

A Summary of EDTA Chelation Clinical Research: All Good!

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The medical community eagerly accepts scientific research buttressing a therapy it already approves. Somewhat more reluctantly, it examines and debates entirely novel approaches. But what it hates worse than poison is reappraising a treatment once rejected. Medicine, after all, is made up of people—people trailing MDs after their names—who, like the rest of us, do not enjoy admitting error.

Someday when chelation therapy is an established part of standard medical care, historians of twentieth century medicine will wonder how so much supportive research on its benefits could have been scrupulously conducted by skillful medical researchers and even more scrupulously ignored by the guardians of our health. By that time, most of the individuals who successfully shifted chelation toward the fringes will not be alive to blush, sparing them extensive embarrassment.

The amount of positive research is certainly formidable. And those studies that purport to demonstrate that chelation doesn't work actually show the opposite. We will now examine much of this research in detail.

In a sense, we're attempting to set the record straight and to tell people who read *Bypassing Bypass Surgery*—especially physicians—where they should look for the scientific evidence. After all, mainstream medical journals engage in unconscionable editorial censorship. They refuse to publish positive research studies on EDTA chelation but are quick to print editorial criticism and anecdotal letters to the editor that are biased against this marvelous therapy. They are also quick to uncritically print highly flawed studies that erroneously allege to disprove chelation, as demonstrated by the Danish and New Zealand studies analyzed below. Journals that do publish supportive studies, although medically excellent, tend to be smaller, less widely read and ignored by the mainstream. Studies supportive of EDTA chelation therapy have consistently been refused inclusion in the MEDLINE computer database by the National Library of Medicine.

Also, academically positioned researchers and professional clinical trialists have been chastised repeatedly by their colleagues, should they be intellectually honest enough to express an interest in research of EDTA chelation therapy for atherosclerosis. They are told behind closed doors that this is not a "politically correct" topic, and that such a research interest would be "career suicide."

Most practicing physicians are entirely unaware that less than 20 percent of the world's total biomedical literature (in all languages) is referenced by the National Library of Medicine in the Index Medicus, and its electronic counterpart, the MEDLINE computer database. Thus, a computer search for positive studies of chelation therapy in the treatment of atherosclerosis will be deceptively negative.

In this chapter, we will discuss several of the most important positive studies, referenced for those who wish to obtain the original articles. Then we will analyze the allegedly "negative" studies. (A very complete listing of all studies thus far published on this topic can be found at the end of chapter 17 of *Bypassing Bypass Surgery* and many are published in complete form as chapters in *A Textbook on EDTA chelation Therapy, Second Edition*.)

Let me make a few points before we begin.

* First, there are no genuinely negative studies. That statement remains true through April, 2002, and applies equally to the Calgary PATCH trial. All the medical research to date on chelation has produced positive results. That is, the data have invariably been positive. This has not prevented medical spinmeisters from misleadingly imposing negative interpretations on positive results—unfortunately this applies to a number of recent studies in widely-read mainstream medical journals.

* Second, financial considerations have limited the size of chelation studies. The drug is no longer patentable and no one has been willing to spend the \$30 million or more that pharmaceutical companies must spend to satisfy the FDA requirements before marketing claims can be made. That's the price tag for a large double-blind, placebo-controlled study to meet FDA requirements. Many of the studies we quote are relatively small. Though they often have less than a hundred patients, they are nonetheless

scientifically significant. Their endpoints are determined by objective numerical measurement of increases in blood flow and are statistically analyzed—conclusions were not determined by merely asking patients how they subjectively felt.

* Third, critics of chelation have frequently suggested that reported improvements are a placebo effect. It is a well-known phenomenon of medicine that when given a completely inactive substance and told that it may help them, many people will show a certain—often impressive—level of improvement for some time after they begin using their new "medicine." Thus, the placebo (as inactive substances used in medical research are called) turns out to have an "effect." But chelation hardly fits the profile of a placebo. A placebo effect begins shortly after its first administration and rarely, if ever, persists for more than three months. Chelation, by contrast, shows its full range of benefits quite slowly. Usually, it requires not only several months of therapy but also an additional several months after a course of therapy for the full benefit of treatment to occur. And the benefits generally persist for years thereafter. Therefore, chelation shows a pattern different from, and indeed opposite to, the pattern of a placebo. Moreover, when studied properly, the benefits are far stronger than a placebo could show. It's nonsense to allege that such dramatic improvements are placebo effects.

* Fourth, statistical analyses of measured improvements in the more carefully performed chelation studies demonstrate that the probability of these changes being due only to random chance is somewhere near the vanishing point of statistical insignificance. That numerical probability ranges from less than one in 1,000 to less than one in 10,000. The reason for such high significance is the magnitude of the improvements measured, despite the relatively small number of patients.

With those general points to guide us, it's time to look at some of the actual studies done on atherosclerosis and chelation.

If you will recall the clinical trials I discussed briefly in chapter of 1 of *Bypassing Bypass Surgery*, they all clearly demonstrated improved circulation after chelation. These are the sort of results that any chelation therapist expects—we not only notice improved exercise tolerance, memory, and mental alertness in our patients but even healthy color returning to their cheeks.

Many other objectively measured indicators of circulatory health tell similar stories. Drs. McDonagh, Rudolph, and Cheraskin took 77 elderly patients with documented narrowing of the peripheral arteries in their legs and measured changes in blood flow after approximately 26 EDTA infusions administered over 60 days. They used the preferred method for such testing: the ankle/brachial Doppler blood pressure ratio. This method compares the blood pressure and flow in the arms with that in the ankles using Doppler ultrasound. In a person with a youthful circulatory system, the normal pressure in the ankles is equal to or greater than that in the arms.

Patients with impaired circulation to the lower extremities have, of course, weaker arterial blood flow and lower blood pressure in their ankles than in their arms. On average, after chelation therapy, the patients' ankle pressure increased from 55 percent of the arm pressure to 71 percent of the arm pressure, a change so significant that the statistical likelihood of its being due to random chance would be somewhere in the neighborhood of one in 10,000.(1) Improvement in Doppler blood pressure reflects only blood flow in larger arteries. EDTA also improves capillary circulation, which is especially reduced in diabetes.

Drs. Casdorff and Farr reported on four patients who had all been recommended to undergo surgical amputation of their gangrenous lower extremities before treatment with EDTA chelation (click here for abstract). Clearly, these were people who had reached end-stage complications of atherosclerosis and poor blood flow. Most of them had deep ulcerations and large areas of dead, necrotic tissue on their feet. In some cases, circulation to the extremities had become so poor and so much tissue had died that the condition was no longer causing significant physical pain. The patients' pain was now mental—in the clear knowledge that they were about to lose a leg.

All four patients chose to postpone amputation (against surgical advice) and receive infusions of EDTA combined with hyperbaric oxygen therapy. Treatment was completely successful in three out of the four cases.(2) In the fourth case, the patient did eventually lose only the tips of his second, third, and fourth toes, but the foot and leg were saved. After chelation, all four patients recovered circulation in their lower extremities sufficient to not only protect them from amputation but to also allow them pain-free walking without limitation or handicap. Several years after chelation therapy, those four patients continued to be alive and well, walking on their own legs and feet. Their recovery—if witnessed by a physician who was unaware of or unwilling to credit chelation's effectiveness—could only be seen as a sort of medical miracle, something comparable to spontaneous remission of an advanced and deadly cancer.

Another study by Dr. Casdorff, as mentioned in chapter one of *Bypassing Bypass Surgery*, contains data showing large, numerically tabulated and objective improvements in blood flow to the brain (click here for abstract and data). Computerized graphs showing improved blood flow are astounding, even to those untrained in medical science. A scintillation counter and computer were used to generate those sophisticated images, which are perhaps the most convincing objective evidence we have for increased blood flow after chelation. I challenge any open-minded physician to review the data in that article and not come away impressed.(3)

Serious students of chelation therapy and health care professionals are referred to a Textbook on EDTA Chelation Therapy, Second Edition, edited by Elmer M. Cranton, M.D., Hampton Roads Publishing Company, Inc., 1125 Stoney Ridge Road, Charlottesville, Virginia 22902, (804) 296-2772, FAX (804) 296-5096. Complete copies of the chelation research studies cited in this chapter, including all of the actual data, are contained in that Textbook. You may also purchase the TEXTBOOK online.

In addition to those smaller studies, there have been other large retrospective studies using a variety of methods to measure changes following chelation therapy.

Drs. Hancke and Flytlie, two Danish doctors with impeccable credentials, published such a study in 1993—a counterblast, as it were, to the Danish bypass surgeons' ineffectual attempt to discredit chelation in the previous year, as described elsewhere on this website. Hancke and Flytlie measured improvements using a several different criteria in a series of 470 patients who were followed for six years following chelation therapy (click here for abstract). Of 265 patients with coronary artery disease and narrowing of the blood vessels to the heart, they reported improvement in 90 percent. Sixty-five of those patients had been referred for bypass surgery before chelation. After treatment, 58 of the bypass candidates improved so dramatically that they avoided the surgeon's knife. Among the 207 angina patients using nitroglycerin to control their pain, 189 were able to reduce their consumption. Most discontinued its use altogether. Of 27 patients awaiting foot or leg amputation, 24 avoided surgery.(4)

These results, remarkable as they may seem, fully correspond with what physicians who administer chelation therapy routinely observe in practice.

Another even larger retrospective study done in Brazil, analyzed the effects of chelation on 2,870 patients with atherosclerosis and related degenerative conditions. Treatment was carried out between 1983 and 1986 (click here for abstract). Nearly all patients were being treated for atherosclerotic vascular diseases. The most serious category, the patients with heart disease, numbered almost one-third of the total; and, in that group following chelation, 77 percent showed marked improvement, 17 percent showed good improvement, 4 percent had partial improvement, and 3 percent were unchanged or worse. Patients with arterial blockage in other parts of the body showed similar improvements.(5)

The researchers, Dr. James Carter, a professor at Tulane University Medical School in New Orleans, together with Dr. Efrain Olszewer, a cardiologist in Sao Paulo, Brazil, decided to follow up these treatment results by conducting a small double blind pilot study on ten patients. Midway through the study they were forced to open up the blind for ethical reasons; five of the patients—these turned out to be the ones receiving EDTA chelation—were doing dramatically better than the placebo group. It was felt to have been unethical to continue giving placebo therapy. The placebo patients were then put on EDTA and they too rapidly began to improve.(6)

One final question is worth asking: Are these diverse studies (impressive though they may be) really typical medical research results on chelation? Drs. Terry Chappell and John Stahl set out to answer that question in 1993. They conducted a meta-analysis of all currently available scientific literature (click here for abstract). This is an eagle-eyed observation and comparison of diverse studies that summarizes, as best it may, the total results achieved by many researchers following chelation therapy. Over the course of the last decade, such analytic overviews have grown more reliable and are more relied upon. Chappell and Stahl identified 19 articles in the medical literature that met their criteria for determining chelation's effectiveness in cardiovascular illness. In combination, the articles provided data on 22,765 patients. The meta-analysis determined that 87 percent of these patients experienced favorable outcomes. Only those improvements measured by objective testing were accepted as evidence in their analysis.

Chappell and Stahl were compelled to conclude that there was very strong published evidence for chelation's effectiveness in the treatment of cardiovascular disease. (7)

There is nothing surprising about such a conclusion. It's very difficult to test real people using chelation therapy and not come away impressed. Nevertheless, some physicians have achieved that feat. Let's look at their research.

Every now and then puzzled patients tell me that a friend, relative, or skeptical physician has told them that chelation was fairly tested and fell flat. I can usually guess what they're referring to. In the last ten years, a small cluster of studies sprouted up in the mainstream medical literature purporting to demonstrate that EDTA chelation was a fizzle when it came to treating cardiovascular ailments.

The curious thing is that those studies—flawed and imperfect though they are—only succeed in offering us still more positive data to support this therapy (click here for analysis).

The most controversial and oft cited study was done in Denmark. It was the handiwork of a group of Danish cardiovascular bypass surgeons. Results of that study were published in two medical journals, the *Journal of Internal Medicine* and the *American Journal of Surgery*. The results were also widely publicized in the news media.

The surgeons had taken 153 patients suffering with intermittent claudication. These were people with such severely compromised circulation in their lower extremities that walking across a parking lot could challenge their fortitude. One measurement of their condition was their maximal walking distance (MWD)—the very longest distance that they could walk before intolerable leg pain brought them abruptly to a halt. The patients were divided into an EDTA group and a placebo group. In the pre-treatment phase, the EDTA group could on average walk 119 meters before colliding with their MWD; the placebo group averaged 157 meters.

Treatment began with the patients receiving either 20 intravenous infusions of EDTA or 20 infusions of a simple salt solution, depending on their group. The study was purportedly double-blinded, that is, neither the patients nor the researchers knew which person was receiving which infusion until after the study was complete. Progress was measured periodically. In particular, we will analyze their results at three months following treatment, when full benefit from chelation would be expected to occur.

Both placebo and treatment groups showed improvement. However, the investigators concluded that the improvement was not statistically significant and—equally important—that the difference in response rate between the EDTA group and the placebo group was roughly similar. Obviously, a drug that fails to achieve more than the placebo effect is presumed to be a dud.

The Danish study impressed many people; but, in rather short order, the integrity of the study was called into question. It was learned that the researchers had violated their own double blind protocol. Not only did they themselves know before the end of the study who was receiving EDTA and who placebo, they had also revealed this information to many of the test patients. Before the study was over the researchers and more than 64 percent of the patients were aware of which treatment they had received.

This was unorthodox and since it had not been reported in the published study, extremely questionable from an ethical standpoint.

Many people had also been struck by the study's relatively small size. Intermittent claudication is a very unpredictable disease, and, unless enough patients are included in a trial, the results tend to be statistically unreliable.

The most interesting aspect of the Danish study, however, was hidden away in the numbers. This is the startling fact that the patients who were given EDTA were certainly a good deal sicker than the patients tested with a placebo. Therefore, the improvements they made were harder earned and more significant. The researchers, who candidly admitted that they undertook the study to convince the Danish government not to pay for chelation, either never noticed that aspect or felt reluctant to reveal it. The evidence is in the pre-treatment MWDs. The EDTA patients' longest average distance before claudication pain stopped them in their tracks was 119 meters, while for the placebo patients it was 157 meters.

Still more significant was the standard deviation. Standard deviation is a statistical abstraction, which reflects the amount of variability among a collection of raw scores. In essence, standard deviation reflects how widely diverse the numbers are in each group. A high standard deviation indicates that measurements were spread out toward the extremes of a wide range, rather than closely clustered near the average. Without going further into the arcane science of statistics, it is enough to say that the plus or minus 38 meters for EDTA patients versus plus or minus 266 meters for placebo group represents an enormous difference in

walking capacity that is heavily biased in favor of the placebo group. The standard deviation numbers show that some placebo patients must have walked half a mile before stopping. The EDTA group's claudication was therefore much more severe. The EDTA group was much sicker. The design of the study was therefore catastrophically biased against EDTA chelation from the outset.

Yet, when the six-month study was completed, the MWD in the EDTA group rose by 51 percent, from 119 to 180 meters, while the mean MWD in the placebo group rose only 24 percent, from 157 to 194 meters. In plain English, looking at all the published data, the chelation group's improvement was more than twice as great as the placebo group's, even though they were significantly sicker at the outset.(8,9)

I believe the Danish study must be interpreted as another solid demonstration of the effectiveness of chelation. If it were not for its relative smallness, I would be happy to quote from its results at any time. I hope the Danish surgeons can be persuaded to undertake another study with five times as many subjects. If they take the trouble to hire an academic statistician to oversee design and interpretation of the study, and refrain from violating the double blind, they may yet do good work, and we shall all be much in their debt.

Another study—also conducted by vascular surgeons—was done at the Otago Medical School in Dunedin, New Zealand, two years later. The subjects of this study were also suffering from intermittent claudication manifested by leg pain and walking difficulties beyond a very limited distance. Chelation subjects were compared to controls. The study extended to three months after 20 infusions of either EDTA or a placebo had been administered. Upon examining the results, the authors of the study concluded that chelation had been ineffective. Once again, that conclusion seems ill founded.

The absolute walking distance of the EDTA group increased by 26 percent; in the placebo group, it increased by 15 percent. This was not considered statistically significant. The study, however, was so small that there were only 17 subjects in the placebo group. One of these was what the statisticians call an "outlier." That is a person whose results differ strikingly from everyone else in the group. That placebo patient's walking distance increased by almost 500 meters. All of the statistical gain in the placebo group was due to this one individual's progress. Without him, their placebo distance decreased slightly.

This illustrates the perils of a small study. A 25-percent gain in the EDTA group compared to no gain in the placebo group would have been very significant statistically.

Meanwhile, even the New Zealand researchers conceded that the improvement in artery pulsatility (measurement of pulse intensity) in the EDTA group's worse leg reached statistical significance. In statistical terms, there was less than a one in a thousand chance that that improvement was not a benefit of EDTA. (10)

I would note only two other things. First, a 26 percent improvement in walking is by no means minor and would attract notice if the agent had been a patentable drug. Second, even that level of improvement is in no sense representative of the much greater improvements claudication patients normally experience after chelation.

There is a simple reason for the difference: smoking.

Smoking so dramatically undermines cardiovascular function, especially in people who are already seriously sick with claudication, that it negates much of the gain that chelation provides. In the New Zealand study, 86 percent of the chelated subjects were smokers. They were advised to quit smoking when the study began, but how many of them actually stopped is, I fear, a subject for skeptical speculation. A demonstration of chelation's full potential requires a much higher percentage of non-smoking subjects at the outset.

Just as this book goes to the printer, another small study alleging to disprove EDTA chelation therapy is being widely reported by the news media. This recent study was conducted by cardiologists in Calgary, Canada, who freely admit their bias against chelation. They seem to have set out to discredit a therapy that they oppose by studying a few patients with heart disease. Because the study has not yet been published in a scientific journal, it is not possible to provide a meaningful critique. I feel certain, however, that when we finally do have an opportunity to conduct a detailed review of that study's design and data, the final assessment will be very similar to that of the Danish and New Zealand studies, as described in detail in this chapter—another hatchet job.

It's relatively easy to design a study specifically to discredit an unpopular therapy, and to make that study superficially appear to be scientific. The United States Congress once commissioned its Office of Technological Assessment to analyze all published medical research for scientific merit. After a careful review of research studies from leading medical journals, they concluded that, "more than 75 percent of all published medical research has invalid or insupportable conclusions as a result of statistical problems alone." The final report to Congress stated, "few published clinical trials are well enough designed to yield valuable results."

And it's not merely intellectual dishonesty. Many doctors who oppose chelation therapy firmly believe that it is ineffective. That is what they have been told. So they attack, with no personal knowledge about what they are attacking. Perhaps they feel threatened because very few doctors have the time to thoroughly read and analyze published studies in medical journals. They usually skim the abstract and jump to the authors' conclusions, accepting them without question.

I have also found medical doctors to be naive and unaware that the peer review process is often used as a form of editorial censorship—a way to maintain the status quo and protect the professional reputations and practices of the reviewers. Also, because medical journals so often depend heavily on advertising by major pharmaceutical companies, studies that are unpopular with that industry are rarely published; while brief letters to the editor and unsupported editorial opinion attacking opposed therapies quickly find their way into print. Journals tend to be reluctant to bite the hand that feeds them.

Powerful psychological defense mechanisms also come into play. If doctors are not taught about EDTA chelation therapy in medical school (and they are not), and if those doctors therefore do not routinely use or prescribe chelation therapy for patients, then they believe one of two things: 1) either their medical educations were deficient and they are not providing the best of care for patients; or, 2) other doctors routinely using and prescribing chelation therapy for medical conditions that are not FDA-approved must be "quacks," exploiting desperate patients. Which do you think their choice will be? It's apparently difficult for many medical doctors to shed an attitude of God-like omniscience and admit that they simply do not know everything there is to know.

One final study that was carried out with what I am forced to call negative intent is such a curious oddity that it also deserves discussion, although it remains unpublished. It is usually referred to as the "Heidelberg Trial" and was conducted at the behest of the German pharmaceutical company Thiemann, AG, in the early 1980s. Once again using patients with intermittent claudication, it compared the effects of 20 infusions of EDTA with 20 infusions of bencyclan, a vasodilating and antiplatelet agent owned by Thiemann.

Needless to say, from a practical commercial standpoint, Thiemann's action was bizarre. If EDTA did well in the trial, Thiemann's own already well established drug could only suffer. Nonetheless, the trial went forward and was reported before the audience in 1985 at the 7th International Congress on Arteriosclerosis in Melbourne, Australia. That study showed that immediately following 20 infusions of EDTA, pain-free walking distance increased by 70 percent. By contrast, the patients receiving bencyclan had increased their pain-free walking distance by 76 percent. The difference between these two results was, of course, not statistically significant, but another result was. It turned out that 12 weeks after the series of infusions was completed, the EDTA patients' average pain-free walking distance had continued to increase, going up by an astounding 182 percent. No further improvement had occurred in the patients receiving bencyclan, however.(11)

A report from Thiemann only mentioned the 70 and 76 percent figures, and press releases stated that chelation was no better than a placebo without mentioning that the "placebo" was a drug that had been proven effective in the treatment of intermittent claudication. Thiemann never released the actual data from the Heidelberg Trial, but some German scientists who had access to it, and who were disturbed at the deception they were witnessing, chose to reveal the data to members of the American scientific community.

The complete data showed that four patients in the EDTA group experienced more than a 1,000-meter increase in their pain-free walking distance following treatment.(12) This highly favorable data from those four patients mysteriously disappeared before the final results were made public. As sponsor and by funding the study, Thiemann had a legal right under terms of their contract to edit the final results and to interpret the data in any way that suited them. An analysis of the complete data showed an average increase in walking distance in the EDTA-treated group of 400 percent at three months after therapy—five times the 76-percent increase of the group receiving bencyclan.

These three ineffectual attempts discredit chelation with flawed research represent pretty much the sum total of scientific involvement that the establishment has had with this extraordinary therapy over the past thirty years.

In January, 2002, the American Medical Association published yet another junk-science study in a seeming further attempt to discredit EDTA chelation therapy. If anything, that so called PATCH study, conducted in Calgary Canada, was one more positive study with a misleading negative conclusion.

However, the darkest moment for chelation actually came way back in 1963. This was when Drs. J. R. Kitchell and L. E. Meltzer co-authored an article reassessing their support for EDTA chelation.

Although it was hardly in widespread use, chelation had been surprisingly uncontroversial up until that moment. Beginning in 1953, Dr. Norman Clarke, Sr., and his associates at Providence Hospital in Detroit began using EDTA chelation to treat coronary heart disease. In 1956, they reported that they had treated 20 patients suffering from chest pain (angina pectoris). Nineteen of the 20 patients had had a "remarkable" improvement in symptoms.(13)

Soon other physicians became interested, among them Drs. Kitchell and Meltzer, who specialized in cardiology at Presbyterian Hospital in Philadelphia. From 1959 to 1963, Kitchell and Meltzer reported on their consistent good results treating cardiovascular diseases with EDTA. Their early reports were all very positive.(14-16)

But in April of 1963, shortly after their last favorable report, they published a "reappraisal" in the American Journal of Cardiology that questioned chelation's value.

That reappraisal article included ten original patients on whom they had previously published data, and 28 patients with coronary heart disease who were treated subsequently. Treated patients in this report were all severely ill. The authors state that the patients were, ". . .referred to us because of severe angina. The patients had previously been treated with most of the accepted methods, and their inclusion in this study resulted from wholly unsuccessful courses. Each of the patients was considered disabled at the start of therapy." This was therefore a very high-risk group with any form of therapy.

Seventy-one percent of patients treated had subjective improvement of symptoms, 64 percent had objective improvement of measured exercise tolerance three months after receiving 20 chelation treatments, and 46 percent showed improved electrocardiographic patterns. Kitchell and Meltzer then went on to conclude that chelation was not effective because some patients eventually regressed more than a year after treatment. However, considering the poor health of the patients, there is no other treatment about which the same statement could not be made. Eighteen months following therapy, 46 percent of those patients remained improved. The results were very favorable even though the authors' conclusions were not.(17)

I believe that this "reappraisal" article was largely responsible for termination of academic research into chelation as a treatment for cardiovascular ills. Rather than analyzing the data for themselves, most physicians simply accepted the mistakenly negative conclusion at face value. We will probably never know what prompted those early researchers to change their position so abruptly. We can only speculate that it was an unrealistic expectation the emergence of bypass surgery would be a final solution.

The years that followed were filled with astonishing demonstrations of surgical inventiveness; and, for at least the next two decades, cure by the knife dominated the medical landscape. Then came balloon angioplasty. Those surgical and high-tech discoveries were splendid in themselves; but what was tragic was to regard them as the preferred, if not the exclusive, approach to complex cardiovascular problems.

As for chelation, its future is now bright because its effectiveness is incontrovertible. Biased or uninformed physicians may call it untested, but no scientifically informed person can read the studies on which this chapter is based without realizing that EDTA chelation therapy is a formidable antagonist to cardiovascular disease.

A major upsurge in demand for chelation is now coming from the many people who have heard first-hand from friends or relatives who benefited from this remarkable therapy.

Periodic research updates will be posted on this website:

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