

Managing Cardiovascular Diseases

Ramblings of a Maniacal Frenetic – Pragmatic Reflections on Helping Patients Understand Their Illnesses and Treatments

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*My father told me, in early 1979, that he was going to see a doctor about doing chelation therapy. I went only slightly berserk, insisting that I would have heard about it in my training or residency if it had any value for cardiovascular diseases. He “wisely” stayed away from **that** charlatan. Then my mother needed drastic surgery for a bleeding ulcer in the fall of 1982. As I needed to fill my days while seeing her in San Francisco, I visited the office of Robert Haskell, M.D. We discussed nutritional medicine and dietary programs ... and then he asked, “Well, you do chelation therapy, of course?” I explained my reservation about doing any treatments that were exaggerated in their claims of helping ... especially with a wide variety of illnesses. He said simply: “Come with me.” We climbed up one flight of stairs. “Here,” he said, “is my nurse. And my charts. And my patients. Have a good day.” And what a day it was! I could barely believe the documented results of patients who had barely been able to walk due to shortness of breath or chest pains or calf pains. And I got to hear their stunning stories, in person – and to lay my hands on their bodies. I was hooked. I spent the next 5 months studying everything I could find on chelation, so that I would “ace” the written exam. At the training, I met Warren Levin, M.D., of New York City, clearly the best lecturer at the meeting. I spent two glorious learning days in his office, the same for Milan Packovich, M.D., of Pittsburgh, also for Charles Farr, Ph.D., M.D., of Oklahoma City, and for another 8 doctors who generously offered to share their best ideas with me, so that I could strive from the start “to be the best.” H. Ray Evers, M.D., of Dothan, Alabama, graciously hosted me for 3 days to see the best of the past. And thus began my saga, to “learn more and do better than anyone else.” At the very least, each of my parents and I myself benefitted greatly.*

Pump, Pipes, and Performance

Cardiovascular diseases (CVD), in order to be adequately evaluated and treated, need to be classified according to the likely etiology or explanation. Simply stated, CVD are associated with the **pump** (the heart), the **pipes** (arteries of whatever size and location), and **performance** (impaired function despite adequate anatomy). One last classification – “pediatric” – will be ignored for this article, since congenital heart diseases, as genetic or developmental irregularities, have their own unique considerations. When the “pipes” involve the *venous* system, such as with thrombophlebitis, this is treated as a special case of inflammation.

Trowbridge's Idiot's Guidelines to Diagnosing Cardiovascular (and Other) Diseases

The following questions are essential:

1. **WHO** is affected?
Environmental exposures, other illnesses/operations?
2. **WHAT** is the change (deviation) from normal structure/function?
3. **WHEN** did these changes start? **WHEN** have they progressed?
4. **WHERE** is the site ... of the ill organ/s? Of the *patient* (home/work/travel, present and past)?
5. **WHY** did the change/s occur (preceding or associated events)?
6. **HOW** did the change/s develop and worsen? **THIS IS OFTEN THE MOST CRITICAL QUESTION**

The principal part of everything is the beginning. By answering these basic “reporter’s questions,” a good start can be made toward diagnosis and effective early treatment. *Every* patient starts here, no matter how complex or easy. Following this list reduces your likelihood of skipping an important factor and heading in the wrong direction.

delivered to the tissues, including inside the brain). Larger arteries are those coming off the heart, going “out” to the organs, up to the head, and down the arms and legs, and these are often more amenable to surgical intervention. In a distressingly large proportion of operations at *any* level, surgeons often imply that “your problem has been fixed, you’re as good as new,” simply because larger or medium-sized pipes have been popped open (ballooned, often with a bracing stent as well), bypassed (skipped over), or reamed out (endarterectomy) and sometimes

Hey, Buddy, Can Your *Really* Treat That?

If we have incomplete or missing diagnoses, should you proceed with treatment? In fact, that complaint has been leveled at chelation therapists for years, that we fail to do “enough” diagnostic workup. If you want the details of your problem delineated down to the molecular level, go to your local university cardiologist. But if you want to feel better now and get on with your life, why not consider a treatment that works for *most* heart and blood vessel problems (and those of many other systems) that plague most people? Problems that don’t improve can continue to be evaluated. The only heart problems that don’t reliably show desired improvement are pediatric, because of their distorted anatomic features. The only peripheral (or central) blood vessel problems that don’t show expected improvement are (sorry, can’t recall any).

What Do People Really Need to Know?

For the most part, medical explanations use technical terms that confuse or oversimplifications that mislead. Using the framework presented here, concepts can easily be offered that lead patients into a fair understanding of the treatments proposed and what to expect. (Much of “doctoring” is *teaching*, which improves compliance dramatically.)

When discussing “heart” disease, many practitioners fail to clarify the distinctions between problems with “**pipes**” and those with the “pump.” The *vast* majority of heart conditions treated with surgery involve the pipes, namely “blockage disease” in the coronary (heart) arteries. In discussing “vascular diseases,” other *small* arteries include those in “end organs” (where blood is finally

“repaired” (patched). In actual fact, operations can be performed on just a few dozen inches of arteries but the underlying problems are widespread, affecting a distressing portion of the 60,000+ *miles* of blood vessels sustaining your body organs.

When patients understand the need to restore better blood flow, distinctions can be made between surgical reduction or removal of *blockage* compared to non-surgical ways to improve *flow*. “Blockage” is a “plumbing” concept, easily grasped. What is harder for many patients to grasp is that better “flow” dramatically relates to incremental reduction of blockage. Increasing the central channel diameter by merely 1/6th (just 16% widening of the vessel diameter) will just about **double** the flow through that vessel. (This tiny difference is difficult to “see” on angiogram x-ray pictures but is easily felt by the patient.) How could such blockage be *gently* removed? “Cardio” exercise sometimes helps. But what about reducing obstructions *naturally* ... through biological changes *induced by* IV chelation therapy. Overly simplified, EDTA chelation appears to dissolve the “mortar” that holds together the gunk that accumulates in the pipes, interfering with flow through the arteries. As the “glue” is removed, the body can safely, easily, and naturally reduce the blockage the same way that ice melts in your water glass without shattering into pieces. In fairness, sometimes very little reduction of blockage itself occurs, but gradual improvements to the nutritional status of cells can markedly improve their function and reduce symptoms earlier attributed to blockage.

When heart disease involves the “**pump**” portion of your heart, we’re looking at three distinct sets of pathologies. *First*, where blood flow has been *completely* interrupted to a small area of muscle, that tissue actually dies (heart attack or “infarct”) and forms a scar. The scar, incidentally, might later stretch and thin out (ballooning out as an aneurysm), with a greater risk for chamber rupture ... so surgery can be advisable. *Second*, the cells in an area can become “sick” from reduced blood flow (“ischemia”) or from nutritional deficiencies (magnesium, B-complex, even calcium), toxic accumulations (lead, mercury, arsenic, other toxic heavy metals), or other adverse changes (such as from organic toxins, pesticides, and so on). Affected muscle cells function less and less well, leading to alterations of normal contraction/relaxation patterns and pumping efficiency. *Third*, heart valve problems (especially for the aortic and mitral valves on the high-pressure left side) and enlargement of the aortic root or thoracic (chest) aorta are distinct anatomical problems often best treated by surgery. Recent advances are unbelievable, where certain heart valve operations (and even some large artery aneurysms) are being performed without “cracking the chest.” One exception is where calcification of valve leaflets *might* be improved by extensive IV EDTA chelation therapy, delaying the need for urgent surgical intervention ... and even improving later operative survival.

Finally, when heart disease affects the *pumping efficiency* of your heart, these are “**performance**” issues. While this category might “blur over” into the second pathological pattern described above, it is distinct in a number of ways. Foremost is where electrical conduction pathway “defects,” for whatever reason, can lead to rhythm disruptions (atrial fibrillation, others) where the pump muscle – although otherwise functionally capable – beats erratically or less efficiently. “Cardiomyopathies” (heart muscle impairments) can result not only from rhythm malfunctions but also from viral infections, nutritional deficiencies, toxic heavy metals such as mercury, decreased oxygen saturations, and even hormonal imbalances (hypothyroidism, perhaps deficiencies of testosterone or progesterone or others).

The Fire Within

Inflammation is a chemical reaction, whether in organic or inorganic systems. What causes fire damage to the “outside” – to any *structures*, from cell organelles all the way up to observable tissues – also wreaks havoc at sub-microscopic levels *inside* biological systems. At the tiniest level, we’re looking at the shifting around (actually, “stealing”) of electrons, with resulting conformational changes of the molecules. The concept is one of “free radicals,” electron-seeking molecules, first proposed by Denham Harman, M.D., in 1955. Other concepts have been advanced, many of which rely upon a basic appreciation of the central role of free radicals. For example, in 1942 Johann Björkstén proposed the cross-linkage theory to explain the “hardening” of tissues as we grow older or sicker (recall the stiff and brittle rubber band found at the back of your desk drawer). Again, electron changes are involved.

The greatest problem with free radicals is that they damage normal molecules in an accelerating pattern, somewhat like a ping pong ball (the “initiating” radical) being thrown into a room full of mousetraps, each “loaded” with another ping pong ball. The resulting “fire” is akin to a nuclear reaction, where it tends to amplify and continue until it is exhausted or quenched. In the body, “anti-oxidants” are essential to interrupt (“quench”) electron free radical damage, known as “oxidation” or inflammation. Virtually *all* degenerative diseases – including cardiovascular – are directly related to free radicals in their initiation and propagation, unrelentingly through cell injury, organ dysfunction, and finally body death. These rampant oxidative changes are *the* common denominator, and the damages to various intracellular organelles or metabolic pathways advance in their interruption to normal function to where they are finally identified as different disease “diagnoses.” Remember: all involve effectively the same inflammation chemistry.

Trowbridge’s Diagnostic Testing for Dummies: Cardiovascular Diseases

The following tests *can* be useful:

(Obviously physical exam with pulse and blood pressure and respiratory rate, CBC with differential and platelets, metabolic chemistry panel, and urinalysis, 12-lead and rhythm EKG, and CXR just to be sure “basics” are covered)

and

ferritin, homocysteine, fructosamine, glycohemoglobin, Vitamin D3, ESR, ANA (quantitative plus pattern), RA (quantitative), fibrinogen, uric acid, LDL low density lipoprotein, Lp(a) lipoprotein cholesterol, small dense LDL, Remnant Lipoprotein RLP cholesterol, HDL or HDL2b cholesterol, apolipoprotein B, triglycerides.

These factors look largely at *genetic* or *epigenetic* issues, to focus treatment on those factors where free radicals matter most.

Anatomic function testing, as described, is directed toward specific “problem” areas.

Since the disease promulgation process is similar in widely variant tissues, this *biochemical* understanding opens the door to treatment programs that can have a *generalized effectiveness* without being *specifically* targeted toward any particular diagnosis. *Enter: chelation therapy.* Clearly chelation is a dominant answer to *most* cardiovascular diseases, bar none. Surgical and drug interventions might still be needed, especially for more advanced disease patterns. But chelation remains the mainstay of treatment.

The Missing Cardiovascular Diagnosis

Repeated (even *annual!* for many “heart patients”) treadmill EKG testing has *minimal* preventive value ... but pays very well. (My closest contact with diagnostic limitations of a resting EKG

was an older gentlemen who presented with uncharacteristic discomforts at two in the afternoon ... a regular cardiogram was normal, but I was still suspicious. Hospitalized at my insistence to a continuously-monitored bed, he suffered his heart attack at midnight. “Instant” coronary care unit response meant a dramatic reduction of otherwise likely heart muscle damage.) Repeated testing of cholesterol and triglycerides has *minimal* preventive value ... but pays very well. Evolving metabolic syndrome changes, once suspected by clinical presentation and slightly elevating fasting or random blood sugars, are best evaluated by merely by clinical monitoring and only periodic testing of blood sugars with concurrent insulin levels. Genetic hyperlipidemias are more ominous and pose substantial survival risks, far more significant than the trivial implication of “your cholesterol is high at 230 and you *NEED* statins!” Even repeated coronary (heart) or aortic and peripheral (belly and legs) angiographic x-ray dye pictures (merely “maps” for surgery) in symptomatically stable patients have *minimal* preventive value and but have attendant appreciable risks. These invasive tests serve primarily a mapping function, to document progression of blockage *advanced* to the point where surgery is now desirable. And again, the angiograms pay well ... and should be reserved for *deteriorating* conditions where salvage surgery is imminent.

Coronary calcium scans (“heart scans,” coronary artery CT calcium scoring, also called EBCT for “electron beam”) are non-invasive and useful predictive monitors for coronary events. Their value is enhanced when a sequentially *rising* calcium count is documented, particularly in a patient who has been asymptomatic. An exciting development is the markedly improved sensitivity of coronary MRA, magnetic resonance angiogram (imaging of heart blood vessels) without (or especially with) use of peripheral intravenous contrast. Among the most accurate “predictive” clinical tests are the carotid neck artery and abdominal aorta ultrasounds (or even CT scans), along with the non-invasive vascular lab tests for leg (peripheral) artery disease. These usually allow tracking of credible blockage and flow patterns, but they don’t reflect the entire range of pathologies hidden inside the vessel walls.

The ideal predictive tests would be those that disclose unsuspected “tendencies” to develop more aggressive diseases. When a particular patient has a number of such genetic or epigenetic (variable gene expression, depending on environment and other factors) proclivities, he or she warrants more attention. Here is your “likely candidate” for more extensive and earlier blockage disease to progress. Since “pipe” problems can be imaged and measured, tests should be proposed as indicated by history, clinical exam findings, and abnormal laboratory patterns. [A simple example serves well: history of visual changes, ophthalmic exam showing blood vessel or retinal changes, with elevated blood sugars – clearly carotid artery studies are appropriate, perhaps even a brain SPECT scan, maybe others.] Unfortunately, the data are unclear regarding how best to predict the development of “*non-pipe*” pathologies – those of the pump or its performance. Cardiac muscle biopsy is *not* something to consider!

One predictive parameter that is grossly *underemphasized* is that of oxygen saturations. Numbers of studies have shown that decreasing nocturnal saturations – which reflect lowering oxygen tension in the blood and, hence, in the tissues and especially inside the mitochondria energy factories of the cells – are directly related to impaired pump function and performance issues. The critical continuous generation of ATP to power cellular processes is absolutely dependent on sufficient oxygen to receive and remove electrons stripped during *oxidative*

phosphorylation in the mitochondria. Energy production is perilously degraded when the anaerobic fermentation pathway is employed. When oxygen saturations are raised toward normal, improvements in tissue functions in *all* organs can be expected, including retarding of “aging” degeneration and even deferred initiation/promotion of neoplastic patterns. Otto Heinrich Warburg, M.D., nominated 47 times for the Nobel prize, finally received the unshared award in physiology in 1931 for discovery of the “nature and mode of action of the respiratory enzyme.” Concluding that cancer (and other deterioration diseases) should be interpreted as a mitochondrial dysfunction, Warburg proclaimed that “**the prime cause of cancer** is the replacement of the respiration of oxygen in normal body cells by a fermentation of sugar.” As noted in [wikipedia](#), “When frustrated by the lack of acceptance of his ideas, Warburg was known to quote an aphorism he attributed to **Max Planck**: Science progresses not because scientists change their minds, but rather because scientists attached to erroneous views die, and are replaced.” Sound familiar?

Incidentally, as mitochondria become “sick” – such as having their chemical pathways poisoned by toxic metals – they swell with and increasing calcium concentration, disrupting their shelf-like cristae fold structures, dramatically interrupting the electron transport chain. Chelation therapy has been shown to repair such injured organelles, restoring more normal energy generation. So here arises the charge that chelation can be criticized for *claiming* that it “*fixes everythin’, jus’ like some kinda snake-oil!*” But ... patients *claim* (and dozens of studies by Edward McDonagh, D.O. and Charles J. Rudolph, D.O., Ph.D., as well as others have *documented*) that chelation *does* “*fix mos’ everythin’!*”

All toxic metals are, to some extent, accumulating in endothelial linings and throughout heart and blood vessel cells as well – mercury, lead, cadmium, arsenic, and so on. Each has a separate contribution to amplify free radical production leading to functional impairment. Rodent studies suggest a marked intensification of damage when two or more heavy metals are present, even in trivial concentrations. But *toxic* heavy metals are not the only culprits. Iron is an essential element that can be present in excess (iron “storage” disorders, even mild polycythemia?), where it also stimulates the generation of free radicals that are especially toxic in metabolically active tissues such as liver and heart, even more so in compromised patients such as diabetics. Neurodegenerative disorders such as Alzheimer’s and Parkinsons have been conclusively linked to excess iron accumulation in brain tissues. Elevated cancer rates are seen in patients with iron overload. These clinical observations confirm Warburg’s contention that mitochondrial decay, mediated through amplified free radical attacks, is the root of disparate and disastrous disease patterns that steal our comfort and, ultimately, our life.

Jukka T. Salonen, M.D., Ph.D., M.P.H., of Finland, reported in 1992 a prospective study of 1,931 men with no symptoms of heart disease. Over the next three years from entry, the lifetime total of cigarettes smoked was determined to be *the* primary risk factor in those 51 patients who experienced acute myocardial infarction (heart attack). The second factor was (*not* cholesterol or blood sugar or blood pressure or obesity!) an elevated blood *ferritin* level (possibly correlated with a shift toward tissue acidosis). Beyond an accurate smoking history, this realization provides an easy laboratory test to identify those at higher risk. Ferritin levels rising ever higher above 100 ng/ml are directly associated with an alarming increasing incidence of coronary events. The iron story is, however, complicated because adequate pools of iron are

essential for life. Ferritin only slowly declines after dozens of EDTA chelation treatments – correction of other metabolic perturbations is essential. Even mild iron-excess patterns might be clinically more significant than earlier appreciated.

A toxic metal side issue is now coming to the forefront: the expanding use of injectable MRI diagnostic imaging contrast agents, such as gadolinium, iron (Feridex), and manganese (Teslascan). Urinary challenge tests with d-penicillamine in some patients have shown very high excretion levels of gadolinium. The clinical significance of these findings is variable, but chelation in patients who have had repeated contrast studies might prove very valuable. Gadolinium use has been linked to onset of rare but often crippling nephrogenic systemic fibrosis, especially in patients with reduced kidney function.

Over-saturated with Facts?

So-called “sleep apnea” is associated with magnified rates and more injurious cardiac events. Sadly, most physicians have only a passing exposure at best to the parameters of at-rest oxygenation. (The awake-in-the-exam-room-chair saturation level obtained in some offices is *almost useless* for prediction, unless the patient is in clinical distress.) “Obstructive” events are routinely blamed for desaturation events, but my experience shows that accusation is *misplaced* in the *vast* majority. Thus, patients are subjected to “scuba-torture” (CPAP pressure mask worn during the night), usually *disrupting* sleep patterns for many. Further, CPAP is notorious as a *failing* therapy within a year of two of starting, due to minimal results (of simply blowing room air) and frustrating discomfort.

Pause to consider: if you use an “*obstructive*” treatment (CPAP) for a “*central*” problem (as interpreted from “low sats”), how likely is it to succeed? *Obstructive* sleep apnea is routinely “diagnosed” by sleep labs – but my observations and clinical results over the past 22 years clearly show that *central* apnea is far more common ... and easily controlled by nasal cannula oxygen from a home concentrator during sleep. Medicare and insurance companies concede that saturations below 89 qualify for “lifetime” oxygen support. In sharp contrast, patients with “sats” from the mid-90s on down show improvements with virtually *all* heart and circulation problems, mentation, arthritis, digestive disorders, strength and vitality, and so on. Recall that robust oxygen availability is essential for mitochondria to meet the metabolic demand for ATP. *My* testing of nocturnal oxygen saturations over the past 22+ years has proven that the vast majority of desaturation patterns can be related to *central* causes. This central “respiratory disconnect” appears associated with past head injuries of any kind and/or toxic insults (heavy metals, organic and inorganic chemicals). Presumably hypoxic/anoxic shock and severe infections (especially meningitis and encephalitis patterns) would qualify, but my experience is too limited to offer those conclusions.

These desaturation issues present a glaring example of “The Missing Diagnosis.” When “modern” medicine doesn’t have (or doesn’t accept) a specific treatment program, then its regimented practitioners routinely miss the accurate diagnosis for one very simple reason ... they don’t *look* for or don’t actually *see* the problem.

Another worthwhile topic to explore would be EECF, enhanced external counterpulsation, compression therapy where air pressure cuffs squeeze on the legs during the relaxation phase of the heart beat (diastole). In a surprising number of cases, augmented boluses of recently oxygenated blood surging through the coronary arteries and vital organs appear to produce a significant improvement in underlying pathology. At present, the cost and complexity of such strategies are prohibitive for many – and, again, the added benefit of supplemental oxygen during EECF treatments is likely overlooked by conventional practitioners.

ABCD ... HFCS

A major change in our food processing has occurred in just the past 40-some years: the introduction of HFCS, **high-fructose corn syrup**, as a *flavoring*. Actually, more as a “*sweetener*.” But it’s not really “*all natural*,” as we think of foods (like glucose or sucrose [table sugar]). And it doesn’t taste just *exactly* like sugar – but it is close enough to substitute in an astonishing number of “sweet” foods and drinks ... *and* even in ketchup, mayonnaise, hamburger and hot dog buns and the like. The worst part is that it acts more like a *drug* than the historical sweeteners such as cane or beet sugar or honey, encouraging you to “seek more” of the HFCS-supplemented foods. *You* probably avoid such foods ... certainly you would recognize the chemical name. But would you tag as the *same* ... “corn syrup solids”? “Natural sweeteners”? “Fructose” (fruit sugar) or “fructose syrup”? “Crystalline fructose”? HFCS intake (often quickly, in soda pop, candies, cookies, “treats,” cereals and baked goods, and junk foods) spikes insulin release and triggers production of triglycerides and cholesterol, let alone aggravating or actually causing intestinal permeability syndrome (“leaky gut”). Elevated insulin levels contribute to all the pathologic damage of metabolic syndrome, the preliminary to adult-onset diabetes, now epidemic in America.

Recent years have shown a 600% increase in daily consumption of HFCS, often unknowingly. Of concern are not merely obesity but also the discovery of interruption of hippocampal function (memory, orientation, even behavioral regulation) and creation of neuroinflammation. HFCS-induced inflammation has also been documented throughout sensitive endothelial tissues lining the heart and blood vessels and in joints. An association with cancer has been shown as well, supporting Warburg’s insistence on mitochondrial deterioration as a primary event. The staggering number of HFCS foods are dangerous *not only* because of their empty-calorie content (lots of calories, devoid of real food value) leading to nutritional deficiencies *and not only* because they encourage increasing intake of sugary/starchy foods, *but also* because *those very foods* sponsor the development and worsening of tooth decay, obesity, cardiovascular diseases, diabetes, and The Yeast Syndrome.

Here’s a provocative observation: HFCS foods entered the food chain in about 1971, and the first book describing The Yeast Syndrome appeared in 1978, *The Missing Diagnosis* by Orion Truss, M.D. “Syndrome X,” cardio-metabolic (or just metabolic) syndrome, was first described in the 1987 Banning Lecture to the American Diabetes Association by Stanford endocrinology professor Gerald Reaven, M.D. This clinical pattern clearly is *the* developmental step toward induced diabetes and preventable cardiovascular diseases. Key pathognomonic features of metabolic (or “insulin-resistance”) syndrome are the curse of our suffering survival as we age: obesity (increasing girth, elevated body mass index), higher blood pressure, elevating

blood sugar (with increasing insulin production), elevating triglycerides, and decreasing HDL cholesterol. *Oh* – and don't even get me *started* on the toxic cellular effects of “other” sweeteners such as aspartame, acesulfame potassium, sucralose, and saccharine. These were approved for *limited* use in foods for diabetics. Now they are widely scattered through the food chain, needlessly exposing millions to chemicals with *demonstrated* toxicity. (You should also be careful of Sapporo *Diet Water* – yes, I've seen it! – and Bernard's *Dehydrated Water* ... an empty tin!) Incidentally, the overwhelming percentage of corn, as used in production of HFCS, is GMO – genetically modified for more robust growth. Some have declared these genetic manipulations by chemical giant Monsanto are the inevitable end of healthy foods. Gives you that warm fuzzy feeling again, right?

GMO crops are engineered to resist herbicides, so higher concentrations can be applied to increase the commercial yield per acre. Are you ready for more and more hidden sources of glyphosate residues (broad-spectrum weed-killer *Roundup*®) – now the world's largest-selling herbicide and another demon-invention from Monsanto? Glyphosate has been connected, among a growing list of other health challenges, to an increased rate of miscarriage, reduction in sex hormone production, and disruptions to endocrine system development. What about autoimmune inflammatory celiac enteropathy – classically described as “gluten intolerance”? Celiac patients experience a two-fold increased risk for coronary artery disease, along with arrhythmias and heart failure. Glyphosphate residues on grains might be the *real* culprit, creating the setting for destructive inflammation throughout body tissues ... and in mitochondria.

Recent reports suggest that glyphosate interruption of cytochrome P450 detoxification enzymes, disruption of aromatic amino acid synthesis by the gut microbiome, and impaired sulfate metabolism could amplify inflammatory pathways, resulting in *many* degenerative diseases ... including those of the heart and blood vessels. Oddly, glyphosate “cages” (chelates?) aluminum in the gut and enhances absorption of this toxic metal. The (controversial! and challenged) speculation of glyphosate-induction of diseases has been suggested by consulting chemist Anthony Samsel and computer science senior research scientist at MIT Stephanie Seneff, Ph.D. (*Entropy* **2013**, *15*(4), 1416-1463) as the “textbook example” of “exogenous semiotic entropy”: the disruption of homeostasis by environmental toxins. But you can rest assured: the Council on Science and Public Health of the American Medical Association has concluded that “it appears unlikely that HFCS contributes more to obesity or other conditions than sucrose.” Keep in mind also that the common high-fat/high-sugar diet creates hyperinsulinemia (part of the metabolic syndrome), a key factor in promoting prostate cancer. Could enhanced *inflammation* (promoted by glyphosate?) along with the *yeast proliferation* induced by such a diet be a major feature in cancer promotion? For years, I have treated elevated prostate specific antigen (PSA) patients aggressively for The Yeast Syndrome ... with uniformly encouraging results. For a provocative review of the crucial interrelationship of fungus (yeast) and cancer, consider this internet video by the television host of *Know The Cause* and my dear friend for over thirty years, Doug Kaufmann: <http://www.knowthecause.com/index.php/cancer>.

Getting on with the Drugs and Stuff

You might think that I'm spending too much time and space discussing the “food issues” – and you'd be wrong. Master Teacher of the American College of Cardiology, Demetrio Sodi-

Pallares, M.D., practicing clinical and electro-cardiology for 60 years with impoverished Mexican citizens, has long *treated* dramatic degenerative heart diseases with little more than radical changes in the diet. His “non-toxic therapy” evolved from low-sodium/high-potassium diet plus infusions of “polarizing” (GIK = glucose-insulin-potassium) solutions to later include a strong electromagnetic field of 200 gauss and even later use of beta blockers, thyroglobulin, and exercise.

In case you haven’t yet grasped the significance of diet in development of disease, refer to the classical findings of Weston A. Price, D.D.S., research director for the American Dental Association who documented the deleterious effects of “foods of commerce” (*Nutrition and Physical Degeneration: A Comparison of Primitive and Modern Diets and Their Effects*, 1939) and those of Francis M. Pottenger, Jr., M.D., regarding uncooked foods (reviewed in *Pottenger’s Cats: A Study in Nutrition*, 1995). [The Price-Pottenger Nutrition Foundation offers tremendous resources at www.ppnf.org.] As the science of nutritional biochemistry advanced during the mid-1900’s, our understanding of health and disease dramatically expanded.

Recall that our populations were told since the 1960’s to avoid salty foods, to lower the tendency to develop high blood pressure, a major risk factor for heart disease. Sure, we have made serious efforts to avoid “salting *at the table*.” But we still eat *salted* peanuts, *salted* pretzels, *salted* chips, *salty* pickles, *salty* deli meats, and so on. And *these* salted sources *don’t* have added iodine. Why is this an issue? Over 100 years ago, public health authorities adopted the addition of iodine to table salt, a product that virtually everyone used, in order to reduce the incidence of thyroid disease (goiter). So now we have three generations of patients who have received a steady *salt* intake but *minimal iodine*. *Bingo!* Broda Barnes, M.D., showed some 40 years ago that low thyroid levels (associated with low iodine intake) are directly associated with a rising risk of heart attack (*Hypothyroidism: The Unsuspected Illness*, 1976). Denis Wilson M.D., has shown that raising thyroid hormone levels (especially *free-T3*) and raising basal body temperatures closer to “normal” can lower heart disease risk, and blood tests are *rarely* reflective of adequate support levels (*Evidence-Based Approach to Restoring Thyroid Health*, 2014). As you *might* predict (note: tongue-in-cheek!), these observations continue to be challenged by the American Thyroid Association.

Magnesium holds a special place for cardiovascular diseases. Lowered intracellular levels of magnesium are difficult to detect but clearly important. When serum levels are normal, intracellular magnesium can have been *scavenged* to maintain that measurement. *Low* serum levels are, therefore, *beyond critical* and *must* be addressed, since they contribute not only to high blood pressure but also both *pump* (CHF, congestive heart failure) and *performance* (contractility and rhythm disturbances) failures, as thoroughly documented by Mildred Seelig, M.D., M.P.H. (*Magnesium Deficiency in the Pathogenesis of Disease: Early Roots of Cardiovascular, Skeletal, and Renal Abnormalities*, 2012). Complementing magnesium certainly are manganese, copper, and zinc – adequate levels of all are essential for the formation of SOD, superoxide dismutases, thought to be the fifth most prevalent enzyme set in the human body, since they serve critical *anti-oxidant* functions in mitochondria, intracellular cytoplasm, and in extracellular fluids. SOD enzymes out-compete the essential tissue production of superoxide, used to defend against invading bacteria, protecting body cells from internally-generated oxidant injury. Back to mitochondria and that inflammation idea, right?

A couple of last points on foods. Bioflavonoids (polyphenols), cell-signaling sugars, and a wide range of other goodies are essential for wellbeing. Colorful vegetables are *the* source of bioflavonoids (and other “live” factors) on the planet. These are critical in biological functions (believe it or *stop* reading the basic science journals!) and virtually all of them are now being also shown to have powerful anti-fungal (and even anti-cancer) activities. Other goodies include items such intracellular glutathione (difficult to absorb unless enhanced by liposomal packaging, synthesis rate limited by scarce availability of L-cysteine, essential for anti-oxidant activity and detoxification), CoQ₁₀ and (induced production of) nitric oxide. These latter two are critical for control of cardiovascular efficiency – *pump* and *performance* issues – while the last aids in dilatation (widening ... or reduced constriction) of the pipes. While these and other factors are not readily “replenished” by direct supplementation, their synthesis and incorporation can be encouraged by specific nutritional support beyond merely the regular “multi-vita/mineral formulas.”

Mitochondrion Basics

Likely derived from prokaryotic ancient “bacterial” invaders into eukaryotic cells, these tiny “power plants” produce the ATP-based (adenosine triphosphate) energy used by virtually every cellular process. Cell life and division – even cell death – relate to mitochondrial status. Think of a “mito” as a “Dagwood sandwich” from the comic strip *Blondie*, with outer bread slices encasing a pile of layers of meats and cheeses. The “bread” in this illustration serves as a limiting outer membrane, through which sugars and fats can enter and ATP compounds can exit. The layers of “meats and cheeses” are equivalent to “shelves” (called cristae) inside the mito, stacked one upon another and separated by an insulating matrix. Enzymes and substrates involved oxidative phosphorylation (processing of sugars through to the end products of the electron-transport system) are aligned along these shelves in specific order, much like you would search for the volume of an encyclopedia from A to Z, not randomly. When all is working well, an innocent “sugar” molecule tumbles its way along, much like a Slinky toy trips its way down stairs, going quickly from one to the next chemical reaction, in order.

Lead and other toxic heavy metals (and even iron, in excess) disrupt in the inner shelf arrangements (apparently by inflammatory changes) and diminish enzyme efficiencies, reducing the rate of energy production. Further, as the mito becomes sickened and less able to perform, it can accumulate calcium ions and swell, distorting the shelf arrangements even more. While specific studies have not been done, EDTA chelation therapy appears a likely prospect to reduce internalized calcium and to restore more normal mito shape and function. Whether lead and other toxic metals are actually *removed* from within the mito is unclear, but *in vitro* laboratory studies demonstrate increased energy production in heart muscle as a result of chelation.

Before settling into the comfort of “modern diagnostics and medications,” be sure that exposures in the patient’s setting are *well* understood. Especially be wary that we have less and less *understanding* about more and more *complexities*. For example, when tetraethyllead was removed from vehicle gasoline in 1976 – a good idea to reduce environmental pollution, to lower blood pressure and heart attack risk, to minimize kidney damage, to improve brain function, and so on – the replacement mineral chosen was manganese. The health hazards associated with

manganese combustion products have now been debated for decades. Might we later find that our replacement is almost as challenging to human health as the original lead that aided octane performance in the gasoline?

Another provocative speculation is that the modern practice of “poly-pharmacy,” where several drugs are prescribed concurrently, might induce or amplify inflammatory processes. Medications are approved for use by the Food and Drug Administration based upon limited clinical testing, where most variables are tightly controlled. The general public, though, offers nothing but variables! The dubious interactions of multiple drugs simply haven’t been studied, and their impact in unwittingly aggravating disease processes – or even in inducing new ones, such as through interference with mitochondrial functions – raise disturbing questions about the bases for “modern” medical practice.

Mitochondrial dysfunctions are at the root of *all* degenerative disease progression. Understanding that optimal cellular and tissue function requires a robust supply of ATP energy leads to the obvious realization that all body activities are impaired whenever this vital component is limited. Any reduced anti-oxidant capability allows for unbridled inflammatory chemistry to wantonly damage cell structures and enzymes. Impairment of energy-dependent production of immune molecules leaves an undefended body increasingly prone to opportunistic attack by uncommon organisms, including those generally considered as non-pathogenic commensals or symbiotes on body surfaces, especially in the intestinal and respiratory tracts. Reduced digestive functions lead to a plethora of chemical and biological insults to gut tissues (and, subsequently, to fragile endothelial cells lining heart and blood vessels), not to mention amplification of nutritional deficiencies.

With rising challenges and diminished circulatory capacity, the physiologic strain on heart performance advances like falling dominos, inevitably resulting in degrees of kidney failure, deterioration of liver functions, accelerating diminution of gastric and pancreatic and hepatic secretions with progressive digestive impairments, peripheral circulatory embarrassment, and even organic brain syndrome. Obviously past lifestyle habits – tobacco and alcohol use, poor food choices, limited sleep, compromised stress adaptation patterns, licit and illicit drugs – create a setting (Claude Bernard’s “internal milieu”) in which all of these results from mitochondrial dysfunction can accelerate more rapidly than in others with more moderate health routines. Clearly the description offered can be observed over a matter of days or weeks in pre-terminal patients – or can be discerned by an astute practitioner some years (even *decades*) before the debilitations become obvious to others.

Putting Together the Bigger Map

While these ramblings might seem to have little to do with “cardiovascular diseases,” ask yourself: “Should I be treating the damage from degenerative diseases while ignoring the environmental factors that persist and aggravate the condition?” That’s rather like allowing the patient to wear the shoe causing the blister perhaps merely an hour or two a day. How foolish! Note that I do not ask whether the problem is with pipes, pump, or performance. The “diagnosis,” in a classical sense, is *almost irrelevant*. The key is to establish the link between the

condition and associated lifestyle choices and exposures so that a more comprehensive (wholistic!) approach can be used.

In effect, I am proposing that virtually *all* treatment for cardiovascular diseases should be aimed at the “utility” level. This example highlights this point: Whoever lives in a house, it matters not ... the house has the “same” utilities as found with all *other* houses. Whether a baker, a banker, a teacher, a postman, *you* depend upon electricity coming in and light and heat going out, depend upon water coming in and drainage and sewage going out, depend upon food and supplies coming in and trash going out. Even a driveway coming in and roads going out!

Consider each “house” to be a single “cell” of the body. Thus, a neighborhood of similar houses would constitute an “organ” and a large cluster of assembled neighborhoods would comprise a “body.” Our current medical paradigm aims at treating problems within specific neighborhoods. I propose that most of our treatments should be aimed at *houses*, at the utilities provided to and functioning within the individual cells. Regardless of *which* utilities or cells are suffering, those are *the* levels at which treatments should be aimed. This viewpoint encompasses a broader explanation of the symptoms and signs seen in illnesses, correlating the expression of disease in *other* organ systems (neighborhoods) that are likewise being affected by the impairment of *their* similar utilities.

Changing the Slope – Oh, *NO!* Not *Calculus!*

As I propose this somewhat different approach to assessment and treatment, let me offer how I describe to patients our assessment and monitoring of their conditions. Our explanation induces patients to stay with treatment programs much longer than they did before. First, let me give homage to indices such as the SF-36 Health Survey. This survey,

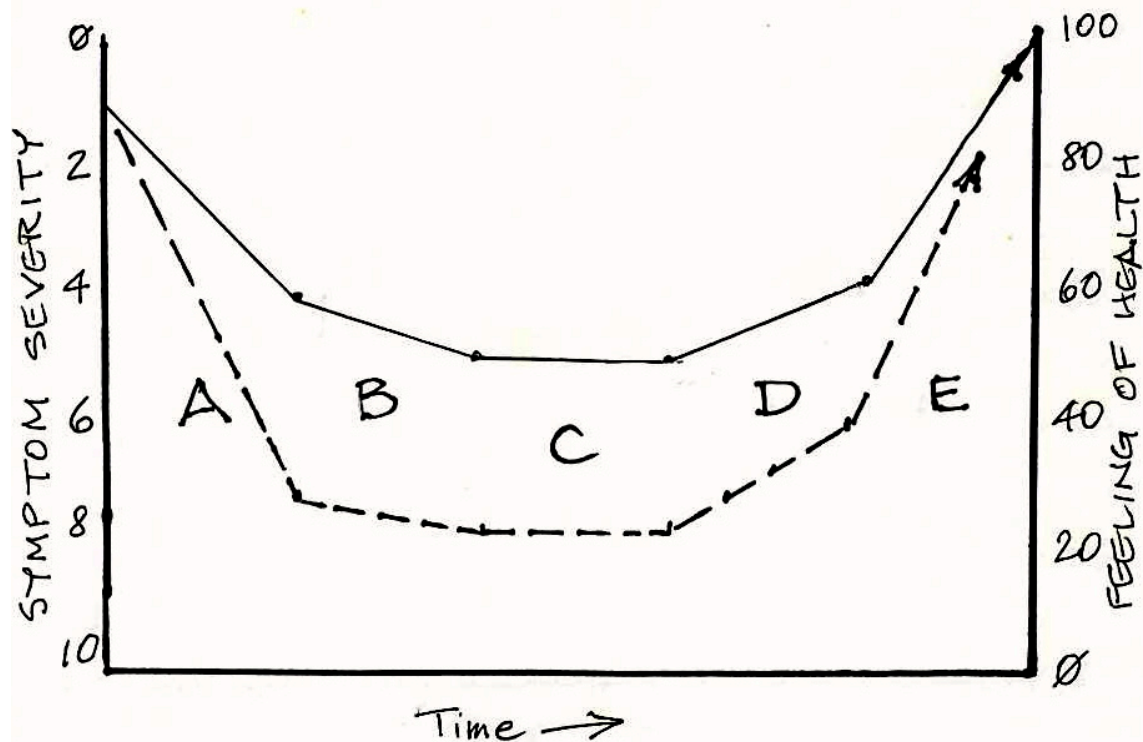
Consisting of 36 items, the SF-36 is a brief survey designed to assess functional health and well-being in a variety of age, disease, and control populations [1]. Each question relates to one of eight domains: physical functioning, role-physical, bodily pain, general health perceptions, vitality, social functioning, role-emotional, and mental health. Results from these subscales contribute to scores for overall physical and mental health. [Shahid A, Wilkinson K, et al. STOP, THAT and One Hundred Other Sleep Scales, 2012, pp. 317-318]

In order to better address our particular patient population, I designed a customized “symptom-rating” form of about 50 items, printed on the flip side of our office visit notes page. At *every* doctor-patient visit, the patient rates his discomforts (from zero to ten) *before* being seen. The various “positives” serve as foci for discussion at *that* meeting. Further, by “flipping up” the chart pages to earlier encounters, the progress of symptom reduction can be easily seen over time, from first visit to present time.

I describe, to a “newbie” patient, that the *first* thing we’re trying to do is to gain some understanding of what *actually* is affecting them. At this point, many patients usually will interrupt to share, once again, their various diagnoses (from other physicians), to be sure that I’m not overlooking their concerns. I acknowledge their statements and say that we understand that

their various disease problems, as a whole, are worsening over time. They agree. I tell them that my first job is to identify the “pinch points,” where doing something simple and deliberate will *slow* the rate at which they’re worsening ... then *level* them off, so that they’re no longer worsening ... then *raise* the slope of the curve, so that they’re steadily *improving* ... with the ultimate goal being to return them to more robust health, often better than that they’ve experienced in recent years.

This explanation encompasses a broader view of “health” more along the lines of Bircher, “a dynamic state of well-being characterized by a physical and mental potential, which satisfies the demands of life commensurate with age, culture, and personal responsibility.” [Bircher J. Towards a dynamic definition of health and disease. Med. Health Care Philos 2005;8:335-41] For those who need a more visual presentation, perhaps this graph might illustrate the concepts easier, since I do draw it “out in space” for patients:



The demonstrative graph is easy for patients to understand. Assume that the patient has symptom complaints, rated on our zero-to-ten scale as shown on the LEFT side. These roughly correlate inversely to the feeling of health and wellbeing, as shown from zero-to-100 percent on the RIGHT side. The patient presents with declining health and worsening symptoms, as noted by the solid line above the “A.” During this first phase, your job is to define the underlying issues well enough to start improving the patient’s condition ... which means *slowing* the rate of *worsening*, as noted by the solid line above the “B.” As you understand underlying causes better and work more with the patient, you work to hold most symptoms “level” or “unchanged,” as

noted by the solid line above the “C.” This, in itself, is a major accomplishment ... now you have the opportunity to make a *real* difference for this patient.

During the next phase, as noted by the solid line above the “D,” you are finally able to achieve some *improvements* and your patient clearly feels healthier. In the final phase, the solid line above the “E,” results from your treatment patterns have become obvious and your patient is benefitting greatly. When you’re able to establish a “maintenance program,” your job is to monitor the hallmark symptoms carefully – along with relevant labs and exam findings – to “stay ahead of the curve,” keeping most symptom severity scores at “3” or often less.

What about the “dotted line” on the graph? Some patients present to you as markedly more acute or chronically worse than anyone would like. “But doctor, I just found out about you ...” The steeper slope above the “A” shows that you have less time to evaluate and find successful treatments. Nevertheless, your job remains the same: slow the rate of worsening, find ways to hold everything “level” while you “buy time” for treatments to work (or to be identified), then work for gradual improvement and then much more aggressive changes.

Patients who are not themselves integrative medicine physicians can have only a brief, disjointed, and even *mythical* view of the roots of their problems, the diagnostic finesse often needed, and the treatment options available. What they clearly understand (or at least hope for) is that doing specific actions *could* lead to particular, desired results. Their motivation to continue their treatment programs comes only from successful responses. Since a patient has completed our same “SF-36-type” questions at each office visit, he or she can readily see the “march” of lower and lower “scores” away from “10” and toward “zero” over the course of several office encounters.

Surprisingly, many improving patients quickly forget how badly they felt or how many limitations they suffered, so their own earlier scores are excellent reminders. In the end, when most of the answers are shifted left, “to the healthy side” (= zero or just 1, maybe 2, occasionally 3), the patient still has a visual reminder of where they started ... and how much better and happier they now feel. And *that* is powerful motivation to continue the *maintenance* programs custom-designed to retain their benefits gained. Repeatedly referring to this “graph” concept during the course of therapy can dramatically aid the patient to understand and comply with the testing and treatment programs, since they can visualize “where they are” in the plan of action.

Trowbridge’s “12-Step Program” of Don’ts and Do’s *

DON’T ...

1. ... Assume that the “other doctors” will appreciate *your* participation or *any* patient improvements
2. ... Assume that the condition has been *correctly* diagnosed or *cannot* be treated
3. ... Assume that the present medications are *not* contributing

to or actually causing symptomatic complaints

4. ... Assume that a failing course is a *likely* outcome for *this* patient at *this* time
5. ... Assume that the treatments earlier prescribed by other doctors are *correct* or even required
6. ... Assume that nutritional support has *little* to contribute
7. ... Assume that any “diet” that has been earlier counseled is *appropriate*
8. ... Assume that oxygen saturation levels are *sufficient*, even when not grossly abnormal
9. ... Assume that an *operation* is the best next choice
10. ... Assume that activity level seen is the *best* that can be obtained
11. ... Assume that your patient *understands anything at all* about his/her condition, treatment, improvement or worsening
12. ... Assume that your patient is at *peace* in his/her soul

DO ...

1. ... Expect to *educate* your patient about the medical and political community as well as about costs and coverages for whatever you do – in person, through books and brochures, with your staff actively supporting
2. ... Expect that effective treatment can be *started*, literally, immediately and then improved upon
3. ... Expect that a *substantial* improvement is within easy reach
4. ... Expect that the rate of worsening can be *slowed* then *halted* for many patients ... and then “*better*” is “within reach”
5. ... Expect that your assessment will reveal *alternatives* that have been missed, disregarded, or ignored
6. ... Expect that proper *supplements* can be, in fact, life-saving

7. ... Expect that radical revision of *food* intake can be life-saving
8. ... Expect that supplemental *oxygen* can be life-saving
9. ... Expect that any patient can be better prepared to *survive* any needed surgery ... or to *avoid* it altogether
10. ... Expect that a gradual *physical* therapy program can be started immediately
11. ... Expect that you will need to *explain* the patient's condition (pipes/pump/performance, mito energy production, interrelated body functions, and so on) and basics of his/her treatment plans ... *often*
12. ... Expect that crucial *spiritual* encounters can be life-saving

* These steps assume that appropriate medical treatment will be pursued concurrently, including detoxification of organic toxins or toxic heavy metals as needed

What More Is There To Learn?

Absolutely *everything*. The practitioner has three *essential* tasks. First, to learn what needs to be known about diagnosing and treating the conditions he/she holds himself out to treat. Second, to establish and oversee proper treatment programs. And third, to effectively explain to the patient and family “what it is and how we’re treating it,” as often as needed.

Reflecting on a lifetime career of treating “all comers,” including those with cardiovascular diseases of *all* kinds (including having had two patients removed from the heart transplant list, due to startling improvements), I find myself struggling to offer comments of value to other practitioners. Most everyone knows the first and second tasks quite well, at least well enough to achieve basic improvements for patients. What matters, then, might be efforts to learn just a little more about how to *explain* the situation to the patient and family. My methods have been successful on many levels, with patients from all backgrounds, with all conditions, and at all levels of presenting severity.

As we strive to learn better to take care of our patients, let us strive also to learn better how to ***make what we do make sense*** to those whose world depends on our “doing it right.”

If you doubt the relevance of these concepts I’ve presented just try them! See whether patients are more receptive to your ideas of diagnosis and treatment. See whether they are more compliant – and whether they continue for maintenance programs moving them toward years of more robust health and longer, more rewarding, and vital independent and comfortable living. If you don’t try them in your practice but instead ignore that they have any value, may

you be blessed by this wonderful quote from our Founding Father Benjamin Franklin: “Any fool can criticize, condemn and complain – and most fools do.”

Enough of my foolishness.

Dr. Trowbridge respectfully dedicates this article to the memory of his recently deceased friend and supportive colleague of more than 30 years, Jimmy F. Howell, M.D., Professor of Surgery at Baylor College of Medicine for over 50 years and one of the distinguished pioneers in vascular surgery. Dr. Howell joined senior colleague H. Edward Garrett in performing the first successful coronary artery bypass operation in 1964. While Director of the Vascular Surgery Training Program at Baylor and The Methodist Hospital in Houston, he oversaw the education of numerous leading national and international vascular surgeons. Dr. Howell graciously shared the podium at our 1996 public chelation celebration, ***The Rumble in Humble: Heart Surgery and All that JAZZ!***

John Parks Trowbridge M. D., has been certified since 1985 as a chelation diplomate by the American Board of Clinical Metal Toxicology, for which he has served as Secretary. A Fellow of the American College for Advancement in Medicine, he has served as director, officer, or president of several varied medical and lay associations. Popular as a professional and public speaker, he co-authored Bantam’s bestselling **The Yeast Syndrome** along with books on chelation and other topics and over 4 dozen CDs and DVDs. An interview published in the just-released book **Chelation and Other Detox Methods to Save Your Life!** presents chelation perspectives gathered over 32 years of offering this superb treatment. He provides a broad array of integrative medical therapies for challenging illness and injury problems at his solo practice, Life Celebrating Health in Humble (Houston), Texas: jptlch@earthlink.net, 1-800-FIX-PAIN, www.healthCHOICESnow.com. (copyright at common law 2015 John Parks Trowbridge)

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