

Chelation Article Misleading

As a former contributor to the *Townsend Letter*, I would like to comment on Dr. Michael Gerber's article "Thirty Years of Progress in Cardiovascular Health" (July 2011). Certainly, it provides useful insight into EDTA chelation therapy, but also lists misleading information that continues to be published in some form or another, and thus is being accepted by chelation therapists as a medical fact. The statements that I oppose may be insignificant when compared with the rest of his manuscript, but they are medically significant. While I do not want to criticize or belittle Dr. Gerber's notable effort, I wish to provide some facts that warrant notification:

1. Available studies indicate that atherosclerotic disease can be successfully treated with intravenous NaMgEDTA. Dr. Gerber's list of research papers well supports his statements.
2. Dr. Gerber refers to mercury detoxification, and while he does not refer to EDTA as a mercury-binding chelator, the reader may get the impression that EDTA chelation removes mercury. Fact is, EDTA has an extremely limited mercury-binding ability (because it has no SH-groups in its molecules).
3. Regarding EDTA in oral or suppository form: if in this form, only 5% of EDTA is actively absorbed, and therefore only this 5% is able to bind metals.
4. The activity of oral EDTA is that of any oral chelating substance: it first detoxifies the digestive tract. The greater the availability of metals found in the digestive tract, the more metal binding occurs. If all binding sites are occupied, additional binding is limited.
5. For systemic detoxification to occur, EDTA must enter the bloodstream. If it is already bound by metals found in the digestive system, *additional* metal binding is further limited.
6. The same principle applies to EDTA suppositories, although a suppository's metal binding is even more limited because its function is limited to the colon.
7. In our laboratory, we have accumulated and statistically evaluated an impressive body of chelation data. In comparison with other chelating data, the data relating to the single use of EDTA suppositories or oral EDTA are limited, mostly because physicians generally use these products in combination with other chelating substances. Hence, we cannot safely say which substance was responsible for whatever metal binding occurred.
8. Very few physicians check fecal or urinary excretion after the *single use* of oral EDTA or EDTA suppositories, and by single I mean administering EDTA (oral or suppository) without the added use of orthomolecular nutrients, algae products, etc.
9. Since EDTA in oral or suppository form primarily detoxifies the

digestive tract, additional detoxification of the vascular system or other body organs is extremely limited. As a point in fact, our data do show an increase in fecal metals. When compared with baseline levels, urine excretion levels after the administration of oral EDTA or EDTA supps are *not* statistically significant.

10. Provided EDTA entered the bloodstream, over 90% is excreted via the renal pathway. Logically, an increase in urine metal concentration can be expected.

Before I state that the use of EDTA in oral or suppository form is a waste of time and money, I propose that the manufacturers of these pharmaceutical products engage into unbiased studies and let data speak for themselves. To this date, I have not seen research data of significance, proving the drugs' usefulness. If they exist, I would appreciate notification.

Most sincerely,
E. Blaurock-Busch, PhD
Wissenschaftliche Leitung/Scientific Management
Scientific advisor, IBCMT
(International Board of Clinical Metal toxicology) & KMT (German Medical Association of Clinical Metal Toxicology)
www.microtrace.eu
www.microtrace.de
www.microtraceminerals.com
P.O. Box 4613
Boulder, Colorado 80306-4613