

Title : INTRAOPERATIVE CHANGES IN HEMOGLOBIN OXYGEN AFFINITY IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS.

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Introduction. We have examined whether patients with ischemic heart disease have changes in P_{50} intraoperatively and postcardiopulmonary bypass. Some data suggest that anesthetic agents may alter the hemoglobin oxygen dissociation curve (1), and Halothane may cause conformational changes in the hemoglobin molecule (2). Others have demonstrated change in P_{50} due to cardiac failure (3), anemia (4), and after cardiopulmonary bypass (5).

Methods. Informed consent and institutional approval for the study was obtained. In 15 patients randomly anesthetized with morphine (2mg/kg) or halothane (0.5 to 1.0% end tidal concentrations) we inserted coronary sinus catheters under fluoroscopic control, thermodilution pulmonary artery catheters, and radial artery catheters before induction of anesthesia. We measured thermodilution coronary sinus flow, cardiac output, pulmonary and arterial pressures, and sampled arterial, mixed venous, and coronary sinus blood to measure blood gases, oxygen saturations, oxygen content, and lactates. From the blood gases and saturations we calculated P_{50} (6) under standard conditions (pH 7.4, BE 0, 37°C) in mixed venous (mv P_{50}) and coronary sinus blood (cs P_{50}) before induction, 10 minutes after intubation, 10 minutes after sternotomy, 10 minutes post bypass and 10 minutes post bypass with the chest closed.

Results. We divided patients into two groups on the basis of preanesthetic mv P_{50} . Those with mv P_{50} greater than 29 torr had even greater cs P_{50} prebypass (mean difference 2.7 ± 2.9 torr). This difference between coronary sinus and mixed venous hemoglobin oxygen dissociation was not evident after cardiopulmonary bypass (mean difference 0.1 ± 3.2 torr). In the patient group with mv P_{50} 's less than 29 torr, cs P_{50} was not elevated. However induction of anesthesia increased P_{50} in both mv (26.1 ± 2.0 torr to 30.3 ± 2.0 torr) and cs blood (26.8 ± 2.1 to 30.2 ± 2.3 torr) for this patient group. This elevation remained throughout the operation.

In the elevated P_{50} group, cs P_{50} correlated with cs oxygen content ($r = -0.75$, $P < 0.01$) and mv P_{50} correlated significantly with two measures of cardiac performance, i.e.

arteriovenous oxygen content difference, ($r = 0.63$, $P < 0.01$) and wedge pressure ($r = 0.61$, $P < 0.05$). Hemoglobin concentration correlated significantly with mv P_{50} ($r = -0.58$, $P < 0.05$).

Discussion. Patients with cardiac failure evidenced by increased wedge pressure and widened arteriovenous oxygen content difference have increased mv P_{50} 's and even greater increased cs P_{50} 's. We speculate that this increase aids peripheral tissue oxygen unloading when oxygen delivery is inadequate because of cardiac failure. Since cs P_{50} 's are higher than mv P_{50} 's it is also tempting to speculate that the heart is a contributor to the elevated mv P_{50} . Cardiopulmonary bypass or the events associated with it remove the mechanism responsible for the elevation since P_{50} returns to normal post bypass. Since ventricular function curves are depressed postbypass when compared to control measurements, it is unlikely that the increased P_{50} is no longer required for tissue oxygen delivery.

The sustained increase in P_{50} after induction in both morphine and halothane anesthetized patients suggest that anesthesia is beneficial for tissue oxygen unloading. Nitrous oxide was the only agent administered to both groups.

References.

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