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Electrocardiographic Changes Associated with EDTA Chelation Therapy

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ABSTRACT: Electrocardiographic data are frequently used as a part of the *diagnosis* of illness. Additionally, its *predictive* use is suggested as an index of movement toward greater health in people considered to be electrocardiographically asymptomatic in conventional terms. Intravenous EDTA treatment of subjects with a demonstrated lead burden is noted to produce statistically significant and clinically desirable changes in QRS duration in lead I EKG scores.

Introduction

The following four points serve as a prelude to, and justification for, this report. First, there is general agreement of the epidemic nature of cardiovascular disease. Second, there is incontestable awareness of the sensitivity of the electrocardiogram to cardiovascular pathosis. Third, current usual and customary treatments for cardiovascular disease leave much to be desired. Fourth, there is at least presump-

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tive evidence of the salutary effects of EDTA treatment upon cardiovascular symptomatology.[1,2]

This report, as far as we can ascertain, for the first time examines one of the specific characteristics of the electrocardiogram (the QRS complex) before and after a series of EDTA treatments.

It is noteworthy that the QRS complex is a measure of ventricular depolarization. As such, with lengthening, it reflects myocardial pathosis. Specifically, it is generally held that a QRS duration in any of the limb leads greater than 0.10 seconds is undesirable.[3-8] Most of the textbooks also provide a lower limit of normality, frequently 0.06 seconds. In general, the presumption is that the shorter the QRS duration, the better is myocardial health, down to 0.06 seconds.

Method

The 28 people who are the subjects of this report were systematically drawn from 127 volunteers who responded to a newspaper and/or television ad requesting the participation of people who fit the following criteria: (1) over the age of 40, (2) ambulatory, (3) demonstrating a 5-fold increase in urinary lead as a result of a 3 gm EDTA challenge, (4) having a fasting serum creatinine level less than 1.7 mg/dL, and (5) having completed an extensive history and screening lab workup.

The therapeutic regimen consisted of a series of 20 EDTA infusions administered at approximately weekly intervals, where possible over a period of 20 weeks. The maximum time span for a very few subjects extended to 38 weeks, with a mean of 30.7 weeks. The point should be underscored that, whenever "EDTA" is used in this document, the content of the infusion is 3 gms disodium EDTA, 15 gms ascorbic acid buffered in sodium bicarbonate, 800 mg magnesium chloride, 40 mg procaine, and 1000 units heparin delivered in 500 cc sterile deionized water and intravenously infused over a period of 3–5 hours. Additionally, each subject was given three multi-vitamin and mineral supplement tablets per day.

For purposes of this report, attention will be directed to the QRS complex as measured in the standard three limb leads. A production Hewlett Packard Model 476A Cardiograph was used for data gathering.

Results

Table 1 summarizes the findings in the 28 subjects before treatment, and following 10 and 20 treatments. Indicated in this chart are the minimum and maximum scores, the means and standard deviations.

When the initial QRS durations are compared to those after 10 and

TABLE 1

Lead I: QRS Length

	Before Treatment	After Ten Treatments	After Twenty Treatments
Sample Size	28	28	28
Minimum Score	0.040	0.036	0.037
Maximum Score	0.113	0.113	0.111
Mean QRS Duration (Sec.)	0.070	0.065	0.064
SD	0.020	0.019	0.018

20 infusions, there are statistically significant improvements between the initial scores and the latter two points ($t = 2.5$, $p = .02$, $t = 2.9$, $p = .01$). In contrast, there are no statistically significant differences when the data from infusion 10 is compared solely to that of infusion 20 ($t = .38$, $p = .71$).

Conclusions

If, as the literature suggests, the shorter the QRS duration, the better the myocardial health, then the experience here described with EDTA is potentially beneficial and clearly not harmful to the myocardium, as indicated by the QRS interval. This conclusion seems reasonable, as indicated by the progressive reduction from .070 to .065 to .064 seconds respectively. This pattern of systematically varying means matched by a systematic decrease in SD is similar to our experience with other improvements following EDTA chelation therapy (eg. blood lead, serum creatinine) with the greatest differences occurring between pretreatment and the 10th treatment. The above is a reflection of a rather small sample and needs to be tested and extended with a larger and better controlled group of subjects. Within the limits of the above data, the evidence suggests that EDTA chelation therapy may offer an effective and measurable therapeutic treatment in heart disease. Because intravenous infusion in the above described manner removes substances from the body other than lead, no intent

is here expressed that the treatment is effective solely because of the removal of significant amounts of lead from subjects treated.

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