Meditation, Protein, Diet, and Megavitamins in the Treatment of a Progressive, Iatrogenic Cardiac and Psychotic Condition

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In dedication to my wife who comforted me all the way.

Doctors *may find it difficult to really understand the inner, subjective world of pain, fear, depression, or any major discomfort, unless they have themselves experienced these feelings. When they have themselves experienced these symptoms they perform a very valuable service by telling the rest of us what it was like. This should make us all better physicians.*

For nearly two years Dr. A. De Liz suffered from physical discomfort due to a cardiovascular problem with angina and high blood pressure. He was treated by conventional medication. Slowly his world began to disintegrate. He developed perceptual changes, primarily visual illusions, and on one occasion auditory illusions. But his insight that these changes were in himself, not in the environment, remained with him most of the time. Once on a flight to Europe he had to check the reality of the auditory illusion by asking his neighbor if he also heard it. When he discovered he had not, he concluded he was insane. In my opinion he was not; he was close to it, but he had not yet reached John Conolly's definition of insanity, "a disease of perception combined with an inability to tell whether the changes were real or not." Dr. De Liz was well able to determine the changes were not real. He suffered, however, from a drug-induced schizophrenic syndrome.

Fortunately when he relinquished the cardiovascular drugs he recovered rather quickly, except that he still suffered from angina. Only after he had placed himself on an Orthomolecular program did he make a recovery. The cause-and-effect relationship is clearly established for him.

Since individuals may react in an undesirable way to any medication, it is essential that when any psychiatric change occurs coincidental with the consumption of drugs, these be examined as possible causative agents.

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A certain degree of soreness on the chest led me to consult an internist in Brooklyn, N.Y., in November of 1973. The physician found an abnormal EKG, high blood pressure (110 diastolic and 160 systolic). He also noticed a decrease in circulation on the left posterior tibial artery. The heart was not enlarged. I had a pulse rate of 95, rhythmic, lungs were clear, blood count and biochemical profile were normal. Mild diabetes was found. In view of the abnormal EKG, the internist suggested hospitalization. However, because I felt well at the time, I refused his recommendation and continued practicing pyschiatry with a daily load of 10 hours, and during about two years I did not have a recurrence of the soreness of the chest. My blood pressure had dropped to diastolic 95, systolic 145 under Aldomet and the salt-free diet that the internist had prescribed.

Two years later, because of a sense of general weakness, I decided to be examined again by another internist who also found an abnormal EKG which looked like a carbon copy of the first one. Just on the basis of this finding, the internist vehemently advised me to enter the coronary unit of the Nassau Hospital in Mineola, L.I., N.Y. Three weeks after I was discharged with Isorbit, 10mg t.i.d., and Inderal, 10 mg t.i.d., I felt for the second time a sharp pain and soreness in the chest. I informed the internist about the situation. His orders were that Inderal should be increased up to 160 mgs per day, during a period of two weeks. I followed his recommendations and got worse. In fact, the anginal pain increased tremendously both in frequency and intensity. To calm the pain, I had to take Isorbid 40 mgs every five hours, day and night. During the night, the pain would wake me up several times and I would seek relief by taking Isorbid.

After six months, new alarming symptoms became manifest, namely agitated depression of moderate degree, irritability, and a sense of unspeakable and shapeless fear, a kind of premonition of imminent death and nothingness in which I would soon be lost. It was a sort of nameless and amorphous threat, a semi-awareness of an insistent but indefinite power. I felt as if torn from the familiar, the reliable, the stable. Shocked, suddenly uprooted, disturbed, faced with the unusual, I looked at me and about me with wild surmise. I was becoming aware of being mentally sick, and this feeling reduced me to a state of complete helplessness. And yet, I wouldn't dare to reveal these feelings to my wife, let alone to a psychiatrist. This situation continued unaltered until the beginning of 1976.

Meanwhile I noticed that things and people about me looked different, both in shape, color, and facial expression. Then, little by little, I felt more confused still and I would dwell in the past and fuse and confuse earlier events of my life with present experiences. Then I had the delusion of being expelled from the clinic where I was working or, for example, that the absence of my Ph.D. title in a scientific program was causally linked with some malignant intention.

During all this time, I kept asking myself what could have been the reason for this progressively altered personality, but I would not understand. There was not a single case of psychosis, neither among my paternal nor maternal ancestors. On the other hand, I had not been subject to emotional stress that might be said to have evoked my abnormal reaction.

While I was reading a scientific paper in a medical convention, I became dizzy, lost equilibrium, and fell to the floor. Next day, physicians made the diagnosis of acute congestive heart failure. I had right lung congestion and blood sputum. The anginal pain increased even more and, in the Emergency Department of the Nassau Hospital, L.I., N.Y., I was told to reduce the Inderal to 40 mgs, t.i.d. and to take Lanoxin and Aldalactazide. After eight days in bed, the heart congestive failure subsided and the internist gave me permission to fly to Wiesbaden (Germany). However, during the flight I had a dramatic pyschotic episode. First and foremost, I developed auditory

hallucinations. In fact I heard a kind of inhuman moaning which seemed to me to come from the plane's engines. I looked around in terror, expecting to detect some similar reaction in the other passengers, but everybody was calm and relaxed.

Yet I asked my companion seated next to me: "Don't you hear that strange rumbling noise?"

"What noise?" he said. "The only noise I hear is the one caused by the normal functioning of the engines."

Frightened to death, I realized that I was insane. It so happened that I had previously decided to land in Paris where my sister was waiting for me. Not having seen her for two years, I had the feeling that her face was not precisely that of my sister but of someone resembling her. She was extremely worried about my appearance and my state of disorientation. I was seen by a French internist who could not understand my physical symptoms nor my pathological condition. He believed that I had been victim of a cerebral hemorrhage.

My anginal pain was as intense as ever when my wife decided one week later to take me to Wiesbaden. When walking in the city, my legs felt very weak and suddenly they gave in and I fell without loss of consciousness. After being observed by a German physician, I was told that my left ventricular's contracting power was below normal and that my blood pressure was 115 diastolic and 222 systolic. He prescribed Aldactone and Digoxin and ordered me to bed as I showed reluctance in going to the hospital.

When after one week my blood pressure had dropped to 100/160, the internist permitted me to take the plane to the U.S. Upon my arrival, I consulted a cardiologist of great reputation at the Long Island Jewish Hospital. He confirmed an abnormal EKG with the following tracing:

Regular sinus rhythm with ventricular rate of 70 per minute, T wave inversion in lead one, AVL, V4, V6 and Q in 2, 3 and AVF suggestive of old inferior wall infarction with anterior wall ischemia. He also found a weaker pulse pressure on the left posterior tibial artery. The heart was not enlarged.

He also stated: "The patient gets, at present time, angina at rest with aggravation nocturnally and whenever he walks a distance of a block." He found a blood pressure of diastolic 110, systolic 200. He also found that I had mild diabetes mellitus which might be compensated by strict diet alone. No heart murmurs were observed, but A2 was markedly accentuated and there was S4 gallop along the left external border.

His final impression was that I had angina pectoris, progressive in nature, and since the medication was not controlling my symptomatology, he felt that I should undergo cardiac catheterization, and according to the findings on cardiac catheterization, he would advise surgical intervention or further medical treatment.

Knowing that by-pass heart surgery results are very debatable and the operation itself still carries the risk of a relatively high mortality rate, I became rather reluctant to go along with the cardiologist's advice. However, my sense of mental and physical agony became unbearable.

One week later, in wild despair and yet full of an ineffable sense of faith, I attended a group meditation at the Society of Friends in Manhasset, L.I. I retreated within myself in an effort to discover my way to salvation. My meditation was an ineffable experience which was immediately followed by an otherwise unknown peace of mind, a subjective feeling that I was on the way to a kind of resurrection. Then next day, in a flash, I asked myself: "Could it be that one or more of the prescribed medications had precipitated an allergic reaction on my central nervous and cardiovascular systems?" My first move was to scrutinize in detail whatever may be known about the side effects of the medication taken during more than two years. Also I couldn't find what I was looking for under the different drugs I

was taking until I found in the Physician's Desk Reference of 1976, page 591, the following on Inderal (Manufactured by Ayerst):

Inderal {beta adrenergic receptor blocking agent) should only be indicated in selected *patients. It should not be used in patients with angina that occurs only with considerable effort or infrequent pre-cipitating factors.* Pharmacologically, Inderal is a relatively new drug about which not too much is known (page 92, Medical Pharmacology, A. Goth, 4th edition, C.V. Mosh Co., 1968).

Inderal competes with beta-adrenergic receptor stimulating agents. When the access to beta receptor sites is blocked by Inderal, the chronotropic, inotropic and vasodilator responses to beta adrenergic stimulation are decreased proportionately. Inderal is useful when excess. *of catecholamines evoke excessive sympathetic activity but not to the point of paralyzing sympathetic activity be-cause this activity is vital to maintain ventricular function. The proper objective of beta blockade therapy is to decrease adverse sympathetic stimulation, but not to the degree that may impair necessary sympathetic support of vital functional balances.*

Propanolol may reduce the oxygen requirement of the heart at any given level of effort by blocking catechola-mine-induced increases in heart rate, systolic blood pressure arid the velocity and extent of myocardial contraction. At the same time, Inderal may paradoxically increase oxygen requirements by increasing left ventricular fiber length and end diastolic pressure and systolic ejection period.

Side effects:

1. *Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of the hands, arterial insufficiency usually of the Reinaud type, thrombocytopenic purpura, and so on and so forth.*

2. **Central Nervous System:** *Lightheadedness, mental depression manifested by insomnia, fatigue, weakness, lassitude, reversal of mental depression progressing to catatonia, errors of perception and cognition, hallucination and acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded consciousness, and decreased performance on neuro-psychometrics.* In view of the confirmation of my insight that my mental symptoms were caused by one of the drugs I had been taking and having discovered that those symptoms had arisen for the first time in my life coterminously with the intake of Inderal, I logically reasoned that if my insight was correct, such a symptomatology would disappear concomitantly with the cessation of Inderal therapy. Consequently, I discontinued Inderal after six weeks of progressively reducing the amount of intake (for if it is done at once, it may precipitate a coronary thrombosis). At that time I wondered if the discontinuation of Inderal alone would abolish the anginal pain and the partial occlusion of the left posterior tibial artery. I looked for a positive confirmation for I reasoned that if my interpretation of the pharmacology of Inderal was correct, then the extreme bradycardia that the drug had provoked in me had in all probability decreased the coronary circulation itself in a coronary system assumed to be atherosclerotic and, consequently, massively leading to a much higher degree of ischemia which in itself evokes the anginalpain.

However, after the discontinuation of the Inderal, though I felt much better, I was still having anginal pain, although less severely and less frequently. The mental picture had not improved much when I reminded myself of the work of Evan V. Shute, London, Ontario (Canada), on the therapy of cardio-vascular diseases with alpha tocopherol. At the same time, as an Orthomolecular psychiatrist, I knew of the completely

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confirmed antischizophrenic therapeutic properties of niacin and of the Linus Pauling contention that vitamin C has anti-atherosclerotic effects. With the unbroken conviction that my spectrum of inferences was relatively well founded, I began my own treatment as follows: niacinamide, 3,000 mg o.d., vitamin E, 100 mg o.d., vitamin C 4 g o.d.

The fact that I started out with 100 mg o.d. of vitamin E was due to Evan Shute's observation that alpha-tocopherol may increase the blood pressure. However, as my blood pressure remained at 100/160, I increased progressively the intake of vitamin E up to 1,000 mg o.d. and still the blood pressure remained at the usual level.

Together with this therapeutic strategy, I committed myself to a strict salt-, sugar-, and starch-free nutrition with a very high amount of fat-free meats and fish.

Final Results and Conclusion

By the end of eight weeks and for the first time in two years I was free from anginal pain, the partial occlusion of the left posterior tibial artery and, of course, my mental condition.

In view of my two years of study of my own illness, the results obtained could not possibly be said to be a chance event, but rather a scientifically validated instance of cause-effect relationship. Having devised a third possibility of therapy to arrive at such a tremendous improvement of my mental and physical health proves without a shadow of a doubt the fallacy of the false disjunction: "Either the medication prescribed controls your symptoms, or we have to operate on your heart." I am sure that the medical establishment would reject this paper on the ground that it could not meet the basic paradigmatic structures of its fossilized thinking. For the medical establishment, Orthomolecular psychiatry has necessarily to be sheer delusion, a passing fad, even when it is known by everybody that the medical

establishment never carried out an unbiased, systematic research on the matter. It is precisely because of this attitude that six cardiologists almost led me close to the gates of death.

This attitude is in part reflected in the tremendous increase of iatrogenesis—diseases caused by the physician—as so profoundly exposed by Ivan lllich in his highly articulate, polemic, recent book **Medical Nemesis—The Expropriation of Health** (1976), published by the Pantheon Editor. He is concerned with the complete lack of medical thought relative to what is meant by individual, concrete, unique psychobiological concept of a sick person. The patient is rather generally assessed as an abstraction. The therapy is not applied to the unique, particular dimensions of the ill individual, but rather to a statistical image of the patient, i.e., the patient is treated as embracing the general characteristics of a class.

Consequently, when a doctor gives to a patient the kind and amount of medication that experience has shown to have been successful in the members of a given class, he commits a serious blunder. For there are an infinite number of variants in the way a human being may react to a certain medication, and it may happen—as it happened to me—that while Inderal has statistically proven to be useful in most cases, it can trigger an allergic reaction in an individual who could not in principle be a member of the class of patients who had responded well to Inderal.

My case is a vivid instance of a general unawareness of the faulty, implicit, fundamental postulates by the medical establishment. These postulates are in urgent need of revision for the detection of thinking in blinders. The disgraceful situation of the medical training is due to the fact that the student in medicine had never been exposed to the insights of philosophy of science.

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Comparing these considerations relative to the most complete clinical study presented in the literature of the subject matter under scrutiny with our own observations of my self-study, the following remarks appear valid:

1. When I was given propanolol I was not suffering from stable severe angina pectoris.
2. The truth is that I only had had one single episode of chest soreness in January, 1973, after considerable physical stress.
3. My history of daily anginal pain episodes began for the first time after I was started on propanolol, 40 mg once daily, and they were massively increased when propanolol was raised to 160 mg daily.
4. Propanolol was not discontinued even after I suffered several acute congestive heart failures.
5. None of the six cardiologists consulted in America, France and Germany had formulated the concept of cardiothoracic ratio, let alone the fact that when in a given patient this ratio is >0, 5, propanolol is contraindicated.

STONE, R. A., and De LED, J.: Relaxation and Hypertension. San Diego's VA Hospital, New England J. Medicine 294: 80-84, 1975.

In this paper a certain number of patients with mild to moderate hypertension were taught meditation techniques of a Buddhist style. After six months of two, brief, daily meditations the blood pressure reductions were correlated with changes in plasma activity of dopamine-beta-hydroxy-lase suggesting that a drop in peripheral adrenergic activity may contribute to improvement. This study focuses on the possible role of the CNS in triggering and maintaining hypertension. It suggests the involvement of adrenergic mechanisms in the etiology of essential hypertension. This means that if autonomic functions can be modified and trained in man and if such training is effective in the early phases of hypertension and more importantly if it can be maintained over a space of time, the positive feedback mechanisms at the level of both the central nervous system and the vasculature may be interrupted, and sustained hypertension may be prevented.

ABSTRACTS

WARREN, S. G. et al.: Long-term propanolol (Inderal) therapy for angina pectoris. Am. Journal of Cardiology 37-420, 1976.

In this study a number of patients with stable severe angina pectoris were given a mean daily dose of propanolol in a prospectively followed clinical observation of five to eight years. The study concludes that propanolol is effective for long-term therapy of severe angina pectoris. However the authors warn that it may increase the risk of cardiogenic shock in acute myocardial infarction, or history of congestive heart failure in which case the drug is contraindicated. It is also contraindicated, the authors stress, in mild asthma and whenever the patient's cardiothoracic ratio is »0, 5.