Title : Oxygenation after Tracheal Obstruction on Air and 100% Oxygen

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Introduction. Arterial oxygenation following apnea on 100% oxygen (O_2) is well known.[1] Mathematical models simulating arterial oxygenation following apnea and breath holding have been developed.[2] There is general agreement that 100% O_2 should be given whenever tracheal obstruction is anticipated, but no direct, experimental data could be found comparing differences in arterial oxygenation following tracheal obstruction while breathing room air or 100% O_2. This study compares physiological, gasometric and metabolic responses in one group of experimental animals breathing room air against a similar group breathing 100% O_2 followed by airway occlusion.

Method. Five canine were anesthetized with sodium thiopental and intubated with a cuffed tracheal tube. Monitoring included pulmonary artery pressure, arterial pressure, right atrial pressure, endotracheal airway pressure and the electrocardiogram. Blood gas samples were taken from the pulmonary artery and the aortic arch. The dogs were ventilated on room air and 100% O_2 and the tracheal tube was occluded with a rubber stopper to simulate airway obstruction. Functional residual capacity (FRC) was taken as the end of expiration. Forced vital capacity (FVC) was simulated by inflation to airway pressured of 20-30 torr. Four tests were run in succession on each dog: 1. room air at FRC, 2. room air at FVC, 3. 100% O_2 at FRC, and 4. 100% O_2 at FVC. Cardiac outputs by thermodilution were done intermittently.

Results. The graph below, presented on semi-logarithmic scale, represents mean arterial oxygenation with time following airway occlusion for all four tests. On room air the slope of the desaturation curves is more gradual than with 100% O_2. In the 100% O_2 tests, the time until hypoxia develops is a function of lung volume and oxygen consumption. Though the times to hypoxia vary in the 100% O_2 tests, the shapes of all the curves once arterial desaturation starts are virtually identical.

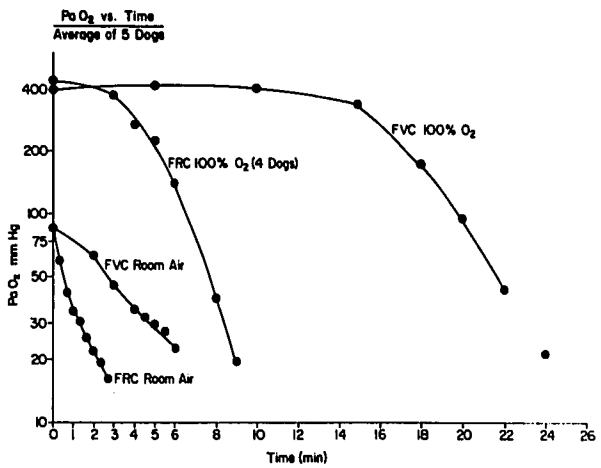
All dogs recovered immediately from the absorption atlectasis associated with 100% O_2 hypoxia and the highly negative airway pressures. It was also noted that three of the five dogs developed pulmonary hypertension.

Discussion. On room air, hypoxia develops more slowly once the lung oxygen tension has been reduced to the mixed venous levels since oxygen is then largely supplied by the desaturation of the oxygen stored in the blood.[3] On 100% O_2, the blood oxygen is the only source during hypoxia because the alveolar PO_2 remains above 500 torr until the alveolar oxygen is virtually gone.

Starting from FRC, dogs developed severe hypoxia (using PaO_2 less than 30 torr as severe) in 1.0-1.7 minutes on room air, while on 100% O_2 this time was extended to 6.0-9.5 minutes. Starting from FVC, severe hypoxia developed in 4.5-5.5 minutes on room air while this time was extended to 18.5-25.5 minutes on 100% O_2. On room air, mild hypoxia (PaO_2 = 50 torr) developed in about one-half the time of severe hypoxia. On 100% O_2 severe hypoxia always developed in less than one minute after mild hypoxia.

Pulmonary hypertension may have contributed to the death of three dogs compared to all the dogs in a previously reported study.[1]

Conclusion. This data documents the need for ventilation with 100% oxygen whenever possible anytime airway obstruction may occur, such as prior to tracheal intubation or extubation.



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