CHELATION THERAPY - AN INFORMAL SUMMARY

The goal of chelation therapy for treating arteriosclerosis (or atherosclerosis) is decreasing the narrowing of arteries by the removal of calcium. Calcium is present in some arteriosclerotic lesions. The drug used, disodium EDTA (disodium edetate, Endrate) grasps or binds calcium, and the hope is that it will grasp calcium from these areas of arterial obstruction.

Such "chelation therapy" is commonly accompanied by other therapies, such as trace element "supplements," large doses of vitamins and dietary modifications. Nevertheless, the central element of "chelation therapy" is repeated intravenous infusions of disodium EDTA.

There is no sound evidence that chelation therapy works -- that it is effective or has clinical benefit.

There is also an important fallacy in this underlying idea. When disodium EDTA is administered intravenously, it encounters calcium everywhere in the blood and it binds calcium from the blood, not from the tiny deposits of calcium that may exist in arteriosclerotic lesions. The bound calcium is removed from the body through the kidneys. The calcium in blood is replenished by calcium from the bone, which is easily accessible to the blood stream, or by calcium from the gut.

The advocates of "chelation therapy" have testimonials from people who feel that their symptoms have been relieved. However, there is no clinical trial or clinical study with scientific merit that has shown the purported beneficial effects. Until August, 1991, the best study was published in 1963. It concluded that "we believe that chelation as used in this study did not benefit patients more than other commonly used therapeutic methods. It is not a useful clinical tool in the treatment of coronary artery disease at the present time". By today's standards, that study has some shortcomings but there is no subsequent study to contradict those results. In August, 1991 and early 1992, reports were published of a large, scientifically sound clinical trial that carefully documents that chelation therapy does not work. Symptoms, clinical findings, x-rays, and other measurements were compared in patients who received the chelation therapy and compared to similar measurements in patients who received a "dummy" therapy instead of chelation. The results were identical; chelation therapy was shown to be of no value.
What about people who have felt better following chelation therapy? In the objective assessment of a variety of medical therapies, including some for arteriosclerotic disease, people have often felt much better in response to sham therapy or "placebos." Indeed, the more dramatic the intervention, the more likely the effect. The more someone has invested in feeling better -- and a course of chelation therapy may be on the order of 30 to 50 intravenous infusions over several months and costs thousands of dollars -- the more likely a person is to be convinced that there has been a benefit.

It might be noted that "chelation" is a chemical term meaning that a compound grasps a metallic element to form a ringed structure. In that sense, some forms of chelation are accepted and appropriate forms of therapy. For example, deferoxamine is a validated therapy in the management of iron overload following repeated blood transfusion and EDTA, similar to that used in chelation therapy of arteriosclerosis, is appropriate and accepted for the treatment of lead poisoning.

This has nothing to do with the alleged benefits of chelation in the management of arteriosclerosis. There is no reason to expect benefit from chelation in the management of arteriosclerosis. More importantly, there has been no scientific evidence of such benefit -- and now there is scientific evidence of no benefit.