

immune function, regulating blood volume and maintaining electrolyte balance.<sup>2</sup> NO is a potent vasodilator. In conditions with renal ischemia, the release of NO occurs as a compensatory mechanism to allow for an increase in oxygenated blood flow to the obstructed area as a result of vasodilation. The fact that the concentration of NO was greater in the EDTA treated groups suggests that EDTA promotes the release of NO by preventing and possibly regenerating endothelial cells in patients with renal ischemia or disease.

Renal disease is a predisposing factor for the development of hypertension.<sup>5</sup> Chronic hypertension increases peripheral resistance in the vasculature contributing to damage and dysfunction of the endothelial lining.<sup>2</sup> Impairment of the endothelium reduces the capacity of the arteries and arterioles to dilate in response to the release of NO,<sup>2</sup> which further increases peripheral resistance. NO levels are reduced in conditions with an increase in molecular adhesion, chemokine and cytokine release and the production of reactive oxygen molecules contributing to systemic inflammation, which can contribute to the development of atherosclerosis and CVD.<sup>2</sup> Therefore, EDTA may be an effective prophylactic for CVD in patients with acute and chronic renal disease.

Although Belloni et al found that the administration of EDTA prior to the induction of ischemic compression reduced the amount of renal damage, a study conducted in Canada by Brant et al evaluating the effectiveness of EDTA plus magnesium sulfate (MgSO<sub>4</sub>) on ischemic cardiac disease concluded that EDTA was ineffective.<sup>6</sup> The study was conducted on 78 patients with the mean age of 65. The patients were randomly selected to receive intravenous infusions of 40 mg/kg of body weight of EDTA or placebo for 3 hours twice a week for 15 weeks and a maintenance treatment once a month for 3 months. The outcome of the treatment was evaluated with a cardiovascular stress test.

The study showed that the therapy group had an average increase in walking time to vascular claudication of 63 seconds while the placebo group had an average time of 54 seconds.<sup>6</sup> The average difference in time to aerobic threshold for the therapy group and the placebo group was 31 seconds and 16 seconds respectively.<sup>6</sup> The category in which the therapy group was determined to be statistically significant and the placebo group failed to achieve significance was maximum oxygen consumption (VO<sub>2</sub> max). The average increase in VO<sub>2</sub> max for the chelation therapy

group was 84 mL/min while the average increase for the placebo group was 40 mL/min.<sup>6</sup>

Although the increase in time to ischemia and aerobic threshold were minimal, the therapy group demonstrated a greater degree of improvement. This suggests that patients treated with EDTA may be utilizing oxygen more efficiently than the placebo group, which can be attributed to the ability of EDTA to preserve or restore endothelial cell function allowing for the vascular to self regulate. This theory would support the significant increase in VO<sub>2</sub> max illustrated in the treatment group. An increase in VO<sub>2</sub> max gives the patients a higher capacity for the transport and utilization of oxygen during physical activity contributing to a higher degree of physical fitness. An elevated VO<sub>2</sub> max reinforces the data obtained by Belloni et al suggesting that EDTA prevents endothelial cell damage and stimulates the release of NO inducing vasodilation. In addition, it may indicate that EDTA promotes pulmonary function. Thus, allowing for higher concentrations of oxygen to be conveyed in the blood.

A study performed evaluating forced vital capacity (FVC) and forced expiration volume (FEV<sub>1</sub>) depicts comparable effects of EDTA on lung function. In this experiment, 38 patients suffering from chronic pulmonary disorders were selected to participate. A Tiffenair Computerized Spirometer was used to analyze FVC and FEV<sub>1</sub> before and after therapy. The treatment consisted of 30 intravenous infusions of 3 g of EDTA, which was administered over a 9 month period. The authors concluded that there was a significant increase in pulmonary function.<sup>7</sup>

The baseline values of FVC and FEV<sub>1</sub> for male patients were 3.50 L and 2.27 L respectively while the values for females were 2.47 L and 1.81 L respectively.<sup>7</sup> After treatment, the FVC and FEV<sub>1</sub> levels for men were 3.91 L and 2.96 L and for females were 2.93 L and 1.89 L.<sup>7</sup> The increase in FVC denotes enhanced pulmonary function, which can be attributed to the ability of EDTA to promote NO release and restore endothelial cell function. Within the pulmonary tissue a high concentration of endothelial cells produce and release NO, which induces vasodilation augmenting the oxygen exchanging capacity of the alveoli and ultimately improving pulmonary function.<sup>8</sup>

FEV<sub>1</sub> measures the maximum amount of air exhaled in 1 second, which is a strong indicator of obstructive pathologies.<sup>8</sup> Although there was a significant increase

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