

## Current Status of EDTA Chelation Therapy in Occlusive Arterial Disease

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**ABSTRACT:** Benefits of intravenous chelation therapy continue to be unknown to most physicians. A series of unfortunate circumstances led to the cessation of clinical research and delayed wide acceptance of this effective non-invasive therapy, which is much less expensive and infinitely safer than bypass surgery. This article addresses those circumstances and supports the use of EDTA chelation therapy in treating occlusive arterial disease and prefaces summaries posted on this website of a series of highly significant controlled clinical studies proving the safety and effectiveness of this therapy.

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Ethylenediaminetetraacetic acid (EDTA), has been used successfully for many years by a small group of innovative physicians for the treatment of occlusive arterial disease. Scientific research has been limited for this use of EDTA because the patent expired many years ago and no funding has been available for research.

The fascinating history of EDTA chelation therapy is presented in Halstead's monograph, *The Scientific Basis of EDTA Chelation Therapy*.<sup>1</sup> For those not familiar with EDTA, the compound is a synthetic dinitrilotetraacetate which forms chelates with calcium, lead and certain other metallic cations. EDTA was synthesized in the late 1930s in Germany. Clinical use in lead poisoning was first reported in 1952.

In the early 1950s, Drs. Clarke and Mosher in Detroit postulated that because calcium composed a significant part of atherosclerotic plaques, EDTA might be of benefit in the treatment of atherosclerotic cardiovascular disease. They were the first to treat occlusive vascular disease with EDTA and observed positive results with marked improvement in circulation. That resulted in the earliest published reports of effectiveness of chelation therapy for occlusive arterial disease.<sup>2-4</sup> Meltzer and other early pioneers in EDTA therapy also reported clear-cut improvement in arterial function; however, the maximum benefit did not occur until approximately three months after a course of treatment. Meltzer and co-workers stopped their study of EDTA in the treatment of atherosclerotic vascular disease because they did not see immediate results, only to resume their study after patients later called to report improvement.<sup>5</sup>

To this day, there are still no published reports in the scientific literature to disprove the effectiveness of EDTA therapy.

A 1963 article by Kitchell, Palmon, Aytan and Meltzer, reappraising their data from an earlier study in 1960 with subsequent additional patients, has repeatedly been cited as disproving effectiveness of EDTA chelation therapy. However, the authors' conclusion is contrary to their published data.

A careful reading of that entire report shows that a majority of patients did improve and maintained that improvement for a considerable time following therapy. After 18 months and with no stated changes in dietary or other risk factors such as smoking, 46 percent of the 28 patients remained improved. Twenty-three of the 28 patients (82%) had suffered previous myocardial infarctions prior to chelation therapy making this a very high-risk group with any form of therapy.

The stated conclusion in that published report, that EDTA therapy was not effective because some patients regressed after treatment was discontinued, is not justified. There is no other treatment about which that same statement could not be made. Patients treated with bypass surgery, balloon angioplasty, and/or medications alone will usually regress over time.

Those 28 patients were evaluated after 20 infusions of EDTA. More than 64% were rated as improved by the authors. As stated above, eighty-two percent had suffered previous myocardial infarctions and were therefore at a high risk of subsequent cardiac

complications or death. Seventy-one percent of patients treated had subjective improvement of symptoms, 64% had objective improvement of measured exercise tolerance three months after receiving only 20 treatments and 46% showed improved electrocardiographic patterns. At the end of 18 months following therapy, 46% remained improved. These results were very impressive and contradict the authors' negative conclusions. In fact, this was a very favorable study for the benefits of EDTA chelation therapy.

Readers are invited to get a copy of that article, available in any medical library, and confirm for themselves what we have written here. Read the entire article, however, not just the summary with its negative interpretation, and form your own conclusion.

In retrospect, the unsupported negative summary in that "reappraisal" article was, to a great extent, responsible for lack of subsequent funding and for cessation of clinical trials with EDTA chelation therapy for occlusive arterial disease.

In addition, that same article referred to ten patients previously described by the authors in a monograph edited by Drs. Seven and Johnson. Dr. Seven was an ardent enthusiast of EDTA therapy, but he suffered an untimely death shortly after publication of the monograph containing the Proceedings of the First International Conference on Chelation Therapy. Dr. Seven's death is another major factor leading to cessation of research into EDTA chelation therapy and to its lack of wider acceptance. More importantly, the Proceedings edited by Seven and Johnson were published in limited numbers and were not readily available to most physicians.<sup>5</sup>

A careful review of the original article in those Proceedings by Meltzer, Ural and Kitchell, which described the ten original patients updated in the Kitchell, et al. "reappraisal" article cited above, reveals significant contradictions. A sentence in the later article states, ". . . The original ten patients received additional courses of treatment and have also been followed continuously." Careful comparison of the two articles reveals that 30% or three of those original ten patients received no additional treatment (or, in one case, only one additional infusion). Thirty percent of the original ten patients were therefore misrepresented in the later paper.

Descriptions of the initial ten patients selected for intravenous EDTA therapy state,

". . . We selected ten patients 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. "Analysis of the two articles makes it obvious that the original ten patients studied were very sick and were high-risk patients. Despite that fact, 50% were still alive, three of whom remained improved while the other two were ambulatory, active, and no worse when compared to their pre-treatment status at the end of 44 months. This is quite a remarkable benefit and further refutes the negative conclusion at the end of the follow-up article.

A practicing physician first exposed to chelation therapy by reading the Kitchell, et al. "reappraisal" article would probably have read only the summary and not the entire report. It was a very time consuming process to extract and summarize the data above. Also, it is unlikely that interested physicians would have had the original data available as published in Seven's monograph in 1960. The contradictions between actual data and the conclusion and the misstatements in the "reappraisal" article are not apparent without time-consuming study and would be missed by most readers. Nonetheless, this report is repeatedly referred to as disproving the effectiveness of the therapy.

It is tragic that acceptance of the remarkable benefits of EDTA therapy could have been seriously delayed by such a flawed report in 1963. EDTA therapy, as used now, is vastly more effective than those pioneering investigations indicated. The outlook has greatly improved as many complexities and problems of chelation therapy have been understood and the Protocol for administration has been upgraded.

In the early days of chelation therapy, magnesium, manganese, zinc, copper, pyridoxine (vitamin B<sub>6</sub>) and other essential nutrients, which are depleted by EDTA, were not replaced during therapy. No attempts were made to modify such risk factors as cigarette smoking and hyperlipidemias. In the current experience of physicians using chelation therapy, patients who continue to smoke experience less improvement or improvement for a shorter duration following treatment than do those who positively improve such well-known risk factors.

Opponents of EDTA chelation therapy repeatedly refer to a brief review article by Craven and Morrelli, based on obsolete studies and written by scientists with little personal expertise in the use of EDTA for occlusive arterial disease.<sup>7</sup> Craven and Morrelli state in their conclusion that because funds have not been provided for large-scale, multivariate, double-blind, placebo controlled studies to conform to satisfy the FDA's requirement for inclusion of atherosclerotic arterial disease in marketing literature, the use of EDTA should be, ". . . conducted under carefully controlled conditions in an academic institution by experienced investigators." Such a statement insults the clinical competence and judgment of the many hundreds of experienced physicians in private practice who have safely been using intravenous EDTA for decades. The implication is that only academically based scientists are competent to conduct research or to form conclusions about effectiveness. What self-serving hogwash!

One case mentioned by Craven and Morrelli describes a patient with rheumatic heart disease who suffered a stroke from a calcium plaque that broke free from a calcified heart valve. This case was published in the days when EDTA was given in doses and rates of infusion 600% greater than rates now known to be safe and currently used. In the early days a dose of 6 grams was given in as little as 30 minutes. Dr. Soffer, a prominent academically based researcher, states in his monograph on the subject that ". . . serious untoward effects rarely occur if disodium EDTA is administered slowly. As now administered, the maximum daily dose of 50 milligrams per kilogram, adjusted downward for age and kidney function, should be given over a period of three hours or more. "The currently approved protocol for administering intravenous EDTA confirms to that dose rate of administration."<sup>8</sup>

Soffer further states that giving the same dose at three times the safe rate results in untoward reactions in only a very small minority of patients. He describes cases in which a full three grams of EDTA were administered in only 20 to 30 minutes, which is six or more times the safe dose-rate currently utilized by physicians who follow the accepted ACAM protocol for chelation therapy.<sup>8</sup> Craven and Morrelli, repeatedly referred to by critics of EDTA therapy, unjustly point to data obtained when these excessive doses were given to warn that EDTA is "dangerous."<sup>7</sup>

There are many theories concerning the mechanisms of action of EDTA, but it has never been clearly demonstrated which of its many effects leads to the observed benefits. Perhaps an action that has not yet been discovered is responsible for patients' improvements.

A review article by Gordon and Vance lists more than 20 physiologic and biochemical actions of EDTA in the human body.<sup>9</sup> One action not mentioned in that article is the fact that prostaglandin synthetase, also called cyclo-oxygenase, which is responsible for production of prostacyclin, is highly sensitive to inhibition by lead. Recent studies document that the average American's skeleton contains as much as 1,000 times more lead than did individuals who lived 1,600 years ago.<sup>10</sup> It could be postulated that EDTA enhances the production of prostacyclin by the removal of lead. In any case, lead is a potent toxin to both the brain and immune system. Removal of lead known to accumulate in inhabitants of all industrialized nations will be of benefit in many ways other than reversal of atherosclerosis.

Animal studies have shown reversal of atherosclerosis in rabbits with EDTA, hyperbaric oxygen and diet.<sup>11</sup> It has also been proven that EDTA chelation therapy exerts a beneficial effect by improving mitochondrial oxidative phosphorylation, enhancing the efficiency of cell respiration, even in the presence of compromised blood flow and diminished oxygen.<sup>12</sup>

The world's scientific literature contains more than 1,000 articles attesting to the many effects of EDTA. It is time-consuming to track down many such scientific references because they are often not listed in the Index Medicus computer database at the National Library of Medicine and are not available to a routine computer search. The situation is further complicated by the many names in the world literature referring to EDTA (Antallin, Aquamollin, Calex, Calsol, Celon, Cheladrate, Chelaplex, Chelation, Comlexone, Disodium edetate, Disotate, Distal 8, Edathamil, Endrate, Havidote, Indranal, Iminol-D, Irgalon, Kalex, Komplexon, Metaquest, Mosatil, Nervanaid-B, Nullapon, Permakleer, Sequestrene, Syntes 12a, Tetracemin, Tetrine, Titraver, Trilon-B, Triplex III, Tychrosol, Tyclarosol, Versene, Warkeelate, etc.).<sup>1</sup>

A consistent observation by physicians using EDTA chelation therapy in cardiovascular disease is that maximum benefit does not occur until approximately three months after a course of treatment. The mechanism of action is not well understood but one possible explanation involves stimulation of parathormone (PTH) and calcitonin production by pulsatile decreases in serum calcium

during EDTA infusion. Osteoporosis is not observed as a consequence of therapy and may even improve somewhat. Early investigators of EDTA speculated that removing calcium from the arterial walls was the most important effect.

In the early days of chelation therapy, incomplete understanding of the action of EDTA on essential trace element cations resulted in deficiencies of zinc. Essential nutritional metallic trace elements are now replaced by oral supplementation both during and after therapy. Zinc has been proven to participate in more than 80 metalloenzymes.<sup>13</sup>

It is not surprising that EDTA therapy, rather than being toxic to the kidney as first reported, can actually be beneficial. Adults with chronic lead exposure experience interstitial nephritis and hypertension.<sup>14</sup> Physicians using chelation therapy consistently observe improved kidney function reflected by improved creatinine clearance and reduction of elevated blood pressure.

EDTA therapy requires additional large, placebo-controlled studies. However, in designing such studies it must be remembered that vascular changes may not be noted arteriographically in spite of significant clinical improvement. Using Poiseuille's Law of hemodynamics one can demonstrate that with perfect laminar flow, a mere 19% increase in the diameter of a blood vessel will double the flow rate. In a vessel with turbulent flow, such as a diseased artery with plaque, this figure decreases to something less than a 10% increase in diameter to cause a doubling of blood flow. In an organ with compromised circulation, an increase in blood flow of 10 to 20% could result in significant functional improvement and relief of symptoms of ischemia. EDTA therapy is known to reduce the number of cross-linkages in connective tissue, improving elasticity sufficiently to allow an artery to dilate, in addition to any effect on existing plaque.<sup>15,16</sup> Arteriography cannot measure vascular changes more precisely than plus or minus 25% with any consistency.<sup>17</sup> With this lack of precision, arteriographic measurements are not valid indicators to prove or disprove the effectiveness of chelation therapy. Noninvasive testing such as visualization of ventricular wall function, changes in injection fraction, and measurements of cerebral or myocardial blood flow using radioisotope imaging, in conjunction with other noninvasive tests of function will lead to more meaningful conclusions.

Clinical studies of that type have been published and do prove that blood flow and function both increase significantly following EDTA therapy.<sup>18-20</sup> Those studies should be repeated by other research workers using a larger number of subjects. The report summarized below by McDonagh, Rudolph and Cheraskin provides a controlled and statistically significant example of independent verification of the previous studies by Casdorph.<sup>18-20</sup> This is good science.

It seems reasonable that future studies should not rely entirely on blood flow measurements, because published observations indicate that improvement in symptoms does not necessarily correlate with improvement in blood flow.<sup>18,19</sup> Metabolic efficiency may improve, even in the presence of compromised blood flow. Patients with little improvement in blood flow do sometimes show marked improvement of function and relief of symptoms. These observations may be explained by removal of toxic heavy metals or by other changes. Future studies should include before and after noninvasive measurements of physiologic function and endurance; including physical stress testing, cognitive tests of cerebral function, distance walked before onset of claudication or angina, requirements for nitroglycerin and other anti-anginal drugs, thermography, plethysmography, Doppler blood flow studies, oculocerebrovasculometry, urinary excretion of various toxic metals and other objective measurements amenable to statistical analysis.

Regardless of blood flow changes, if claudication and angina are relieved with reduced use of prescription drugs, and if physical endurance or mental ability improves, such benefits would be quite enough to justify treatment with EDTA. All published studies indicate that those improvements do occur in a high percentage of patients treated with EDTA chelation therapy.

Hundreds of thousands of coronary artery bypass procedures have been performed without benefit of controlled studies to prove safety and effectiveness. Medical insurance companies, including Medicare, have traditionally paid for those very expensive and risky surgical procedures without question. An assumption has been made that angiographic evidence of occlusion with follow-up evidence of improved blood flow was sufficient justification for surgery. It has never been demonstrated that this is the major or only reason for relief of symptoms. A number of years ago cardiac sympathectomy was used to effectively relieve angina. Perhaps cardiac sympathectomy (myocardial denervation), which inevitably accompanies coronary artery bypass surgery, leads to subsequent improvement of coronary vasospasm and is an important factor in post-surgical improvement of angina.<sup>21</sup>

EDTA is a generic substance. Its patent expired many years ago. Without patent protection no pharmaceutical company will spend the many millions of dollars required to fulfill stringent requirements of the FDA to add the indication of occlusive arterial disease to marketing literature. Practicing physicians cannot afford the time or money to perform such studies. This leaves only the federal government or wealthy foundations to fund further research into EDTA therapy.

The U.S. Food and Drug Administration (FDA) formerly utilized a standard letter in response to requests for information concerning EDTA therapy. Such requests frequently come from senators and congressmen in response to complaints from their constituents who have been refused payment by medical insurance. A year ago the FDA repeatedly stated that EDTA was unsafe and could be "fatal". The same statement could be made concerning water, oxygen, table salt and many other benign substances, if administered in large concentrations or too rapidly. Subsequently that same FDA totally reversed itself. In recent years, when they issued Investigational New Drug Licenses (INDs) to researchers of chelation therapy, FDA scientists reviewed the literature and concluded that safety was not an issue. EDTA is a safe, inexpensive, outpatient procedure that avoids the need for hospitalization. The FDA also mailed a letter to medical licensing boards in all states asking for information about complaints of harm caused by EDTA chelation therapy. Not a single response of harm was returned.

Opponents often state that the use of EDTA for arteriosclerosis should only be administered out under an IND because the treatment "is investigational". This statement is untrue. The FDA, federal and state courts, the AMA, and state medical licensing boards have all ruled that a physician is free to use any approved drug for any use that in the physician's professional judgment is in the best interests of the patient. EDTA is a FDA-approved drug. A physician is in no way legally constrained to restrict the use of a drug to those indications approved for marketing claims by the FDA and published on the package insert. Disodium EDTA is FDA approved for uses other than occlusive arterial disease. It is a little known fact that the FDA does not regulate what a doctor can or cannot use in his or her practice. By law, the FDA can only regulate marketing and advertising claims for drugs and medical devices sold across state lines.

FDA regulations require full disclosure of all evidence of adverse effects on the package insert that accompanies each bottle or vial of a prescription drug, no matter how minor that evidence may be. However, evidence for proof of effectiveness cannot and will not be listed until many millions of dollars have been spent for very extensive and complicated studies.<sup>23</sup> Many highly competent, ethical, and respected doctors routinely administer intravenous EDTA chelation therapy in their clinical practices for symptoms of cardiovascular disease, with consistently good results. Available information indicates that almost as many patients have been treated with this therapy as have been treated with coronary artery bypass surgery and/or balloon angioplasty.<sup>1</sup>

The National Center for Health Care Technology (NCHCT), a division of office the U.S. Congress, administered by the U.S. Public Health Service, evaluated intravenous EDTA therapy, when used for the treatment of atherosclerotic arterial disease, for possible reimbursement by federally-funded medical insurance (such as Medicare). The initial report of the NCHCT classifies use of EDTA in the treatment of occlusive arterial disease as investigational, which prevents reimbursement by Medicare.

In retrospect it appears that the NCHCT based its conclusions on statements by cardiovascular surgeons, the American Heart Association and from the American College of Cardiology, on the assumption that those groups represent experts in the field of EDTA therapy. The opposite is true. Most practicing cardiologists and surgeons have little or no professional expertise in the use of EDTA therapy for cardiovascular disease. Professional associations of cardiologists and cardiovascular surgeons have consistently opposed chelation therapy, despite their lack of first-hand knowledge concerning its effectiveness and safety in the treatment of occlusive arterial disease. They have consistently voiced strong prejudice and opposition against allowing chelation therapy to encroach on their turf.

Politically powerful and traditional medical organizations represent large groups of physicians who make their livelihood with catheterization studies, balloon angioplasty, and bypass surgery. That is a \$5 billion per year industry in the United States alone. Many hospitals would be in financial difficulty and many vascular surgeons and cardiologists would be forced to find other applications for their medical skills without these procedures. To call on those segments of the medical profession using surgery to make recommendations concerning the use of EDTA is comparable to asking "the fox to guard the hen-house."

While cardiologists and vascular surgeons enthusiastically promote the utilization of very expensive, highly invasive and dangerous catheterization and bypass surgery, there are more than two thousand physicians in the United States, and others overseas, who routinely and successfully treat cardiovascular intravenous EDTA. Medical insurance companies are consistently advised by those same self-styled "experts" not to pay for chelation therapy but to pay instead for much more expensive cardiovascular surgery and angioplasty.

Benefits last for many months following a course of therapy with intravenous EDTA, while the effects of prescription drugs are short-lived and they must often be taken several times each day at considerable expense. EDTA has behind it 40 years of clinical experience with proven low toxicity when properly administered.<sup>1</sup> Treatment with EDTA has more benefits than the well-publicized calcium blockers and other drugs. EDTA removes toxic metals, which contaminate everyone, and EDTA improves elasticity of connective tissues throughout the body.<sup>1,15,16</sup>

John Olwin, M.D., a respected cardiovascular surgeon and retired medical school professor at the University of Illinois, has been using EDTA chelation therapy in his practice for more than 20 years, often in preference to vascular surgery. He has had extensive experience in the treatment of occlusive vascular disease using intravenous EDTA and he reports a very high percentage of successful results.<sup>25</sup> Ralph Lev, M.D., M.S., a respected vascular surgeon, retired medical school professor and formerly Chief of Cardiovascular Surgery at the teaching hospital of the New Jersey Medical School, also uses EDTA chelation therapy extensively in his practice, often in preference to surgery or as an adjunct to surgery.

Using EDTA, many ischemic limbs have been saved from amputation and cerebrovascular symptoms of dementia and ischemia have been reversed. Strokes have been arrested and prevented. Angina is often relieved without surgery. EDTA therapy is more effective than other non-surgical therapies in a large majority of patients treated.<sup>1-6,9-16,18-20,25-32</sup>

Improvement occurs throughout the whole body, not just in one small segment of a blocked artery.<sup>1,9,15,16,33-35</sup> Depending upon modification of lifestyle and risk factors of disease, treatments may have to be repeated from time to time, but the same applies to bypass surgery or to any other form of therapy. The underlying disease usually progresses, despite aggressive therapeutic procedures. With chelation therapy extension of quality life is predictable.<sup>16</sup>

An additional long-term benefit of EDTA was reported from Switzerland in 1980 by Blumer and Reich.<sup>31</sup> A ten-year follow-up of 59 patients treated with EDTA therapy showed a 90% reduction in the death rate from cancer, when compared with 231 control patients. Statistical analysis confirms that these findings are highly significant. This report has important implications for the practice of preventive medicine.

Double-blind studies of EDTA chelation therapy that will satisfy all opponents are will probably be impossible because of the multifactorial nature of the ideal total therapy. When studying occlusive arterial disease the scientific community must be convinced to revamp its collective thinking and accept a new scientific method with positive functional results as proof of effectiveness.

As stated by Watts, ". . . a basic criterion of whether a method is scientific or not is whether it works for the purpose intended.... It is difficult, often impossible, to control all of the variables in a given study or experiment, and the linear process often takes time, too much time. Results are needed sooner, not later, to be useful in medical practice."<sup>37</sup> Watts argues for a new scientific method in medicine which will ". . . produce timely results in terms of the life span of both physicians and their patients."

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