Dr. Cranton's Current Thoughts about EDTA Chelation Therapy, February 2017

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There are many theories that attempt to explain the mechanism of action by which EDTA chelation therapy benefits atherosclerosis and other age-related diseases. However, we still do not really know how it works. I discuss below my latest thoughts on that subject.

Atherosclerosis as been shown to have has multiple contributing causes. Infection is one such potential cause for which there exists an extensive amount of evidence. Benefits of EDTA chelation therapy in treatment of atherosclerosis may be, at least in part, a result of its unique antibacterial and anti-infective properties, which are concentrated on blood vessel walls and developing plaque. Very positive benefits are reported in the recently published Trial to Access Chelation Therapy, which could be interpreted to support the theory of infection as an important cause.

A review of the large amount of published data implicating *Chlamydia pneumoniae* as a likely cause of atherosclerosis and coronary heart disease is very convincing. You are invited to search this topic further. Other infective organisms have been implicated, but the evidence for *Mycoplasma pneumoniae* is by far the most convincing. This includes serological data, epidemiological studies, physical detection of the *Chlamydia* organism in plaque, data from PCR studies, and histopathology all point to *Chlamydia* as a causative organism.

Treatment with conventional antibiotics in several large clinical trials was not consistently beneficial, and subsequent interest in *Chlamydia* diminished. We now know, however, that *Chlamydia* has a very complex life cycle and periodically converts to a metabolically inert, intracellular body, somewhat like a virus, that becomes resistant to antibiotics. Those antibiotic trials might have been expected to fail because the inert stage periodically reactivates to reinfect the host.

Unlike other antibiotics, the antimicrobial activity of EDTA does not require metabolic activity. It acts more like an antiseptic. EDTA strips essential trace elements from microorganisms, including zinc, iron, manganese, and others, without which further bacterial growth and replication is not possible.

EDTA remains extracellular in the body and does not cross healthy cell walls. The potent antibacterial properties of EDTA are thus concentrated in circulating plasma and focus largely on plaque and blood vessel walls—where *Chlamydia organisms* sequester. Much higher chelating activity acts on the inner surfaces of blood vessels and plaques. EDTA avidly binds and inactivates essential trace metals

—without which microorganisms cannot grow or replicate. That is one reason that EDTA is used as a food preservative. It is self-sterilizing in solution.

Although oral EDTA is somewhat absorbed, it is also rapidly excreted in urine and blood levels never become high enough to exert a significant chelating effect on plaque. Oral EDTA has never been shown effective in a clinical trial.

Plaque is a diseased form of tissue, resembling scar, and does not possess a normal cell membrane. It thus seems reasonable to believe that high levels of infused intravascular EDTA can diffuse into plaque and attack any hidden microorganisms. Disodium EDTA also removes calcification from plaques, which would increase permeability to EDTA. That does not occur with calcium EDTA. Calcium EDTA has never been shown effective in a clinical trial, although anecdotal evidence does support partial benefit.

It is herein proposed that antibacterial action against *Chlamydia* is an important mechanism of action for benefit against atherosclerosis following EDTA infusion. That theory does fit the data.

This mechanism of action would also explain why full benefit following EDTA chelation takes several months to occur and also explains why a prolonged infusion over several hours results in more benefit. It would take some time for plaque to regress and scar tissue to shrink following elimination of the infective organism.

A temporary drop in plasma pH occurs when infusing disodium EDTA (without calcium). EDTA releases acid into the circulation when it binds to intravascular cations, such as calcium, magnesium, and many others. Two hydrogen ions are released for each polyvalent cation as it is chelated. The resulting acid shift in plasma pH increases the chelating properties of EDTA. This may be another reason that disodium EDTA is more effective than calcium EDTA. That acid shift does not occur when infusing either calcium EDTA or magnesium EDTA.

Clinicians have come to prefer calcium EDTA because it can be infused more rapidly without warning signs of hypocalcemia. They forget, however, that renal toxicity may occur silently, without warning any symptoms in susceptible patients when a full dose of calcium EDTA is infused in less than three hours.

Because disodium EDTA (without calcium or magnesium) can cause pain at the infusion site (venous spasm as a result of the acid shift in pH), lidocaine is added to the infusion. If discomfort is still a problem, the use of a larger antecubital vain, application of moist heat, or infusion of a bit more lidocaine directly into the IV tubing will usually solve that problem. If patients ae informed that the discomfort is related to beneficial effect, they will be reassured.

In some locations it is difficult to find disodium EDTA (without the calcium). In that case it may be necessary to have a compounding pharmacy make the necessary supply.

When an isotonic carrier solution of either normal saline or 5% dextrose and water is used, osmolality of the infusion is not a problem.

The TACT study showed EDTA chelation to be significantly more effective in patients with diabetes. It is well known that bacterial infection is more of a problem with diabetes, providing more evidence for the infection hypothesis.

The TACT study used a complex infusion solution with magnesium EDTA and many other additives. Personal observations over the years seem to show that benefits were greater with the simpler protocol used for many earlier years, from 1955 through 1995, using only disodium EDTA and lidocaine in an isotonic carrier solution. Many earlier published studies using that simple protocol reported results that appear better than TACT results.

MISLEADING INTERPRETATION OF TOXIC HEAVY METAL DATA

The "*linear toxicity with no threshold (LNT) model*" is a highly misleading scientific error. That model assumes that if a substance is toxic at high levels, decreasing toxicity continues in a linear fashion all the way down to a zero level. In actual fact, a safe threshold level almost always exists below which no further toxicity occurs.

Every living person has some small amount of toxic heavy metals in their body's'—usually at low and safely tolerated levels. Scientific instruments have become so sensitive that extremely low levels can now be measured. Because EDTA is rapidly excreted in the urine while chelating metal ions from the body, a chelation infusion greatly magnifies the concentration of many metals in urine. Chelation magnifies urine levels many fold above baseline, even when baseline excretions are well below the threshold for toxicity. This often leads to a false diagnosis of 'heavy metal toxicity' in chelation patients. An accurate diagnosis of toxicity requires testing of blood levels.

EDTA binds and removes nutritional metals, such as zinc and manganese, 10 to 15 times more aggressively than its effects on purely toxic metals. It seems quite possible that those much greater actions explain subsequent benefit.

There is no evidence that heavy metal toxicity causes atherosclerosis or coronary heart disease. It is merely a coincidence that EDTA is used to treat atherosclerosis and is also used to treat heavy metal toxicity.

To dispel another myth, heavy metals do not commonly promote free radical damage. It is only when ionic metals in solution change easily by one valence state, i.e., ferrous to ferric, cuprous to cupric, manganese, chromium, and perhaps others, that they can act to catalyze an increase of free radicals.

There is no scientific reason to believe that heavy metal toxicity or removal of toxic metals is a reason for benefit in chelation for cardiovascular disease. That seems a 'red herring' and represents a classical error of logic: *Post hoc ergo propter*

hoc (after this therefore because of this). That logical fallacy is based on a mistaken notion that because one thing happens after another, the first event was a cause of the second. Post hoc reasoning is the basis for many superstitions and erroneous beliefs.

These musings are best read in association with the following links. (You may need to copy and paste these links into your browser.) Many references in those links will take your further.

http://drcranton.com/chlamydia.htm

http://drcranton.com/chelation/recommendedingredients.pdf

http://drcranton.com/chelation/TACT2A.pdf

http://drcranton.com/chelation/EDTAstudies.pdf

Until data becomes available that demonstrates equivalent benefit, it is strongly urged that the use of calcium and magnesium EDTA be avoided and that patients be treated only with disodium EDTA, in an isotonic carrier solution with lidocaine to prevent pain. The many highly favorable clinical trials published between 1955 and 1995 used only that simple protocol with disodium EDTA. There has never been a published study showing benefit from calcium EDTA, although partial benefit probably does occur. The TACT study used magnesium EDTA but overall results were less than reported in the earlier studies with only disodium EDTA (without calcium). Although Calcium EDTA is probably of some benefit, it is thought to be of significantly less benefit that disodium EDTA.