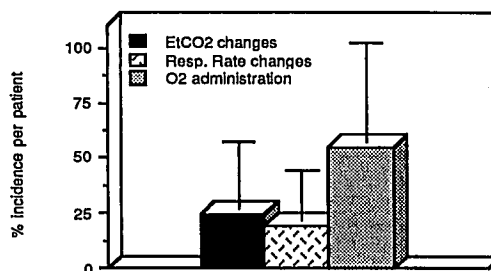


**TITLE:** HYPOVENTILATION AND THE MIXED ETIOLOGY OF POSTOPERATIVE HYPOXEMIA  
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Postoperative hypoxemia is secondary to a variety of causes, including increased venous admixture, decreased functional residual capacity, upper airway obstruction, and respiratory depression and hypoventilation. In a preliminary phase of a study, approved by the institutional clinical investigation committee, we evaluated 34 recovery room (RR) patients for postoperative arterial oxyhemoglobin desaturations and the association of these desaturations to hypoventilations. Desaturations measured by a Nellcor N200 or N1000 were defined as a SaO<sub>2</sub> ≤ 92% for ≥ 30 sec. Nasal EtCO<sub>2</sub> was measured (10% change was significant) by infra-red analysis using a Nellcor N1000 or a Beckman LB3 capnometer. Recordings of the EtCO<sub>2</sub>, SaO<sub>2</sub>, and pulse intensity were made. SaO<sub>2</sub> decreases associated with uneven pulse intensity signals were discarded. Patients were compared by sex (16M), age (48±18y), weight (78±23kg), history of cardiac (9) or pulmonary (10) disease or smoking (12), ASA status (1.9±0.7), site of surgery (thorax 1, abdomen 21, periphery 12), type of anesthetic (regional 7, general 27), duration of anesthesia (1.7±1 hr), narcotic dose (equivalent to 6.4±5.8 mg MSO<sub>4</sub>), premedication, temperature, intravenous fluid volume (1.8±1.2 L), and whether O<sub>2</sub> was administered during desaturation. Patients were transferred to the RR spontaneously breathing room air. During RR stay oxygen was administered by facemask (23) or facemask (2) (FIO<sub>2</sub> 0.4) at 12 lpm, or nasal cannula at 2-3 lpm (7). 2 patients breathed room air. Personnel

were not blinded to SaO<sub>2</sub> values and routine care was not altered. Data was analysed by ANOVA and Student t-tests.

On RR admission mean SaO<sub>2</sub> was 98±2.5%. During RR stay 14 pts. (41%) desaturated to 88.2±2.2% for 45±30 sec. at 30.5±42.5 min. after admission. There were 4.9±5.1 desaturations per patient (69 total episodes). 32 desaturations (46%) were associated with changes in EtCO<sub>2</sub> and 23 (33%) with changes in respiratory rate. 54% of desaturations occurred during oxygen administration. The mean incidence per patient of desaturations with changes in EtCO<sub>2</sub>=24.6% and respiratory rate=19.3% (figure). The same patient often exhibited separate desaturations with or without signs of hypoventilation. Desaturations were positively correlated with age and lower admission SaO<sub>2</sub>. Hypoventilations, as documented by nasal EtCO<sub>2</sub> and respiratory rate, are associated with slightly less than half of the arterial desaturations in postoperative patients. Also of note was the mixed etiology of desaturations within individual patients.



## A1058

**TITLE:** Malignant Hyperthermia Simulator  
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We have previously described a simulator for general anesthesia that includes a graphical user interface and a mathematical model of cardiovascular and respiratory physiology and pharmacology of 50 drugs to predict patient responses during simulated anesthesia and surgery<sup>1</sup>. The pathophysiological changes associated with malignant hyperthermia (MH) were added to provide an environment for anesthesiologists to observe and manage case scenarios involving this potentially life-threatening crisis.

**Methods** Following the administration of a potent volatile agent or succinylcholine, the physiological model produces marked changes in vital signs, acid-base balance and temperature similar to those described by Rosenberg<sup>2</sup>. In the simulation, muscular rigidity may be seen, followed by greatly increased CO<sub>2</sub> production and O<sub>2</sub> consumption which increase ventilatory requirements. Tachycardia is in response to the metabolic and respiratory acidosis. Temperature rises as fast as 1°C every five minutes. In addition, hyperkalemia, dysrhythmias, and cardiovascular collapse may follow. Twelve other cardiovascular and respiratory critical incidents are included in the simulator, so the diagnosis of MH is not immediately obvious, much as in the OR. Treatment on the simulator includes removing

the offending agent, cooling the patient, and the administration of appropriate doses of dantrolene<sup>3</sup>.

**Results** Four case scenarios were developed for the MH simulator. The four patients have different susceptibilities to a reaction and differing manifestations of the reaction. Reactions will be triggered depending on the agents administered, and can be avoided altogether with the proper technique.

**Discussion** Early diagnosis of MH is vital to successful treatment. Anesthesiologists that observe the sequence of physiological changes and practice the treatment protocols for MH on a simulator may be better prepared for this event in the operating room. Anesthesiologists can also practice simulated administration of general anesthesia to patients known to be MH susceptible.

### References

1. Anesthesiology 72: 191-197, 1990
2. Br J Anaesth 60: 268-273, 1988
3. Anesthesiology 70: 625-629, 1989