Title : CARDIOVASCULAR EFFECTS OF EXTUBATION

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Introduction. Cardiovascular stimulation during tracheal intubation and laryngoscopy has been well described in recent literature. Avoidance of these changes in the ischemic heart has been stressed in order to avoid changes in myocardial oxygen consumption. Surprisingly little data has been published for cardiovascular changes resulting from tracheal extubation, while no data exists from patients with known coronary artery disease. We have investigated the cardiovascular changes subsequent to tracheal extubation in this group of patients, and the effect of prior intravenous lidocaine on these changes.

Methods. Eighteen ASA Class III patients were studied 18 to 24 hours post-coronary artery bypass grafting. Direct arterial blood pressure was recorded via a radial artery catheter and a flow directed Swan-Ganz catheter was inserted and connected to a pressure transducer and Edwards cardiac output computer. Heart rate was obtained by standard electrocardiogram.

All patients received morphine, pancuronium bromide, and 50% nitrous oxide in oxygen for anesthesia; pharmacologic reversal was not attempted for any agent. No patient received any drug or blood transfusion for at least six hours prior to extubation.

Eighteen patients were randomly assigned into two groups of nine patients. One group received lidocaine (1.5 mg/kg i.v.) approximately 90 seconds prior to extubation, while the other group received no treatment.

Parameters measured were heart rate, systolic, mean, and diastolic blood pressures, right atrial pressure, mean pulmonary artery pressure, pulmonary capillary wedge pressure, and cardiac output. Cardiac index, systemic and pulmonary vascular resistance and rate pressure product were calculated from standard equations. Arterial blood gases were also analyzed. Statistical significance was calculated using Student's t-test for paired and unpaired data. Differences with a probability of 0.05 or less were considered significant.

Results. No significant differences were noted in any parameters between the groups before extubation. At one minute post extubation, significant ($p < 0.05$) increases were measured in CI, PCWP, PVR, PAP, HR, MAP, SVR, and rate-pressure product index in both groups. These changes remained significant beginning at one and continuing until 10 minutes following extubation; at 15 minutes all parameters did not differ significantly from pre-extubation values. No significant difference in any parameter was noted between groups when compared at the same time intervals. Arterial blood gases measured at 5 and 10 minutes after extubation were not significantly different from those measured before extubation. There were no EKG changes suggestive of ischemia post extubation.

Discussion. These data demonstrate that tracheal extubation in the post coronary artery bypass graft patient is followed by intense cardiovascular stimulation persisting for at least ten minutes, and that intravenous lidocaine (1.5 mg/kg) does not alter these changes.

While plasma lidocaine levels were not measured, previous reports have shown similar dosages of lidocaine to result in therapeutic plasma levels for 10-20 minutes, the time interval over which significant cardiovascular changes were observed in this study. That extubation only is responsible for the cardiovascular changes observed seems likely, since lidocaine in this dosage range has been reported to have minimal cardiovascular effects.

Even though no patient in our study developed arrhythmia, angina, or infarction during or following extubation, their potential would seem apparent from our results. Thus, cardiovascular monitoring would be appropriate following extubation in these patients. Also, evaluation of other pharmacologic means of attenuating this response would seem warranted.