disturbances, both sexually and mentally. The mental symptoms may mimic those of schizophrenia.

In 1928, studies showed that the anterior pituitary of the cow had a definite factor which would cause milk secretion in the rabbit. In 1933, Riddle gave the name "Prolactin" to the factor. The human prolactin molecule has been isolated and its peptide structure is similar to that of growth hormone which is, however, more abundant in both the blood and the pituitary. Franz and Kleinberg (1970) established that prolactin circulates in both men and women fluctuating with the menstrual cycle, rises with pregnancy and lactation, and the blood level also rises with all the potent anti-psychotic drugs. The common denominator in the relationship between prolactin and antipsychotics is the neuro-transmitter dopamine. Many antipsychotics act centrally to decrease dopamine levels, the major inhibitor of prolactin secretion.

The natural purpose of prolactin is to cause milk secretion and to regulate calcium and other minerals. At the time of childbirth, the mother's prolactin level is 3X normal, and this high level continues on in the first months of nursing. After six months of continued nursing, the prolactin levels are only slightly elevated. The initial high prolactin levels block ovulation and normal menstruation so that the mother is relatively non-fertile during this period. Extended breast-feeding is the one way of spacing children which is used in undeveloped countries.

In 1972, Franz et al. found that prolactin levels rise

during sleep and return to normal after awakening. Their second finding was that any type of physical or emotional stress can cause a large rise in prolactin blood levels. Tyson and Zachi (1976) suggested that stress might cause tumor formation in the anterior pituitary which could persist after the stress is relieved. This proved to be the case!

These tumors are now frequently diagnosed in both male and female patients who are presently labeled schizophrenic. In women, breast secretion occurs with a disturbed menstrual cycle and loss of sexual libido. Late symptoms are disperceptions, infertility, facial hair, chemical diabetes, and headaches. In men, the main effect of such a tumor is loss of libido and impotence. Of course, some of the anti-psychotic drugs will produce these symptoms in some patients. I recall that the large doses of chlorpromazine (Thorazine) that we used in patients at Manteno State Hospital in the 1950s produced breast secretion in about 5% of the female patients.

The best differentiating key to the presence of a prolactin-secreting tumor is the level of blood prolactin. R. C. Smith and his group at Houston, Texas, (1984) found that continued use of haloperidol at the oral dose of 10 mg to 20 mg per day produced a greater increase in prolactin in females (85 mg/ml) than in males (30 mg/ml). The decision must rest with the physicians as to whether the measured prolactin level is due to a possible tumor or to the effect of the anti-psychotic drug. Fortunately, we have an effective drug to treat the tumor called bromocriptine (Sandoz Parlodel).

Bromocriptine is one of several ergot compounds which are known to be an effective treatment for acromegaly, a rare pituitary tumor which produces gigantism. Prior to the discovery of bromocriptine the patients, when accurately diagnosed, had their tumors removed surgically via the nasal approach. Many tumors recurred

and now respond to bromocriptine in doses of 2.5 to 7.5 mg per day. Bromocriptine can also be used in a therapeutic trial to determine if the prolactin level decreases and if the symptoms subside within two to three weeks of daily dosing at the 5 mg level.

Prolactin affects salt and water balances so that many mysterious illnesses may be owing to the normal fluctuations in prolactin secretion. This is greatest at the menstrual period and may cause a weight gain of two or more pounds at menstruation. The remedy is nutrients to control the edema, but when this is not effective, then bromocriptine (2.5 to 5.0 mg taken at bedtime) may provide a logical remedy.

The side effects of bromocriptine are sleepiness and postural hypotension which is a drop in blood pressure which causes dizziness when the patient stands up suddenly.

Bromocriptine is effective. M. O. Thorner of the University of Virginia Medical School reported that bromocriptine induced ovulation in 13 of 21 hyperprolactinemic women, restored menstruation in 15 and abolished abnormal breast secretion in 20 of the 21. Bromocriptine is also standard therapy for suppressing lactation in women who do not wish to nurse their babies.

There have certainly been patients with documented hyperprolactinemia whose schizophrenic symptoms have improved with bromocriptine administration. They also represent a small portion of the schizophrenic population. The underlying, neurological explanation for the prolactin-induced psychiatric disturbances remains elusive at best.

HOMOCYSTEINURIA MASQUERADES AS SCHIZOPHRENIA

Homocysteinuria is a metabolic disorder in which the patient excretes in the urine an abnormal amino acid called homocysteine. Homocysteinuria is found in children often labeled schizophrenic. Homocysteinuria is also found in adults who are vitamin B-6 deficient. In man, the homocysteine leads to rapid development of calcified arteries.

In teenagers, the mental symptoms of homocysteinuria mimic exactly those of schizophrenia. If untreated with vitamin B-6, the disorder may produce seizures. Other manifestations include osteoporosis, scoliosis, pectus carinatum or excavatum, and mental retardation. Acute problems usually surround the propensity for thromboemboli formation resulting in myocardia, pulmonary, and cerebral infarcts. The usual cause of the biochemical defect is a lack of the enzyme cystathionine B synthase, inherited as an autosomal recessive, but other enzyme defects such as a lack of methylene tetra hydrofolate reductase can cause the homocysteinuria and the symptoms of schizophrenia. Freeman et al in 1975 reported one such 15-year-old female patient who was diagnosed as schizophrenic with catatonic reaction and finally diagnosed as simple schizophrenia.

Fortunately, they did the cyanide nitroprusside reaction test on her urine and found this to be highly positive for the presence of homocysteine. She responded to both pyridoxine and folic acid and she probably needed zinc as well since she had at one time a severe abdominal pain probably of spleen or liver origin—hemolysis of red cells as in zinc and B-6 deficiency! Brachen and Coll (1985) review the literature on homocysteinuria and schizophrenia and report an additional case which failed to respond

to folic acid or pyridoxine.

In addition to a low methionine diet, B-6, the vitamins betaine, B-12 and folic acid have been found useful in individual adult cases. Any methyl donor will probably help when enough zinc pyridoxine are present to promote the methylation reaction.

Homocysteinuria is a rare disease probably affecting only one in 10,000 of the patients labeled schizophrenic. If treated early, the schizophrenic symptoms are reversible so we do need more tests with cyanide nitroprusside reagent on the urines of all patients admitted to psychiatric hospitals. The Sandare Chemical Company of Dallas, Texas 75208, sells a kit which allows for the accurate testing of homocysteinuria and cysteinuria. The kit uses the well-established silver-nitroprusside reagent.

OTHER BIOCHEMICAL THEORIES

THE PROSTAGLANDIN THEORY CORRELATES WITH HISTAMINE THEORY

Prostaglandins (P) are fairly ubiquitous lipid compounds postulated to have hundreds of different cellular control functions, most still awaiting real proof. Peripherally, prostaglandins are effective vasodilators, while centrally, prostaglandin E elicits fever when present in the hypothalamus. The neuronal pain response to chemical, mechanical, and thermal stimuli is greatly enhanced by the local presence of prostaglandins. These diverse implications of prostaglandins as a modulator of nervous activity has led Horrobin (1977, 1978) to postulate that a loss of prostaglandin homeostasis may be the biochemical imbalance responsible for the schizophrenias. With further investigation of these findings, it appears that the prostaglandin theory by Horrobin and the histamine theory by Pfeiffer (1972) may be measuring the same physiological phenomena, but merely using different biochemical parameters of the disorder. The common denominator is probably that both prostaglandin and histamine levels are affected adversely by excess copper in the tissues.

A number of studies have demonstrated evidence of a deficiency of prostaglandin E-1 and low prostaglandin E-2 and low prostaglandin E-1 (PGE-1) in the direct measurement of cerebrospinal fluid of schizophrenic patients. (Prostaglandins are differentiated into series 1, 2, and 3 and such differences are denoted by a number following the term). In 1985, Heleniak and LaMola noted the marked similarity between Horrobin's low PGE-1 and Pfeiffer's low histamine biotype of schizophrenia. Both biotypes manifest identical symptoms—thought disorders, overarousal, grandiosity, paranoia, ideas of reference, hallucinations and mania. The high copper/low zinc

ratio diagnostic to histapenia is also apparent in the low PGE-1 syndrome. Methionine which exacerbates the symptoms by lowering blood histamine also worsens the low PGE-1 type schizophrenia. Methionine excess gives rise to taurine which blocks the release of dihomogamalinolenic acid, a precursor of PGE-1. Perhaps most enlightening is the observation that the levels of histamine and prostaglandins move concurrently in the body in response to different physiological challenges. Although the exact relationship between these two compounds has yet to be delineated, many believe that Prostaglandin E-1 may serve as a second messenger in the activation of histamine. Cabut and Vincenzi (1967) demonstrated that PGE-1 stimulated the release of heparin and histamine from mast cells in tissue, which would tend to support this "second messenger" hypothesis; however, more data are needed.

Despite the lack of knowledge concerning the interrelationship of prostaglandin and histamine, those described as deficient in these compounds respond favorably to the same treatment regime—yet another clue that the same physiological phenomena are responsible. Folic acid, vitamin C, pyridoxine (vitamin B-6), niacin, zinc, penicillamine (a copper chelating drug), and a high protein diet result in significant improvement in patients with respect to both biochemical keys. The oils whose digestion ultimately results in the precursor gamma linolenic acid and prostaglandin E-1 should be consumed in sufficient amounts to avoid a nutritional deficiency. Usually these are supplied to a large extent by the cooking and salad oil used in food preparation and the seafood we eat.

Safflower oil, olive oil, and sunflower oil are preferred due to their content of polyunsaturated fats. The oily fish, sardines, herring, bluefish, etc., are the most beneficial in the seafood category. These provide omega 3 linolenic acid. If such products are not present in the diet,

they may be supplemented with evening primrose oil and mega-EPA (a fish body oil product).

Thus, in conclusion, an imbalance of prostaglandin metabolism certainly has proven to be a factor in the occurrence of some of the schizophrenias. The syndromes described, however, appear to be those previously differentiated by Pfeiffer as histapenic (low blood histamine) and histadelic (high blood histamine). Treatment for both low histamine and low prostaglandins is the same. Prostaglandins are simply another index against which these patients can be differentiated, diagnosed and treated. The proof of the pudding is in the arrest of the symptoms of the schizophrenias. The Princeton Brain Bio Center has had great success in treating the schizophrenias according to their blood histamine levels. The clinic which determines the blood levels of prostaglandins has not yet arrived.

DRUG INDUCED PSYCHOSIS

Any agent which directly or indirectly influences the nervous system has the inherent potential for profound mental or behavioral symptoms. The pervasiveness of the nervous system as the controller of body functions precludes that few pharmacological compounds can be eliminated from this neuro-active category.

The existence of drug-precipitated schizoid breaks have been common since the discovery of the first plant hallucinogens. An illustrative record of a common New World hallucinogenic agent, jimson weed, as reported in 1705 from Virginia follows:

The James-Town Weed (which resembles Thorny Apple of Peru, and I take to be the Plant so call'd) is supposed to be one of the greatest Coolers in the World. This being an early Plant, was gather'd very young for a boil'd Salad, by some of the Soldiers sent thither, to pacifie the Troubles of Bacon; and some of them eat plentifully of it, the Effect of which was a very pleasant Comedy; for they turn'd natural Fools upon it for several Days. One would blow up a Feather in the Air; another wou'd dart Straws at it with much Fury; and another stark naked was sitting up in a Corner, like a Monkey, grinning and making Mows at them; a Fourth would fondly kiss, and paw his Companions, and sneer in their Faces, with a Countenance more antick, than any in a Dutch Droll. In this frantick Condition they were confined, lest they should in their Folly destroy themselves; though it was observed, that all their Actions were full of Innocence and good Nature. Indeed, they were not very cleanly; for they would have wallow'd in their own Excrements, if they had not been prevented. A Thousand such simple Tricks they play'd and after Eleven Days, return'd to themselves again, not remembering any thing that had pass'd.

During the Revolutionary War, the British troops frequently made a tea of the jimson weed, and their antics were equally as colorful.

Certainly in our society of a federally regulated pharmaceutical industry, a schizophrenia-like disorder in response to a legally obtained drug is more the exception than the rule. Drugs are rigorously tested before being released in the marketplace, and a propensity for psychotic reactions would lead to certain rejection by the F.D.A. One, however, cannot abandon the possibility of a drug-related disturbance, especially when the course of an acute psychotic illness follows the implementation of a pharmaceutical. Due to inherent differences in the patient population, one patient may be therapeutically aided for years without adverse side effects, while seemingly minor factors may predispose another to mental disaster.

A list of drugs with documented cases of schizophrenia-like complications appears below. Since new reports are constantly surfacing, we do not guarantee the completeness of this list, but hope it will provide a general overview of the potential for drug-induced psychoses.

Atropine Psychosis

Drug or Pharmaceutical	Common Symptoms
Amitriptyline (Elavil, others)	Confusion; memory
Atropine, Belladonna alkaloids	loss; disorientation;
Benztropine (Cogentin),	depersonalization;
Cyclopentolate (Cyclogyl)	delirium often with
Desipramine (Pertofrane),	high fever; auditory,
Doxepin (Adapin, Sinequan),	visual and tactile
Imipramine (Tofranil, others),	hallucinations; fear;
Nortriptyline (Aventyl),	paranoia; incoherent
Protriptyline (Vivactil),	speech; flushed, dry
Scopolamine (Hyosine),	skin.
Tricyclic antidepressants,	
Trimipramine (Surmontil)	

OTHER BIOCHEMICAL THEORIES

Amphetamine Psychosis

Drug or Pharmaceutical	Common Symptoms
Amphetamines, Dextroamphetamine, Diethylpropion (Tenuate), Fenfluramine(Pondimin) Methamphetamine, Phenmetrazine (Preludin), Phentermine (Fastin) Cocaine, "Crack"	Bizarre behavior, hallucinations, paranoia, depression (on withdrawal)

Other Agents

Drug or Pharmaceutical	Common Symptoms
Amantadine (Symmetrel)	Visual hallucinations, nightmares
Aminocaproic acid (Amicar)	Acute delirium with auditory, visual and kinesthetic hallucina- tions
Anticonvulsants, Ethosuximide (Zarotin), Phenytoin (Dilantin, others), Primidone (Mysoline)	Tactile, visual and au- ditory hallucinations; delirium, agitation, depression, paranoia, confusion, aggression
Antihistamines	Anxiety, hallucina- tions, delirium
Asparaginase (Elspar)	Confusion; depres- sion; paranoid, bizarre behavior
Baclofen (Lioresal)	Visual and auditory hallucinations, paranoia, insomnia, nightmares, mania, depression, anxiety, confusion
Bromocriptine (Parlodel)	Mania, delusions, vis- ual hallucinations, paranoia, aggressive behavior

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Chlordiazepoxide (Librium)	Probably same as diazepam
Chloroquine (Aralen)	Confusion, agitation, violence, personality change, delusions, hallucinations
Cimetidine (Tagamet)	Visual and auditory hallucinations, paranoia, bizarre speech, confusion, delirium, disorientation, depression, seizures, insomnia, amnesia
Corticosteroids (prednisone, cortisone, ACTH, others)	Mania, depression, confusion, paranoia, visual and auditory hallucinations, catatonia
Cycloserine (Seromycin)	Anxiety, depression, confusion, paranoia, visual and auditory hallucinations, paranoia
Dapsone (Aviosulfon)	Insomnia, irritability, confusion, disorientation, hallucinations, paranoia
Diazepam (Valium), Clonazepam (Clonopin), Clorazepate (Azene, Tranxene)	Rage, excitement, hallucinations, depression, suicidal thoughts
Digitalis glycosides	Nightmares, euphoria, confusion, delusions, amnesia, belligerence, visual hallucinations, paranoia
Disopyramide (Norpace)	Agitation, depression, paranoia, auditory and visual hallucinations, panic
Disulfiram (Antibuse)	Delirium, depression, paranoia, auditory hallucinations, mania, catatonia

OTHER BIOCHEMICAL THEORIES

Ephedrine	Hallucinations, paranoia
Ethchlorvynol (Placidyl)	Agitation, confusion, disorientation, hallucinations, paranoia
Indomethacin (Indocin)	Depression, confusion, hallucination, anxiety, hostility, paranoia, depersonalization
Ketamine (Ketalar, Ketaject) "Angel Dust"	Nightmares, hallucinations, crying, delirium, changes in body image
Levodopa (Dopar, others)	Delirium, depression, agitation, hypomania, nightmares, night terrors, visual and auditory hallucinations, paranoia
Methyldopa (Aldomet)	Depression, hallucinations, paranoia, amnesia
Methylphenidate (Ritalin)	Hallucinations
Methysergide (Sansert)	Depersonalization, hallucinations
Metrizamide (Amipaque)	Confusion, disorientation, hallucinations, depression
Nalidixic acid (NegGram)	Confusion, depression, excitement, visual hallucinations
Niridazole (Ambilhar)	Confusion, hallucinations, mania, suicide
Pentazocine (Talwin)	Nightmares, hallucinations, disorientation, agitation, bizarre behavior
Phenezine (Nardil)	Paranoia, delusions, fear, mania, rage, aggressive behavior

THE SCHIZOPHRENIAS: OURS TO CONQUER

Phenobarbital, Barbiturates	Excitement, hyperactivity, visual hallucinations, depression, delirium-tremenslike syndrome
Phenylephrine (Neo-Synephrine)	Depression, visual and tactile hallucinations, paranoia
Procainamide (Pronestyl)	Paranoia, hallucinations
Procaine Penicillin G	Terror, hallucinations, disorientation, agitation, bizarre behavior
Propoxyphene (Darvon)	Auditory hallucinations, confusion
Propranolol (Inderal)	Depression, confusion, nightmares, visual and auditory hallucinations, paranoia
Quinacrine (Atabrine)	Bizarre dreams, anxiety, hallucinations, delirium
Thiabendazole (Mintezol)	Hallucinations
Vincristine (Oncovin)	Hallucinations

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B12/FOLIC ACID DEFICIENCY PSYCHOSIS

Perhaps one of the most deceptive and tissue-destroying of the schizophrenias is B12/folic acid deficiency. The inter-relationship between vitamin B12 and folic acid is such that a deficiency in either of the components will result in the appearance of the full deficiency syndrome. B12 is required for the utilization of the folates, especially in blood formation. Striking rapidly, often without warning, the deficiency attacks the adult eroding both the spiritual and financial foundation of family. The condition does not respond to treatment by antipsychotic drugs. Thus, the prognosis is poor unless the correct diagnosis is reached initially.

The symptoms of B12/folic acid deficiency include all of the classic symptoms of schizophrenia, as well as some unique ramifications on the peripheral nervous system and blood formation. Perhaps the best diagnostic feature, although not always present, is a pernicious or macrocytic anemia unresponsive to iron treatment. Nervous involvements may include encephalopathy, cerebellar atrophy or dysfunction, myelopathy, and peripheral neuropathy. Behavioral manifestations of nervous dysfunctions include headaches, sleeplessness, forgetfulness, irritability, depression, and paranoia. Bachevalier and Botez (1978) have documented severe learning impairments in rats suffering from folic acid deficiency. Kariks and Perry (1970) report B12/folic acid deficiency, with or without a resultant anemia, to be associated with skin diseases, recurrent abortions, celiac disease, gout, tropical sprue, liver disease, rheumatoid arthritis, tuberculosis as well as mental illness, and organic brain disease. Additionally, some pharmacological agents have been linked to B12/folic acid deficiency, including phenytoin sodium,

phenylbutazone, nitrofurantoin, phenobarbital, barbiturates, analgesics, pyrimethamine, primidone, and alcohol.

In 1954, Bodenoch was the first to draw attention to anti-convulsant drugs used in the treatment of epilepsy and a subsequent folic acid deficiency. Prolonged treatment with phenytoin derivatives may in some epileptics lead to a macrocytic anemia and paranoid psychosis. Supplementation with folic acid will improve the psychosis and anemia. Anticonvulsant therapy other than phenytoin should then be used.

Kane and Lipton, in an early review, point to five clinical problem areas where altered folate metabolism has been found. In addition to the metabolic and psychiatric complications previously mentioned, the population at risk include mentally retarded children, pregnant and postpartum women, and women taking oral contraceptives. Stone and his colleagues found twenty-two percent of the pregnant women surveyed to be folate deficient. Combined with Shojania's study, reporting thirty percent of the women taking oral contraceptives to be deficient, this data seems to suggest a role for the endocrine system, particularly estrogen and copper in the metabolic balance of B12 and folic acid. In a report with more profound implications, Read and his coworkers reported eighty percent of the admissions to an old age home to be folate deficient, possibly explaining the symptoms of apathy, withdrawal, and depression common in geriatric patients. More than likely these folate deficiencies in the elderly can be attributed to nutritional deficits and malabsorption syndromes.

The data suggests routine monitoring of folic acid and vitamin B12 status for selective patient groups. Certainly the users of anticonvulsants must be surveyed to avoid psychosis. Psychiatric patients, mentally retarded children, pregnant women, and oral contraceptive users may avoid psychotic complications through this

monitoring. However, the group which would appear to benefit the greatest from an awareness of B12/folic acid balances is the geriatric community. The implementation of a comprehensive survey and supplementation program may increase longevity, productivity, and the overall quality of life.

SCHIZOPHRENIA AND WATER IMBALANCE: POLYDIPSIA, POLYURIA, AND PSYCHOSIS

A 21-year-old female university student was brought to the emergency room confused, with bizarre behavior. The young woman was found in an agitated state...She reacted to vocal stimuli by staring, screaming incoherent words. Neurological examination did not reveal any meningeal irritation signs...No other abnormality was found in the physical examination...

The patient's mother reported that her daughter had drunk 30 glasses of tap water, one after the other, after she was asked to drink 6 and another 6 glasses before ultrasound examination of an ovarian cyst.

The patient assumed that the more she would drink the more informative the test would be...After the test....she said she was dying, not knowing how to explain her feelings.

—Kott, 1985

The above case of intoxication from water alone is outstanding not only because it occurred in a physically and mentally normal young woman who voluntarily consumed an excessive amount of tap water (preparing herself for an ultrasonic gynecological examination), but also because her acute state completely returned to normal following conservative treatment (Kott, 1985). Acute water intoxication is a medical emergency. The psychotic state can be immediately induced. Any delay in starting the appropriate treatment may cause irreversible brain damage and, occasionally, death. Because water at no cost can produce altered mental states, one might say cynically, why pay good money for street drugs?

Earlier work by Rowntree demonstrated that water

ingested in excess of the subject's excretion ability leads to water intoxication manifested by "restlessness, asthenia, polyuria, frequency of urination, diarrhea, salivation, nausea, writhing, vomiting, muscle tremor and twitching, ataxia, tonic and clonic convulsions, frothing at the mouth, helplessness, stupor, and coma." Self-induced water intoxication was first recognized to be associated with schizophrenic disorders in 1938 when Barahal reported on a 31-year-old woman, hospitalized with paranoid schizophrenia, who developed seizures and coma following the ingestion of large quantities of water (Rae, 1976).

Excessive drinking of water (polydipsia) and excessive volumes with urination (polyuria) are common among schizophrenic patients and combine with severe hyposthenuria (which refers to a urine specific gravity of 1.003 or less), hyponatremia (low serum sodium), seizures, coma, and cerebral and visceral edema, to describe the syndrome called self-induced water intoxication and schizophrenic disorders (SIWIS). Severe hyposthenuria is the silent biological marker that always antedates the complications of SIWIS. Major motor seizures which can result from a serum sodium concentration of less than 120 mEq/l (normal = 142) are the most commonly recognized presentation of SIWIS (Vieweg et al, 1984a).

The prevalence of compulsive water drinkers in state mental hospitals ranges between 6.6% (Jose et al, 1979) and 17.5% (Blum et al, 1983), with half of those patients suffering the complications of water intoxication. Approximately 70% to 80% of all compulsive water drinkers have a schizophrenic disorder manifested by, among other classic schizophrenic characteristics, inappropriate affect, auditory hallucinations, paranoid delusions, and looseness of associations (Jose et al, 1979). The most frequent and important clinical findings are lethargy,

disorientation, confusion, semi-coma or coma, and seizures (Smith et al, 1980). Because many have died from the complications, SIWIS is increasingly included in the differential diagnosis of unexplained death among psychiatric patients.

Vieweg and his associates (1985c) claimed that some patients' daily intake of water is as high as seven or eight gallons. Hariprasad (1980) reported that patients drank between 7 and 43 liters of water a day. They frequently arise at night to urinate and consume additional fluids. When water faucets and fountains were not available, patients consumed water from showers and toilets. Surprisingly, few patients described feeling excessively thirsty.

Although the biochemical mechanism behind the need to drink large quantities of water is still unclear, patients who are aware of their excessive drinking habits offer a variety of explanations. Some attribute their polydipsia to delusions, hallucinations, or anxiety. Others more specifically spoke of washing out worms, or trying to free themselves of creatures and other foul material. Many believed they were cleansing themselves of sin including one patient who informed researchers that God had told her to drink large quantities of "holy water" (Smith, 1980).

A 29-year-old woman with a diagnosis of chronic schizophrenia died as a result of drinking 4 gallons of water daily "to cleanse her body of cancer" (Rosenbaum et al, 1979). Another young woman who died from the complications of water intoxication was unfortunately blamed for her compulsive behavior. Having learned of the great quantity of water she ingested, the medical examiner attributed her death to intentional suicide. The situation was later clarified by a doctor well versed in the SIWIS syndrome who informed reporters by adding that "She...was not able to control her water intake" (Clinical

Psychiatry News, 1977).

Many hypotheses have been offered to explain the compulsive water drinking behavior and the resultant psychosis. Kissileff (1933) proposed that the drinking is a "response to increased levels of transmitter substances accumulating in the limbic system during the experience of frustration." Other concepts emphasize the antidiuretic hormone, arginine vasopressin, and its widespread role in learning, memory, behavior, as well as its critical role in water metabolism (Nemeroff et al, 1973). Histamine is also involved in thirst regulation in the limbic system and has been shown to be particularly important in the schizophrenias (histadelic and histapenic schizophrenic) (Pfeiffer, 1972).

Work by Snyder (1984) and his associates has focused on the amygdala and other parts of the limbic area where opiate receptors are highly concentrated. They demonstrated that opiate agonists are up to 50 times stronger when sodium is absent. As previously mentioned, low sodium levels are common in psychotic patients. Perhaps some patients "seek" to enhance the opiate receptor effect by excessive drinking which decreases the surrounding sodium concentration. If this were true, it might follow that in a sense hyponatremic patients are actually addicted to the water. Hyponatremia is often associated with psychosis, seizures, and coma. Patients with low serum sodium levels and resultant psychoses have been invariably reported in Bartter's syndrome (Desmit et al, 1970).

Robertson (1985) has pointed out that not all psychotic patients who drink excessive amounts of water have low serum sodium levels. Some people with low serum sodium levels never develop associated complications. (Men are less likely to develop the complications associated with hyponatremia.) Individuals ingesting vast amounts of liquid each day (e.g., 20 liters) generally are

able to secrete the load (Barlow et al, 1959). Psychotic patients who consume and excrete large quantities of water but maintain normal serum sodium levels are referred to as having primary polydipsia. Chronic compulsive water drinkers often have serum sodium levels lower than normals. Psychotic patients who consistently maintain low serum sodium levels seem to have reset osmostats and are often confused with a third group of psychotic patients with PIP syndrome (psychosis, intermittent hyponatremia, and polydipsia) who manifest hyponatremia during 4-63% of serum sodium determinations (Vieweg, 1986). What seems most crucial is the "concentration of sodium and water, not the absolute levels of either. Subjects with hyponatremia have been reported with high, normal and low total body water and sodium levels. Regardless of sodium levels, people who excessively drink should be watched for psychotic symptoms.

Once all this water has been ingested into the system, it often leaves as nearly equal quantities of urine (polyuria). In 1933, Hoskins and Sleeper reported that schizophrenic patients on the whole excreted almost twice the average daily urine volume (average 2,602 ml - minimum 510 ml and maximum 8000 ml) over emotionally healthy controls (average 1,328 ml—minimum 655 ml and maximum 2805 ml). Other studies which focused on the high volume excretors reported several patients who voided as much as 30 liters of fluid in one day (Vieweg et al, 1985e). Many more recent studies come close but are unable to exactly replicate the urine volumes recorded by Hoskins and Sleeper because the former, more accurate techniques do not conform to current standards which control the use of human research subjects. Most seem to underestimate daily urine output by as much as 25-50% (Vieweg, 1986).

If much of the excess water remains in the body, it may lead to severe hyponatremia and brain and gut swelling. The syndrome of inappropriate antidiuretic hormone

(SIADH) secretion causes the body to retain water and, thus, may produce a water imbalance with normal ingestion (Bartter et al, 1967). Urinary sodium and osmolality are inappropriately elevated in the presence of water intoxication and hyponatremia. The condition is present in virtually all patients after surgery. An inability to properly excrete an ingested water load is one of the characteristic metabolic abnormalities in Addison's disease (Lever, 1983). Other clinical situations in which SIADH secretions have been reported include malignant tumors of the bronchus, gut and thymus; neurological disorders, for example meningitis, trauma, and various intracranial tumors; bacterial lung infections; and drugs (Bartter et al, 1967). Myxedema (low thyroid function) and acute intermittent porphyria, two more entities associated with schizophrenia (Pfeiffer, 1970) have been related to the syndrome of inappropriate antidiuretic hormone. Sometimes the ADH secretion is sufficiently raised for patients to become intoxicated after ingesting normal amounts of water (Thorn, 1970).

In one of Dubovsky's patients (1973), the occurrence and disappearance of the inappropriate secretion of antidiuretic hormone (ADH) was concurrent with the presence and resolution of acute psychosis on two separate occasions. Reports of this nature have led some researchers to suggest that the inappropriate hormone release syndrome is psychogenic. Smoking is known to elevate ADH levels and experimental evidence suggests that ECT elevates ADH (Jose, 1980). Pain, exercise, and perhaps acute psychosis itself can cause the syndrome of inappropriate ADH secretion and impairment of water excretion.

While excessive drinking may solve well-known dehydration problems, endurance athletes must be careful to avoid water intoxication. Diluted drinks appear to be more dangerous than water alone. Exercise-induced ADH secretion can be significant enough to interfere with the

proper excretion of ingested water and, thereby, cause a psychotic break. Having already suffered psychotic breaks due to water intoxication, three endurance athletes were advised to drink less while running in the future. All three have subsequently completed prolonged endurance courses uneventfully (Noakes, 1984).

Excess fluid intake is particularly dangerous for the psychotic patient on medication. Unfortunately, many of the most useful pharmaceutical agents also are capable of inducing inappropriate release of ADH. Psychotherapeutic drug administration should be strictly limited to patients in control of fluid consumption. It is considered to be one of the iatrogenic causes of SIADH (Matuk, 1977). Thiazide diuretics, which are often prescribed to reduce the sodium content in hypertensives, are risky because further reductions in sodium content can add to the problem of hyponatremia. Antidiuretics may cause further damage, on the other hand, because they help to retain water and, thereby, contribute to a reduced sodium concentration in the blood. Any drug that increases water retention is dangerous: phenothiazine, desmopressin acetate, vasopressin, prednisone, propanolol, narcotics (meperidine, morphine, hydromorphone), ACTH, oxytocin, are just a few. Hypnotic post-operative fluid administration may be more dangerous than water ingestion.

Notable neurological abnormalities appear both with hyponatremia and with increased fluid retention, presumably as a result of brain swelling (Rendell, 1978) and often progress rapidly to convulsions, coma, and finally death. Patients usually recover quickly and completely unless ADH-releasing drugs are also administered. With ADH-releasing drugs, the pressure in the brain can reach such high levels that the brain actually herniates towards the spinal canal and the patient dies. Behavioral changes with increased body fluid retention, short of convulsions and coma, include irritability, decreased

concentration and increased psychotic symptoms. Certainly the brain is a sensitive "bioassay," and thinking and behavior over time must be influenced by the brain's physiologic milieu (Rosenbaum, 1979).

Some of the most tragic cases of permanent brain damage and death due to complications of hyponatremia occur in hospital settings with female patients who arrive essentially healthy, prepared to undergo elective surgery. Not only is SIADH secretion likely in the post-operative state, but medications with water retention side-effects are commonly administered. Arief (1986) found that even with no serious underlying medical conditions, 15 women developed grand mal seizures within 49 ± 7 hours following elective surgery. For some, there were early warning signs and psychiatric consultation was sought for "psychological symptoms" including hostility, depression, disorientation, and hallucination. For others, it all happened within a period of less than 10 minutes. At the time of seizure activity, the plasma sodium concentration for all 15 patients was 108 ± 2 mEq/dL (normal = 142). All developed respiratory arrest within 60 minutes after the onset of seizures. Four patients died. Nine women remained in a persistent vegetative state and were institutionalized for custodial care.

The greatest problem for these women was the average delay of 16 hours before therapy was begun for the hyponatremia and the slow rate of correction even then. (The appropriate correction protocol appears to include increasing the plasma sodium level by about 2 mmol/L per hour to a level of 128 to 132 mmol/L.) For ten of the patients, hyponatremia was not even suspected as a cause of their seizures and respiratory arrest. Instead, subsequently unconfirmed diagnoses included: acute stroke, sagittal sinus thrombosis, arteriovenous malformation, herpes encephalitis, migraine with vascular occlusion, rupture of cerebral aneurysm, skull fracture with subdural

hematoma, and coma of unknown origin. Forty-two consultants and many diagnostic studies later (including CAT scan, EEG, angiography, lumbar puncture, and open-brain biopsy), hyponatremic therapy was begun. The longer the hyponatremic state, the more severe the irreversible brain damage and the more likely death will result. This suggests that many of the managing and consulting physicians were not aware that hyponatremia could lead to the observed symptoms (Arieff, 1986).

It has long been known that dehydration can lead to altered mental states; it is now clear that excessive hydration can also alter mental function. Not only hypernatremia, but now clearly hyponatremia can be associated with psychosis (Farley et al, 1986). Both can, in extreme cases, be life-threatening. Clinicians must be encouraged to be constantly aware of the problem.

In-patient psychiatric units where large volumes of water and coffee are ingested, where cigarette smoking is heavy, where a variety of medication is prescribed, and where psychotic patients are hospitalized, are classic sites for disturbances in water regulation. Even though the syndrome is common in psychiatric patients, the physical aspects of the condition are often overlooked and left untreated by the hospital professionals who focus on psychiatric complaints. The chemical imbalances in the bodies of excessive water drinkers and/or excessive urinator may not only cause schizophrenic symptoms, but may also lead to life-threatening complications broadly divided into those due to hyponatremia and those due to fluid retention. This condition can be easily diagnosed with simple tests of serum and urine osmolality. The treatment for the majority of cases is simple and consists of water and fluid restriction. The therapeutic response can be dramatic (Matuk, 1977).

NATURAL OPIATES, DEPRESSION AND COMPULSIVE RUNNING

Since their discovery in 1975, great enthusiasm has surrounded the natural role and possible therapeutic value of the body's endogenous opioid transmitters in the brain. Concurrently, there has been much contemplation concerning abnormalities of opioid transmitters and their role in mental illness, namely schizophrenia. Three biologically related groups have been identified as possessing a morphine-like activity in the brain. The enkephalins, methionine enkephalin (met-enkephalin) and leucine-enkephalin (leu-enkephalin) were the first to be discovered. These five amino acid peptides are derived from the precursor molecule, proenkephalin. The second group consists of dynorphin, a 13 amino acid peptide derived from the precursor prodynorphin. The third and longest opioid transmitter is B-endorphin, a 31 amino acid peptide. B-endorphin is derived from B-lipotropin which, in turn, is derived from proopiomelanocortin. Proopiomelanocortin serves as a precursor for a number of other chemical modulators, namely adrenocorticotropin (ACTH) and melanocyte stimulating hormone (MSH).

B-endorphins, dynorphins, and enkephalins act pharmacologically like morphine and the opiates. Injected into the ventricles of the brain, they produce a marked analgesia and a state of euphoria. Enkephalins exhibit only a weak opiate effect, probably due to their small size. However, B-endorphin is 48 times as potent as morphine, while dynorphin is 200 times as potent as morphine! Large doses of such agents may produce respiratory depression, sedation, and muscle rigidity—all morphine-like symptoms.

Endogenous opiates are released in response to stress in anticipation of life-threatening situations and

possible painful stimuli. The stress may be of a physical or mental nature. This stress-induced, natural analgesia may explain how soldiers wounded in battle and athletes injured in competition often report no feeling of pain. David Livingstone, the Scottish Missionary and explorer of Africa, illustrates a century-old example of stress-induced analgesia as he describes being attacked by a lion.

“...I heard a shout. Starting, and looking half round, I saw the lion just in the act of springing upon me. I was upon a little height; he caught my shoulder as he sprang, and we both came to the ground below together. Growling horribly close to my ear, he shook me as a terrier does a rat. The shock produced a stupor similar to that which seems to be felt by a mouse after the first shake of the cat. It caused a sort of dreaminess in which there was no sense of pain nor feeling of terror, though quite conscious of all that was happening. It was like what patients partially under the influence of chloroform describe, who see all the operation, but feel not the knife...The shake annihilated fear, and allowed no sense of horror in looking round at the beast. This peculiar state is probably produced in all animals killed by the carnivora; and if so, is a merciful provision by our benevolent creator for lessening the pain of death.”

—(David Livingstone, Missionary Travels, 1857)

Studies of schizophrenics have suggested increased, normal and decreased natural opiate activity. Certainly the inclusive nature of the definition of schizophrenia, as well as methodological problems concerning the characterization of the opioid being measured contribute to the contradictory data.

As a result, three hypotheses and subsequent therapeutic approaches have been forwarded in the literature.

Based on the hypothesis that increased opioid activity is responsible for schizophrenic conditions, naloxone, an opioid antagonist, has been administered producing decreased hallucinations (David et al., 1981). Decreased opioid activity in schizophrenics has been treated with intravenous administration of B-endorphin with varied results (Berger et al., 1981). De Wied et al., (1978) have suggested schizophrenic symptoms result from an imbalance in endorphin metabolism, specifically the inability to convert B-endorphin to des-tyrosine γ -endorphin. They find that des-tyrosine γ -endorphin functions as a neuroleptic agent and good therapeutic results have been reported with des-tyrosine γ -endorphin treatment in diagnosed schizophrenics.

The Dutch scientists of Utrecht, headed by Verhoeven et al, compared two peptides—desencephalin gamma endorphin (3 mg) and ceruletide (40 mcg) for their ability to modify schizophrenic behavior. Six injections were given over a period of two weeks. Each peptide produced significant improvement compared to the placebo. The beneficial effect of the peptides lasted two weeks after the treatment which was essentially without side effects. At present, these peptides are expensive (@ \$300 per dose), but electroshock therapy costs that and is more damaging. The Japanese headed by Yamagami of Osaka found that ceruletide in doses of 1 to 2 mcg per kg is an effective treatment. We have had a father who can afford it invest \$3000 in an effective peptide which has been used in treatment of his schizophrenic daughter.

Although endorphins, dynorphin, and enkephalins were originally praised as possible non-addictive pain medications, subsequent studies have shown them to be dependence and habit forming. Thus, individuals may be addicted to endorphins or even natural liberation of endogenous opioids. A strenuous daily workout may free large amounts of endogenous opioids, resulting in a

euphoric feeling. Habitually, such exercise may lead to an addiction to one's daily "opioid fix." Additionally, if a population suffers from inherently depressed opioid levels as some studies suggest, exercise may elevate levels to normal standards. This addiction is exceptionally illustrated by a runner and patient of the Brain Bio Center.

Compulsive Running
by Jean

"I run. In a sense it is synonymous to saying I am, I exist. For in running I feel I have control of many aspects of my life. I run for me. It is a totally selfish act. Yet, because I run, I am able to live and give to others.

To date, I have run 3,328.5 miles over a period of 3 1/2 years. Although to some that may seem like an exorbitant amount of miles; compared to many runners, it is hardly a beginning.

None of my accomplishments are unique, exceptional, or even noteworthy, for many people have run further and faster. What is perhaps out of the ordinary about my running is the amount of injuries I have had. Although injuries such as shin splints, sprained ankles, chondromalacia (runner's knee), achilles tendonitis, neuromas, and stress fractures are as common to runners as chicken pox to a child, I would venture to say that most have not had eight stress fractures and continued to run.

With an injury record like mine, some might question why I would continue running. Stated simply, "It makes me feel good." Socrates once said, "How singular is the thing called pleasure, and how curiously related to pain, which might be thought to be the opposite of it...yet, he who pursues either is generally compelled to take the other." It is true the injuries I have had have been painful, extremely so at times, and I find no pleasure in that. But to me, the positive aspects of running have far outweighed

the negative.

I ran my first mile on January 3, 1983 because a friend invited me to run with her. I had been basically inactive for over 10 years, and at 32 was extremely out of shape and overweight. At first it took a great deal of "grit" and determination to run every day. I enjoyed the freedom I experienced from the routine and responsibilities of wife and mother of four young boys. But I honestly did not relish pushing my legs till they ached, gasping for breath, or drowning in sweat.

In five weeks I progressed from one to five miles, lost six pounds, and sustained my first stress fracture. I had exceeded my body's ability to adapt to the stress of running. The podiatrist I consulted said it was an overuse injury—too much, too fast, and too soon. The story of my life.

As I was put in a cast and faced with several weeks without running, I became nervous, agitated, anxious, irritable, and had more trouble than usual sleeping. I tried to keep busy, but time dragged, and nothing helped relieve these negative symptoms until I was able to run again.

Three weeks after having the cast removed I ran my first 10K, finished in excruciating pain, but filled with ecstasy. I loved the competition, not only against others, but myself. In completing the race I felt a sense of satisfaction, strength, and power that I had not felt for years. I felt like a kid again, ready to conquer the world.

The next day, I sat in my podiatrist's office having another cast put on (my second stress fracture, 3rd metatarsal of my right foot), and he asked me if running the race was worth it. Without hesitation, I said yes.

Again I experienced symptoms of withdrawal, such as: headaches, tension, and depression as the weeks of recuperation crawled on. However, once able to run again, it was easy to forget how bad the injury, and resulting negative emotions, felt.

I ran for three months before I began to experience severe pain in my left foot, a stress fracture of the second metatarsal. Although in considerable pain, I continued to run until I completed a local 5K road race (placing third overall female) I had desperately wanted to compete in. Having been able to run the race made the following two weeks without running more bearable. As soon as my doctor consented, I began running again.

Three weeks later due to continued pain in my left foot, I had another X-ray. It showed a stress fracture of my heel. This time I continued to run, reducing both distance and speed, and it eventually healed.

Then in December of 1983, after running with severe shin splints for several weeks, I stress-fractured both tibias. I put off going to my podiatrist as long as possible because I wanted to complete 1,000 miles before the end of the year. But it became so painful that I could barely walk, let alone run.

The doctor who read the bone scan told me that I would never run again. At first he thought I was the victim of spouse abuse and told my podiatrist, who promptly assured him that my injuries were simply due to my running. He said in all his years of medicine he had never seen both legs stress fractured simultaneously.

My right leg was put in a cast for six weeks with the threat of another cast being put on my left leg for an additional two to three weeks. I never had the second cast put on.

I think a lot of people had their doubts that I would run again, but I knew I would. There was never any question to me. Three months later, I was back on the roads again.

All this may sound irrational, but at the time I was running to survive. Had I not been running that year, I seriously doubt that I would have lived. I needed to run. It was a time when my life seemed to be falling apart.

Running gave me a sense of stability. At times it was the single bright spot without which destruction was certain in the blackness that surrounded me.

By deliberately stressing my body through running, I was able to relax my mind. The constant repetition and rhythm of movement had a tranquilizing effect. The power and confidence I felt as I ran helped me fight the emotional turmoil I experienced, knowing I could still control some aspects of my life. The success I attained in running encouraged me, creating confidence in my ability to deal with difficult situations.

Fortunately, once I recovered from stress-fracturing my legs, I was able to run nearly a year without any major setbacks. I hurt from time to time, but nothing I couldn't tolerate and run through.

In September, 1985, injury struck again as I stress-fractured my left tibia. I anticipated trouble this time. I had developed shin splints and my shoes were badly worn. But, faced with the choice of not running until I could afford new shoes or run with mine as they were, I ran. And I broke.

Again, I continued to run even after I knew I had another stress fracture. My life situations were such that the physical pain was far less than the mental anguish I felt, and in a strange way, one made the other bearable. Finally, however, the pain increased until I could barely walk, and I knew that I had to seek medical aid once more.

Being completely inactive was unbearable. I 'climbed the walls' and sank into a deep depression. I tried walking several miles at a time on my crutches, but all that was accomplished was to get my arms sore.

By the time I got the cast off, I was so distressed from the extended inactivity that I ignored the pain that was still present, and ran. In December, after another bone scan showed the fracture was more extensive than in September, I again had to stop running. Without a cast I tried

biking and walking. By the end of February I was still experiencing a great amount of pain, so I gave up and let the doctor put another cast on.

I went eighty-one days without running—the longest time since I began three and a half years ago. In April, 1986 I started a vigorous program of rehabilitation, which my family practice doctor had outlined for me. Slowly, the lethargy I felt disappeared, and I began to feel stronger. I pushed myself as hard as I could, following my doctor's advice to the letter.

With all the physical activity and exercise, it would seem that I wouldn't miss running, but I did. Nothing comes close to the enjoyment and fulfillment I get from running long, and hard, and fast. There is something about the repetitive, rhythmic pounding during running that produces a calmness, a peacefulness, a sense of well being, not equaled in any other activity I've tried.

Sometimes, usually after an injury, I have considered not running any more. There are days when I feel terrible while running, when pain and fatigue seem overpowering. But I keep going, and eventually feel better.

Running has changed my life physically—not only a healthier cardiovascular system, but losing twenty-five pounds, going from size 14 to size 3; socially meeting many new people on the road, at races and in the running club to which I belong; intellectually—producing greater creativity and concentration; spiritually—providing quiet solitude in which to pray and meditate on things of God; and emotionally—creating more patience, stability, strength, confidence, and a sense of self-worth.

Will I continue to run? Yes, definitely. Why? Because I am what I am and running is what it is."

Jean, aged 34, is histadelic, blood histamine 73, 78, absolute basophil count is 58, 75 per cu mm. First visit, Spring 1986. She is depressed, compulsive and fearful.

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She is tolerantly married, and they have a family of four boys. This all-male family occurs naturally in high histamine women since their vaginal secretions are more dilute, and the male sperm wins in the race to fertilize the ovum. Her blood was low in manganese and molybdenum and high in copper and iron. Her hair analysis showed low manganese, magnesium, and chromium, but she was not hypoglycemic by the blood spermine test. As with many compulsive patients, she has micrographia. She had severe insomnia partially induced by the 30 mgm per day of Deseryl needed to temper her depression. On the standard treatment program for histadelia, she is now much better and, furthermore, she understands the biochemical nature of her illness.

OTHER PEPTIDES AND THE SCHIZOPHRENIAS

In addition to the endogenous opioids, other peptides have been implicated in the etiology or therapy of the schizophrenias. Two such peptides, substance P and cholecystokinin and its derivatives have been studied due to their effects on dopaminergic pathways as possibly related to schizophrenic symptoms.

The "dopamine hypothesis" attributes the psychiatric symptomatology of the schizophrenics to an impaired regulation of the neurotransmitter dopamine. Dopamine is concentrated within the basal ganglia and limbic system of the brain, thus centrally acting with other neurohumors to coordinate body movement, memory, emotions, and learning. An overreactive dopaminergic system has been implicated in schizophrenia; however, despite numerous studies, evidence remains intangible. L-dopa, the precursor of dopamine used in the treatment of Parkinson's disease, has been found to exacerbate symptoms in schizophrenics. Some Parkinson's disease patients actually develop schizophrenia-like dysfunctions if L-dopa is used in excess.

Originally identified as the neurotransmitter involved in the conduction of pain, subsequent studies have delineated a greatly expanded role for substance P in the central nervous system. High concentrations of substance P have been localized in the basal ganglia, hypothalamus, substantia nigra, and the central grey region of the brain. The distribution of substance P is markedly similar to that of dopamine suggesting interactions. Substance P plays a facilitory role in dopaminergic pathways, stimulating release of the dopamine neurotransmitter. Studies of schizophrenia patients have noted significantly elevated levels of substance P fragments in the cerebrospinal fluid

implying an overabundance of this neuro-transmitter in the brain. Excess production and release of substance P would lead to an overabundance of dopamine resulting perhaps in schizophrenic difficulties.

Cholecystokinin (CCK) was originally thought to be simply a digestive hormone stimulating the release of pancreatic enzymes and the contraction of the gall bladder. Recent research, however, has found CCK receptors to be widely distributed throughout the brain with particular enrichment in the limbic and cortical areas. Cholecystokinin is antagonistic to the release of dopamine from limbic neurons and, thus, may serve as a regulatory system controlling hyperfunctioning dopaminergic neurons. Consistent with the dopamine hypothesis, postmortem studies of schizophrenics have found depressed levels of CCK both in cortical and subcortical regions. Treatment of schizophrenics with cholecystokinin and its derivatives, ceruletide and caerulein has ameliorated symptoms in some, while others remained unresponsive. The methodology of these studies has since been severely criticized.

Although evidence for the involvement of dopamine, substance P, and CCK remains inconclusive, then numerous discoveries in the physiology and pharmacological production of peptides seems to suggest expanded research and therapeutic use of such agents in not only schizophrenia, but all chronic disorders of the body.

MONOAMINE OXIDASE AND PARANOIA

Monoamine oxidase (MAO) is a key copper-containing enzyme involved in the degradation of the biogenic amines, most notably noradrenalin or norepinephrine. Since the early seventies, numerous studies have concentrated on correlations between monoamine oxidase activity and the schizophrenias. MAO, commonly measured in blood platelets, varies to some degree in the human population and such deviations seem to be largely genetic in origin. Studies of monozygotic (identical) twins show striking similarities between monoamine oxidase activities in the two offspring. Murphy and Wyatt in 1972 first documented significantly reduced platelet MAO in chronic schizophrenics as compared to normals. Chronic schizophrenics were shown to have only 41% of the monoamine oxidase activity of normals. Subsequent studies have further differentiated this low MAO activity in the mentally ill with the most distinct difference being found in the chronic paranoid schizophrenic. Additionally, researchers have found decreased MAO activity in hallucinating schizophrenics but increased MAO in schizoaffectives. Much controversy still surrounds these latter two findings. However, of more than thirty studies completed, only six have failed to find significant reductions in MAO activity in chronic paranoid schizophrenics. This is, therefore, a real key to paranoia.

Fischer et al. (1960, 1972) and Potkin et al. (1979) have postulated phenylethylamine as the ultimate cause of the paranoid psychosis. Phenylethylamine is the ultimate cause of the paranoid psychosis. Phenylethylamine is a natural substrate for the enzyme monoamine oxidase. Therefore, a reduction in MAO would logically result in a buildup of phenylethylamine which is structurally similar

to amphetamine. The "phenylethylamine theory" states that phenylethylamine produces an amphetamine-like psychosis. Amphetamine psychoses are frequently characterized by paranoid delusions. Three of five studies have documented increased urinary excretion of phenylethylamine in diagnosed schizophrenics. However, chronic paranoids were not differentiated from the general schizophrenic population.

A second theory centers on the overstimulation of the brain due to the depression of monoamine oxidase's degradative activity on the catecholamines. Farley et al. (1978) noted significantly elevated levels of norepinephrine in the limbic system while performing post mortem studies on paranoid schizophrenics. An unusually high concentration of norepinephrine was localized in the ventral, septum, stria terminalis, nucleus accumbens, and mammillary bodies. The limbic system has been consistently implicated in the etiology of schizophrenia due to its role in learning and emotions and such findings further accent the limbic system and its MAO content as factors in paranoia.

As with any enzyme, a number of physiological factors will alter levels of MAO. Alcoholics have consistently lower platelet MAO; however, the behavioral repercussions of this decrease are unknown. Robinson et al. (1971) have noted a gradual increase in platelet MAO activity with age. This may explain why paranoid schizophrenics slowly improve with age without any effective treatment. Females have less paranoia and have consistently higher MAO platelet levels than males. Progesterone may cause this as it appears to increase MAO activity; however, estrogens and androgens may be MAO inhibitors. Adrenalin increases platelet MAO activity. Iron deficiency anemia is associated with a decline in MAO activity which is reversed with iron administration. Pharmacological agents inhibit MAO activity,

specifically antihypertension and antidepressant medications. Certainly, the aforementioned factors may cause an imbalance in monoamine oxidase levels and production; whether they alone may precipitate a psychotic episode remains unknown.

Testing for monoamine oxidase levels previously involved the assaying of platelets found in the blood. The process is rather involved and, thus, its usefulness as a clinical test is questionable. Recently, however, Sullivan (1980) discovered that low monoamine oxidase levels strongly correlated to elevated urinary excretion of tryptamine. As previously discussed, others have also found elevated excretion of phenylethylamine. Thus, it appears that a simple urine test may soon be devised as an indicator of MAO activity and, hopefully, paranoia.

We can conclude that a definite link exists between low platelet monoamine oxidase and chronic paranoid schizophrenia. This may be owing to the high copper level in most paranoids. Although the details of this relationship remain speculative at this time, the reduction in monoamine oxidase activity probably represents a predisposition or sensitivity to paranoid schizophrenia. Other stresses combine with this genetic predisposition resulting in the manifestation of paranoid symptoms.

MANY SCHIZOPHRENICS DESERVE A THERAPEUTIC TRIAL OF THYROID

In the course of a hypothyroid psychosis, a young man committed murder. He was later judged to be not guilty by reason of insanity, although he was clearly sane at the time of his trial.

Thyroid replacement alone restored the subject to normal physical and emotional health...the formerly psychotic subthyroid person (became) emotionally competent again with thyroid maintenance...he was then judged competent to stand trial.

—Easson, 1980

The normal thyroid (a ductless gland located at the base of the neck around the Adam's apple) manufactures thyroxine (T4) out of iodine and the amino acid tyrosine and secretes the hormone thyroxine into the blood. Through the actions of thyroxine, thyroid function determines growth and protein synthesis, controls body temperature, regulates the metabolism or the burning of food in the body and influences, to a great extent, mental and emotional balance. Because thyroxine levels are readily available, thyroid function is accurately measured according to blood thyroxine levels. The range for normal thyroxine is between 5.5 and 11.5 mcg/dL.

One of the first studies on thyroid and the schizophrenias was done by Hudson Hoagland and Gregory Pincus on patients at Worcester State Hospital in Massachusetts. In the general use of thyroid in patients who presumably had normal thyroid activity, they found that 1 in 200 patients became entirely normal in behavior on thyroid alone. This is only 1/2 of 1%, but if you are that patient, then the relief is complete and most welcome.

Infants whose thyroid glands are atrophied at birth

(iodine deficiency) are said to have "cretinism" and are characteristically mentally defective, lethargic and dull, with scanty hair and pasty, thick skin. Many do not survive to adulthood. The well-recognized clinical syndrome of cretinism testifies to the dependency of the central nervous system on thyroid hormone for differentiation and organization. Thyroid function can become decreased as a side effect of medication. Patients on lithium often develop low thyroid function and need thyroid. Lithium has a greater affinity for iodine than sodium, so the iodine goes out in the urine as lithium iodide.

Myxedema (low thyroid function) is also found in the adult and is often presented with mental dysfunction and other symptoms which parallel childhood cretinism. In 1949, Asher highlighted the association between myxedema and psychiatric abnormalities by creating the neologism, "myxedematous madness." In his clinical article, Asher points out that "Myxedema is one of the most frequently missed causes of organic psychoses." Unlike the patient mentioned above, most psychotic schizophrenics are not dangerous. However, if left untreated, scary hallucinations combined with extreme paranoia can carry a severely ill patient through criminal actions.

In the case quoted above, the young man became severely (subthroid) psychotic after having two-thirds of his thyroid removed surgically because of hyperthyroidism (excessive thyroid function). Fortunately, violent cases are not common. Unfortunately, many people who have never had thyroid surgery suffer schizophrenic symptoms due to low thyroid function. Mrs. N., a 48-year-old "hypothyroid" married mother of two was diagnosed as schizo-affective with paranoid delusions and "suicidal preoccupations." On the evening prior to her admission to a psychiatric hospital, she was found by her family to be "confused and talking funny" (Granet and Kalman, Cornell Medical Center, New York). Doctors found her

acute psychosis to be a symptom of myxedema.

Ever since the first descriptions of myxedema by Gull (1874) and Ord (1878), emotional symptoms and especially paranoia have been repeatedly included in low thyroid function syndrome descriptions (Asher, 1949; Easson, 1966; Whybrow et al, 1976; Davidoff et al, 1977). The 1888 report on myxedema by the Clinical Society of London sums the psychiatric manifestations of myxedema as follows:

Delusions and hallucinations occur in nearly half the cases, mainly where the disease is advanced. Insanity, as a complication, is noted in about the same proportion...It takes the form of acute or of chronic mania, dementia or melancholia, with a marked predominance of suspicion and self-accusation.

Effective treatment for myxedema was first described by George Murray in 1891. With sheep thyroid extract, which could bring thyroid function within normal levels, dimwittedness turned to laughter, slow thinking became quickened, hearts speeded up, and body swelling (edema) and associated heavy facial wrinkles gave way to firm muscles. At long last, a cure seemed to be found for senile dementia. Although the thyroid substance gained much attention as a miracle anti-aging "drug," it soon became apparent that thyroid therapy only brought "youth" to those who were initially thyroid deficient.

For anyone with clear signs of hypothyroidism including hypersensitivity to cold with cold hands and feet; chronic depression and overall lack of energy; dry, thick skin; abnormal hair loss; chronic constipation; inability to lose weight even on a strict diet; excessive drowsiness and slowness to thinking; thick and brittle nails; or overall body swelling, the treatment of choice is still desiccated thyroid therapy, starting with one grain and increasing

toward four grains until symptoms subside. Because a slight degree of hypothyroidism can easily go unnoticed, a therapeutic trial of thyroid is recommended even when tested thyroid levels (T4) appear to be normal.

Sometimes it helps to record the body's basal temperature (thermometer in the armpit first thing in the morning before getting out of bed). If the basal body temperature is low (97.8 is average, 96.3 is a typical low value), then a therapeutic trial of thyroid should be administered. By the time the basal body temperature has risen .4 degrees, one can assume that a significant thyroid effect has been achieved (Atkins).

We repeat, a few schizophrenic patients (about 1/200) who actually do have normal thyroid function will find their mental symptoms relieved with subsequent thyroid treatment. Although 1 in 200 may not seem like a worthwhile percentage, to the few who can be helped by thyroid therapy, a therapeutic trial will prove invaluable. Because adjunct thyroid can be dangerous to patients with already high thyroid function, only schizophrenic patients with a normal to low level of T4 (below 8 mcg/dL) truly deserve a therapeutic trial of thyroid.

Psychiatric manifestations of hyperthyroidism (which can, among other ways, be induced by oral intake of too much thyroid) result in hypomania, exhilaration or euphoria in some persons; however, a more common manifestation are symptoms that mimic psychiatric disease. Anxiety neurosis, delirium, manic behavior, paranoid states, and schizophrenia are all diagnosed with underlying hyperthyroidism. Motor signs, such as tremulousness or hyperactivity can yield a state of exhaustion and pronounced fatigue with accompanying emotional lability, heightened emotional and physical responses to seemingly trivial anxieties, irritability, and insomnia (Swanson et al, 1981). In less energetic and older patients, apathy and depression may replace nervousness (McGee

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et al, 1959). Clearly those who already have high blood thyroxine levels should avoid additional thyroid intake.

SERINE ABNORMALITIES AND THE HYPERMETHYLATION & HYPERDOPAMINERGIC THEORIES

In searching for clues to the etiologies of the schizophrenias, some researchers have focused in on abnormalities in plasma amino acids. Although no correlations were found for the majority of the twenty-two amino acids, patients suffering from psychosis did have significantly elevated plasma serine and depressed plasma glycine. One would expect an imbalance in these instrumental amino acids to have serious implications upon neuronal function as well as on behavior, because serine and glycine are crucial to both nerves and the brain. Serine, via its metabolites phosphatidylserine and phosphatidylethanolamine, plays an important role in the fluidity of nerve membranes, while glycine is the major supplier of one carbon units of methyl groups and an important inhibitory neurotransmitter to the brain.

To completely understand the data compiled by researchers, one must first understand the relationship between serine and glycine. If glycine itself is not present in sufficient quantities nutritionally, serine will be converted into the deficient amino acid. In the presence of tetrahydrofolate (THF), the enzyme serine hydroxymethyltransferase (SHMT) can convert serine into glycine and N⁵, N¹⁰ methylene tetrahydrofolic acid.

Pepplinkhuizen and his associates (1980) were the first to note abnormal serine/glycine metabolism in psychotic patients. After loading psychotic patients with serine, Pepplinkhuizen noted that a strangely low excretion of serine was followed five hours later by an exacerbation of psychotic symptoms. In a subsequent study, Waziri (1983) found significantly higher plasma serine levels in psychotics when compared to levels in

nonpsychotics. In a study the following year, Waziri and his coworkers (1984) reiterated the earlier findings on elevated serine, this time expressing the data as an elevated serine/cysteine ratio. Yania et al. (1986) noted elevated serine in studies of cerebrospinal fluid. Recovered psychotics were found to have elevated plasma serine levels despite the remission of psychotic symptoms (Waziri, 1974). This same report also documented a decreased activity of the enzyme serine hydroxymethyltransferase.

Two contradictory theories have been forwarded, each attempting to explain the role of serine-glycine metabolism in psychotic symptom development. The first is merely an extension of the overmethylation theory first proposed by Osmond and Smithies (1952). Poppelinkhuizen (1980) hypothesized that the decreased excretion of serine in psychotics resulted from the increased conversion of serine to glycine before it has a chance to leave the body. This results in a concurrent increased production of N⁵, methylene tetrahydrofolic acid which ultimately produces more S-adenosyl methionine (SAM), a strong methylating agent which increases the methylation of various neuroactive compounds. Because methylation increases activity, the final outcome of the increased conversion of serine to glycine, is greatly increased neuromodulator activity in specific neural pathways.

It is this overstimulation of neural pathways which is thought to result in psychotic symptoms. Although logical, this theory cannot explain the finding that serine hydroxymethyltransferase (SHMT) activity is reduced in psychotic patients. If glycine and methylene THF are being overproduced, then the activity of SHMT should instead be elevated.

Baldessarini (1978), in his critical review of clinical and laboratory studies, found no support for the

hypermethylation theory. If the hypermethylation theories were accurate, then supplementation leading to high levels of SAM would result in psychotic symptoms. Such supplementation, however, does not produce psychotic symptoms.

The hyperdopaminergic theory of serine psychosis was proposed by Levi and Waxman in 1975. This theory is supported by the findings of reduced enzyme SHMT activity and presumes a depressed conversion of serine to glycine. With impaired serine to glycine conversion, less glycine is produced, which leads to less SAM production, because SAM, among other things, inactivates dopamine at normal synapses, and the final outcome of the decreased conversion of serine to glycine is increased activity of dopaminergic neurons. This hyperdopaminergic state is hypothesized to be responsible for psychotic breaks (as previously discussed in "Other Peptides and the Schizophrenics").

Thus, these two theories are polarized with respect to the amount of serine to glycine conversion in psychotic patients. The hypermethylation theory is founded upon the increased conversion of serine to glycine, while the hyperdopaminergic theory is based upon the reduced activity of SHMT and thereby a reduced conversion of serine to glycine. Although the latter is more highly supported by the enzymatic studies documenting decreased SHMT activity, a single study is far from conclusive. Until more data are presented, both theories must be pondered. Regardless of which metabolic theory proves more accurate, serine/glycine ratios would be of greater interest if they pointed to a more effective treatment for any of the schizophrenias.

CALCIUM, MAGNESIUM AND MENTAL ILLNESS

Just as scientists have probed the amino acids looking for clues to the cause of mental illness, so too have the major elements been investigated. Two such elements, calcium and magnesium, have been implicated as playing a role in schizophrenia, and since these cations move concurrently in the body, they should be considered together. Both are fairly ubiquitous in the human body, and for reasons of brevity only their neurological effects will be reported. Magnesium is the most plentiful cation in intracellular fluids and, thus, is instrumental in the establishment and potentiation of osmotic balance.

Magnesium Relaxes; Calcium Stimulates

Magnesium, antagonistic to calcium, inhibits synaptic nervous transmission by depressing neurotransmitter release. Calcium is the primary cation in the transmission of nervous signals throughout the body. Calcium influx across the nervous membrane leads to the release of neurotransmitters and the potentiation of the action potential across the synapse. Elevated extracellular calcium concentrations enhance neurotransmitter release. Calcium and magnesium also play similar roles in muscle contraction, calcium as a stimulant and magnesium as an inhibitor.

There are two body compartments which form the environment of the nervous system. Centrally, the brain and the spinal cord are bathed by the rigidly-controlled cerebrospinal fluid (CSF), while, peripherally, the nerves are permeated by serum. Between these two fluid pools lies the blood-brain barrier selectively allowing substances to cross into the cerebrospinal fluid and, thus,

provide a stable environment for mental and behavioral functioning. Changes in serum calcium and magnesium usually produce concurrent, but less severe changes in CSF calcium and magnesium. Carman and Wyatt (1979) reported data that suggest a fundamental difference in the blood brain barrier of psychotics. Shifts in serum calcium produced a reduced shift in the opposite direction in cerebrospinal fluid. Far from conclusive, this may identify a small subset of psychotic patients who have abnormal calcium homeostasis due to blood brain barrier dysfunctions.

High Calcium = Psychosis

More direct and fruitful research has surrounded the identification of psychotic symptoms associated with abnormal levels of calcium and magnesium and subsequent methods of treatment. The most clearly defined relationships exist with calcium, although even these studies are far from conclusive. Depressed levels of calcium are correlated with depression, while elevated levels of calcium are correlated with agitation, mania, and psychosis (Carmen and Wyatt, 1979). These relationships appear logical since calcium is essential to the production and secretion of neurotransmitters. A deficiency in calcium would result in sharply-reduced activity in the neuronal pathways and subsequent depression. To the contrary, elevated calcium would amplify nerve impulse to the point where organized patterns would deteriorate. Harris and Beuchemin (1956) noted elevated CSF calcium in psychotic patients as compared to nonpsychotics. Additional support for such imbalances is formed by manipulation of the calcium hormones, namely calcitonin and parathyroid hormone. Produced by the parafollicular cells of the thyroid, calcitonin decreases the circulating levels of calcium, and patients supplemented with

calcitonin have noted significant exacerbation of depression (Carman and Wyatt, 1979).

Parathyroid hormone, produced by the parathyroid glands, antagonizes the effects of calcitonin by mobilizing the calcium in the bone, thus increasing serum levels. Patients suffering from hypoparathyroidism (decreased secretion of the parathyroids) were noted by Snowden and colleagues (1976) to manifest paranoid psychosis with decreased serum calcium levels.

Excess Magnesium = Confusion

Magnesium paints a slightly more clouded picture, possibly due to its less permeating role in nervous modulation. Hypo-magnesia has been associated with convulsions, tremors, ataxia, nystagmus, fasciculations, paresthiasias, tetany and EEG changes. Psychiatric disturbances include depression, agitation, disorientation, confusion, hallucinations, and irritability. Hypomagnesia is consistently found in conjunction with hypocalcemia, accentuating the concurrent movements of these two cations in the body. Hypomagnesia has been noted secondarily in patients receiving mercurial diuretics or ammonium chloride, primary aldosteronism, tetany, diabetes, hyperparathyroidism, hypoparathyroidism, porphyria and inappropriate ADH secretion. Both calcium and magnesium imbalances have been linked to alcoholism, possibly explaining the etiology of alcohol's association with mental dysfunctions (Ananth and Yassa, 1979).

The extensive use of parenteral magnesium in patients without psychotic reactions would argue against any direct action of magnesium, i.e. 10 cc of 20% of Mag. SO₄ given intravenously in eclampsia. Also, Mg Cl₂ has been used as a test of circulation time without any side effect of psychosis.

We know from hair analysis of the patients with the

schizophrenias that those who are hypoglycemic have very high calcium and magnesium levels and low zinc, manganese and chromium levels. The trace metals needed for processing sugar are zinc, manganese and chromium but when these are deficient we believe that the more abundant calcium and magnesium are mobilized for sugar transport and utilization. Hence, this explains the very high levels of calcium and magnesium in the hair of those patients who are in the end-stage of trace element deficiency. Those who have studied all the trace elements liken the situation to that of a spider web with each radius represented by a trace element. A tug on one radius trace element will distort the others.

The actions of many commonly-used psychiatric drugs may influence this Ca/Mg ratio. Neuroleptics have been shown to decrease both calcium and magnesium levels in humans (Athanasena et al, 1983), possibly explaining their effect on nervous activity. The phenothiazines inhibit calmodulin activity and thus decrease circulating calcium levels. Lithium has also been shown to reduce circulating calcium levels (Dubovsky and Franks, 1983). Another study has documented an unexplained rise in magnesium following lithium administration (Christiansen et al, 1976). Other non-drug treatments appear to have similar effects on the effective circulating levels of calcium. Sleep deprivation, used experimentally in the treatment of depression, was reported by Bojanovsky and colleagues (1974) to increase serum levels of Ca, thus ameliorating depression. Electroconvulsive therapy was shown (Carman, 1977) to alter calcium metabolism and, thus, may explain its usefulness in behavioral abnormalities.

Calcium Blockers May Help

The accumulation of knowledge in the area of Ca

and Mg has led to the proposal of new and novel treatment for psychiatric difficulties. Dr. Jay Goldstein (1984) reports improvement in neuroleptic-resistant schizophrenia when a calcium channel blocker is added to the treatment regime. Although a double-blind study is lacking at this date, certainly this could signify a new treatment approach to some of the schizophrenias. This remains an avenue to be explored as we struggle to explain and treat the resistant schizophrenias.

**IMMUNOGENETICS OF THE
SCHIZOPHRENIAS**

UNFORTUNATELY THE SCHIZOPHRENIAS ARE INHERITED

The experience of any of the schizophrenias, either directly as a sufferer or indirectly as a friend or relative, is dramatic enough to send anyone searching for a cause. The parents of a schizophrenic often are the first to notice the change in personality and usually are the ones who feel the greatest need for an explanation. Out of desperation, some parents come to believe certain events cause the illness—an automobile crash, the death of a parent, or a move to a new town. Many parents blame themselves.

Theories tracing the onset of some schizophrenias to parental upbringing patterns were once common, but are no longer given much credence or validity. The old concept that “schizophrenogenic mothers” cause the disease by their overly-protective, or hostile and cold attitude toward the child has largely been abandoned, along with other notions that pathological family interactions are at fault. As the late Dr. Frances Cheek of the New Jersey Neuropsychiatric Institute, in a very exhaustive family study, concluded, family reaction to the patient is more likely to be the result, rather than the cause, of the illness. Although families with a high incidence of the schizophrenias do have different rules of family interaction, it now seems more correct to speak of “skewed parental relationships” and “schism” type conflicts simply as coping problems. Of course, families with schizophrenic members act differently from other families; they are different!

It even makes sense that parents treat their schizophrenic children differently. Schizophrenics are different and can be very difficult. The extent to which the differences and difficulties can or should be overridden by sympathetic understanding of the illness is limited. Mothers necessarily find it extremely taxing to express only

tender feelings to a child who continually rebuffs, ignores, and threatens. Untreated schizophrenics often have no love for others in any mature sense and, correspondingly, are often not very loveable. This fact alone may seriously threaten a woman's image of herself as a loving mother. Throw in the emotional storms, indifference, negativism, lack of perception, and monstrous or monotonous behaviors, and the result is even more intra-family distress. The inescapable adverse emotional reactions of schizophrenics understandably provoke ambivalence and even outright hostility; much of the discord found in schizophrenic families may be linked directly to the patient's symptomatic acts.

Patients, relatives, friends and therapists who accuse and blame parents of schizophrenics for causing the disease only add insult to injury. Those who understand that schizophrenics can be very frustrating help to reduce the anxiety in the family and allow the remaining tenderness and compassion to surface. Support also reduces resentment. It can help a very troubled mother open up to her child.

Typically, when one child in a family has schizophrenia a number of other children, raised by the same parental child-rearing methods, show no sign of illness. Studies of these normal children in schizophrenic families were the first to show that parenting techniques were clearly not directly responsible for the development of any of the schizophrenias. Although many felt relieved of their feelings of guilt, additional anxiety was triggered when other studies proposed a genetic link. Parents who are aware of the genetic theory now fear the appearance of the disease in future children and grandchildren.

To these people we offer the following chart to be used as a guide. It shows how often the disease is found in the relatives of schizophrenics. It should be noted that the information relates to populations and not to

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individuals. The information is compiled on undifferentiated schizophrenia—as behaviorally defined. The numbers, therefore, have no true predictive value when applied to individuals with accurately diagnosed biotypes of the schizophrenias.

<u>Familial risk</u>	<u>Lifetime prevalence</u>
No relation	1%
Uncles, aunts, etc.	3%
One parent	6%
Both parents	39%
Children	13%
Twins	
Identical	50%
Non-identical	10-13%
Siblings	10%
Half-siblings	4%

The figure shows that for second-degree relatives (aunts, uncles, nieces, nephews, grandparents, grandchildren, and half-brothers or sisters), the risk is three times greater than for the general public (1% risk in general public, up to three percent for second-degree relatives). Children with one schizophrenic parent have about a six percent chance (others say it's more like ten to fifteen percent) of having the disease at some point in their lives; whereas, those with two schizophrenic parents have an increased 39 percent chance. The chance that brothers and sisters of a schizophrenic either already have it or will get it is about 10%. The highest risk, as the genetic theory suggests, is found among identical twins. In half of the identical twins of schizophrenics both twins have the disease!

Even though the family studies did not necessitate a genetic theory, the studies of identical twins are conclusive. For fifty percent of the identical twins of schizophrenics to also have the disease, there must be a role for

genes to play. Dr. Kidd cites the very same twin studies, however, to remind us that whatever the genetic factor is, it cannot tell the whole story of schizophrenia. The observation that half the identical twins of schizophrenics do not have the disease on its own is "proof that genes are not sufficient for the development of schizophrenia. Nongenetic factors—copper intoxication, zinc deficiency, viruses, prenatal health, and many other factors—affect the way the brain operates" (Science 82, August 1982, p. 90).

Other twin studies support the notion that nongenetic forms of schizophrenia may exist alongside genetic forms. Reveley and colleagues (1984) found that only twins without a family history of schizophrenia had enlarged brain ventricles—a common finding in some schizophrenics. They concluded that:

Some common environmental factor, possibly perinatal damage, may have led to the increase in ventricular size in schizophrenic-discordant pairs, with schizophrenia developing in the more severely affected twin.

And so most scientists have come to focus on possible biological or medical models as causes of schizophrenia. Since the times of the "schizophrenogenic mother" model, the goal of most research has been to link behavior and biology, to discover easy-to-manage, predictable and consistent biological markers that will permit earlier diagnosis, more selective treatment, and accurate prediction of the outcome of treatment (Science 82, August 82, p. 90). We have an edge over most of the research presented here because we understand that there can be many independent causes of schizophrenia. After a clearly familial variety has been identified, individual genetic studies should prove quite fruitful in their search for why some of the schizophrenias run in families.

THE INHERITED SCHIZOPHRENIAS

In at least some cases, genetic causes cannot be reasonably disputed. Much doubt, however, remains about the extent of the genetic component and whether or not organic or environmental explanations are also needed. In the cases where genes are clearly involved, some basic factor not sufficient for the development of one of the schizophrenias is probably transmitted. What is inherited is probably some sort of predisposition or vulnerability to a schizophrenia and not the disease itself. Although it remains possible that some types of schizophrenia are actually carried on a dominant "disease gene," further studies focusing on the schizophrenic families that show a particularly high prevalence of the disease will have to be conducted before accurate conclusions can be drawn. We need more studies that look for similarities among similar, biologically defined subgroups. Studies which lump all schizophrenics together into "simplistic schizophrenia" as if all shared a common cause, are not capable of yielding the desired informative results.

In Biochemical Individuality, Dr. Roger J. Williams considers the many "sub-clinical" genetic variations in the population. With significant variation imposed with every generation, it seems logical to assume that each of us is unique not only in our outward physical features, but also in our inner metabolic, biochemical and anatomical "features." These differences have not been highlighted in the past and, therefore, have not seemed important to the clinical setting. However, such subtle differences, though not enough to cause spontaneous abortion of a fetus, can lead to significant, otherwise unexplainable clinical problems. Dr. Williams makes such a case for the schizophrenic when he asks "If individuals differ at birth in the susceptibility to schizophrenia, for example, of what does

their individuality consist? . . .

“Aside from anatomical differences with respect to brain and endocrine glands especially, there is the possibility that they differ in their nutritional requirements. One may have need, in order to meet the stresses of life and keep his brain metabolism functioning, a larger amount of a certain crucial nutrient than the other. Lacking these nutrients, his brain metabolism gets out of joint and mental disease results.”

We at the Brain Bio Center have long assumed that there are many disorders that can surface as schizophrenia. In our observations of over 10,000 so-called schizophrenics, we have found that fully one-third have a specific nutritionally-based inherited metabolic disorder. They have kryptopyrrole in their urine. These “pyroluric” patients are genetically prone to zinc, manganese and B-6 deficiency. They lose excess zinc and B-6 in their urine. In these pyroluric families, most males are miscarried or stillborn. It seems that the male fetus needs more zinc for proper development than the female fetus. The mother’s deficiency, or his own inherited deficiency or increased need, is sufficient enough to preclude normal development. The males that are born have birth defects. The all-girl pyroluric families go on to produce more defective males. In some kindreds, normal males did not occur in four consecutive generations! Studies are needed on this subgroup of schizophrenics to learn more about the genetic factors involved.

In contrast to these all-girl families, Brain Bio Center studies show that the high histamine (allergic) schizophrenic woman is apt to conceive only males. Although this fact may be owing to her characteristically thinner vaginal secretions which give the male sperm an advantage in reaching the ovum, allergies are certainly familial.

The genetics of the high histamine disorder presently included under the schizophrenic label also needs to be studied.

Although studies have found some correlations between schizophrenia and celiac disease, most were dismissed because of the low number of cases. While it remains true that celiac disease is not the cause of all cases of schizophrenia, wheat gluten enteropathy, which is similar to celiac disease, can lead to schizophrenic states. The few cases of schizophrenia in the wheat gluten population were likely symptomatic as a direct result of the sensitivity to wheat. This fact was overlooked as the researchers sought an all-encompassing cause. Wheat gluten enteropathy is certainly familial and is probably the disease responsible for 5 to 10% of all patients presently labeled schizophrenic. Studies should be done with these patients to see why the schizophrenic symptoms surface. Genetic studies on this subgroup are also long overdue.

Geneticists have often wondered why schizophrenia has remained in the population for so long and at such a high frequency given that schizophrenics are often suicidal or emotionally incapable of reproducing. Sir Julian Huxley, Professor Earnst Mayr and Drs. Humphrey Osmond and Abram Hoffer suggest that the high prevalence of schizophrenia may at least in part lie with certain advantages in life which accrue to patients with some types of schizophrenia. Histapenic (high copper) schizophrenics (low histamine level) have an increased resistance to shock. Their low level of histamine in the body keeps them free of allergies and resistant to viral infections. In addition to better survival, the females of this type of schizophrenia may prove to be more fertile. Although much of this is speculative, it is suggestive of possible research frontiers.

In conclusion, then, it seems fair to say that genetic studies lumping all schizophrenics together are now

obsolete and will soon be superceded by genetic studies on specific biotypes of schizophrenia. Not only will such studies show familial disorders of a metabolic or anatomical nature, but, as Roger Williams suggests, individuals alone may have such disorders resulting from the mutation and random variation that naturally occurs in the genetic shuffle between generations. The facts of biochemical genetics and biochemical individuality make inescapable the conclusion that each individual must be distinctive in his responses to stresses of all kinds and that vulnerability to mental disease must vary from individual to individual.

Once it is accepted that a variety of disorders can independently lead to the same schizophrenic state, many otherwise unaccountable facts found through research should prove illuminating. Research will soon turn its focus from searching for a narrow, single cause to identifying all the biological disorders that can result in schizophrenic symptoms.

SOME OF THE SCHIZOPHRENIAS ARE INFECTIOUS

It has long been known that an infection by the Spirochete *Treponema Pallidum* leads to syphilis, and that one patient in about 200 who contracts syphilis suffers from brain infection with symptoms which can closely resemble schizophrenia. Samuel Henry Kraines offers the best example of this in his book, The Therapy of the Neuroses and Psychoses, where he explains that the symptoms of paresis (brain infected by syphilis) are varied, so that almost any type of mental disease is simulated: 1) simple dementia, 2) manic or grandiose states, 3) simple depressive states, 4) agitated depressive states, 5) schizophrenic states, 6) psychopathic personality states, and 7) psychoneurotic states. These symptoms result from the fact that various parts of the brain are damaged and not from the kind of etiologic agent.

Other infectious diseases such as pneumonia, typhoid fever, and erysipelas, may also lead to altered mental states. (The temperature rise itself alters brain metabolism by affecting various enzyme systems differently.) In addition, toxic substances produced by infective agents may directly affect brain metabolic function. Schizophrenia-like symptoms surfaced in association with the influenza epidemic of 1918 (Menninger, 1926) and other viral infections can affect the brain like herpes virus, and encephalitis lethargica (Crow, 1983). As far back as 1928, it had been noted that encephalitis and schizophrenia are different diseases, but it seems that some factor common to both is sometimes present and accounts for the coincidence of identical symptoms.

Current infectious disease theories of some schizophrenia claim that "the factor common to both" (schizophrenia and encephalitis) is a virus. The rabies virus and

herpes zoster virus attack very localized areas of the brain, specifically the limbic system (see chapter on the Limbic System). They can alter the function of a cell by deforming the structure, and thereby, the action of some enzyme. This approach often leaves no visible sign of their destruction. Compatible with a viral etiology is both a tendency toward a seasonal onset distribution and a season-of-birth effect. Although there has never been hard evidence suggesting that any schizophrenia has been transmitted from one individual to another, virus-like particles have been discovered in the spinal fluid and the nasal secretions of some schizophrenic patients (Marazav, 1954a and b; Scarlato et al, 1956; and Malis, 1959).

Rabies is a virus known to be easily transmitted which can seriously affect the brain and cause victims to "go mad." In some cases, the rabid madness looks quite similar to schizophrenia. Hattwick and Gregg have divided the typical course of clinical signs and symptoms of rabies into five stages, and report the third "acute neurological stage" as "characterized in most, but not all cases, by both behavioral aberrations such as hallucinations, disorientation, and fits of "furious" hyperactive behavior, and finally by paralysis." Although we do not see the paralysis in schizophrenia, it sure seems likely that a virus similar to the rabies virus may be found which leads to at least some cases of schizophrenia. We do know that rabies goes to the brain via the peripheral nerves and that, although the infection first locates where it arrives, it then seems to preferentially infect the limbic area.

In one reported case, a 15-year-old girl who had been bitten by a dog ten years earlier, was found at autopsy to have unequivocal evidence of rabies virus in the hippocampus (another structure in the limbic system associated with schizophrenia). Other reports which focus on the high affinity of rabies virus for the acetyl-choline receptor (Tignor, Smith, 1984) another unrelated receptor

(Reagan, Wunner, 1985), and on the selective impairment of membrane receptors in rabies virus infections may also prove very helpful in search of a virally-induced form of schizophrenia.

The discovery of latent viruses which live in the nervous system opened new areas for viral research (Gajdusek and Gibbs, 1977). Such viruses are believed to play a role in many neurological diseases including herpes simplex encephalitis, Creutzfeldt-Jacob disease, Parkinson's disease and multiple sclerosis (Bower, 1985). Researchers are excited because a latent virus-role in one type of schizophrenia could not only explain the influence on the nervous system, but also the waxing and waning intensity of symptoms, and the altered brain cell function without signs of other brain cell changes. Theory holds that a latent virus whose shape adequately mimics a protective HLA antigen will itself be protected from an immune attack. Since the immune system cannot destroy the virus, it lives in the nervous system throughout the life of the victim.

Because such a "slow-acting" virus can proceed to subtly trigger a number of altered immune states over a long period of time, a person showing their first signs of schizophrenia at twenty or thirty years of age, could have theoretically become infected while still in utero or shortly after birth. Perhaps the exact timing of the original infection is the crucial factor. Certain viral diseases cause brain damage if introduced at one stage of fetal brain development but not at another stage. German Measles (rubella) is well known for its ability to cause mental retardation, heart and other defects if it infects the baby in the first three months of pregnancy. The effect is extremely specific and focuses its destructive measures only on the organs developing at the time of infection. Herpes simplex virus and coxsackie virus can also lead to fetal brain damage.

Some structural changes described in occasional schizophrenic brains at the time of autopsy look very much like virally-induced changes. Alterations in the protein portion of some schizophrenics' spinal fluid are also compatible with a viral infection theory. The strongest support for a latent virus role in schizophrenia came when Crow et al (1976b) and Tyrrell et al (1979) found that the virus-like particles in the spinal fluid of schizophrenic patients had a marked cytopathic effect. Elevated serum or CSF levels of interferon, a substance produced by viral-infected cells, were found in low-dose or not medicated schizophrenic patients with recent onset or recent worsening of psychotic symptoms (Libikova et al, 1979) (Preble and Torrey, 1984, 1985) strongly suggesting a viral involvement. Studies that were unable to confirm the results did not control for drugs (Rimon, 1985) (Vaheri et al, 1985). Moises (1985) believes that drug-free schizophrenic population studies will clarify the conflicting interferon results.

Other studies are searching for a viral role in altered neurotransmitter metabolism (Koos, 1984) and trying new computer tomography techniques (described earlier) to help visualize possible viral effects (Crow, 1983) (Rimon, 1983) (van Kammen and DeLisi, 1984). Masterson (1986) is reporting links between lysine, a known herpes viral antagonist, and schizophrenia. Although many are hopeful, Charles A. Kaufmann of St. Elizabeth's Hospital in Washington, D.C., believes that research attempting to demonstrate a link between viral infections and some schizophrenias "has provided only indirect evidence and is fraught with pitfalls" (Science News, Vol. 128, p. 346, 1985). Perhaps the gaps in reasoning will be filled once it is accepted, that not all schizophrenics must show signs of viral infection for some to have virally-induced disorders.

Strongly against a viral cause of the schizophrenias is the fact that when the biotypes of the schizophrenias are

IMMUNOGENETICS OF THE SCHIZOPHRENIAS

determined and corrected, 90% of the patients are socially rehabilitated with specific nutrient therapy. None of these nutrients are specifically virocidal.

IMMUNOGENETIC RESEARCH IN SCHIZOPHRENIA

The immune system is our security against particular diseases such as the viruses mentioned previously. As a result, any defect in the system, resulting from a mutation or the inheritance of "defective" genes, can seriously increase our overall vulnerability to these diseases. We know that many schizophrenics seem to enjoy a greater resistance to infections of all kinds, while others are particularly susceptible to colds and allergies. Because immune defect mutations are probably common and frequently inherited, much research has focused on the transmission of those factors from generation to generation. This area of research is called immunogenetics.

Immunogenetic studies have significantly contributed to our understanding of many familial diseases which, like schizophrenia, have complex poorly understood causes. Because of these reported successes, researchers have looked to immunogenetics for data which will unlock the complexities of schizophrenia's presumed environmental and inherited transmission.

Human Leukocyte Antigens and Schizophrenia

One of the most interesting developments in medical immunogenetics has been the discovery of strong associations between specific proteins (human leukocyte antigens) found on white blood cells and a wide range of chronic disorders. The human leukocyte antigen (HLA) system participates in the immune process. Following the rules of genetic inheritance, the HLA genes code for certain HLA proteins to appear on the surface of all cells in the body. This process occurs in utero and uniquely labels the fetus's own cells so they can be recognized and

preserved later as the immune system proceeds to destroy foreign substances.

As with other inherited genes, HLA protein configurations are more similar among genetically related individuals. HLA genes are often located close to disease genes. As a result, the two genes are usually inherited together and the easy detection of one (the HLA gene) suggests the presence of the other (the disease gene). The HLA protein is rarely itself involved in the disease process. It is a mere 'marker' for the disease—a 'visible' sign (in this case a protein which one can test for) which is inherited along with the more 'invisible' disease. Using genetic marker techniques, one can detect a genetic predisposition or absence thereof before the disease itself surfaces. HLA markers can also distinguish between genetically different forms of a disease.

Major diseases for which genetic markers have been found include ankylosing spondylitis (HLA-B27 aids diagnosis), juvenile-onset 'insulin-dependent' diabetes (which can be distinguished from adult-onset 'insulin-dependent' diabetes using HLA methods) sickle cell anemia (prenatal diagnosis), Huntington's disease (diagnosis before children are born), and multiple sclerosis. Hoping to learn more about the genetic transmission of schizophrenia, or to aid in the complicated diagnostic procedure, some 20 or 30 studies have searched for HLA markers in schizophrenia. Although associations between schizophrenia and more than the 20 antigens of the HLA system have been reported, there is little concordance in results, either between studies or between behavioral subgroups of patients within a single study. Several groups found consistent results delineating paranoid (HLA-A1) and hebephrenic (HLA-A9) 'subtypes' but a closer look revealed differences in their original diagnostic criteria.

In addition to the HLA-A, -B, -C, and -DR antigens

studies by numerous researchers, Dr. Christina Ruddick of Sweden recently analyzed many other possible genetic markers including complement factors Bf, C3, C4 and C6, and Gc serum groups with subtypes, haptoglobin, transferrin C subtypes, and alpha-1-antitrypsin including M subtypes. After comparing her results to those of others, Dr. Ruddick felt compelled to conclude a 'weak at best' association between HLA types and schizophrenia. Her other genetic marker studies showed no significant results or were so recent she could not confirm them with other data. To date, no conclusive evidence supports an HLA marker association for schizophrenia.

The major problem with these HLA studies is the presumption that all cases of schizophrenia share a common cause. The fact is that many factors can cause schizophrenic symptoms and only some of those factors are likely to have biological correlates as specific as HLA markers. Because HLA markers are found in genetically-transmitted disorders, it might prove fruitful to repeat HLA-type studies using schizophrenic subgroups with demonstrated familial inheritance patterns.

IMMUNE GLOBULINS AND THE SCHIZOPHRENIAS

A variety of studies have searched for other immunologic abnormalities in schizophrenic patients. Those that focused on the overall levels of antibodies by immunoglobulin class (IgG, IgM, IgA) in the blood stream and CSF of schizophrenic patients led nowhere (Durell and Archer, 1976). Some workers found a marked increase in the levels of IgM (Pulkkinen, 1977) while others found a decrease in IgM and no change in IgG or IgA (Bock et al., 1971). The concentrations of all three classes were shown to increase by Amkraut et al. (1973), Domino et al. (1975), and Zarrabi et al. (1979). At the Brain Bio Center, where studies focus on the individual subtypes of schizophrenia, research found pyroluric schizophrenic families to be consistently low in IgA.

Since promising results had been obtained using brain-specific proteins as antigens (Vartanian et al., 1978), the neurospecific protein A-100 was tried in allergy skin tests with schizophrenics. A positive reaction of delayed type seemed to suggest to Jankovic et al. (1980) that not only the humoral half of the immune system (antibodies, immunoglobulins, etc.), but also the cellular half of the immune system (peripheral blood T-lymphocytes) was involved in the pathogenesis of schizophrenia. Kolyaskina (1983) and Prilipko and Liedman (1982b) confirmed the link between schizophrenia and cellular immunity with an extensive systematic study of peripheral blood lymphocytes.

Investigations suggesting that antibodies against the O-antigen on nerve cells and T-lymphocytes may be one of the factors capable of altering the lymphocyte population in schizophrenics were supported by seven international research centers in biological psychiatry as part of

the World Health Organization Collaborative Program. Although these and other studies reported altered levels of T-lymphocytes and B-lymphocytes in schizophrenic patients (Loseva, 1977; Vartanian et al., 1978; Zarrabi et al., 1979; Duorakova et al., 1980; Nyland et al., 1980; Mach et al., 1983), DeLisi et al. (1982) now of NIMH explains that the clinical relevance of such altered immunity studies in schizophrenics must be questioned. Significant changes in immunity are to be expected in a population of hospitalized chronic patients. Long-term hospitalization itself significantly increases exposure to various infective agents and carcinogens, which quite naturally can lead to adaptive changes in the immune system.

COULD SOME SCHIZOPHRENIAS BE AUTOIMMUNE DISEASES?

One particularly exciting area of research in immunogenetics considers the possibility that schizophrenia is an autoimmune disease. A systematic study of schizophrenia as an autoimmune disease was first stimulated when Lehmann-Facijs (1937-39) found antibodies against brain antigens in the blood serum and cerebrospinal fluid of patients with schizophrenia. The immune system was actually attacking the body's own brain cells. In 1967, Dr. Robert Heath of Tulane University found that EEG abnormalities comparable to those of schizophrenia could be produced in rabbits by making the rabbit immunologically sensitive to its own brain. They also found that sheep anti-monkey brain and sheep anti-human brain antibodies could induce EEG changes when injected into monkeys. Heath and his associates (Heath and Krupp, 1967; Heath et al., 1967a, 1967b, 1970; Garey et al., 1974) found the human anti-human brain antibody to be an hallucinogenic protein that attacked the septal region of the brain. They named the protein antibody 'taraxein.'

Although some investigators have found it difficult to repeat the specifics of the Heath et al. experiments (Fauman, 1982), many have helped to establish the association between brain antigens and antibodies in the blood stream and/or CNS of a significant percentage of schizophrenic patients (Kutzetzov and Semenov, 1961 - 21.2%; Yokoyama et al., 1962 - 28.5%; Rubin, 1965 - 25%; Heath and Knapp, 1967 - 85.7%; Baron et al., 1977 - 63%; and Pandey et al., 1981 - 48.1%). The antibodies which occur in schizophrenics at an elevated frequency over that of normals, appear to be directed against the alpha-2 glycoprotein (Vartanian et al., 1978 or Liedeman and Prilipko, 1978), and to correlate with the acuteness of the

disease and the rate and severity of its course (Kolyaskina and Kusher, 1969; Semenov and Glebov, 1969). A report appeared in the Chinese International Medical Tribune (Sept., 1984) indicating that 70% of their schizophrenic patients had serum with antibrain antibodies. No such antibodies were found in any control serum. Again, the results showed positive correlations with length of illness, severity of symptoms, past history of illness and family history of illness.

Dr. Prilipko and his associates propose that the appearance of the antibrain antibodies is likely related to stress secondary to illness. They point out that the well-known specific proteins of the brain tissue (especially protein S-100) which are naturally not tolerated by the immune system, and are meant to be protected from the immune system by the low permeability of the blood-brain barrier, can be released into the blood stream following a stress-induced change in the permeability of the blood-brain barrier.

According to their theory, the emotional pain and stress of illness activates lipid peroxidation (LPO) which then increases the permeability of the barrier cell membranes and allows the brain-specific proteins to leak into the blood stream. The body responds to these intolerable brain proteins by producing antibodies specific to them. After a definite time interval required for production, the immune system sends antibodies to these brain-specific antigens into the blood stream to trigger the autoimmune process. Support came when antibodies to brain antigens were found in the blood sera of stress-exposed animals and when the administration of certain chemicals known to prevent the activation of LPO caused by emotional-pain stress was shown to simultaneously block the autoimmune response (Prilipko et al., 1983).

Research efforts focused on these antibodies are increasing at the National Institute of Mental Health,

where DeLisi, Weber and Pert are sure they saw signals of antibrain antibodies at work. Pert and her co-workers are developing new techniques with which they hope to pinpoint the aberrant antibodies. "There's been so much bad work in this field that scientists are a little depressed about it and don't expect much from new studies," explains Candace B. Pert of NIMH. "But the theory for antibrain antibodies in schizophrenia is more visible than ever. The trick will be to get immunologists to start working with biological psychiatrists so the theory can be more rigorously tested" (Science News, Vol. 128, p. 347, Nov. 30, 1985).

NON-BIOCHEMICAL THEORIES

BODY TYPE AND THE SCHIZOPHRENIAS

The lean, gangling male or asthenic individual has frequently, in the past, been pinpointed as the body type preferred as a nest for the schizophrenic process. In contrast, the fat man was supposed to be lazy, jolly, and sleepy. We now know these generalizations to be in error. This error is apparent in the activity of at least two types of drugs—namely, Thorazine and Reserpine—and will sometimes increase the weight of thin, chronic male schizophrenics without improving their schizophrenia. These are the rare occasions when the antipsychotic drugs work mainly on the body and to a lesser degree on the mind. The drug-induced increase in weight may be so great as to necessitate a reducing diet to control the weight gain.

Compared to the underweight individual, the overweight individual has the advantage of a large mass of fat, which can act as a sponge or "buffer tissue" to absorb and slowly release the fat-soluble drugs and biochemicals which act on the brain. Two examples are the anesthetics, such as ether, alcohol, or pentothal, and the stimulants, such as amphetamines. Obviously, the fat man can drink much more alcohol than the thin man and not get as drunk, and the anesthetist learns to use more anesthetic to allow for saturation of the fat of the patient. The fat man or woman can also take more stimulant whether coffee, "speed," or "Bennie pills" and not get as stimulated, maniacal, or "turned on."

Since we postulate a stimulant from the environment (copper) or from the cells of the body which overstimulate the brain to cause schizophrenia, the brain-stimulant effect is based on the fat-soluble nature of this stimulant and is undoubtedly modified by the amount of body fat available.

Thus, the fat paranoid patient is apt to be a productive insomniac, who can be exceedingly active and may be more hypomanic than paranoid. The same degree of abnormal stimulation, if occurring in the thin individual, results in severe insomnia and, perhaps, hospitalization for paranoia or other schizophrenic symptoms.

Some people, not ordinarily classified as schizophrenic, have learned for themselves, by crash diets, that the thinness does not produce the best mental state for them. Obviously, their fatty tissue acts as a buffer to pick up abnormal stimulants and gives them a more productive and livable personality. The productivity and originality of some of our uncouth, large, overweight, eccentric characters is, thus, positively amazing to their friends, but the secret may really be that a mild schizophrenic stimulant process plus body fat makes them eager, productive, and creative.

SEX AND THE SCHIZOPHRENIC

Sexual disturbances have long been associated with schizophrenia. Although it remains true that the illness often begins with the upsurge of sexuality in puberty, sexual maladjustments are more likely the result than the cause of schizophrenias. Characteristic sexual maladjustments of schizophrenia are excessive masturbation, male impotence, female frigidity and/or promiscuity. Because of their inability to form close relationships, schizophrenics do less dating, are less likely to marry, and have more divorces than the rest of the population.

Dr. Garfield Tourney calls the varied schizophrenic sexual disturbances, often odd or shocking, "disorganized sexuality" because they follow no set pattern. Before the disease is evident, the patient may show confused sexual identity, incestuous problems, compulsive masturbation, sado-masochistic behavior, asceticism, a bizarre preoccupation with sexuality, compensatory hypersexuality, or a belief in self injury through sexual activity. With the onset of psychosis, the patient may suddenly use obscenities, appear nude, or become flagrantly seductive or exhibitionistic. The patient's guilt about his sexual impulses may be projected into aural hallucination, voices telling him "You are bad and dirty," or "You are queer." Conversely, the patient may try to manage his guilt by falsely accusing others of sexual misdeeds. Severe schizophrenics are frequently unable to feel sexual pleasure. The schizophrenic's sexual symptoms, like other disperceptions, may alarm and disturb the patient. Homosexuality is frequently assumed by the public to be a symptom of schizophrenia. Both active and passive homosexuality are more common in paranoid schizophrenics. Male schizophrenics are usually more preoccupied with homosexual aspirations and concerns than female schizophrenics.

Their basic emotional difficulty, however, is not strictly homosexual; feelings of inferiority and failure, power struggles, dependency longings, and desires for nonsexual affection all contribute to their seemingly homosexual concerns. Although homosexuality often occurs with the schizophrenias, the two are probably independent conditions.

Because the sex hormones do not control erotic response, they have minimal effect on sexuality. Instead, effective nutritional or drug therapy is used to make abnormal sexual symptoms disappear.

For the recovering patient who desires normal love and sex interests, supportive counseling should emphasize healthy ways of initiating and building relationships with others. Having missed the experience of dating and other social activities, the recovering schizophrenic often lacks confidence in his ability to make friends and to enter into sexual relationships. Counseling should, of course, include discussion of effective methods of birth control.

MANY SCHIZOPHRENIAS AFFECT THE LIMBIC SYSTEM

The limbic system is a part of the brain which lies deep in the center, and it is composed of contiguous portions of the frontal and temporal lobes. The main structures of the limbic system include the amygdala and the hippocampus which have direct connections to many structures. Not long ago, the limbic system was thought to be merely an ancestral remnant of the primitive sense of smell. We now know, from people like Dr. Paul MacLean, the modern father of the limbic system, that it is "able to correlate every form of internal and external perception." It has "selective, integrative, and unifying functions by which raw experience is harmonized into reality and coherent activity is organized." An abnormality anywhere within the system can throw the whole system off.

A look at a variety of brain diseases shows that the particular symptoms result from the fact that the system is damaged and not from the particular kind of etiological agent. We know that brain tumors located in the limbic system are more likely to produce schizoid symptoms. Cases of encephalitis which produce schizoid symptoms have been found in several studies to involve the limbic system, and epilepsy, when it originates in the limbic area, is more likely to be accompanied by schizoid symptoms.

Some of the strongest evidence linking schizophrenia to the limbic system came from studies by Robert Heath and his coworkers. They found abnormal limbic electrical activity in schizophrenic patients. Three other research groups followed Heath's lead. One group found the abnormal electrical impulses in sixty-one of sixty-two schizophrenic patients, and what's more, the impulses were less frequent as the electrodes were moved further from the limbic area. A second group noticed that the

abnormal electrical activity coincided with bizarre patient behavior.

Many structural changes have been described in the limbic systems of schizophrenics or in neighboring areas directly involved in limbic system activity. According to Cutting, perhaps the most significant findings are gliosis in the mid-brain and changes in the thickness of the corpus callosum. Several investigators have reported glial proliferation (evidence of a previous inflammatory process) in the mid-brain, around the "junctions and the major pathways of the limbic system" (Stevens, 1982). Other studies found abnormalities of the upper brain stem and suggested an increased number of dopamine receptors. In fact, histamine, acetylcholine, dopamine, and norepinephrine neurotransmitter changes are all specifically linked to the limbic system. Torrey theorizes that the anatomical location of the schizophrenias is in the left limbic area and cites many findings which could be explained by such a theory.

Viruses are known to attack very specific areas of the brain while leaving other areas intact. The rabies virus and the herpes zoster virus will attack only one kind of cell in one part of the central nervous system. Studies at the Wistar Institute in Philadelphia seem to suggest that the rabies virus locates in the hippocampus (one of the limbic system structures). We know that the rabies virus can certainly send one mad. Cytomegalovirus is a member of the herpes family of viruses and is known to have an affinity for the limbic system.

It is also within reason that specific dietary deficiencies or successes might selectively affect the limbic system. The highest level of zinc is normally in the mossy fibers of the hippocampus. This zinc connection needs further study. In the meantime, it seems certain that the limbic system is the part of the brain primarily affected by many, if not most, of the schizophrenias.

STRUCTURAL AND FUNCTIONAL DIFFERENCES IN THE BRAINS OF SOME SCHIZOPHRENICS

Various claims that the brains of schizophrenics are different in structure from the brains of nonschizophrenics date back to 1910. Over the years, these claims gradually fell into disrepute because the differences could be found in some schizophrenic brains but not in others. Since schizophrenia is not a single disease, the structural differences could not be found in all cases. Also, a nutritional deficiency which has gone on for 10 to 20 years should leave anatomical footprints.

Both the structure and the function of the brains of schizophrenic patients have been shown to differ from those of nonschizophrenics. Aside from the major structural differences found in the limbic system (see chapter), other well-known findings came from CAT-scans (computerized tomography). These include enlargement of the fluid-carrying ventricles, atrophy (loss of brain substance), and/or abnormalities of the cerebellum. These results have been replicated by many without study of the patient who has schizophrenia for the first time or patients who have periodic schizophrenia.

The functional differences can be even more impressive. The abnormal electrical impulses described previously were centered on the limbic system. Differences have been found both with evoked potentials (a special electrical impulse elicited by auditory, visual, or sensory input) and EEG potentials (electroencephalograms). Chronic schizophrenics have significantly different EEG profiles than matched groups of normal volunteers, and these differences are hyperregulation of the brain waves as shown by Dr. Leonid Goldstein. In 1982, Monte S. Buchsbaum and colleagues at the National Institute of

Mental Health in Bethesda, using the positron-emission tomography (PET) scanner, showed decreased utilization of oxygen and glucose (suggesting decreased brain function and decreased blood flow) in the frontal lobes of schizophrenics.

PET works by coating brain food, namely the sugar glucose, with radioactive tracers. Detectors surrounding the patient's head record the levels of radioactive material as the brain cells make use of the food. The resulting computer-generated pictures reveal unique patterns of activity in some schizophrenic brains. During hallucinations, the speech and hearing centers of the brain burn the glucose (brain food) more rapidly than do those areas of non-schizophrenics. In the frontal lobes of the brain, where planning and organizing abilities are focused, activity appears to be reduced. Another developing technique called xenon inhalation tomography shows decreased blood flow in the frontal lobes of the brain of schizophrenics. Differences are apparent and these may well be owing to excess of copper, deficiency of zinc and other nutrients.

PSYCHOLOGICAL AND EMOTIONAL STRESS TRIGGERS SOME SCHIZOPHRENIAS

Dr. Prilipko implicates emotional pain and stress as the events triggering the appearance of antibrain antibodies. This theory may explain the well-documented exacerbation of schizophrenic symptoms often associated with stress.



Figure 13: Become a student of the stress avoidance school.

Stress is understood to be that factor which pushes a vulnerable individual over the edge. Schizophrenia most

often appears in young adults (15-30 years of age) just at the time when the stresses associated with coming of age and becoming a responsible adult in society start to peak. Stress from family pressures, work or school, social pressures and pressures associated with significant individual events all appear to most seriously impair those people who are in some way already predisposed to schizophrenia. Both positive and negative life-changing events can bring about such stress and precipitate symptoms in unduly sensitive individuals.

It is only in this way that stress within the family can seem to "cause" schizophrenia. It must remain clear, however, that even moderate levels of stress, easily handled by most family members can be enough to offset one predisposed to schizophrenia. Even "good" stresses such as a new, enjoyable job or a newborn baby can trigger the imbalances associated with schizophrenia. It seems a shame that these families are often blamed for maliciously causing the disease. Clearly such events cannot be completely avoided in the fear of causing schizophrenia. An untreated schizophrenic will do best to avoid stress whenever possible. One who can get proper treatment, on the other hand, should be able to stand increasing levels of stress with no sign of symptoms.

ENVIRONMENTAL STRESSES CAN TRIGGER SCHIZOPHRENIA

Henry Shroeder was the first to postulate that some schizophrenics are victims of environmental poisons. Stress sufficient to surface schizophrenic symptoms in a predisposed individual can come from ingestion or absorption of toxic materials from the environment. Most often heavy metals such as lead, copper, mercury, silver, and cadmium are found to be the intoxicating agents. Copper is frequently presented in the tap water used for drinking and a switch to pure spring water is all that is necessary to reduce the paranoia and hallucinations that can come with copper intoxication. Less common cases report patients who are prone to intoxication by the pure water itself—a seemingly harmless substance.

We have long known that mercury poisoning made hatters mad. (Hatters make hats.) We now know that lead and copper can make children autistic and/or schizophrenic and that deficiencies can be equally stressful. Lack of manganese, zinc, and chromium makes patients hypoglycemic. Finally, fully one-third of the so-called schizophrenics are simply zinc deficient. It seems a shame that these folks are treated psychotherapeutically when nutritional supplements would ease their symptoms.

THE KNOWN BIOCHEMISTRY OF STRESS

We all realize that stress is bad, but few of us know how to counteract nutritionally the many nutrients which are lost from the body in stressful situations. This knowledge is important to make the medical model of the schizophrenias come alive and breathe sanity.

In the late thirties, we stressed our trained dogs to see what elements were lost when a hungry dog saw another dog eating. The two elements which increased twofold in the urine were calcium and potassium. We tried calcium and potassium as a combination tablet for the treatment of headache and had such success that we still recommend salt substitute for potassium plus any calcium salt for migraine.

Swedish researchers have shown that the stress of insomnia in humans is accompanied by a rapid drop in the plasma zinc level, which is not corrected by a full night's sleep. Drs. Walter Pories and William Strain have noted an increased need for zinc following traumatic injury and, therefore, treat their surgical patients pre- and post-operatively with zinc salts. Studies performed in stressed rats have demonstrated a concurrent rise in copper as zinc levels fall (Flos and Balasch, 1977). Remember, zinc is a copper antagonist (see Histapenia). Stress has been reported to decrease body stores of vitamin C, especially with respect to the adrenal cortex, the body's producer of adrenaline. Increased requirements of vitamin B-6 have been noted due to stress, probably as a result of increased production of kryptopyrroles (see Pyroluria). We predict that most of the essential trace elements will be found to be dissipated by stress and with these elements the body will lose Vitamin E, and all the other water soluble vitamins. Nutrient supplements of vitamins and minerals are, thus,

very important to replace biochemicals lost in states of stress.

Much recent attention has been focused on the effect of stress upon the immune function and the protein balance of the body. Studies show that zinc, manganese and magnesium are needed to form antibodies and actuate immune cells. Logically, the body's caloric requirements increase when subjected to stress; however, a large proportion of this increase appears to be due to increased protein needs, especially for branched chain amino acids (BCAA)—leucine, isoleucine, and valine. If these needs are not met nutritionally, the body will enter into a period of catabolism, breaking down existing muscle tissue to meet metabolic demands. Loading surgical patients with amino acids (BCAA) has resulted in shorter and less eventful recoveries in many operations.

Life's stressful mineral emergencies can be summarized as follows: 1) diarrhea or fever in childhood, 2) the rapid growth spurt of adolescence, 3) dieting for weight loss at any age, 4) pregnancy in adulthood but even more stressful in teenagers, 5) emotional turmoil, 6) surgical operations, 7) the menopause, 8) and changes in habits with aging and the accumulation of copper, aluminum, and lead with age.

When these mineral emergencies are recognized and supplements taken to correct the deficiencies, the individual will live longer and happier.

SUMMARY: NON-BIOCHEMICAL THEORIES

No single clear model presently exists for the pathogenesis of schizophrenia. Classic organically based studies of schizophrenics which begin by assuming that the schizophrenic population is organically homogeneous, are riddled with gaps and contradictory experimental facts. The inability to overcome these difficulties is best explained by their misleading assumptions and their routine behavioral classification of patients.

The need is for more solid biochemical endpoints or keys to the disease which can then be searched for in the large organically heterogeneous population labeled schizophrenic. The truth is that the schizophrenic population is neither biologically nor behaviorally homogeneous. As a result, studies looking for behavioral correlations in schizophrenic patients will continue to prove disappointing.

The situation is further aggravated by the difficulties inherent in patient based biological experimentation. The hospital setting is not ideal because of the overlay of heavy neuroleptic medications. Furthermore, the expense and politics of the hospital are against prolonged observation. The outpatient clinic wherein the scientist actually sees the patients personally will provide the most valid observations. In this setting, the patients are used as their own controls. Many nutritional treatment regimes do not work on certain schizophrenic patients. Some, like folic acid which increases the body's histamine levels, even make some patients worse. (Folic acid makes the histadelic patient worse.) Roger Williams sums up the preferred approach to patient study as he points out that such studies necessarily involve "repeated observations on the same individuals, in contrast to a series of single observations

on representative populations.”

In 1985, the NIMH selected schizophrenia as one of its five major targets, elevating it to a new level of importance. Perhaps in due time some of the studies suggested in these chapters will be pursued and more will be learned about the biological parameters whose changes in the body reflect the patho-physiological processes which accompany the development and relief of schizophrenias.

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SELF-EVALUATION: THE PSYCHOANALYSTS LOOK INTO THEIR COMPLACENT MIRRORS

The American Psychoanalytic Association, in the October 1976 issue of its Journal, released findings of a long-term sociological and statistical study of psychoanalytic practices and the comparative results of treatment by psychoanalysts and psychotherapy respectively. This is summarized in the A.P.A. News.

As judged by Dr. Weinstock and Dr. Hamburg, who headed the evaluation committees, the group of 10,000 patients treated by psychoanalysts were 96.6 percent satisfied with their improvement obtained over one- to four-year periods. The analysts judged that 97.3 percent of the patients were "improved in total functioning." While this sounds good, one should note that the relief of symptoms in chronic male schizophrenics was only 9 percent, and the overall rate of symptom cure was only 27 percent in schizophrenia. In contrast, the spontaneous remission rate from the first attack of schizophrenia is 25 to 30 percent in males, and the hospital discharge rate for male schizophrenics treated by all available methods is a whopping 76 percent. The 9 percent, therefore, confirms the impracticality of treating schizophrenia by psychoanalysis. No mention was made of patients who got worse under analysis and had to be hospitalized. Neither was any mention made of the suicide rate—an unavoidable contingency which always plagues psychiatric practice.

The investigators were forthright in pointing out the limitations of the findings and the methodology employed. Nevertheless, some new sociological information is available. For example:

Fifty-seven percent of the patients studied had incomes under \$10,000; 25 percent from \$10,000 to

\$20,000; and 15 percent more than \$20,000. (The cost a year for orthodox analysis is often well above \$5,000, with anywhere from one to several visits weekly.)

Nearly all (98.8 percent) were white and included Protestants and Jews in about equal numbers; only 10 percent were Catholic or of other denominations.

Twenty-two percent had been to high school, 41.9 percent to college, and 35.8 percent to graduate school. Since this adds to 100 percent, one can infer that at least a high school education is needed for a patient to understand the workings of the mind as disclosed by psychoanalysis.

Forty-four percent were in analysis for less than a year; 33 percent for two years; and 11.3 percent for four years or more.

They state that psychoanalysis was found to be significantly more effective in adjudged, improved character structure than was psychotherapy. However, no similar group of patients receiving drug therapy or only psychotherapy was included, nor was the evaluation done by "controlled tests."

The authors on the report venture that this first evaluation will lead to further studies with sharper focus, more certain methodology, and with investigators who will have more adequate time to complete the job. Such a study would be of greater significance if another group receiving drug therapy and psychotherapy were included.

TYPES OF PSYCHOTHERAPY

1. **Listening therapy:** Some analysts of the Carl Rogers School believe that the patient's flow of talk should not be interrupted. This may be called the sympathetic ear, and frequently the only feedback is a mild "continue!" or "tut, tut!" or as the old therapy said, "Ach! Who listens!" This is not measurably effective in schizophrenia.

2. **Talking therapy:** Active interchange of a counseling nature between therapist and patient. This is useful, but in the treatment of schizophrenia it must always be of a supportive rather than of a probing nature. It is of secondary importance compared to nutrient therapy.

3. **Psychoanalysis:** An expensive form of listening and talking therapy which is cultivated and perpetuated by the disciples of Freud, Jung, Adler, and a few later and lesser saints. It is expensive and may continue for years without measurable results. It is not recommended for the schizophrenias.

4. **Group therapy:** At an appointed time a group of patients meet to discuss their problems and seek possible solutions. The similarity of their many problems becomes quickly evident and benefits usually result—depending on the training and ability of the group leader, who is usually a clinical psychologist, psychiatrist, social worker, or trained counselor, for example, of the ministry.

When the patients congregate on the basis of their disabilities, then the group is called Schizophrenics Anonymous, or Alcoholics Anonymous. The AA group has demonstrated tremendous value over the years.

5. **Other types of psychotherapy** are useful, but certainly not specific. These are occupational and usually include music, dancing, games, nature walks, work, etc.

Any physician who undertakes to treat a patient must

apply recognized and logical procedures. These are:

1. Diagnosis of the condition. In mental disease this entails the elimination of brain tumor, psychomotor epilepsy, etc., as the possible cause of the symptoms. A psychometric test such as the MMPI, EWI, or HOD is mandatory. A careful history is essential.

2. Treatment by use of the nutritional therapy and the use of those drugs with which the doctor has had the greatest success.

3. Follow-Up. In the case of mental patients, a program of careful follow-up is necessary to assure continued social rehabilitations.

4. The patient may need allergy tests, but the nutrient and drug therapy should not be stopped during allergy testing.

If the patient has serious mental disease, time is of the essence and should not be squandered by delayed diagnosis, inadequate treatment, or trying the latest fad.

TALKING THERAPY IN SCHIZOPHRENIA

Attempts to treat patients with a serious degree of schizophrenia by means of talking therapy have been dismally unsuccessful. This conclusion is based on the fact that when this approach is adopted, specially trained psychotherapists, who probably have to have a certain kind of personality make-up themselves, are needed; the amount of time and persistence required are forbidding, and the results are dubious.

Conventional psychodynamic formulations explain everything from orifice to anus and can also explain to some degree how the mental functioning of a patient with schizophrenia is altered. Such formulations are also applied to patients who have cancer or other serious illness. But a fundamental difficulty with such formulations is that they are after the fact and not predictive. They detail the form of the illness as it has become manifest, but they cannot predict this beforehand and, hence, are of little value.

Although Sigmund Freud occasionally delved into the histories of schizophrenics to gain further understanding of psychopathology, he was firmly convinced that psychoanalysis is not an appropriate form of treatment for such patients, and he steadfastly refused to accept patients with schizophrenia. He based this on the belief that their disease rendered them incapable of forming the kind of intense emotional relationship (transference) with a psychotherapist which he regarded as essential to a favorable outcome of the treatment.

More recently, Drs. Erich Fromm and Frieda Fromm-Reichmann showed that transference could be elicited from a schizophrenic patient, but this required an extraordinary effort in terms of time and dogged refusal to

be discouraged. No matter how dedicated the therapist, attempts to treat such patients by these methods may commonly require five or more hours a week for ten or more years. Even then, the improvements may be minimal and can be separated from the natural fluctuations of the illness only with difficulty. Other therapies are more positive and quicker.

There have been variations on the psychoanalytically based approach. One of these was that of Dr. Harry Stack Sullivan, who viewed mental illnesses as "failures in relationships between people" rather than disruptions of processes within people. Time has not shown this to be true, nor can we evolve a successful scheme for the treatment of schizophrenia from this approach.

Dr. J. N. Rosen of Philadelphia tried another modification which is very close to what Freud called "wild" analysis. Where Freud always insisted that formulations about the patient's thinking and feelings should always be derived from the patient himself without any preconceptions, Dr. Rosen adopted certain interpretations of schizophrenic behavior and ideation as universal and proceeded to batter his patients verbally with these, regardless of their context in terms of the patient's current clinical status. Early remissions were reported, but early remissions are frequent in the so-called "stormy" or "noisy" phase of the illness. This fad has passed with the advent of drug and nutrition therapy. The foregoing examples are not complete, but have been selected to give representative examples of types of talking therapy which have been tried.

That talking therapy is not an appropriate treatment for schizophrenia is suggested by a study made at the University of Southern California. Here patients were given a combination of talking therapy and medication. Even those who showed the most improvement demonstrated almost no psychological insights relevant to their illness. Nor could it be shown that their degree of

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improvement was any greater than what might have been expected from medication alone. In another study, a male nurse in an underdeveloped country treated 2,000 male patients with drugs and had the same "success rate" as that published for modern American mental hospitals, namely, social rehabilitation in about 75 per cent of the patients.

Psychiatry and the patients have suffered in the past from numerous fads because of the lack of any specific treatment for schizophrenia. As medical science has progressed, it is now possible to adhere to specific therapies and treat each disease or symptom on the basis of a rational understanding of its causes and course. Today it is increasingly believed that schizophrenia has a biochemical basis. Talking therapy might in certain cases relieve some of the suffering, as it can for patients with other illnesses; but to expect talking therapy, any more than faith healing, to correct basic organic malfunctioning (biochemical imbalances) is, of course, futile. Such a misapplication does an injustice to the method and underestimates the disease.

Some therapists may be likened to the old barnstorming pilot who always flew his airplane by the seat of his pants rather than by modern instruments. This worked well for the pilot on sunny days, but not in clouds or fog. A similar fog is engendered by the two variables, such as adolescence and schizophrenia when they occur together in the teenager. Under these circumstances the therapist needs the best and most accurate instruments available. The psychometric tests such as the MMPI, EWI, and HOD card-sort tests are available, can be administered and easily evaluated, and should be used since early drug treatment for the schizophrenic teenager is essential to prevent chronic disability.

The teenager and the parents should avoid a therapist who is not interested in a different diagnosis, since only an accurate diagnosis can lead to adequate therapy. Modern

society cannot afford to tolerate the treatment of juvenile schizophrenia by talking therapy alone when better therapy is available.

When to Use Talking Therapy

Wise counseling and verbal supportive therapy is useful in the following circumstances:

1) The early adult or teenager may need continual counsel because, if he has been sick, he has missed the maturing effect of the teenage rat race.

2) The schizophrenic returning to society after several years in a hospital needs counsel. Part of this therapy consists of a constant check on and adjustment of the drug therapy. The patient may benefit from psychodrama in which he relives some of the situations he will face in the outer world.

3) Talking therapy of a supportive or reassuring nature may be helpful at any time. Problems of social interaction can be solved or procedures outlined to minimize these problems and, thus, reduce environmental stress. Hence, talking therapy if expertly directed will help solve the problems of everyday life. If inexpertly directed, talking therapy may dig too deeply and put salt in old wounds, increasing mental stress and the degree of schizophrenia.

4) Talking therapy can bring the patient's attention to his strained relationships with people who are important. If the patient learns to handle these situations better, then mental stress is reduced.

WHEN TO HOSPITALIZE

The four clinical conditions in the schizophrenias which usually require hospitalization are: 1) When the patient attempts or threatens suicide, 2) When the patient is paranoid with agitation and threatens bodily injury to others, 3) When the patient's behavior interferes with the normal life of the family, 4) When the patient is uncooperative and will not take medicine of any kind, he usually must be hospitalized for initial treatment, and then can be given weekly injections of one of the long-acting preparations.

Aside from these conditions most other schizophrenic states may be treated at home if the family will cooperate. The four instances for hospitalization listed above do not necessarily require state, county, or private psychiatric hospitalization. With proper medical supervision, the medical section of a general hospital can give effective and rapid treatment with a resultant rapid return of the patient to his home for continued treatment.

Home treatment may be better for many schizophrenics. This decision must be made by the physician in charge. A study of 152 state hospital patients by Dr. Benjamin Pasamanick in the Louisville, Kentucky, area provides some thought-provoking data. The patients were divided into three groups: 57 were given anti-schizophrenic drugs while living at home, 41 were given dummy capsules while living at home, and 54 as a control group were hospitalized for drug therapy. The two home-care groups were visited regularly by a public health nurse from the community mental health center. These visits were made weekly for the first three months, every two weeks for the next three months, and monthly thereafter for up to thirty months in some instances. The nurse renewed the patients' supply of drugs.

At the close of the study, 77 per cent of the home-care patients were socially rehabilitated. Only 34 per cent of those on dummy capsules were able to stay at home. The hospital control group needed 83 days on the average for the social rehabilitation of their patients which meant that the 57 drug patients treated at home had saved almost 5,000 hospital-patient days.

According to the report, the hospitalized group required hospitalization more often after discharge than did the home-treated group. The number of readmissions are 25 out of 54 (46 per cent) for the hospital group and 13 out of 57 (23 per cent) for the home-treated group. Dr. Pasamanick suggests that early home drug treatment is the only type of prevention for schizophrenia presently available. One cannot easily humanize the hospital, but the dehumanizing effect of large institutions can be prevented by home treatment. Long-term hospitalization increases the patients' withdrawal from society, makes them more dependent and less able to function normally.

Obviously, the family must be willing to have the patient stay at home and must be willing to tolerate the strange behavior of the patient before the antischizophrenic drugs start their good effect.

As a clinching argument, Dr. Werner M. Mendel reviewed the hospital records of 3,000 hospitalized schizophrenics and found that the discharge rate was 76 per cent, regardless of whether the patient had been hospitalized 7, 30, 60, or 90 days. The discharge rate of 76 per cent is, thus, independent of the number of days spent in the hospital. With drugs and nutrients, the rate for social rehabilitation is 90%.

Since many of our psychiatric hospitals, such as private and veterans' hospitals, have a waiting list for admission, the physician should not allow nutrient or drug treatment to be delayed simply because a psychiatric bed is unavailable.

How Does the Patient Get Out of the Mental Hospital?

The exit varies with the type of hospital (whether private, veterans', or public) and the reversibility of the legal or signing process that got him into the hospital. If he is over age 21 and admitted to a private hospital on his own signature, then he is a voluntary patient and has the legal right to sign himself out. He is foolish to do so, however, unless his schizophrenia is under good control and unless he plans to continue the regime of therapy which has relieved his symptoms.

Getting out of a public hospital is usually much more difficult than the equally laborious process of getting into a public hospital—so the patient must have tolerance and patience. If he is over 21 years of age and has signed himself into the hospital, the exit process may be equally simple; that is, he may sign himself out. But he should not be foolish and leave before he is well enough to cope with the stresses of the outside world. He should plan to take advantage of the halfway house or clinic which may be affiliated with the hospital. He should also plan to join a local Schizophrenics Anonymous group or similar group, such as Recovery, Inc., if one exists in his locality (Chapters 18, 51).

If he has been committed to a county or state hospital, then his stay in the hospital may be longer, but not necessarily more tedious. By actively engaging in the work and occupational therapy programs of the hospital, he will have ample opportunity to impress the hospital staff with his cooperation and stability. Needless to say, he should cooperate completely in the medical and group therapy programs.

He may have been committed because society thought at the time that he would be safer and get well more quickly under enforced hospitalization. It is the purpose of hospital therapy to provide treatment until adequate

social rehabilitation occurs. At this point, a committee of the medical staff goes on record to the effect that the patient will be able to maintain his mental and physical health in the outside world. Usually a careful follow-up on his health is planned.

Discharges from public hospitals are made under the strictest controls. The physician in immediate charge makes the recommendation after careful evaluation of the patient's condition. The patient is then presented at a weekly staff conference. After his committee approves of the recommendation, the entire record comes before an administrator who makes the final decision. In some states, such as New York, a legal staff member must also approve.

It is not the purpose of a public institution to keep a patient beyond what is considered to be his maximum degree of social rehabilitation, but release does take time.

A useful mechanism for obtaining quicker release from a state or public hospital is the transfer to a private hospital. The private hospitals are more adequately staffed and, frequently, can more rapidly complete the numerous interviews, questionnaires for departure, and arrangements for continued therapy which are necessary for a patient's discharge. The political sensitivity and vulnerability of a public hospital may, frequently, make the decisions in the release of a patient most difficult.

When the Patient Returns from the Hospital

1. The doctors' orders should be carefully followed (and this is extremely important).

- a. Continued medication. Be sure to have an ample supply.
- b. Regular visits to the clinic should be continued.
- c. Refusal to take medicine, or side effects

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from medicine, should be reported to the doctor.

- d. Specific vitamin intake and high nutrient diet should be encouraged.

2. Socializing should be tactfully encouraged, but not insisted upon.

3. Work plans of an occupational therapy type should be planned: painting, cleaning, gardening, etc.

4. Daily exercise is helpful: walks, jogging, pushups, knee bends, games.

5. Encourage attendance at selected motion pictures.

6. Suggest hot baths (soaks) prior to bedtime as a means of inducing sleep.

7. Plan meals that are high in nutrients, low in sugar, and free of caffeine.

8. In the family, discuss the illness in an open fashion at all times; after all, a repaired brain is as good a topic of conversation as a recovery from double pneumonia.

9. The inner locks of the apartment or house should be made inoperative before the patient's return. This is particularly important if the patient was in the habit of retiring to the bedroom or bathroom and locking the door. Room doors, of course, may be closed, and all members of the family will respect privacy by learning to knock before entering.

10. A patient quickly learns that some degree of relief from the profound brain stimulation can be provided by the excessive use of nicotine, usually in the form of cigarette smoking. As he uses it, nicotine is not a stimulant but a tranquilizer. This habit must thus be tolerated while he is severely ill.

Some Things the Patient Must Remember Now That He Is Out of the Hospital

The patient must:

1. Realize that he is sick, cooperate with the doctor and follow advice, and above all, continue the medicines.
2. Realize this illness is no one's fault, including his own.
3. Not act or be influenced by hallucinations.
4. Not discuss hallucinations with anyone except the doctor.
5. Eat well and regularly.
6. Get plenty of rest and avoid fatigue.
7. Avoid stressful emotional reactions whenever possible.
8. Never use the illness as an excuse for ugly behavior.
9. If he has a relapse or a letdown, he should realize that this is a temporary condition. He recovered before, and he will recover again. The next relapse will be less severe.
10. When well, write down all reasons for living, so when he feels badly, he can read his list again and be comforted.

Options for Non Hospital Orthomolecular Treatment

Hospitals do not follow the directions of orthomolecular practitioners. Thus, the essential nutrients are invariably stopped on entering hospitals for psychiatric study or even for allergy testing. (The patient may be allergic to the vitamins!) Without the essential nutrients which are known to be needed the patient rapidly relapses and the hospitalization is prolonged. Suitable alternatives to hospitalization must be provided or the patient may be

shifted from allergy to the State psychiatric hospital. These may range from halfway houses (pensiones) to treatment in the home under the care of an operative nurse trained in the orthomolecular approach. The orthomolecular physician can thus continue to provide the medical care. This situation is similar to childbirth under the care of a wise midwife with the trained doctor available on call. The overall result is better and the patient will make continuous progress toward recovery from the allergy or mental disease.

The halfway house takes years of effort to establish. The usual population of patients at any one time will vary from 6 to 20 patients. Plans must be made for the orthomolecular food, non allergenic housing and orderly admission, daily care and dismissal of the patients. The house will not make a profit so it should be organized as a not-for-profit unit and should receive help from the governmental bodies or annual donations from benefactors. The insurance companies should pay for this care as they do for hospital care. Some volunteer help can be used to ease the great financial burden.

The late Rosalind LaRoche, who ran the Earth House in East Millstone, N.J., was the first trained operative in the field of orthomolecular medicine. She heard of a recluse schizophrenic brother of a famous psychiatrist at a party. She suggested that with her orthomolecular nutritional knowledge she could socially rehabilitate the recluse brother. She traveled to the mid-west with the packets of nutrients and within two months she had the recluse exercising daily and working at the family factory. He is now totally rehabilitated and is a member of the board of directors of the business. Such activity needs courage, strength, self assurance and adequate training on the part of the orthomolecular operative. This training can be had at some of the orthomolecular centers around the world. Patients who cannot

be left alone or patients who have withdrawn from society are the prime targets for therapy supervised by operatives.

Some drug crutches may be needed to get the patient in the mood for nutritional therapy. The operative is trained in the use of lithium (in small doses) and haloperidol drops which are tasteless and odorless. Haloperidol can, thus, be used covertly in the initial stages of therapy. As the patient gets better on the nutritional therapy, the dose of lithium and haloperidol can gradually be reduced. Dr. Paul Janssen of Beerse, Belgium, who discovered haloperidol, once told me confidentially that he believed that one half of the paranoid patients of Europe were on covert haloperidol treatment. The covert drops do work and we at the Brain Bio Center have had great success in the treatment of recluse, uncooperative, schizophrenic patients. I usually place one drop of haloperidol on my tongue and then on the parents' tongue to demonstrate that the medication is tasteless and odorless. The haloperidol liquid has been placed in milk, orange juice and, in one instance, under the plastic wrapper of frozen pizza pies for the treatment of a paranoid boy who would not eat any of the food his mother cooked. He cooked and consumed the pizza pies and got progressively better day by day!

As an alternative to the halfway house, a trained psychiatric nurse may agree to take a patient into her home for continued therapy. We have found this type of orthomolecular therapy to be highly successful and perhaps the least expensive of any arrangement. The new family milieu is particularly conducive to rehabilitation. It is obvious that any of these options are better than the psychiatric hospital or the rented bare apartment which is frequently suggested to get the patient away from the family.

THE ECONOMICS OF SCHIZOPHRENIA

There is no question as to whether or not this illness is expensive. It is! The major question is, "How expensive?" While no survey of charges is known, some personal experience with charges for hospitals, doctors, medication, special treatment, travel, and other items may provide useful guides as to what to expect. These costs were incurred during 1983-1985 and need adjustment upward to reflect current prices.

Hospitals:

In general, hospitals, with teaching, training, and research facilities, are the most expensive. They have costs running from \$300 to \$400 per day. These high costs are, in part, due to the high cost of personnel. These hospitals tend to have the highest number of nurses, attendants, specialists, and doctors per patient.

There are also special purpose clinics catering to mental illness. These have both in- and out-patient service. They are designed to provide quick, restorative treatment. In two of these we have experienced rates of about \$400-1000 per day with medication. Doctors' fees and charges for electric shock therapy (EST) or other special tests and treatment may be extra.

Least expensive, but also least able to provide attention to the patient, are the state hospitals. These may be free to state residents if the family income is low. When charges are billed to those who can pay, the rates have been low. This charge includes everything. Parenthetically, a state hospital is no place to take good clothes or personal belongings—clothes and other belongings may be lost or stolen.

Also, it is worth noting that if patients are allowed

home for weekends or brief stays, there is no charge for such days.

Hospital costs depend greatly on the length of stay. When the patient is ill enough to require state hospitalization, then a lengthy stay in this secure and different environment is needed for the patient to recover sufficiently to return to a more normal society. This stay is seldom less than one month but can extend into many months or even years. In northern New Jersey, many county hospitals are better than the state hospitals. The war veteran should make arrangements to receive the excellent care provided in the Veterans Hospitals. A similar system exists for merchant seamen.

Doctors:

When the patient is not in a hospital he will have doctors' fees. In our experience, psychiatrists' costs have varied from \$100 to \$125 per hour visit. We have found doctors willing to discuss charges if they felt the costs were so burdensome that the patient might be unwilling to keep up recommended treatment. As in hospitals, the costs are a matter of time and frequency of visits. Psychiatrists have suggested twice a month or even longer intervals. The length of time under the doctor's care varies to some degree with the approach of the doctor, the nature of the illness, and the doctor's busy schedule. Doctors seeking to discover causes and those who use psychotherapy or analysis require the longest treatment time.

Somewhat lesser fees are charged by psychologists. These have been about \$50 to \$75 per hour with something less for group therapy sessions. As in the case of psychiatrists, there is sympathetic concern for the family's ability to pay.

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Travel

All of these forms of treatment require travel. Travel costs can mount up if visiting is maintained during hospitalization. Arrangements for babysitters or home care also may be required. Expenses of this sort are not eligible for reimbursement under most hospitalization or "major medical" policies. It may, however, be claimed as a medical expense on your income tax.

Medication

The antipsychotic drugs can be expensive. One family we know spent \$350 on drugs per week. Some prescriptions have cost as much as \$1.00 per tablet, and dosages can be as much as six tablets per day, or six dollars per day. Des-tyrosine endorphin, an experimental drug in the treatment of schizophrenia, costs approximately \$300 per injection. Each injection lasts only one week. It is refreshing to note that niacinamide, vitamin C, B-6, zinc, and manganese tablets can be purchased directly, so that a 3.0 gram per day dosage of each can be as little as 40 cents a day.

Insurance

Insurance can help to pay for many of these costs. Under Blue Cross contracts, the patient is eligible for 20 to 30 days' hospitalization in one contract year. This certainly helps, but seldom can a hospitalization be completed within this period. Many employers provide major medical insurance for further protection. This covers medical costs not covered under Blue Cross or other hospitalization insurance. While they certainly help, most of these contracts have limits. These are usually total dollar and time limits. Virtually all of these require an

initial payment by the insured—from \$50 to \$500, plus a percentage payment of all subsequent costs.

Those patients who have paid into Social Security should investigate the possibility of permanent or temporary disability payments. Under the 1965 law, any insured worker whose disability is expected to last at least 12 months may qualify for disability benefits at the beginning of the seventh month. Chronic schizophrenia qualifies as a disability.

Other Costs

There are special problems and costs if the patient is the breadwinner. He may have some limited sick leave at work, but this is seldom enough to cover the period of hospitalization. At this point, the question of family income becomes an overpowering problem.

Besides hospitals, there are other types of "away from home" living arrangements—day care, halfway houses, and other possibilities. Halfway houses provided by the state are considered extensions of the hospitals, and the rates are the same. We have also investigated social camps and have been quoted rates of as much as \$1000 per month.

If a community mental health center is nearby, the patient can frequently be treated at home after the diagnosis is established. For this purpose the visiting nurse is extremely valuable. The public health nurse may also be able to give relatives valuable advice in regard to local treatment facilities.

In New Jersey, under the Beadleson Act, the homebound patient is entitled to continued schooling. This must be asked for and, frequently, insisted on by the patient. This service is obtained by application through the local school board.

THE HISTORY OF ELECTROCONVULSIVE THERAPY AND ITS PLACE AMONG OTHER PHYSICAL THERAPIES IN THE TREATMENT OF THE SCHIZOPHRENIAS

Prior to 1933, the only physical treatment available to psychotic patients was sedative therapy. The sedation was produced by hot baths or packs, paraldehyde, sodium amytal, or morphine-scopolamine. Every means was needed to keep the patient from dying of acute psychotic exhaustion. In spite of rotating methods of sedative therapy, tube feeding, and hot packs, many patients died.

From 1915 to 1935, Norway had an intensive program of nursing care and occupational therapy for the schizophrenic, but the effect on the patients' discharge rate was not noticeable. Dr. Odegard, in a study involving Norwegian hospitals, says:

"This was the period of mental hygiene: prevention was stressed, because the prognosis was so bad for fully fledged mental disorders. The high hopes of a prevention have hardly been fulfilled even today, whereas the dramatic improvement in therapeutic results was quite unexpected. Starting from 1935, the discharge rate was doubled in five years, and in ten years it was trebled."

The discharge rate for mild schizophrenias was always low, but the stay was shortened. The real change was in the severe schizophrenics who might otherwise have remained hospitalized for years or their lifetime.

Although they have not completely fulfilled Dr. Odegard's "high hopes" for preventative measures, three noteworthy advances are responsible for significant increases in hospital discharge rates.

In 1933, Dr. Manfred Sakel introduced insulin coma therapy. In 1934, Dr. Ladislav J. Meduna introduced metrazol convulsive therapy. Metrazol convulsive therapy was rapidly discarded when Dr. U. Cerletti and Dr. L. Bini (1938) introduced electro-convulsive therapy. They found that a single convulsion could be safely produced by the application of an alternating current to the head.

After the year 1940, ECT and insulin coma were the standard therapies for the schizophrenias. The general rule was to use insulin coma in the younger patients and ECT in the older patients. Both of these therapies shortened hospitalization or produced complete remission of the disease in some patients. I can recall at Manteno State Hospital in the period 1948 to 1952 that on the active treatment ward for female schizophrenics 25 to 30 patients were given maintenance ECT treatments twice weekly. When given in moderation, no more often than every other day to the nondominant side of the brain (the right side in right-handed and the left side in left-handed patients), the treatment may be well tolerated but it can also get out of hand. With refinements such as electronic control of the current, thiopental to produce amnesia, and other drugs to relax the muscles, ECT has lost some of its side effects, but many remain. We know from treating epileptics that the seizures of epilepsy will decorticate (produce a vegetable) a young patient by the age of six years. The goal in epilepsy is to reduce the harmful seizures to no more than one a week. Electrically-induced seizures are just as debilitating to the brain as is grand mal epilepsy, and so should be limited. The effect of ECT may be transient or totally ineffective so the patients may live out their lives with the original depression or psychosis, and an impaired memory.

It is written that informed consent is obtained from each patient (or relative) before ECT is used. However,

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this informed consent is frequently in the batch of papers which must be signed on admission to the psychiatric ward of the hospital. Even if the informed consent paper were read slowly aloud to the befuddled patient, the consent would be signed without anyone being informed of the alternatives to ECT and its effect. With the introduction of reserpine drug therapy in 1952, the use of ECT decreased remarkably.

The use of ECT has shifted from the back wards of state hospitals to private psychiatric hospitals where the limited days of hospitalization, paid for by insurance, dictate quick cures (usually within three to four weeks). In state hospitals, time is less important, so ECT therapy is rare. As a result, private psychiatric hospitals often overuse ECT merely because it is expedient. It is indeed sad that the middle-class, insurance paying, employed patients with allergies and depression are getting a dubious quick fix rather than adequate diagnosis and treatment.

In the U.S.A. where safe, effective nutritional treatments are available and often preferable, each year an estimated 50,000 to 100,000 patients still receive a series of ECT treatments (Bowen Mathup, Wall Street Journal 8-11-86). In this same article, Harold H. Sackheim, M.D., recalls "five schizophrenic patients who were catatonic and not eating. After ECT therapy they started eating." Catatonia is very rare these days because of the use of neuroleptic drugs by most therapists. Dr. Sackheim's catatonic patients would have responded in 24 hours to an injection of Prolixin enanthate or decanoate. Instead of receiving gentle drug treatments, these five patients had five points taken off their I.Q. by ECT.

Today we know that many psychotic patients are merely that way due to biochemical imbalances. Treatment should first aim at nutritionally correcting those imbalances. For those who do not respond to such nutritional therapies and do not respond promptly to

antipsychotic drug medication, ECT therapies can be effective in preventing hospitalization. For those already in hospitals, the length of stay is usually shortened. Moreover, ECT may be the only form of treatment to which some suicidal patients will respond rapidly. In these cases, ECT is truly life-saving.

Today, one must adopt a philosophy of treatment wherein all therapeutic measures are used in a progressive fashion to either prevent hospitalization or restore a hospitalized patient to community living. The first move should be talking therapy of a supportive nature, reduction of environmental stress, and mega-nutrient therapy. The second step is to determine the allergies of the patient. Is the patient allergic to wheat or other foods? Is the patient high in copper or histamine? Only after these have been tried, should antipsychotic drugs be included alongside the nutrients. Finally, ECT should be used only as the last resort.

In conclusion, we suggest the following tests and trials be completed prior to any use of electroconvulsive therapy (ECT). The lack of the patient's response to psychotherapy, lithium therapy, or anti-depressant drug therapy is not enough evidence to warrant ECT treatment. Severe suicidal depression may have several causes:

1. Is the patient taking basic nutrients at doses that will combat environmental stress?
2. Is the patient depressed as a result of allergies to Wheat Gluten? Corn? Beef? Milk?
3. Is the patient naturally high in histamine? What is the absolute basophil count and blood histamine level?
4. Is the patient pyroluric? Mauve factor? Serum zinc? Erythrocyte-GOT level?
5. Is the patient vegetarian or malnourished? Plasma amino acids? Serum folate and B-12 level?
6. Is the patient intoxicated with heavy metals?

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Blood aluminum, lead? Blood serum copper level? Hair test for heavy metals?

7. Does the paranoid patient have a low platelet MAO level or a high urinary tryptamine excretion?

8. Does the patient respond to pyridoxine, tyrosine, zinc and manganese therapy? This frequently is effective when anti-depressant drugs are not.

If these questions are answered positively, then the need for ECT may vanish. Only if none of these alternative diagnoses prove positive, and severe suicidal depression or severe psychotic symptoms remain, should electroconvulsive therapy begin. In such cases, it may be the only treatment available and then may be ineffective!

SPECIFIC DRUGS ARE AVAILABLE FOR SOME OF THE SCHIZOPHRENIAS

“Almost all symptoms and manifestations characteristic of schizophrenic psychosis improved with drug therapy, suggesting that the phenothiazines should be regarded as ‘antischizophrenic’ in the broad sense. In fact, it is questionable whether the term ‘tranquilizer’ should be retained.”

This is an exact quote from the report of the Collaborative Study Group, National Institute of Mental Health, U.S. Public Health Services, published in Archives of General Psychiatry, Vol. 10, p. 246, March, 1964.

Psychotropic drugs of this class have been called ataractic and neuroleptic. For some time they were described as the “major” tranquilizers to distinguish them from the “minors” of the antianxiety type. These agents are unrelated chemically to previously known sedatives or sleep-producing compounds. Pharmacologically, psychotropics differ in these respects: they do not (in overdose) produce anesthesia; they tend to increase muscle tone of the tongue and face and lower the convulsive threshold; and they have a negligible production of drug dependence or habit formation. As the group name implies, drugs of this kind have striking effectiveness in the treatment of psychoses, such as the schizophrenias. They are frequently useful in small doses as antianxiety agents, although their muscle side effects limit their general usefulness.

In the early fifties, the first of these new drugs, reserpine (Serpasil) and chlorpromazine (Thorazine) were introduced. These are specifically antipsychotic in their effect. The best of the new drugs normalize some patients without producing sleepiness. They have revolutionized the treatment of one type of schizophrenia and have

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slowly, but so positively, improved patients that most can now be treated at home or in the medical sections of general hospitals. Let us summarize the effect in man of the three chemical classes of drugs now available.

The Oral Antischizophrenic Drugs

Chemical Class	Phenothiazine Type	Butyrophenone Type	Reserpine Type
(100)	Thorazine*/ Largactyl (chlorpromazine)	(2) Haldol (haloperidol)	(2) Serpasil (Reserpine*)
(100)	Mellaril* (Thioridazine)		
(50)	Compazine (Prochlorperazine)		
(50)	Serentyl (Mesoridazine)		
(10)	Moban (Molindone)		
(10)	Loxitane (Loxapene)		
(10)	Trilafon (Perphenazine)		
(8)	Navane (Cis-Thiothixene)		
(5)	Stelazine (Trifluoperazine)		
(2)	Prolixin (Fluphenazine)		
(2)	Permitil		
(X)	Equivalent antipsychotic dose. For example: 100 mgm. Thorazine = 2 mgm. Prolixin.		

*Most sedative or sleep producing

The Intramuscular Antipsychotic Drugs

I.M. Prolixin enanthate** I.M. Haldol I.M. Reserpine
I.M. Prolixin decanoate Haldol decanoate

** (Long acting—single injection lasts 2 weeks)

Thorazine or Trilafon are used routinely in the muscle to calm agitated patients. Intravenous Valium, Sparine or Sodium Amytal may sometimes be used to calm agitation. These drugs are not perfect and any one or more of the following side effects may occur.

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1. Tightening and contractions of the muscles of the eyes, tongue, face, or neck.***
2. Restlessness, such as floor pacing (apparent agitation).***
3. Tremor of the hands and arms.***
4. Blood disorders, such as decreased red or white count.
5. Yellow jaundice.
6. Psychiatric depression.
7. Malignant neuroleptic syndrome.
8. Convulsions
(These symptoms 4 through 8 are serious symptoms.)
9. Increased sensitivity of the skin to sunlight.
10. Increase in appetite and body weight.

***All of these muscle symptoms are relieved by Benadryl or Atropine.

All of these drugs are more or less effective in hallucinations and paranoia which is the type of schizophrenia with high copper and low blood histamine. However, the drugs do not correct the biochemical cause of the hallucinations. Only when the excess copper is avoided will the patient stay free of hallucinations without drugs.

For the agitated, hallucinating patient (when nothing else is available) the oral use of 50 to 100 mgm. Mellaril every hour is highly effective. Oral Thorazine is not nearly as effective. Some of these symptoms are simply annoying and will disappear with time. However, let the physician be the judge, since he has antidotes for many of the symptoms.

In addition to the above the antiepilepsy drugs, such as Dilantin, may be used in those patients who have a high absolute basophil count, are histadelic, or are allergic. All patients labeled schizophrenic are vitamin and mineral deficient and are, therefore, apt to respond abnormally to the anti-schizophrenic drugs and even to the protein in their foods. Therapists are apt to give a large dose of

Haldol or Prolixin to these nutrient-deficient patients so that a seizure may result. The patient is then classed as allergic to the drug, whereas the actual cause was that too big a dose was given to a malnourished patient. The malnourished teenager may become an obligate vegetarian because vitamin B-6 deficiency causes a bad reaction to all animal protein. Both protein and drugs depend on adequate vitamin B-6 for their normal destruction and utilization.

Megavitamin Therapy

Niacin (nicotinic acid; B-3) was discovered as the anti-pellagra factor in 1935. Before 1950, Dr. Anita Washbourne of Wisconsin reported that students with emotional difficulties did better when given large doses of niacin. The first study on schizophrenics was done by Drs. Abram Hoffer and Humphrey Osmond in 1952 to 1954. A controlled study was completed in 1954, which showed either niacin or niacinamide therapy to be better than dummy capsule therapy. In the next five-year period, the results showed:

	No.	No. Hospitalized	No. Suicide
Control patients	98	47	4
Niacin patients	73	7	0

The dose of vitamin needed is large, namely 3.0 grams per day. Larger doses are, however, used by internists to lower the blood cholesterol. The physician can also use niacinamide, since these large doses of niacin produce a reddening of the skin in the blush area. This flush is more severe in blonde patients and children, who may also complain of itching in the reddened area. In these patients, the doctors recommend niacinamide, which does

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not produce the reddening of the skin in the blush area. In older patients, however, niacinamide may produce sedation and psychiatric depression; and in all patients with large doses, some degree of irritation of the stomach may occur. In its extreme form, this irritation produces nausea and vomiting.

Since these initial studies of the schizophrenias and nutrient therapy, more effective treatment regimes have been devised. Our accumulation of knowledge concerning the nutritional balance of the body and the various biochemical types of schizophrenia have allowed the implementation of various specific nutrient therapies tailored to the demands of their mental illness and its subsequent biochemical imbalance.

Fashionable Tegretol Therapy

The use of drugs in psychiatry runs in fashionable trends like narrow or wide neck ties. When enthusiastic therapists are not doing expensive undiagnostic tests such as the dexamethasone suppression test or having patients buy expensive rose-tinted plastic glasses, they are trying Tegretol (carbamepazine) for all types of illness. This anti-epilepsy drug can produce lethal depression of the bone marrow so informed consent forms must be used for protection. Blood counts are done daily or weekly and when the white blood cell count nosedives the drug is discontinued. Tegretol is the very last resort of the anti-epilepsy drugs to be used for seizures, but now it is the first drug to be tried in many hospitalized psychotic or manic patients. When Tegretol substitutes successfully for Lithium, the patient is in a precarious situation on a medication which is much more dangerous than Lithium.

Reserpine: The Essence of India Snake Root

Reserpine was introduced in 1952 and produced a remarkable relief of symptoms in chronic, female, backward schizophrenics. We had a 50% social rehabilitation rate with reserpine at Manteno State Hospital. Doctor J. L. Beilant of Southern California Medical School in the J. Clin. Psychopharm. 6 p. 180 (1986) reports similar results in patients at Canyon Manor in California with daily doses of 0.75 to 5.0 mgm. Twelve of 18 females while 6 of 18 males responded dramatically. As we used reserpine, doses above 5 mgm. could produce pulmonary edema and sudden death. However, used carefully the chronic, non-responsive patient may deserve a trial of reserpine if all else is unrewarding.

Drugs such as the major tranquilizers should be considered as temporary crutches which can be used until the biochemical imbalances are slowly corrected by nutrient therapy. Tranquilizers, if continued at high doses for many months, may produce tardive dyskinesia—a persistent movement of the muscles. Manganese taken daily in doses of 50 mgm is helpful as is also the daily use of deanol which builds up acetylcholine—the normal working hormone in muscle contraction.

Another problem is polypharmacy. Many psychiatric patients take many drugs simultaneously. All this adds up to a potentially dangerous constellation of pharmacological interaction and personal neglect of the patient which might prolong suffering and delay rehabilitation.

Studies have indicated that only a few patients on antischizophrenic drugs require an anti-Parkinson drug for a prolonged period. And while psychopharmaceuticals can have great beneficial effects, the lethargic, asocial, odd behavior of some patients which is usually attributed to illness may well be the result of medication. In these patients, when dosages are reduced or drugs



Figure 14: Drugs are like skyrockets. They have a great display, great impact, many side-effects, and possibly a final burn-out.

discontinued, a favorable transformation occurs, the patients become more sociable and much of the odd behavior disappears.

Irrational psychopharmacology may have any number of factors at its root—understaffing in hospitals, lack of interaction between patient and doctor, discrepancies between scientific understanding and clinical use, and the effects of drug advertising, which doesn't always serve the best interests of responsible medical practice. Whatever the cause, it is important that the patient know that there are viable alternatives from which to choose. Adequate diagnosis, improvement of diet and treatment with specific nutrients is the first step toward a more effective and tolerable treatment. If needed, a drug such as Haldol or Prolixin may be substituted for Thorazine. These produce fewer side-effects and can be used as "holding drugs" until the nutrients take effect. A "pharmacological lobotomy" is not at all necessary, nor is the frustrating disruption of the patients' imaginative resources.

Chronic Effects of Haldol or Prolixin

Prolonged use of Haldol or Prolixin (without extra nutrients) can result in tardive dyskinesia (TD)—chronic muscle movements of the tongue, facial, neck and arm muscles. The nutrients which will help prevent this chronic illness are manganese with choline or deanol. Once the disorder has appeared, the use of choline, deanol and manganese may take weeks and months to correct the abnormal movements of TD.

The neuroleptic malignant syndrome (NMS) is yet another sometimes lethal side effect of antischizophrenic medication. More than 20 publications have depicted the sad effects of prolonged use of the antischizophrenic drugs. Patients may get elevated temperature, sweating,

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rapid pulse, panting, soiling, rigidity, dazed mutism, stupor, and coma. If the drug is not withdrawn, death can occur within 24 hours.

Summary

Since antischizophrenic drugs prescribed by the therapist can be disabling and even lethal, the therapist should explore nutritional approaches to therapy which will prevent side effects of the drugs and allow for the correction of the diagnosed biochemical imbalance. Nutrients do not have lethal side effects.

ANTI-ANXIETY DRUGS IN SCHIZOPHRENIA

Drugs, such as alcohol, Restoril, Ativan, Halcion, Serax, Xanax, Tranxene, barbiturates, Miltown, Librium, Valium, Doriden, Noludar, chloral hydrate, paraldehyde, and many others, have a strong sedative or anti-anxiety effect. They may be useful in an emergency, and the physician may use them in conjunction with the more effective antipsychotic medication. Anti-anxiety drugs should not be used as the only medication for treatment of the schizophrenias. Fully one-fourth of all alcoholics are chronic schizophrenics, who have found that alcohol as a sedative in gross overdosage will give them a weekend of relief from their profound overstimulation.

As with alcohol, all anti-anxiety drugs have a varying degree of drug abuse (habit-forming) liability, which is not true of the antipsychotic drugs. Schizophrenics who are hooked on drugs, such as the short-acting barbiturates, have a double disorder, which may be difficult to diagnose and treat unless these patients are completely frank with their physician.

Intravenous Valium will make the pre-psychotic patient lose all control so that mania may result.

THE USEFULNESS OF LOW DOSE LITHIUM

Although lithium therapy for overexcited patients was discovered way back in 1949 by Dr. John F. J. Cade of Australia, its medical acceptance has been slow. To this date, lithium therapy is legal only for the treatment of the manic stage of manic-depressive disorders. This remains the only legal use of lithium. Numerous publications have appeared indicating that lithium therapy is also useful in chronic depression, premenstrual depression, excess thyroid secretion as in hyperthyroidism, treatment of alcoholics, and anorexia nervosa. Because of the excellent studies of a Danish investigator, Denmark and other countries have allowed lithium therapy for the treatment of other disorders since 1960.

At the Princeton Brain Bio Center, we have used lithium in schizophrenia and other patients for 25 years. Although lithium has no effect on hallucinations, it does allow the non-hallucinatory patient to reduce his effective dose of major tranquilizers. This reduces the side effects of large doses of drugs such as Prolixin. The patient is also made better able to tolerate hallucinations while on lithium therapy. Delva and Letemendia (1982) estimate that 1/3 to 1/2 of all schizophrenic patients may benefit from the use of low dose lithium therapy. Because their study shows no patients with signs of clinical deterioration, the implementation of low dose lithium treatment appears to be an almost no risk situation.

Lithium has few side effects because its actions are specific. According to Dr. B. S. Levy (1968), "The patient receiving lithium treatment is alert without lethargy or sedation. It seems clear that lithium is the ideal therapeutic agent for acute and chronic mania. It is also very effective for the hypomanic states whose frequent recurrence leads

to deterioration of the patient's social situation. In this type of patient, lithium is superior to other drugs which produce only brief symptomatic improvement and a large amount of sedation."

As Reilly and colleagues (1984) hypothesize, the selectivity of lithium in comparison to other neuroactive compounds results from its regional and specific binding to receptors in the brain. Lithium has been shown to influence receptors in many areas of the brain, including the acetylcholine receptors in the caudate nucleus, the opiate and dopamine receptors in the corpus striatum and the cerebral cortex, the serotonin receptors in the hippocampus, and the GABA receptors in the striatum and in the hypothalamus. Many of these areas, in particular the basal ganglia and the limbic system, have been continually implicated in the etiology of the schizophrenias.

Levy continues, "What has caused even more interest in lithium is that it appears to be active as a prophylactic agent against recurrent psychotic depression. Studies have shown that lithium given prophylactically to patients with recurrent depression is able to substantially diminish the depressive attacks. This effect holds true whether the patient has shown only depression in the past or has had alternating phases of mania and depression. If used prophylactically, lithium requires a dosage with few side effects and causes no restriction of normal emotional expression." Alexander and colleagues (1979) in a study of lithium use in schizophrenics and schizoaffectives heralded lithium not only for its actions as an antipsychotic, but also as an antimanic and an antidepressant. Few other agents can tout such broad effectiveness.

Some professionals, motivated by inexperience (and their desire to fill hospital beds), tell patients that "lithium therapy can only be started in a hospital where daily lithium levels will be run." This is untrue! We and others have found that adults ranging in age from 12 to 50 years

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can be started on two 300 mg tablets of lithium carbonate per day. On this dose, lithium levels can safely be determined at monthly intervals. Patients frequently do well on only one or two tablets of lithium per day. The mean lithium level produced by this low dose therapy is 0.4 meq/L. The posted therapeutic level for mania is 0.6 to 1.3 meq/L. Therefore, at 0.4 meq/L, patients are well below the level that might produce any untoward reaction. Sometimes, we suggest that the yet unimproved patients start lithium therapy (2 tablets per day) two weeks prior to their next scheduled visit, and do a blood serum lithium level upon their arrival.

In animal studies, Manfred Anke, Ph.D., of the Karl Marx Veterinary School of Leipsig, Germany, found lithium to be an essential element needed by the goat and the miniature pig. Deficient animals lie dormant with no muscle tone. In humans, patients on lithium therapy manifest an unusually elevated white blood cell count. These findings suggest that lithium may be a limiting factor in the production of white blood cells which act as infection fighters. Lithium, easily measured via hair tests, may actually be deficient in individuals plagued by a chronically low white blood cell count, although no studies of this kind have been performed to our knowledge. Lithium levels between 0.2 and 2.0 meq/L increased growth in domestic fowl (Ocanoff et al, 1985), again suggesting that lithium may be an essential trace element in all mammals.

Although lithium therapy at low doses has minimal side effects, they become more severe as the dose increases. Serum levels up to 0.4 meq/L (1-2 tablets/day) may produce a fine tremor of the hands and slight tiredness. Serum levels between 0.5 and 0.8 meq/L (3-4 tablets/day) can produce nausea, diarrhea, polydipsia, polyuria, and hypothyroidism. High dose lithium therapy with serum levels between 0.9 and 1.5 meq/L include the

previously mentioned symptoms, gross tremors, lethargy, tiredness, and memory loss. Such high dose therapy is not warranted as lithium may be combined with other neuroactive therapies.

Lithium is compatible with all tranquilizers, vitamins, nutrients, and antibiotics. Some reports indicate that haloperidol (Haldol) or thioridazine (Mellaril) in combination with lithium therapy may be neurotoxic and often lethal. One must remember, however, that this is probably true of any major tranquilizer when used in conjunction with large doses of lithium. With tranquilizers, only small doses of lithium are justified. Lithium should be avoided in patients on digitalis or diuretic pills.

In addition to its neurological effects, lithium offers three advantages which are often overlooked by therapists. Because lithium is excreted slowly from the body, it allows for a prolonged therapeutic effect. Second, because lithium can be easily measured in the hair or blood, the level provides a useful parameter against which one can measure patient compliance. Third, an overdose of lithium is unlikely due to the fact that as few as three tablets will cause nausea and vomiting.

As with most new therapies, the medical community has been slow to recognize the usefulness of lithium therapy. Much of the confusion surrounds high dose lithium therapy which appears to be unwarranted in even the most severe cases. Despite the confusion, low dose lithium has proven to be a powerful adjunct to many accepted neuroactive treatments. Reports concerning the possible essential nature of lithium may conclude that the majority of the population, not merely psychiatric patients, could benefit from low dose lithium supplementation. We anxiously await forthcoming research in this area.

CONCLUSION/PHILOSOPHY

THE HYPNAGOGIC STATE: A NATURAL PHENOMENON

‘Suspensions amongst thoughts are like bats,
amongst birds, they fly ever by twilight.’

— Francis Bacon

The term hypnagogic is derived from *hypno* meaning sleep and *agogos* meaning induce. The hypnagogic state refers to the numerous sensory and motor responses which characterize the drowsy interval between waking and sleep. We all have experienced motor effects as our brain goes to sleep at night; a leg or an arm will shoot out or our entire body twitches and rudely awakens us. A similar event occurs in the sensory part of the brain resulting in visions and hallucinations as we fall asleep. These hypnagogic phenomena have been described many times and are quite normal.

One of the earliest printed references to hypnagogic events is found in the autobiography of astrologer Simon Forman, written in 1600. Commenting on his childhood, Forman noted, “So soon as he was always laid down to sleep he should see in his visions always many mountains and hills come rolling against him, as though they would overrun him...” Just as dreaming is seen to be healthy and normal, one should accept these states as healthy, despite their sometimes striking vividness.

The literature labels a variety of psychological phenomena as hypnagogic including among others, spontaneous visual, auditory, and kinesthetic images, qualitatively unusual thought processes, verbal constructions, tendencies toward extreme suggestibility, and symbolic representations. Although little is known about the mechanisms of memory storage and retrieval of such experiences, many have studied the qualitative nature of the hypnagogic state. Visual hallucinations consistently

follow a pattern of sequential development beginning with flashes of color, light and geometric shapes, followed by faces and static images, and finally more complex scenes. Auditory images are usually composed of the person's own name being called, visual figures speaking, and music or chimelike sounds. The perception of time is greatly accelerated while in a hypnagogic state, unlike in dreams where time is perceived as relatively normal. Physiologically hypnagogic experiences have characteristic brain wave recordings represented by a decreased frequency and a depressed amplitude.

Original studies suggested that hypnagogic images decrease in frequency and intensity as a function of age. However, recent findings have found this to be a gross misconception. Certainly a number of social and environmental factors influence the hypnagogic state; however, its presence should be a sign of normality at any age.

Drugs, particularly LSD and mescaline derivatives, greatly intensify hypnagogic images. Certainly this is due to the hallucinogenic nature of these pharmacological agents. It is not yet clear whether the production of the drug-induced imagery and the hypnagogic imagery represent similar physiological mechanisms.

Despite the potentially distressing nature and their frequently disturbing "deja vu" aspects, such experiences represent a state of mental health rather than mental illness. Clearly, one should not be concerned about the appearance of hypnagogic phenomena.

HOW ABOUT ME AND THEE AND OUR SCHIZOPHRENIAS?

If we consult the books on psychology or psychiatry we find two difficulties: one is that many behavioral signs of schizophrenia are described without distinguishing how these are different, if at all, from the same signs in normal people. The other is that great disagreements exist among psychiatrists about the significance of the clinical behavioral features. The critical reader of these books usually ends up with real doubt as to just how schizophrenia can be recognized in patients, let alone in me and thee!

For example, the schizophrenic has disperceptions and hallucinations, but we also may have mild hallucinations when disperception overtakes us while driving a car at dusk before our eyes are dark-adapted or, even worse, while driving in a fog or snowstorm when visibility is impaired. This disperception can produce illusions and hallucinations which are most uncomfortable and dangerous. This may be similar to sensory isolation, which is known to produce hallucinations. Also, the sleep-deprived subject, after two or three days, starts hallucinating and may become very paranoid. Paranoia may occur even in the normal individual when sleep-deprived.

For example, one common claim is that schizophrenic patients suffer from a "disorder of thinking." While this is often invoked in making the diagnosis, the exact definition is hazy since the disorder may involve only abstract thinking on a single subject matter, such as radio waves or the police. Moreover, to us and the patient, a disorder of thinking may vary with the time of day, since we know that, on awakening, our thoughts may be disordered. Some people think more clearly in the morning, while others are mentally more alert in the late hours of the evening.

What aspects of schizophrenics are easy to recognize and important to the relatives? In other words, what are the manifestations which distinguish the illness, so that people without technical training can distinguish it from other disturbances? There are several telltale areas in which untreated schizophrenics have difficulty which can be recognized easily. One is their inability to adjust to different circumstances.

In dealing with changing circumstances, we can always be most effective if we have at our command a diversity of ways of acting and reacting. In addition, we need mobility in switching from one approach to another so that we can adopt the most effective way quickly. While doing this, we must be able to weigh each solution in turn to see if it will work, or whether another should be used. This testing and learning process provides diversity and mobility to our thoughts and acts. The untreated schizophrenics lack this elasticity altogether. They will only deal in original stereotypes and behave the same regardless of how inappropriate or maladaptive the action becomes. They do not modify or learn from experience. How well or how badly they get along in life depends on 1) how severe the incongruity becomes between this stereotyped behavior and the real circumstances, and 2) how well the nutrients, drugs and somatic therapies restore the normal process. This abnormal process cannot be modified by talking therapy.

Secondly, the untreated schizophrenics act as if they are entirely selfish and unable to love or even be fond of a dog, let alone a human being! The mark of a sound and mature personality is caring for other people, being able to love others. Normal people growing up come to recognize their own mortality and imperfection. One real solution to the stress and the other trials of life is the formation of intense emotional relationships usually with one's family or friends, in which one comes to care as much for others

as one's self. This brings enrichment of life with warmth, closeness, and mutual good deeds, which can be achieved in no other way. Like other mental patients, but to a far more severe and intractable degree, the untreated schizophrenics are hampered in their capacity for forming such close relationships. If untreated, they may live entirely alone on a little island of self-absorption or grandiosity. Attempts to elicit feelings of friendship are met with indifference, contempt, or hostility. Dealings with others are strictly on an even exchange basis, oftentimes more or less bizarre in nature. While other mental patients also are often like this, they are not so completely rigid.

Once the talking therapist gets past the defensive fears of the schizophrenic, the intensity of the available feeling and loyalty of the patient can be overwhelming. The therapist must be utterly honest and unafraid. The patient demands more naked honesty than some guarded therapists can give. If the therapist cannot be utterly honest, the patient will remain cold and withdrawn. The schizophrenics, when they consider it safe, are usually much too frank and loving.

Humor combines these two aspects of human living. Humor requires complex and rapid rearrangement of ideas, which is a difficult process for the fixedly rigid mental processes of the untreated schizophrenic. In addition, humor basically concerns people and their interactions with each other. In this respect, it often serves as a useful safety valve for tensions. The ability to laugh off things that are distressing and yet cannot be changed is also completely absent in the untreated schizophrenic. These patients are likely to perceive humor as insulting, and instead of smiling, they react like Tam O'Shanter's wife: "Gathering her brow like gathering a storm, nursing her wrath to keep it warm."

Normal appreciation of humor returns with treatment. For example, a paranoid schizophrenic on the mend

related to us how jealous he was of his wife. When he was on the 4:00 p.m. shift he called home frequently to try to catch her out, which would mean she was out with another man, of course. We made him laugh at himself when he realized that he never called home when he was on the 8:00 a.m. to 4:00 p.m. shift. He was suspicious of his wife only when he worked on the evening shift, but, if unfaithful, she had the same eight hours to cheat in the morning as in the evening.

Thirdly, we would like to re-emphasize that the schizophrenic suffers from a distortion of the world around him, which is called *disperception*. This is like looking in a shattered or distorted mirror in a house of horrors. The *disperception* may involve all of the five senses, the sense of time, the size of the body, and perception of those around. These distortions may occupy the mind and give rise to inappropriate grimaces. Personal space may be distorted so that the patient talks too loudly with the head only 12 inches away, instead of the usual more comfortable two to three feet. Lack of vocal volume control occurs. Some of these *disperceptions* are measured accurately in the HOD and EWI tests for schizophrenia.

Finally, as we have previously noted, the schizophrenic is frequently overstimulated, perhaps from some chemical in his body. This results in insomnia, fatigue, and restlessness.

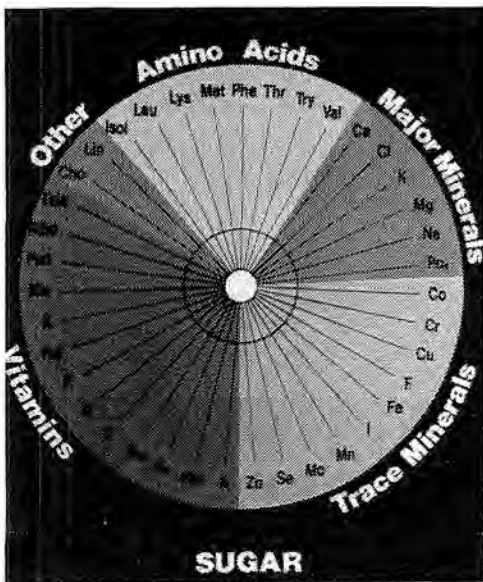
Thus, schizophrenia can be recognized by these four signs: 1) stereotyped behavior, 2) the ability to make enemies rather than friends, 3) the various *disperceptions* which alter behavior, and 4) the overstimulation which may be so severe as to turn day into night and night into nightmares.

How about thee then? Looking at yourself, what do you find? Do any or all of these four marks of schizophrenia operate so strongly as to interfere with your normal

CONCLUSION/PHILOSOPHY

day-to-day living? Are you one of the walking wounded, the non-hospitalized two percent who might benefit by modern day treatment? As you ask yourself, so I will ask myself. I hope that for both of us these signs are not present, but if they are we now know what to do about them.

SWEET BUT NOT NUTRITIOUS



Sugar is man made... Honey is bee made... there is little difference in nutrient value.....Sugar nothing, Honey a tiny amount. All the natural ingredients which made the sugar cane plant grow green and tall have been removed by man. There are no proteins, vitamins or minerals... only EMPTY CALORIES which rob the body of important nutrients.

On these charts, all 39 nutrients are essential to human life. There are no nutrients in sugar.

The average American consumes $1\frac{1}{3}$ teaspoon of sugar every 3 minutes, 24 hours per day, 200 pounds a year. Sugar makes all worse. Sugar leads to imbalance in the calcium-phosphorous relationship. Sugar can make you fat. Sugar can contribute to diabetes, arthritis, tooth decay, nervous disorders, PMS, acne, hypoglycemia.

**REFERENCES AND BOOKS FOR
FURTHER READING**

BOOKS FOR FURTHER READING

The average layman involved in schizophrenia, because of self-interest or the illness of a relative, will only pursue reading matter that pertains to his immediate interest. Therefore, if the relative is hospitalized, he is more interested in the new media that may tell of new advances. Such a reliable magazine is the "Journal of Orthomolecular Medicine," the only scientific periodical devoted entirely to nutritional treatment. This is available with a participating membership in the American Schizophrenia Foundation. Other important articles may appear in the "Journal of Nervous and Mental Diseases," "Biological Psychiatry," and "The Schizophrenia Bulletin."

For background reading, these older books are recommended and may be found in the public library.

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The exact references are available from The Princeton Brain Bio Center and even updated information continues to be filed each week on all of the topics discussed in this book.

A letter to the P.B.B.C. on your professional stationary will result in up-to-date references on any given topic. Depending on the time involved and the nature of the request, a donation to the P.B.B.C. will be suggested. We are a not-for-profit charitable organization with tax exempt numbers as follows:

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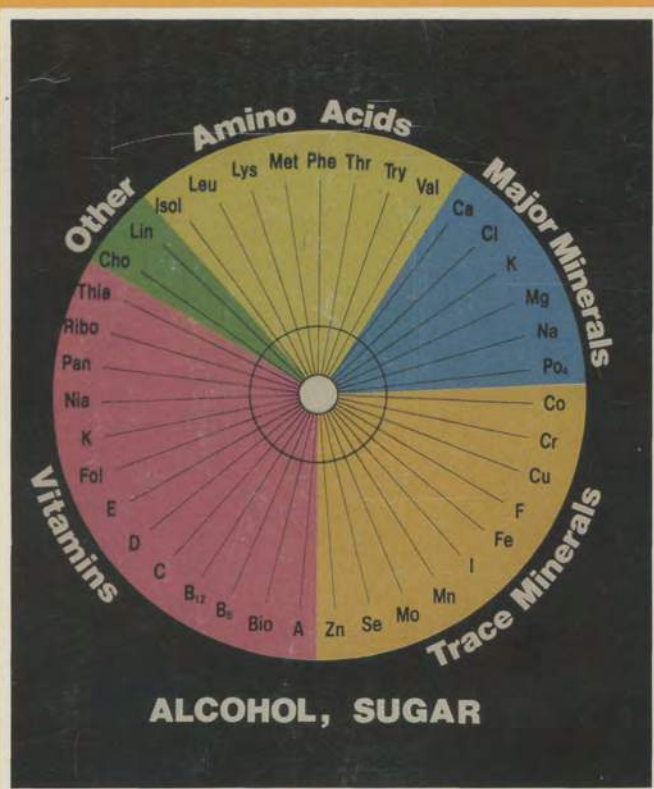
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WARNING! THESE TWO ARE DANGEROUS TO YOUR HEALTH



ALCOHOL

- Can cause malnutrition
- Destroys nerve cells
- Damages the liver
- Interferes with digestion
- Can cause heart disease
- Can cause seizures
- Can cause headaches
- Can cause ulcers
- Dehydrates brain tissue
- Is habit forming
- Is a man-made and dangerous sugar

SUGAR

- Is an "empty" calorie
- Starves brain cells
- Can cause diabetes
- Can cause hypoglycemia
- Can cause skin problems
- Can cause hyperactivity
- Can cause nutrient deficiencies
- Can cause tooth and gum disease
- Is habit forming
- Comes from cane, corn, or beet sugar and is man-made!