Noninvasive Treatment for Sequellae of Failed Coronary Blood Circulation: 100% Occlusion of Left Anterior Descending Coronary Artery, 30% Stenosis Right Coronary Artery, and Left Ventricular Contractility Deficit

E.W. McDonagh, D.O. and C.J. Rudolph, D.O., Ph.D.

2800A Kendalwood Parkway, Kansas City, MO 64119, USA

Abstract. Following a myocardial infarction, a symptomatic 59-year-old male Caucasian was found by coronary arteriography in 1990 to have 100% closure of the left anterior descending coronary artery and 30% stenosis of the right coronary artery. His left ventricle was hypokinetic and exhibited dilatation and abnormal contractility. He refused coronary artery bypass surgery and angioplasty because of risk and instead chose the non-traditional ethylenediaminetetraacetic acid (EDTA) chelation therapy. The disease process, as seen by comparing pre- and posttreatment arteriograms in 1990 and 1992, respectively, has been significantly reversed.

Key Words: Ethylenediaminetetracetic acid—EDTA—Chelation therapy—Coronary artery disease.

Introduction

The death rate for cardiovascular disease has dropped 51% since 1950, yet this disease will claim more than one million American lives this year. According to the American Heart Association the cost for 1992 was \$117.4 billion in physician and nursing services, hospital costs, medication costs, and lost productivity [1].

Much of this expense can be attributed to two procedures that are being recommended with increasing frequency: coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA). In seven years, the number of CABGs performed has increased from 180,000 to 380,000, and the number of PTCAs has increased from 30,000 to approximately 285,000 [2]. These procedures are often repeated, and the question must be considered whether or not we are reducing the incidence of heart disease or merely providing an expensive, temporary solution. When the CABG pro-

cedure was thoroughly investigated in a multi-center study and published as the Coronary Artery Surgery Study (CASS), it was demonstrated that the surgery (CABG), when compared to traditional medical and drug therapy, offered no additional benefit in terms of morbidity, mortality, or reinfarction rate [3].

In this paper we describe how treatment with intravenous ethylenediaminetetraacetic acid (EDTA) can offer unique advantages beyond those provided by treatments routinely utilized today.

The use of EDTA chelation therapy, originally patented in the late 1930s for the treatment of lead poisoning, has advanced significantly since Clarke first reported on its effectiveness in coronary artery disease [4]. Since then, intravenous EDTA has been shown to be effective in a wide variety of chronic degenerative diseases. It improves renal function [5,6], carotid artherosclerosis [7-9], peripheral vascular disease and intermittent claudication [10,11], plasma lipid abnormalities [12-14], diabetes [15], osteoporosis [16], pulmonary function disorders [17], and platelet aggregation [18,19]. Serum calcium levels, which might be expected to drop during this therapy, have remained within normal range throughout and after treatment with EDTA [20]. A study of over 2,800 patients has shown chelation therapy to be of significant benefit in treatment of cardiovascular, cerebrovascular. and peripheral vascular disease [21]. In addition, the authors have published several papers on the improvement in physiologically measurable variables during submaximum exercise stress testing, such as heart rate [22] and systolic blood pressure [23], in patients who have undergone chelation therapy.

It has been demonstrated in animal studies that intravenous EDTA therapy will remove calcium from the arterial wall in atherosclerotic rabbits as well as remove calcified atherosclerotic plaque ([24] and F.M. Walker, pers. comm.), but the reduction in atherosclerotic plaque in human coronary arteries has not been demonstrated until now.

Materials and Methods

The patient, C.L., is a 59-year-old male Caucasian commercial airline pilot who suffered a myocardial infarction in April 1990, followed by cardiac catheterization. The patient had a total occlusion of the left anterior descending coronary artery (LAD) just below the bifurcation of the left main coronary artery (LCA), and a 30% lesion in the right coronary artery (RCA). In addition, his left ventricle was hypokinetic and exhibited dilatation and abnormal contractility. Hence, CABG was advised. The patient refused because of the risk of the procedure and instead chose the nontraditional intravenous EDTA chelation therapy.

When first seen at this facility in September 1990, he complained of shortness of breath, increasing fatigue, and anginal pain which had been gradually increasing in severity while under previous medical care. He was given 35 treatments (as described later) at this facility. His symptoms ceased and function improved as a result of the therapy. He returned home and continued with additional treatments.

C.L. received a total of 72 chelation treatments. In October 1992, the second coronary arteriogram was performed showing the LAD now to be 60–70% stenosed. In addition, the previous 30% stenosis of the RCA was eliminated. Only a mild hypokinesis of the ventricular wall persisted.

A thorough history and physical examination including a comprehensive blood chemistry profile was performed. A solution composed of 3 g of the disodium salt

of EDTA in 500 cc of 5% dextrose with 3 cc of 50% magnesium sulfate, 20 meq (10 cc) potassium chloride, 1 cc B-complex vitamins, and 7.5 g of sodium ascorbate was then infused intravenously over a 3 h infusion time. The patient received 30 identical infusions in the initial treatment phase. Blood chemistry results and electrocardiograms were repeated after each ten infusions. Oral broad-spectrum nutrient supplements were provided.

Pre- and post-treatment coronary arteriogram films were examined, measured, and evaluated by a board certified cardiologist.

Results

Figures 1a and 2a, and 1b and 2b represent the angiograms of patient C.L. in 1990 (pretreatment) and 1992 (posttreatment), respectively.

Figure 1a and b depict the left coronary artery system. Figure 1a shows the LAD which is totally occluded near its origin (see arrow 2). The LCA is patent (arrow 1), and an anomaly, the intermediate ventricular branch of the left circumflex coronary artery, appears superior (cranial) to the left anterior descending artery (arrow 4). The circumflex coronary artery appears caudal to the left anterior descending artery (arrow 3). The LAD is totally occluded near its origin, while other vessels have atherosclerotic luminal irregularity present without high-grade obstruction.

In Fig. 1b, the LAD is patent with luminal irregularity

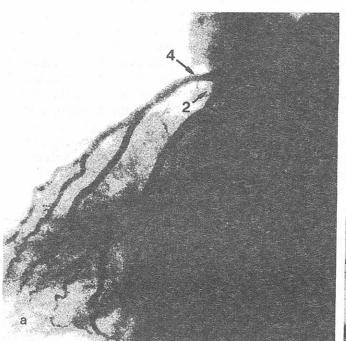




Fig. 1. Coronary arteriogram of patient's left anterior descending coronary artery prior to (a) and after (b) treatment with EDTA chelation therapy.

and recanalization (arrow 2). The intermediate ventricular branch (arrow 4) is seen cranial to the left anterior descending carotid artery. The main body of the left circumflex coronary artery (arrow 3), is seen caudal to the LAD and the LCA (arrow 1). The residual stenosis within the left anterior descending carotid artery is estimated at 65% with a complex plaque preceding the obstruction.

Figure 2a and b are angiogram prints of the patient's RCA both prior to (Fig. 2a) and after (Fig. 2b) the patient had been treated with EDTA chelation therapy.

In Fig. 2a, the trunk of the RCA is denoted by arrow 1. The 30% occlusion in that same artery is indicated by arrow 2. In Fig. 2b, the proximal right coronary is again indicated by arrow 1 but the area of the 30% occlusion (arrow 2) shows no luminal defect. In addition, multiple areas representing lower grade (less than 30%) lesions of atherosclerotic plaquing in the area proximal to the lesion are indicated by arrow 3. These are notably absent or greatly reduced in Fig. 2b. The increased intensity of the angiographic dye column is much more dense in the posttreatment angiogram (Fig. 2b arrow 4), representing significant improvement in perfusion distal to the original lesion. Figure 3a and b show, respectively, an artist's representation of the original coronary architecture and the posttreatment results. The 100% original occlusion and the 65% posttreatment lesion of the LAD are denoted, as is the original 30% lesion in the RCA.

This improvement in coronary arterial occlusion status was accompanied by an improvement in the patient's left ventricular contractility as measured by pre- and posttreatment ejection fraction calculations.

It is estimated that the ejection fraction of 30% prior to therapy was increased to 55% after therapy. If one assumes that a normal ejection fraction is 70%, and normalizes these results to that figure, then the overall improvement in the patient's left ventricular contractility is about 36%. This increase significantly improved the quality of life for this patient.

Discussion

Intravenous EDTA therapy has been shown to be a viable alternative in cerebrovascular and peripheral vascular disease, but as far as we have been able to determine, no coronary obstruction has been proven to be regressed with this therapy. This case report demonstrates that EDTA chelation therapy can reverse atherosclerotic plaque in the coronary arterial system. There are several possible explanations as to how this has occurred.

This patient could have developed a thrombus which occluded a previously partially stenosed LAD. It is possible that the body's internal process of autolysis dissolved the blockage. While this theory is worthy of consideration, it does not explain the great improvement in contractility of the ventricular musculature, dissolution of obstructing atheromatous disease throughout the coronary vasculature, and great improvement in myocardial blood flow. Also, the extended period of time that the LAD was occluded before beginning EDTA treatment (April 1990 to September 1990) would tend to discredit the autolysis theory. This patient was symptomatic during this time.



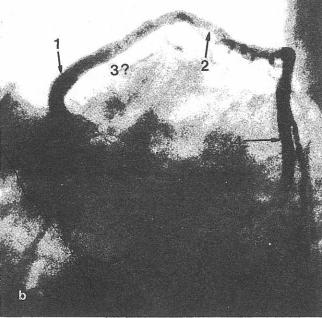


Fig. 2. Coronary arteriogram of patient's right coronary artery prior to (a) and after (b) treatment with EDTA chelation therapy.

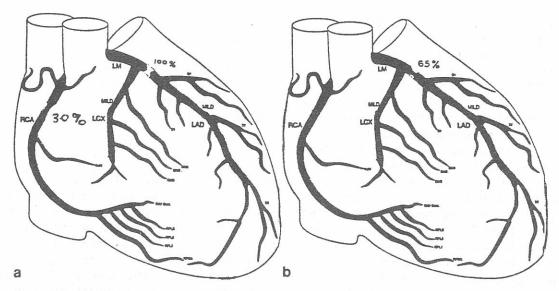


Fig. 3. Pre- (a) and post- (b) treatment coronary artery architecture.

The longer the ventricular muscle is ischemic, the poorer the prognosis for the normalization of pumping efficiency, and the more likely is failure, inefficiency, and aneurysmal dilation. His left ventricle shows post-treatment remodeling, normalization, and improved pumping action.

The autolysis theory would not explain the great improvement in inside diameter of the RCA and other, smaller, previously stenotic coronary branches and collaterals. The 30% stenosed RCA was never totally occluded, therefore, thrombus formation was absent and autolysis would not account for its improvement. In addition, there was a significant increase in distal runoff in the terminal branches of the RCA. There appeared to be a significant amount of diffuse disease in the area proximal to the moderate grade (30%) right coronary lesion, which resolved post treatment.

Previous studies have questioned the feasibility and frequency of bypass surgery and angioplasty. Graboys et al. [25] concluded that in many instances a significant number of angioplasties could be delayed or avoided, pending appropriate medical therapy. It was also concluded that approximately 50% of patients undergo inappropriate CABG. These recent findings underscore the findings of the Coronary Artery Surgical Study (CASS) that, when compared to medical therapy, CABG does not appear to prolong life, nor does it prevent future myocardial infarctions in patients experiencing mild angina or who are asymptomatic after infarction following coronary angioplasty [3].

Additional published case studies showing regression of coronary disease in patients treated with EDTA chelation therapy will provide additional proof of its efficacy.

References

- American Heart Association. Heart & Stroke Facts. American Heart Association, 1993
- 2. Washington, DC: National Center for Health Statistics and the Commission of Professional Hospital Activities, 1992
- CASS principal investigators and their associates. Myocardial infarction and mortality in the coronary artery surgery study (CASS) randomized trial. N Engl J Med 1984; 310(12):750-758
- Clarke NE, Clarke NE Jr, Mosher R. Treatment of occlusive vascular disease with disodium ethylenediaminetetracetic acid (EDTA). Am J Med Sci 1960; 239:732–744
- McDonagh EW, Rudolph CJ, Cheraskin E. The effect of EDTA salts plus supportive multivitamin/trace mineral supplementation upon renal function: a study of serum creatinine. J Holist Med 1982; 4:146-151
- McDonagh EW, Rudolph CJ, Cheraskin E. The effect of EDTA chelation therapy plus supportive multivitamin mineral supplementation upon renal function: a study in blood urea nitrogen (BUN). J Holist Med 1982; 4:21–23
- McDonagh EW, Rudolph CJ, Cheraskin E. An oculocerebrovasculometric analysis of the effect of EDTA chelation upon vascular stenosis. J Holist Med 1982; 4:21–23
- Rudolph CJ, McDonagh EW. Effect of EDTA chelation and supportive multivitamin/trace mineral supplementation on carotid circulation: case report. J Adv Med 1990; 2(1): 5-11
- Rudôlph CJ, McDonagh EW, Barber RK. A nonsurgical approach to obstructive carotid stenosis using EDTA chelation. J Adv Med 1991; 4(3):157–166
- McDonagh EW, Rudolph CJ, Cheraskin E: The effect of EDTA chelation therapy plus multivitamin/trace mineral supplementation upon vascular dynamics (ankle/brachial systolic blood pressure). J Holist Med 1985; 7(1):16–22

- 11. Olswewer E, Sabbag FC, Carter J. A pilot double blind study of sodium-magnesium EDTA in peripheral vascular disease. J Natl Med Assoc 1990; 82:173-177
- McDonagh EW, Rudolph CJ, Cheraskin E. The homeostatic effect of EDTA with multivitamin/trace mineral supplementation upon high density lipoproteins (HDL). J Osteo Phys Surg Calif 1982; 8:34
- 13. McDonagh EW, Rudolph CJ, Cheraskin E. The influence of EDTA salts plus multivitamin/mineral therapy upon total serum cholesterol/high density lipoprotein cholesterol. Med Hypoth 1982; 9:643–646
- McDonagh EW, Rudolph CJ, Cheraskin E. The effect of ethylenediaminetetraacetic acid (EDTA) upon blood cholesterol in a private practice environment. J Int Acad Prev Med 1982; 7:5-12
- McDonagh EW, Rudolph CJ, Cheraskin E. The glycohemoglobin (HbAlc) distribution in EDTA-eligible patients. J Orthomol Psychiat 1984; 12(1):1–3
- McDonagh EW, Rudolph CJ, Wussow DG. The effect of intravenous disodium ethylenediaminetetraacetic acid (EDTA) upon bone density levels. J Adv Med 1988; 1:79–85
- McDonagh EW, Rudolph CJ, Barber RK. Effect of EDTA chelation and supportive multivitamin/mineral supplementation on chronic lung disorders: a study of FVC and FEV1. J Adv Med 1989; 2:553-561
- Rudolph CJ, McDonagh EW, Barber RK. An observation of the effect of EDTA chelation and supportive multivita-

- min/trace mineral supplementation on blood platelet volume: a brief communication. J Adv Med 1990; 3(3):179–184
- Van der Schaar DC (ed) Some in vivo effects of EDTA on platelets, presented at the annual meeting of the American Academy of Medical Preventics. Washington, DC: 1985.
- McDonagh EW, Rudolph CJ, Cheraskin E. The effect of intravenous ethylenediaminetetraacetic acid (EDTA) plus supportive multivitamin/trace mineral supplementation upon fasting serum calcium. Med Hypoth 1983; 11:431–458
- 21. Olszewer E, Carter JP. EDTA chelation therapy in chronic degenerative disease. Med Hypoth 1988; 27:41–49
- 22. Cheraskin E, Wussow D, McDonagh E, Rudolph C. Effect of EDTA chelation and supportive vitamin/trace mineral supplementation with and without physical activity on heart rate. J Int Acad Prev Med 1984; 8(6):5-9
- McDonagh EW, Rudolph CJ, Cheraskin E, Wussow DG.
 The effect of EDTA and supportive multivitamin/trace mineral supplementation with and without physical activity upon systolic blood pressure. J Orthomol Psychiat 1984; 13:1-9
- Kaman RL, Rudolph CJ, McDonagh EW, Walker FM. Effect of EDTA chelation Therapy on aortic calcium in rabbits on atherogenic diets: quantitative and histochemical studies. J Adv Med 1990; 3(1):13-21
- Graboys et al: Results of a 2nd opinion trial among patients recommended for coronary angiography. JAMA 1992; 268(18):2537–40

Visual Field Evidence of Macular Degeneration Reversal Using a Combination of EDTA Chelation and Multiple Vitamin and Trace Mineral Therapy

C.J. Rudolph, DO, PhD, FACAM, R.T. Samuels, OD, and E.W. McDonagh, DO, ACGP, FACAM

ABSTRACT: A 59-year-old white, partially disabled female patient presented with a history of visual field defects and a diagnosis of Map-Dot-Fingerprint dystrophy. Examination further revealed retinal changes associated with moderate macular degeneration. The patient was treated with a combination of intravenous chelation therapy, using a series of 3 gram infusions of ethylene diamine tetracetic acid (EDTA) and multivitamins and trace minerals. After treatment, the patient's visual field defect improved. Her vision was restored to 20/25 in the right eye and 20/20 in the left eye and the quality of her central macular vision was greatly enhanced as measured by autoperimetry. The patient remains symptom free with restored vision one year later.

Introduction

This continues a series of papers analyzing the effects of intravenous ethylene diamine tetracetic acid (EDTA). In previous papers the main focus has been on the improvement in circulation in the carotid arte-

Dr. Rudolph is a partner and co-owner of McDonagh Medical Center, 2800-A Kendalwood Parkway, Kansas City, MO, where Dr. McDonagh is co-owner and founder. Dr. Samuels is a Doctor of Optometry in private practice at 2700 Kendalwood Parkway, Kansas City, MO.

Address correspondence to Dr. Rudolph, 2800-A Kendalwood Parkway, Kansas City, MO 64119.

204

ries (1-3), the lower extremities (4) and in functional cardiac improvement as measured through sub-maximal treadmill testing (5,6) and coronary angiography (7). In treating the patients that were subjected to these studies, it was noted that those with vascular disease and concomitant visual field deficits had significant subjective visual improvement, especially those with macular degeneration.

Map-Dot-Fingerprint dystrophy is also known as epithelial basement membrane dystrophy or Cogan's dystrophy. This entity is probably a dominantly inherited trait which involves the corneal epithelium and its basement membrane. It often causes recurrent corneal erosions and intraepithelial microcysts with sub-epithelial ridges. With a biomicroscope, these defects resemble a map, or fingerprint, thus the name which relates to its physical appearance (8,9).

Senile macular degeneration (SMD), now called age-related macular degeneration (AMD) was first described as a visual disease by Haab in 1885 (10). Although there is some disagreement as to what constitutes AMD, certain common phenomena seem to be present in the majority of patients who suffer from this visual impairment. The Framingham eye study (11) illustrated that it is a condition related to age. The prevalence rate is: 1.6% for those in the age group of 52 to 64 years; 11.0% for those in the age group of 65-74 years; and 27.0% in those patients 75 years of age and older. Since the elderly are the fastest growing segment of our society, health care professionals will be confronted with more and more of these patients. At present, it is the leading cause of severe visual loss for people over 65 years old. It is conventionally considered to be irreversible since no treatment is known.

Other major causes of visual loss in patients over 65 years, namely cataract, diabetic retinopathy, and glaucoma, all have some form of treatment which has at least some success in sparing visual acuity.

To our knowledge, this represents the first study showing objective data demonstrating the improvement in macular degeneration and is the focus of this article.

Case Report

A 59-year-old white female patient presented to our clinic with a history of Map-Dot dystrophy. She had previously been diagnosed with this condition by her ophthalmologist'in 1992 and was told that there was no effective therapy for this disorder. When first seen at this clinic in July of 1993, she was

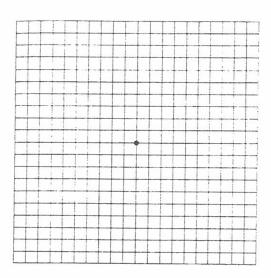
subjected to a series of diagnostic tests on her vascular system. In addition, she was referred to Dr. Robert T. Samuel for consultation and autoperimetry studies.

The Dicon TKS 4000 Auto Perimeter (Visamed) was utilized for perimetry studies performed initially on July 23, 1993 with a follow-up on November 5, 1993. After the initial examination, the patient received a course of 30, 3 gram infusions of intravenous EDTA, together with multivitamins and trace mineral supplementation according to the protocol of the American Academy for Advancement in Medicine (12).

BERNELL M-R-M GRID TEST RECORDING CHART

(Maculopathy-Retinopathy-Metamorphopsia)

USE FOR
M-R-M Grid Test Book, 553M Slide And Amsler Grid Test



Date:	Doctor's Name:
Reproduct	ion of Amsler and M-R-M Grid Book Test No. 1

1985 Bernell Corp., South Bend, IN

FIGURE 1

Form No. BC / F 553M

Amsler grid used as a quick screen for macular degenerative disorders.

She received no concomitant therapy other than that provided by this institution, and excellent results were obtained. Currently her visual problems are better. She has noted improvement in her visual acuity, peripheral vision and central macular vision. Studies of her macula were obtained both before and after our treatment with direct ophthalmoscopy and auto perimetry (Dicon TKS 4000). Amsler grids (Figure 1) presented to the patient before and after treatment indicated a reduction in visual disorder after treatment. An Amsler grid is a photograph of a narrow lined grid at which the patient stares. If there are macular disorders present, the patient will see wavy lines, whereas if there is no macular disease, the lines will appear straight and perpendicular to each other.

Results

Figures 2a and 2b represent the details of the pre- and post-treatment retinal studies done on this patient. The test performed on July 23, 1993 was a full threshold 80/30 and is represented as Figure 2a. The test performed on November 5, 1993 was the superthreshold 120/60 and is depicted in Figure 2b.

The peaks and depressions in Figures 2a and 2b illustrate the visual sensitivity to light stimuli. The exact center on these visual fields indicates the fovea centralis. This portion of the figure should exhibit the highest peak illustrating the area of vision with the greatest sensitivity to light. This is cone vision. As the topography rises and falls farther to the periphery from the center peak, these figures indicate greater or lesser sensitivity to light. The deep depression lateral to each central peak coincides with the optic nerve or physiological blind spot.

These two separate tests are not exactly the same because the instrument was upgraded by the Visamed company in the interim between testing. Even though not identical, they point to a significant improvement in the visual fields. This patient represents the first in a series of patients tested for improvement in visual fields before and after chelation therapy. Refinements have been made in subsequent studies which will undoubtedly produce more uniform findings. The patient was subjectively elated by her newfound vision.

Table 1 depicts the patient's visual acuity before and after therapy.

TABLE 1
Visual Acuity Studies

Eye	Pre-Therapy Visual Acuity	Post Therapy Visual Acuity
Right Left	20/60 20/30	20/25 20/20

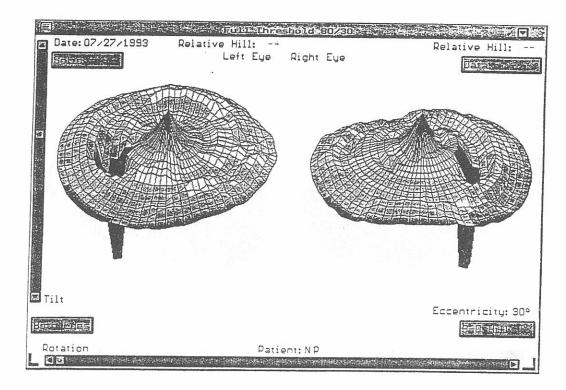


FIGURE 2a

Pre-treatment auto-perimetry studies on patient, NP.

Peripheral vascular studies were negative. The carotid arteries showed mild plaque development at the bifurcation bilaterally.

Discussion

These data indicate that chelation, in combination with multivitamin and trace mineral therapy, had a dramatic effect upon the patient's visual acuity. From past clinical experience with nutritional supplementation in the treatment of macular degeneration, this result was much faster and there was a more significant improvement in the patient's visual acuity than with nutritional therapy alone.

Although it is not possible in this one case report to separate the effectiveness of nutritional therapy from that of chelation, the fact remains that highly significant clinical improvement occurred in a condition which is traditionally considered to be intractable. Our experience (2,3) is that chelation has a marked beneficial effect on small

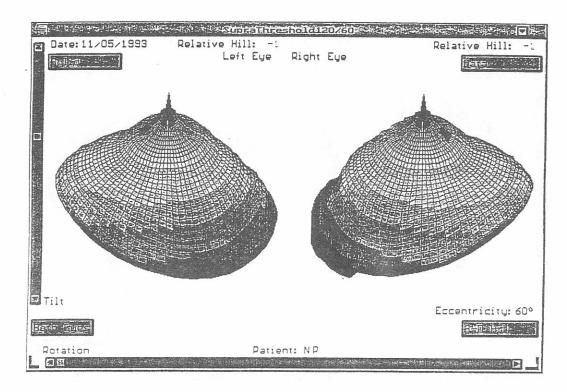


FIGURE 2b

Post-treatment auto-perimetry studies on patient, NP.

blood vessels in the carotid distribution. It is therefore postulated that it has a similar effect on the terminal arterioles in the retina, thus enhancing oxidative perfusion in the tissues. It is to be noted that the vascular studies performed on this patient did not show evidence of peripheral vascular disease. The carotid arteries showed some mild plaque development at the bifurcation bilaterally (15-30% occlusion) but obviously the study did not visualize the arterioles in the retina. We are left with some degree of speculation which can only be confirmed by further study of the effects of chelation therapy in the disease.

To understand AMD, one must first consider the physiology of the retina, especially the macula (13), the central area of the retina inside the vascular arcade which has at least two areas of nuclei in the ganglion cell layer. Inside the macula is the fovea, a zone of slightly greater pigmentation defined by the rim of the concave slope and approximately 1.5 mm in size. Under this area is the thicker choroid or vascular layer, and at its center is the foveola (350 um), which is free

of cells except for the outer segments, containing red and green cones. The fovea contains the highest concentration of "macular yellow." The foveal light reflex (UMBO) is produced by a small depression in the center of the foveola. The macula is closely associated with the retinal pigment epithelium, Bruch's membrane and the underlying choriocapillaries from which the macula receives its nutrition (14,15).

The normal aging process begins to produce changes in the layers beneath the macula that results in AMD. It is usually bilateral in nature and makes its first appearance as sharply defined drusens which are thickened patches of hyalinization from Bruch's membrane. As these drusens spread, fine granular deposits form, thus causing the clinically visible appearance of disturbances of the pigment and increased visibility of the choroidal vessels.

Bruch's membrane continues to thicken and impinges more on the choriocapillaries. At this point, the drusens have less distinct margins and seem to grow together. Pigment clumping becomes more evident, causing greater distortion and atrophy of the photoreceptors. The blood vessels in the choroid are narrowed, thus reducing the supply of both oxygen and nutrients to the macula. Neovascularization follows, forming a fragile vascular network under the macula. These changes lead to very distinct areas of depigmentation referred to as geographic atrophy. Vision in this type of AMD is called "dry" or atrophic AMD and may survive in this state for a long period of time (16).

The second type of AMD is called "wet" or exudative AMD. This type causes detachment of the retinal pigment epithelium and hemorrhages from the fragile sub-macular neovascular net. This causes rapid, severe visual loss. According to the Framingham eye study, 80% are dry or atrophic AMD, 8% wet or exudative AMD and 12% are questionable (11). Certainly any treatment that provides improved circulation to the choroid has a hope of delaying the onset of AMD.

Macular degeneration spares no race. Some studies have indicated that it is more prevalent in whites than blacks (17), although other studies have pointed to an equal distribution between races (18). Orthodox medicine has very little to offer. The nutritional approach has, in recent years, been considered a viable alternative as the cones of the eye contain disproportionately high levels of zinc when compared to the rest of the body. It has been demonstrated that zinc therapy, using doses of ionic zinc in the range of 50-100 mg per day has a beneficial effect on the disease (19). Although zinc supplementation was used in this patient, we do not believe that the effects on the

macula could be attributed wholly to zinc supplementation. As a matter of fact, it has been our experience and that of others (20,21) that zinc excretion increases 6,170% after EDTA chelation therapy, thus creating a transient zinc deficiency which must be replaced by supplementation. We believe that the clinical improvement in this patient was directly related to chelation therapy since zinc supplementation is unlikely to create a positive balance with the degree of loss due to EDTA.

It has been reported that dietary supplementation of anti-oxidants, including vitamin A, beta-carotene, vitamin E and vitamin C as well as zinc, copper, selenium, manganese and riboflavin may help AMD (22). It is a known fact that the elderly are often deficient in these substances. The therapy is tolerated well and has low risk.

Since the initiating factor is not well defined, it is postulated that if the vascular integrity of the choriocapillaries was maintained, then this age-related sequence of events that leads to macular degeneration might be prevented. Any treatment that would restore the circulation in the very distal branches of the macula, thus allowing more nutrient delivery to it, would be logical. The evidence is that chelation has this effect.

It has been previously established that infusions of EDTA combined with a multi- and trace vitamin mineral therapy could reverse atherosclerotic occlusive disorders in the larger, more proximal trunk branches of the ophthalmic artery, namely the common and internal carotid arteries (2), but its effect on the terminal branches of these arteries has never previously been demonstrated.

To our knowledge, this is the first article in which autoperimetry demonstrated improvement in AMD after the combination of EDTA chelation and multivitamin, trace mineral therapy. The patient was relatively free of visual problems at its conclusion. She has been followed for 1 year and has suffered no relapse in her visual acuity at the time of this writing.

Conclusion

Macular degeneration is one of the leading causes of visual loss in the elderly in this country. Annually, it is estimated to cause serious visual loss in approximately 30% of people over the age of 75. While not associated with a high mortality, it can cause great disability and morbidity by robbing the elderly of their vision when it is so impor-

tant to the quality of life in retirement. With today's escalating, if not astronomical, health costs, it is reassuring that there exists a therapy that has a multi-fold potential. This technique is a relatively low-cost therapy for this and other degenerative visual disorders. It may offer a solution to the chronic problem of visual loss in the elderly.

Renal Artery Stenosis Reversal in a Hypertensive Individual, Using a Combination of EDTA Chelation and Multiple Vitamin and Trace Mineral Therapy

C.J. Rudolph, DO, PhD, FACAM, and E.W. McDonagh, DO, ACGP, FACAM

ABSTRACT: A 70-year-old white male patient presented with a history of severe hypertension and left renal artery stenosis. Doppler duplex scanning revealed a peak systolic velocity blood flow in excess of 200 cm/sec in the renal artery. The renal artery to aorta flow velocity ratio was greater than 3.5. The patient was treated with a combination of intravenous chelation therapy using a series of 3 gram infusions of ethylene diamine tetracetic acid (EDTA), together with orally administered multivitamin and trace minerals. After treatment, the patient's blood pressure was reduced, requiring less medication. Blood flow in the left renal artery and the renal artery/aorta flow ratio were within normal limits.

Introduction

This case report continues a series of papers analyzing effects of intravenous EDTA infusions. We have previously published data on the improvement in circulation in the carotid arteries (1–3), the lower extremities (4) and in functional cardiac improvement as measured through sub-maximal treadmill testing (5) and coronary angiography (6). We have also reported an individual with macular degeneration whose retinal artery perfusion was improved as measured by retinal autoperimetry studies (7).

Address correspondence to C.J. Rudolph, DO, PhD, FACAM, 2800-A Kendalwood Parkway, Kansas City, MO 64119.

In treating the patients that were subjected to these various studies, it was noted that if hypertension were present with vascular disease, there was improvement in blood pressure after EDTA chelation. There was improvement in systolic blood pressure in all stages of submaximal treadmill testing in patients undergoing the treatment (8).

Case Report

C.M., a 70-year-old white male patient presented to our clinic with a long-standing history of hypertension due to left renal artery stenosis. He had previously been diagnosed with this condition by a vascular surgeon in 1997, using real-time ultrasound, color, duplex doppler. He had been told that the only effective therapy was surgical, either angioplasty or bypass. When first seen at this clinic in June of 1997 he had been subjected to a series of diagnostic tests on his vascular system that showed moderate aorto-iliac occlusive disease. A stress electrocardiogram was performed but, while negative for ischemia, was limited in value due to the development of claudication.

The patient then received a course of 70 intravenous infusions, each containing 3 grams of EDTA. He also received supplements of multivitamins and trace minerals according to the protocol of the American College for Advancement in Medicine (ACAM) (9). Blood pressure medication included verapamil extended release 240 mg. in the morning and doxazosin mesylate 2 mg. at bedtime. They were continued throughout the study at the same dose, but no additional treatment was given other than the chelation and nutritional supplements. The patient was re-tested with a second renal ustrasound study after receiving 70 EDTA chelation treatments.

Results

Table 1 shows the flow velocities in the renal arteries as determined by diagnostic ultrasound performed in the radiology department in Heartland East Hospital, St Joseph Mo. The equipment used for the duplex scan was an Acuson 128XP diagnostic ultrasound. The velocity of flow in a healthy renal artery is in the range of 100 cm/second (10) and increases proportionately with the degree of stenotic narrowing of the lumen. This flow velocity is also dependent upon concomitant flow in the aorta as it varies. It is therefore helpful to express the flow of blood in the renal artery as a ratio to that in the aorta. Thus, peak flow in the renal artery is reported together with the renal artery/aorta flow ratio. This allows the flow measurement to be standardized.

When this patient was first examined, the peak velocity in his left

TABLE 1

	Flow Velocity	Renal Aortic Ratio
Pre-Treatment	206 cm/sec	3.75
Post-Treatment	113 cm/sec	1.69

Table 1 shows the flow velocity in the renal artery before and after patient was treated with 70 infusions of EDTA chelation. The renal aortic ratio is a measure of the flow velocity in the renal artery divided by the blood flow in the aorta and indicates stenosis if it is > 3.50.

renal artery was 206 cm/sec. After the chelation treatment, the test was repeated and the peak flow was recorded as 113 cm/sec. The renal artery/aorta blood flow ratio initially in this patient was 3.75 and, when the test was repeated, it was recorded as 1.69. The normal for this ratio, as published by Kohler et al. (11) is less than 3.5. In this publication, a table illustrates the distribution of non-stenotic and stenotic patients with reference to the renal artery/aorta ratio.

By using angiography and comparing it with doppler readings, 60% occlusion of the renal artery, considered to be the threshold where a lesion becomes hemodynamically significant, is equivalent to a renal artery/aorta ratio of 3.5 (11). Using the table published by Kohler et al. it is possible to conclude that our patient had an occlusion in the left renal artery in the 60–70% range. By the same token, it is possible to conclude that the occlusion was reduced to 20% or less after chelation and nutritional supplementation. This obviously represents a significant hemodynamic improvement in renal artery blood flow.

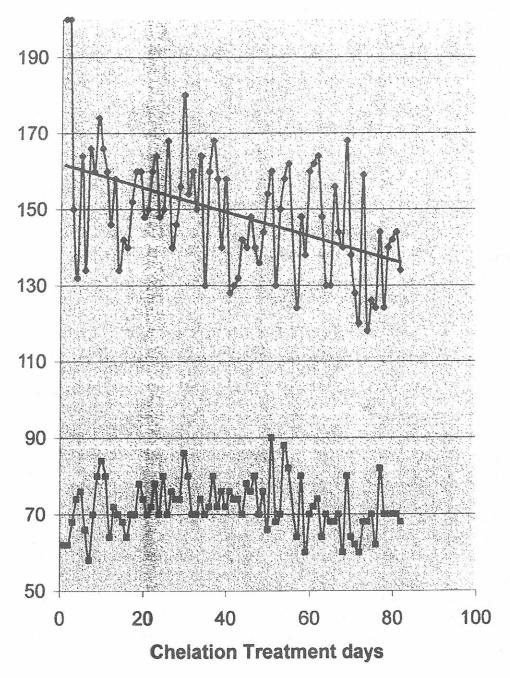
Since the presenting symptom of this patient was hypertension, the improvement in renal artery stenosis should hopefully lead to a clinical change in blood pressure. The patient's blood pressure was measured each day prior to his intravenous infusion of EDTA. Figure 1 shows a plot of these measurements. The upper plot represents the systolic and the lower one shows the diastolic pressures respectively. The trend line clearly shows a decrease in the systolic pressures, whereas the diastolic readings remained relatively constant in the range below 90 mm Hg.

The true time interval between infusions varied, but on the average the patient received infusions three times weekly for the first 30, twice weekly for the next 10 infusions, weekly for the subsequent 10 and every other week for the remainder.

196

FIGURE 1

A plot of the patient's systolic (upper graph) and diastolic (lower graph) blood pressure versus the number of chelation therapy infusions. The line through the systolic measurements is a trend line showing the general direction the blood pressure is heading.



Discussion

These data indicate that EDTA chelation, in combination with nutrient supplements, had a dramatic effect upon the renal artery stenosis and the hypertension that was secondary to it. From past clinical experience, the use of low fat and low sodium diet and nutritional supplementation may improve hypertension, but they have never been demonstrated to have a therapeutic effect on renal artery stenosis. Angioplasty and renovascular surgical treatment are the current method of choice, but risks include embolization, restenosis, anesthetic reactions and even death.

Renal artery stenosis is a definable cause of hypertension. A fixed obstruction in a renal artery causes an elevation of serum renin, angiotensin and, ultimately aldosterone. Intrinsic lesions can be either atherosclerotic (67%) or fibromuscular (33%) in origin. Patients with atherosclerotic lesions tend to be older, have higher systolic blood pressures, and are more likely to develop end-organ damage than those with the fibromuscular type of lesions that are found usually in younger people, more commonly females.

Hypertension from this cause is generally only amenable to surgical intervention. Angioplasty in the atherosclerotic type has a success rate in the 60–70% range and is invasive. If renovascular surgery is required, the cure rate is in the 40–50% range. If a patient has had hypertension for more than 5 years, only 25% have a favorable response to surgery (12). Although only a single case, the response from EDTA chelation in this patient strongly suggests that it should be first choice before risking surgical intervention, particularly with the indifferent results. Neither is it possible to separate the effectiveness of nutritional therapy from that of EDTA chelation. The fact remains, however, that highly significant clinical improvement occurred in a condition that is traditionally considered to be only amenable to surgical intervention. It is also highly unlikely that nutritional supplementation would do this on its own.

Our experience (2-6) is that EDTA chelation has a marked beneficial effect in all areas of the circulatory system and that the best approach to a systemic problem is a systemic solution. Even if the renal artery stenosis is only the "tip of the iceberg," there may be a significant presence of other vascular lesions that have not yet resulted in expression.

There are numerous causes of hypertension besides that related to kidney dysfunction (13). Heavy metals are also known to be powerful pressor substances. Patients with both hypertension and reduced renal function (serum creatinine > 1.5 mg/dl) had significantly larger amounts of mobilizable lead than did patients without renal impairment, suggesting that lead may play a role in essential hypertension (14).

As EDTA chelation is the treatment of choice in lead poisoning, it may be the treatment of choice if lead can be shown to be an etiologic factor (14). Cadmium, another toxic pressor, is thought to displace zinc in tissues, especially the kidney. This is most likely to be responsible for the effect of cadmium in blood pressure (15). Since cadmium has been used in tobacco products, it may be the mechanism that affects human health and cardiovascular status (16). Mercury binds strongly to sulfhydryl groups in the renal parenchyma and can cause toxicity and result in hypertension (17). A screening test by hair analysis in this patient did not reveal any increase in these heavy metals and there was no indication to pursue this further as a possible etiol-

The improvement seen in renal artery flow in this patient might be confusing to some since the earlier studies suggested that EDTA was nephrotoxic. Cranton (18) reported on this subject in a comprehensive review. He noted that the initial studies used much higher doses (9 grams versus 3 grams) and much higher infusion rates (1/2 hr. versus 3 hrs). This resulted in an assault on the renal tubular system that was, on occasion, irreversible. McDonagh and associates (19) followed that up with a 383 patient study using current protocol. They found that this treatment did not result in renal damage and kidney function actually improved about 15% as measured by pre- and posttreatment serum creatinine. These effects were later confirmed by a similar study showing a reduction in blood urea nitrogen in patients treated with EDTA chelation therapy (20). As the kidney is perhaps the most important tissue for regulation blood pressure, we originally assumed that the beneficial effects in hypertensive patients were related to removal of heavy metals. The fact that there was actual improvement in the renal function was, however, suggestive that the mechanism of blood pressure reduction might actually be in improving renal circulation.

Hypertension, high cholesterol, diabetes mellitus and obesity are significant risk factors for vascular occlusive disease. Untreated hypertension may result in thrombotic or hemorrhagic cerebrovascular accidents, renal insufficiency, congestive heart failure and coronary heart disease with subsequent myocardial infarction (21,22).

To our knowledge, this is the first article in which hemodynamically demonstrable improvement in a renal artery lesion has been shown after treating a patient with the combination of EDTA chelation and multivitamin, trace mineral therapy. The patient remains well and his blood pressure is well maintained with reduction in the dose of his medication.

Conclusion

Hypertension is one of the leading causes of vascular disease in the elderly in this country. While not associated directly with a high mortality, it is a direct precursor to the first (heart disease) and third (stroke) ranked causes of death in the United States. With the escalating, if not astronomical, health care costs of today, it is reassuring that there exists a therapy that has a multi-fold potential. This technique is a relatively low-cost therapy for this and other degenerative disorders. It may offer a solution not only to the chronic problem of hypertension, but also to the feared lethal conditions to which it can lead.

References

- 1. McDonagh E W, Rudolph C J, Cheraskin E. An oculocerebrovasculometric analysis of the effect of EDTA chelation upon vascular stenosis. J Holist Med 1982;4:21–23.
- 2. Rudolph C J, McDonagh E W. Effect of EDTA chelation and supportive multivitamin trace mineral supplementation on carotid circulation: case report. J Adv Med 1990;3:5-11.
- 3. Rudolph C J, Mc Donagh E W, Barber R K. A nonsurgical approach to carotid stenosis using EDTA chelation. J Adv Med 1991;4:157-166.
- 4. McDonagh E W, Rudolph C J, Cheraskin E. The effect of EDTA chelation therapy plus multivitamin trace mineral supplementation upon vascular dynamics (ankle/brachial systolic blood pressure). J Holist Med 1985;7:16-22.
- 5. Cheraskin E, Wussow D G, McDonagh E W, et al. Effect of EDTA chelation and supportive multivitamin/trace mineral supplementation with and without physical activity on heart rate. J Int Acad Prev Med 1984;8(6):5–9.
- 6. Mc Donagh E W, Rudolph C J. Non invasive treatment for sequella of failed coronary blood circulation, 100% occlusion of left anterior descending coronary artery and 30% stenosis of right coronary artery and left ventricular contractility deficit. J Neuro Orthoped Med Surg 1993;14:169–173.
- 7. Rudolph C J, McDonagh E W, Samuels R. Visual field evidence of macular degeneration reversal using EDTA chelation therapy and combined multivitamin trace mineral therapy. J Adv Med 1994;7:231-240.
- 8. McDonagh E W, Rudolph C J, Cheraskin E, et al. The effect of EDTA and supportive multivitamin/trace mineral supplementation with and without physical activity upon systolic blood pressure. J Orthomol Psych 1984;13:1–9.

- Cranton E M. Protocol of the American College for Advancement in Medicine for the safe and effective administration of EDTA chelation therapy. J Adv Med 1989; 2:269-306.
- 10. Avasti P S, Voyles W F, Green E R. Non-invasive diagnosis of renal artery stenosis by echo doppler velocities. Kidney Int 1984;25:824-829.
- 11. Kohler T R, Zierler R E, Martin R L, et al. Non-invasive diagnosis of renal artery stenosis by ultrasonic duplex scanning. J Vasc Surg 1986;4:450–456.
- 12. Wyngaarden J B, Smith L H. eds: Cecil's Textbook of Medicine, 18th edition, 1988:280-291.
- 13. Conn R B, ed: Current Diagnosis. Philadelphia, W B Saunders. 1991:409-411.
- 14. Batuman V, Landy E, Maesaka J K et al. Contribution of lead to hypertension with renal impairment. N Engl J Med 1983;390:17-21.
- 15. Perry H. Hypertension and the geochemical environment. Ann NY Acad Sci 1972; 199:202–206.
- 16. Friberg L, ed: Cadmium in the Environment. Cleveland, CRC Press. 1971.
- 17. Pier S M. The role of heavy metals in human health. Tex Rep Biol Med 1975;33:85-106.
- Cranton E M. Kidney effects of ethylene diamine tetracetic acid: a literature review. J Holist Med 1982;4:152-167.
- 19. McDonagh E W, Rudolph C J, Cheraskin E. The effect of EDTA chelation therapy plus supportive multivitamin-trace mineral supplementation upon renal function: a study in serum creatinine. J Holist Med 1982;4:146–151.
- 20. McDonagh E W, Rudolph C J, Cheraskin E. The effect of EDTA chelation therapy plus supportive multivitamin-trace mineral supplementation upon renal function: a study in blood urea nitrogen (BUN). J Holist Med 1983;5:163–171.
- 21. Kannel W B. Some lessons in cardiovascular epidemiology from Framingham. Am J Cardiol 1976;37:269-282.
- Kannel W B, Wolf P A, Verter J et al. Epidemiologic assessment of the role of blood pressure in stroke. The Framingham study. JAMA 1970;214:301-310.

References

- 1. McDonagh EW, Rudolph CJ, Cheraskin E: An oculocerebrovasculometric analysis of the effect of EDTA chelation upon vascular stenosis. J Holist Med 1982; 4:21-23.
- Rudolph CJ, McDonagh EW: Effect of EDTA chelation and supportive multivitamin trace mineral supplementation on carotid circulation: case report. J Adv Med 1990; 3:5-11.
- Rudolph CJ, McDonagh EW, Barber RK: A nonsurgical approach to carotid stenosis using EDTA chelation. J Adv Med 1991; 4:157-166.
- 4. McDonagh EW, Rudolph CJ, Cheraskin E: The effect of EDTA chelation therapy plus multivitamin/trace mineral supplementation upon vascular dynamics (ankle/brachial systolic blood pressure). J Holist Med 1985; 7:16-22.
- McDonagh EW, Rudolph CJ, Cheraskin E, Wussow DG: The effect of EDTA and supportive multivitamin/trace mineral supplementation with and without physical activity upon systolic blood pressure. J Orthomol Psych 1984; 13:1-9.
- Cheraskin E, Wussow DG, McDonagh EW, Rudolph CJ: Effect of EDTA chelation and supportive multivitamin/trace mineral supplementation with and without physical activity on heart rate. J Int Acad Prev Med 1984; 8:5-9.
- McDonagh EW, Rudolph CJ: Non-invasive treatment for sequella of failed coronary blood circulation, 100% occlusion of left anterior descending coronary artery and 30% stenosis of right coronary artery and left ventricular contractility deficit. J Neuro Orthoped Med Surg 1993; 14:169-173.
- 8. Starck T, Hersh PS, Kenyon K: Corneal dysgenesis dystrophies and degenerations. In: Albert DM, Jakobiec FA, eds. Principles and practice of ophthalmology, vol 1, Philadelphia, W B Saunders Company 1994; 26.
- Mandel E, Wagoner MD. Atlas of corneal disease. Philadelphia. WB Saunders Company 1989; 3.
- Duke-Elders, ed. System of Ophthalmology. London, Henry Kimpton 1966; 609-616.
- 11. Kahn HA, Leibowitz HM, Ganley JP, et al. The Framingham eye study, I,. Outline and major prevalence findings. Am J Epidem 1977; 106:17-32.
- Cranton EM: Protocol of the American College for Advancement in Medicine for the safe and effective administration of EDTA chelation therapy. J Adv Med 1989; 2:269-306.
- 13. Lovie-Kitchin J, Bowman KJ: Senile macular degeneration-management and rehabilitation. Stoneham, Mass. Butterworth Publishers 1985; 19-52.
- Bressler S, Bressler N, Gragoudas ES. Age related macular degeneration: drusen and geographic atrophy. In: Albert DM, Jakobiec FA, eds. Principles and practice of ophthalmology, vol 2, Philadelphia, W B Saunders 1994; 826-833.
- Sahel J, Brini A, Albert D. Pathology of the retina and vitreous. In: Albert DM, Jakobiec FA, eds. Principles and practice of ophthalmology, vol 4, Philadelphia W B Saunders 1994; 2251-2254.
- Green WR, Key SN. Senile macular degeneration. a histopathological study. Trans Am Opth Soc 1977; 75:180-250.
- Ferris FL. Senile macular degeneration: review of epidemiological features. Am J Epidemiol 1983; 118:132-151.
- Gregor Z, Joffe L. Senile macular changes in the black African. Br J Opth 1978; 62:547-550.
- van der Schaft TL, de Bruijin WC, Mooy CM, et al. Element analysis of the early stages of age related macular degeneration. Arch Opth 1992; 110: 389-394.
- Cranton EM, Liu ZX, Smith IM. Urinary trace and toxic elements and minerals in untimed urine specimens relative to urinary creatinine. part I: concentrations of elements in fasting urine. J Adv Med 1989; 2:331-349.
- Cranton EM, Liu ZX, Smith IM: Urinary trace and toxic elements and minerals in untimed urine specimens relative to urinary creatinine. part II: provoked increase in excretion following intravenous EDTA. J Adv Med 1989; 2:351-397.
- 22. Alexander LJ: Ocular vitamin therapy—a review and assessment. Optometry clinics, vol 2, 1992; 4:1-34.