Title: Influence of hyperbaric oxygen on left ventricular contractility, total coronary blood flow, and myocardial oxygen consumption in the conscious dog

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Abstract: It is known that hyperbaric oxygenation (HBO) decreases total coronary blood flow (TCBF) and cardiac output (CO). To determine whether this is related to an alteration in myocardial contractility, 10 chronically instrumented conscious dogs were studied during pharmacologic autonomic blockade. Left ventricular (LV) volume was measured with ultrasonic transducers, LV transmural pressure with micromanometers, TCBF with Doppler-flow probes, and coronary AVO2 difference (A-CSO2) was calculated from direct LV and coronary sinus (CS) sampling. To evaluate the effect of increased oxygenation, data were obtained during resting control conditions and during dynamic vena caval occlusions (VCO), at 1 atmosphere of pressure, while breathing air (1 bar/0.21); at 3 atmospheres, breathing compressed air (3 bar/0.21), and at 3 atmospheres breathing 100% oxygen (3 bar/1.0). Because of autonomic blockade, heart rate (HR) was not statistically different in the three conditions. With increasing oxygenation, arterial oxygen tension (PaO2) increased from 85 +/- 5 mmHg (mean +/- SD) at

	1 bar/0.21, to 1374 +/- 201 mmHg at 3 bar/1.0 whereas arterial carbon dioxide tension (PaCO2) and pH values were not statistically different. Arterial oxygen content (AO2 content) and CSO2 content increased significantly (both P < 0.05) with increasing PaO2. LV stroke volume (SV), CO, coronary blood flow, and myocardial oxygen consumption (MVO2) were all significantly reduced (P < 0.05) with increasing levels of oxygenation. Intrinsic myocardial function, as measured by the stroke-work/end-diastolic volume relationship was unchanged from 1 bar/0.21 to 3 bar/0.21, and to 3 bar/1.0 (P < 0.20). Thus, the diminished TCBF, CO, and MVO2 associated with HBO do not seem to be associated with a primary alteration in myocardial contractility, but rather may result from a physiologic autoregulation of the myocardium to increasing levels of PaO2.
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