Additional Table 1: Cardiovascular indications for O_3 therapy

Study	Pathology	Concentration and route of O_3 administration	Type of study	Measured parameter(s)	Results	Side effect(s)	Mechanism of action
Martínez-Sánchez et al.[14]	Coronary artery disease	57 patients with massive cerebral infarction	Ungrouped; cocktail therapy: nimodipine (10 mg) intravenously, once per day, for 10 consecutive days	Prothrombin time	Significantly improved ($P < 0.001$)	None	Upregulation of adenosine A_2 receptor
				Oxidative stress levels	Increased antioxidant activity	None	Increased SOD and catalase enzyme activity
Hernandez et al.[40]	Previous myocardial infarction (3 months to 1 year)	200 mL of blood subjected to O_3-AHT, for a final concentration of 50 mg/L; treatment was given 5 days a week for up to 15 sessions	Pretest-posttest design (n = 22)	Serum lipid pattern	Cholesterol and low-density lipoprotein were significantly reduced with no changes in high-density lipoprotein and triglycerides	Not reported	Initiating radical formation which increasing lipid peroxidation
				Activity of antioxidant defense system	Biologically significant increases on erythrocyte GPx and glucose-6-phosphate dehydrogenase	Not reported	O_3-AHT stimulates ROS scavenger enzymes

Note: O_3: Ozone; O_2: oxygen; O_3-AHT: O_3 autohemotransfusion; GPx: glutathione peroxidase; SOD: superoxide dismutase; ROS: reactive oxidative species.