

Effect of EDTA Chelation and Supportive Multivitamin/Trace Mineral Supplementation on Chronic Lung Disorders: A Study of FVC and FEV₁

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ABSTRACT: Thirty-eight subjects with chronic degenerative disorders were treated with intravenous infusions of disodium ethylene diamine tetraacetic acid (EDTA). These patients were evaluated objectively for maximum expiration lung volumes before and after EDTA chelation. Each subject had 30 treatments over a period of approximately 9 months. Forced Vital Capacity (FVC) increased an average of 12.1% ($p < 0.001$) and Forced Expiration Volume in one second (FEV₁) increased an average of 12.8% ($p < 0.010$). Patients with lung disorders increased 18.9% ($p < 0.001$) in FVC and 20.8% ($p < 0.05$) in FEV₁. Overall, 34 of the 38 subjects (90.5%) improved in pulmonary function after EDTA infusions.

Introduction

Chelating agents, particularly EDTA, have been available for years. This study continues a series of papers analyzing the effects of intravenous EDTA therapy. Past publications include research in renal function^{1,2}, vascular occlusive disease³, osteoporosis⁴, various analyses of biochemical blood serum levels⁵⁻⁹—atherosclerosis and related disorders^{10,11} to mention only a few. As far as can be determined, this is the first attempt to examine the effect of EDTA therapy plus multi-

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vitamin-trace mineral supplementation upon pulmonary expiration volumes. Specifically this report was designed to answer four questions.

- 1). What are the post therapy effects of EDTA chelation upon Forced Vital Capacity lung volume (FVC)?
- 2). What are the post therapy effects of EDTA chelation upon Forced Expiration Volume in one second (FEV1)?
- 3). How do the above results compare to predicted normal lung volumes adjusted for height, sex, age, and race?
- 4). What are the post therapy effects of EDTA chelation on subjects with abnormally reduced lung expiration volumes?

Materials and Methods

Thirty eight patients suffering with chronic degenerative disorders participated in this experiment in a private practice environment. Included were 24 males ranging in age from 32 to 84 years old (with a mean and standard deviation of 66.0 ± 10.5) and 14 females ranging in age from 49 to 83 years old (with a mean and standard deviation of 65.0 ± 10.2). At the initial examination, each patient underwent a detailed history, physical examination, and a comprehensive battery of biochemical tests. Pulmonary expiration lung volumes were measured on a Tiffenair Computerized Spirometer. Volumes analyzed included Forced Vital Capacity (FVC) and Forced Expiration Volume in one second (FEV1). These results were then compared to predicted normals according to subjects' age, race, height, and sex¹². Following the initial studies, each subject received 30 intravenous EDTA infusions over a period of approximately 9 months. EDTA is administered intravenously because the human digestive system does not readily accept the synthetic amino acid. Due to the leaching effect of chelation therapy on the essential trace metals, a multivitamin trace mineral supplement accompanied the infusions. The dosage was 3 grams of EDTA per treatment. After the 30 treatments, each patient again underwent a physical examination, a comprehensive battery of biochemical tests, including another measurement of pulmonary expiration.

Results

Question 1: The post therapy effects of EDTA chelation upon Forced Vital Capacity lung volume (FVC). After EDTA intravenous infusions the improvement in Forced Vital Capacity was statistically significant in both male and female subsets. Table 1 demonstrates that the FVC averaged 12.1% higher for all ($t = 7.081$, $p < 0.001^*$), 10.5% higher for men ($t = 5.889$ $p < 0.001^*$), and 15.7% higher for women ($t = 3.067$ $p < 0.01^*$).

Question 2: The post therapy effects of EDTA chelation upon Forced Expiration Volume in one second (FEV1). As shown in Table 2 the 12.8% improvement in Forced Expiration Volume in one second was statistically significant overall ($t = 3.333$ $p < 0.01^*$).

Question 3: How do the above results compare with predicted normal lung volumes adjusted for height, age, sex, and race¹²? After EDTA infusions plus multivitamin/trace mineral supplementation both FVC and FEV1 increased significantly* in percentage of expected volumes (Tables 3 & 4). In the initial examination the mean FVC was 80.0% of the predicted expiration with a range of 49.0–110.0%. After EDTA chelation the average FVC was 92.5% of prediction with a range of 61.0–169.0% ($t = 4.872$ $p < 0.001^*$). Before treatment the average FEV1 measured 88% of predicted with a range of 45.0–127.0%. After 30 treatments the mean FEV1 was 6.7% higher at 96.3% with a range of 66.0–133.0% ($t = 2.896$, $p < 0.01^*$)

TABLE 1

Influence of Intravenous EDTA plus Multivitamin/Trace Mineral Supplementation upon Forced Vital Capacity (FVC)

	Sample size	Initial FVC mean	Final FVC mean	Change (L)	Change (%)	Statistical evaluation
Entire group	38	3.12L	3.55L	0.43L	+ 12.1%	$t = 7.081$ $p < 0.001^*$
Men	24	3.50L	3.91L	0.41L	+ 10.5%	$t = 5.889$ $p < 0.001^*$
Women	14	2.47L	2.93L	0.46L	+ 15.7%	$t = 3.067$ $p < 0.01^*$

*statistically significant difference between the means

TABLE 2

Influence of Intravenous EDTA plus Multivitamin/Trace Mineral Supplementation upon Forced Expiration Volume in One Second (FEV1)

	Sample size	Initial FEV1 mean	Final FEV1 mean	Change (L)	Change (%)	Statistical evaluation
Entire group	38	2.24L	2.57L	0.33L	+ 12.8%	t = 3.333 p < 0.01*
Men	24	2.27L	2.96L	0.69L	+ 23.3%	t = 3.249 p < 0.01*
Women	14	1.81L	1.89L	0.08L	+ 4.2%	t = 1.667 p < 0.05

*statistically significant difference between the means

Question 4: What are the post therapy effects of EDTA chelation on subjects with abnormally reduced lung expiration volumes? For this report abnormal lung expirations included patients with no more than 70.0% of predicted lung expirations adjusted for age, height, sex, and race¹². Table 5 demonstrates again statistically significant results in both FVC and FEV1. In the initial study 26.0% of the patients demonstrated abnormal FVC's. After treatment only 2 subjects (5.0%) remained abnormal and those two subjects still increased an average of 16.5%. Before treatment there were 5 patients with abnormal

TABLE 3

Distribution of Change in Percentage of Predicted Lung Volumes Adjusted for Age, Height, Sex, and Race after EDTA plus Multivitamin/Trace Mineral Supplementation on Forced Vital Capacity (FVC)

	Sample size	Initial FVC mean %	Final FVC mean %	Change	Statistical evaluation
Entire group	38	80.0%	92.5%	+ 12.5%	t = 4.872 p < 0.001*
Men	24	82.9%	93.4%	+ 10.5%	t = 3.903 p < 0.001*
Women	14	77.2%	92.0%	+ 14.0%	t = 2.746 p < 0.01*

*statistically significant difference between the means

TABLE 4

Distribution of Changes in Percentage of Predicted Lung Volumes Adjusted for Age, Height, Sex, and Race after EDTA plus Multivitamin/Trace Mineral Supplementation on Force Expiration Volume in One Second (FEV₁)

	Sample size	Initial FEV1 mean %	Final FEV1 mean %	Change	Statistical evaluation
Entire group	38	89.6%	96.3%	+ 6.7%	t = 2.896 p < 0.01*
Men	24	92.0%	101.0%	+ 9.0%	t = 2.308 p < 0.05*
Women	14	84.5%	88.0%	+ 3.5%	t = 1.292 p < 0.05

*statistically significant difference between the means

FEV₁'s. The initial FEV₁ mean was 58.0% of predictions with a range of 45.0–70.0%. After EDTA therapy the mean was 26.4% higher at 78.8% of predicted with range of 63.0–107.0% (t = 2.66 p < 0.05*).

Discussion

Forced Vital Capacity (FVC) and Forced Expiration Volume in one second (FEV₁) are common procedures used to aid the diagnosis of lung disorders. FVC is defined as the maximum volume of air a sub-

TABLE 5

Influence of EDTA plus Multivitamin/Trace Mineral Supplementation upon Abnormal Lung Expiration Volumes

	Sample size	Initial mean % of predictions	Final mean % of predictions	Increase	Statistical evaluation
FVC	10	60.5%	80.4%	+ 18.9%	t = 5.923 p < 0.001*
FEV ₁	5	58.0%	78.8%	+ 20.8%	t = 2.66 p < 0.05*

*statistically significant difference between the means

ject is able to expire after a maximum inspiration of the total lung capacity^{13,14}. Reduced FVC can be associated with a restriction in the lung's ability to expand either due to changes in the pulmonary parenchyma or because of diseases of the pleura, chest wall, or the neuromuscular apparatus regulating respiration¹⁵. Pneumonia, pulmonary fibrosis, and pleurisy are examples of restrictive disorders¹⁶. FEV1 is the amount of air expired in the first second of FVC^{13,14}. Thus, the FEV1 measures the speed of the airflow. A dramatically decreased FEV1 demonstrates upper airway resistance or obstructive problems in the lungs. Examples of obstructive diseases are emphysema, chronic bronchitis, and asthma¹⁶. In the normal lung the FEV1 is at least 80.0% of the FVC. A lower FEV1/FVC ratio suggests obstruction in expiratory airflow. However, if the lung is restricted (reduced FVC) and obstructed (reduced FEV1), the FEV1/FVC ratio might appear to be normal (80.0% or higher); in this case one would need to investigate further to examine lung function. For this reason, it is important to compare the volumes with predicted normals and examine the actual amount of gas expired.

Historically, EDTA has been used to treat lead poisoning, digitalis intoxication and certain collagen disorders¹⁷. Many physicians have successfully used EDTA chelation therapy to treat atherosclerosis and related disorders. Theoretically the chelate removes calcium deposits from the vessels, subsequently excreting bound calcium ions in the urine. As a result of this action the body's metabolic functions of calcium and other minerals are altered¹⁷. Specifically, a turnover of calcium may be produced through the stimulation of the parathyroid glands¹⁷⁻²⁰, which may therefore contribute to the breakdown of atherosclerotic lesions by the removal of metastatic calcium from the plaques^{17,21}.

From a simplistic viewpoint, "The concept of chelation involves the use of a family of chemicals that are able to grasp metals with a claw-like action. The metal then becomes, in turn, incorporated into a multimembered ring structure and in doing so loses its physiological and toxic properties. Thus, a metal or mineral such as calcium or lead, that comes in contact with a chelating agent is imprisoned in the chelating chemical and is excreted from the body in this bound and inert form¹⁷." The human body has many natural chelators that assist in vital functions. Some of these carriers are amino acids, globulins, polypeptides, and proteins; without them we would cease to exist. EDTA is a synthetic amino acid. It forms strong soluble complexes with cationic minerals such as calcium, zinc, magnesium, lead, and cadmium.^{17,22}

If EDTA is administered correctly, with appropriate dosage, rate, and concentration, it should produce no deleterious side effects. During the early stages of development, intravenous EDTA chelation was hindered by overdoses that resulted from not understanding the effective therapeutic range of this drug. The result of this incorrectly administered dosage was kidney, liver, and spleen damage, and even death^{17,19,23,24}. As Walker reports, "The nature of EDTA nephrotoxicity is not known, but there is a definite association of increased vacuolar changes in the tubular epithelium of the kidneys with a large dose of the chelate¹⁷."

However, various studies have found that safe levels of the chelate produced no renal damage¹⁷. In fact, McDonagh^{1,2} has even reported improved renal function with intravenous EDTA chelation. Foreman²⁵ concluded that a dosage of 50mg per kg per day would be safe for humans, and Meltzer¹⁹ reported that 3 grams of disodium edetate per dose to be without danger of nephrotoxicity. If we have improvement in the function of one tissue as a result of EDTA chelation then certainly there may be improvement in other tissues.

The air we breathe is poisoned with some 40 toxic metals²⁶. Tobacco smoke, dust, motor exhaust, and pollution drape our air with metals such as lead, cadmium, and nickel. Over time these small particles accumulate in the lungs. Cadmium has been linked with pulmonary emphysema and nickel with lung cancer²⁶. As this foreign matter collects, it damages the fragile alveoli, resulting in tissue calcification. It is suggested that the improved lung function seen in these patients was due in part to the removal of these accumulated materials.

Conclusion

The evidence presented in this report indicates that statistically significant improvements in abnormal FVC and FEV1 occur in patients receiving intravenous chelation therapy, together with multivitamin/trace mineral supplementation. It is suggested that the mechanism of action of chelation is removal of calcium and heavy metals precipitated in the pulmonary parenchyma.

References

1. McDonagh EW, Rudolph CJ, Cheraskin E: The effect of EDTA salts plus supportive multivitamin-trace mineral supplementation upon renal function: A study of

2. McDonagh EW, Rudolph CJ, 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 Holistic Med* 1983; 5(2):163-71.
3. McDonagh EW, Rudolph CJ, Cheraskin E: An oculocerebrovasculometric analysis of the effect of EDTA chelation upon vascular stenosis. *J Holistic Med* 1982; 4(1):21-3.
4. McDonagh EW, Rudolph CJ, Wussow DG: The effect of intravenous disodium ethylene diamine tetraacetic acid (EDTA) upon bone density levels. *J Advanc Med* 1988; 1(2):79-85.
5. McDonagh EW, Rudolph CJ, Cheraskin E: Serum cholesterol and the aging process. *Med Hypoth* 1981; 7:685-93.
6. McDonagh EW, Rudolph CJ, Cheraskin E: The effect of intravenous disodium ethylene diamine tetraacetic acid (EDTA) upon blood cholesterol in a private practice environment. *J Int Acad Prev Med* 1982; 7(1):5-12.
7. McDonagh EW, Rudolph CJ, Cheraskin E: The homeostatic effect of EDTA with supportive multivitamin-trace mineral supplementation upon high density lipoproteins (HDL). *J Osteopath Phys Surg Calif* 1982; 8(2):34.
8. McDonagh EW, Rudolph CJ, Cheraskin E: The influence of EDTA salts plus multivitamin-trace mineral therapy upon total serum cholesterol/high density lipoprotein cholesterol. *Med Hypoth* 1982; 9:643-6.
9. McDonagh EW, Rudolph CJ, Cheraskin E: The effect of intravenous disodium ethylene diamine tetraacetic acid (EDTA) plus supportive multivitamin/trace mineral supplementation upon fasting serum calcium. *Med Hypoth* 1983; 11:431-58.
10. 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(4):1-9.
11. 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 Holistic Med* 1985; 7(1):16-22.
12. Morris JF, Johnson LC: Spirometric standards for healthy non-smoking adults. *Am Rev Resp Dis* 1971; 103:57-67.
13. Levitzky MG: *Pulmonary Physiology*. New York:McGraw-Hill Book Company, 1982.
14. Sackner MA, edit: *Diagnostic Techniques in Pulmonary Disease*. New York:Marcel Dekker, Inc. (Pts. I,II), 1980.
15. Guyton AC: *Textbook of Medical Physiology*. 6th ed. Philadelphia:WB Saunders Company, 1981; pp 482-5.
16. Welton AF, edit: Physiology and biochemistry of a diseased lung. *Adv Clin Chem* 1987; 26:293-383.
17. Walker FM: The effects of EDTA chelation therapy on plaque calcium and mineral metabolism in atherosclerotic rabbits. Thesis for Doctor of Philosophy at North Texas State University, Denton, May 1980.
18. Perry HM, Jr: Chelation therapy in circulating and sclerosing diseases. *Fed Proc* 1961; 20(2, Suppl 10): 254-7.
19. Meltzer LE, Kitchell JR, Palmon F: The long term use, side effects, and toxicity of disodium ethylenediamine tetraacetic acid (EDTA). *Am J Med Sci* 1961; 242:51-7.
20. Boyle AJ, Mosher RE, McCann DS: Some in vivo effects of chelation-I on rheumatoid arthritis. *J Chron Dis* 1963; 16:325-8.
21. Lamar CP: Calcium chelation in atherosclerosis, nine years clinical experience. Presented at the Fourteenth Annual Meeting of the American College of Angiology, San Juan 1968 (reprinted in *Reprints on Medical Literature on Chelation Therapy* compiled and self published by HW Harper and GF Gordon 1975).
22. McDonagh EW: *Chelation can cure*. Kansas City:Platinum Pen Publishing, 1983.
23. Holland JF, Danielson E, Sahagian-Edwards A: Use of ethylene diamine tetraace-

- tic acid in hyper-calcemic patients. *Proc Soc Exp Biol Med* 1953; 84:359-64.
24. Dudley HR, Ritchie AC, Schilling A, Baker WH: Pathological changes associated with use of sodium ethylene diamine tetra-acetate in treatment of hypercalcemia—Report of 2 cases with autopsy findings. *New Engl J Med* 1955; 252:331-7.
 25. Foreman H, Vier M, Magee M: The metabolism of C-14 labeled ethylene diamine tetraacetic acid in the rat. *J Biol Chem* 1953; 203:1045.
 26. Schroeder HA: *The Poisons Around Us*. Bloomington:Indiana University Press, 1974.

