

**TITLE:** HEMODYNAMIC EFFECTS OF DESFLURANE VERSUS ISOFLURANE IN PATIENTS UNDERGOING CORONARY BYPASS GRAFT SURGERY

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**Introduction.** Desflurane (D) is an inhalation anesthetic structurally similar to isoflurane (I), yet less soluble in blood.

Previous studies in healthy volunteers have characterized the hemodynamic effects of D to include increased heart rate (HR), increased cardiac output (CO) and decreased systemic vascular resistance. We report the hemodynamic effects of D versus I in patients undergoing coronary artery bypass surgery.

**Methods.** After institutional review board approval, randomization, and informed consent, 22 ASA III patients aged 33-85 and scheduled for elective coronary artery bypass graft surgery received either D or I. Induction was accomplished with thiopental 2 mg./kg. and fentanyl 5 mcg./kg. Muscle relaxation achieved with pancuronium .1 mg./kg. D or I was administered up to the level of 1 MAC and maintained for at least 5 min. Thereafter, the level of inhalation agent was titrated to the patients hemodynamics. Hemodynamic

measurements were made at nine intervals: baseline (B), end induction (IND), 1 min. after intubation (ET), skin prep, incision, sternotomy, prior to bypass, after bypass and end of surgery. Variables measured included systemic, pulmonary, central venous and pulmonary capillary wedge pressures; CO; HR; and end tidal anesthetic agent.

**Results.** Most measured and calculated variables were similar between I and D groups. A significant difference in mean PA pressure and wedge pressure was found at end induction with higher pressures in the D group.

Table I (Mean  $\pm$  SD); \*p < .05

|      |   | B           | IND         | ET          |
|------|---|-------------|-------------|-------------|
| ART  | D | 86 $\pm$ 12 | 73 $\pm$ 13 | 71 $\pm$ 13 |
|      | I | 84 $\pm$ 8  | 66 $\pm$ 10 | 80 $\pm$ 14 |
| PA   | D | 19 $\pm$ 4  | 22 $\pm$ 4  | 21 $\pm$ 3  |
|      | I | 17 $\pm$ 4  | 16 $\pm$ 3* | 20 $\pm$ 5  |
| PCWP | D | 13 $\pm$ 3  | 16 $\pm$ 5  | 14 $\pm$ 3  |
|      | I | 12 $\pm$ 3  | 11 $\pm$ 2* | 14 $\pm$ 5  |

**Discussion.** The above differences in PAP and PCWP may represent a greater cardiac depression by D, secondary to a more rapid equilibration of D than I. Alternatively, despite randomization, dissimilarity of the groups was suggested because baseline stroke volume index was significantly lower in the D group (45  $\pm$  10) than I group (56  $\pm$  10).

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**TITLE:** EFFECT OF ACUTE AORTIC CONSTRICTION ON SYSTEMIC AND CEREBRAL ADENOSINE [ADO] IN MAN

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**INTRODUCTION:** THIS STUDY INVESTIGATES THE EFFECTS OF ACUTE AORTIC CONSTRICTION ON PLASMA AND CEREBROSPINAL METABOLITES OF HIGH ENERGY PHOSPHATES IN PATIENTS UNDERGOING THORACIC [TAA] AND INFRARENAL [AAA] AORTIC ANEURYSMECTOMY.

**METHODS:** AFTER INFORMED CONSENT AND INSTITUTIONAL APPROVAL ARTERIAL [A] AND PULMONARY-ARTERIAL [PA] ADO, HYPOXANTHINE [HYPO] AND LACTATE [LAC] WERE ANALYZED IN 18 PATIENTS (66  $\pm$  11 YEARS (SD)) DURING AAA BEFORE [C], DURING INFRARENAL CLAMPING, AND DECLAMPING. BESIDE OTHERS BLOOD PRESSURE [SBP], CARDIAC OUTPUT [CO] AND STROKE WORK [SW] WERE DETERMINED.

IN 7 PATIENTS (68  $\pm$  5 YEARS (SD)) WITH TAA THE EPIDURAL SPACE (L3/L4) WAS CANNULATED AND THE PERIOPERATIVE CHANGES OF INTRACRANIAL PRESSURE [ICP] AND CEREBROSPINAL [CS] ADO AND HYPO DETERMINED.

**SUMMARY:** 1.) DURING AAA ADO, BUT NOT LAC PARALLELS THE CHANGES IN SBP, CO, AND STROKE WORK INDUCED BY ACUTE AORTIC CONSTRICTION.

2.) DURING REPERFUSION ADO, HYPO, LAC AND H+ ARE WASHED OUT INTO THE VENOUS BLOOD INDICATING A SIGNIFICANT IMPAIRMENT IN TISSUE OXYGENATION UNDER CLAMPING CONDITIONS. 3.) DURING TAA ICP IS SIGNIFICANTLY INCREASED. THE ENHANCED ADO AND HYPO MOST LIKELY REFLECT THE DEVELOPMENT OF CEREBROSPINAL ISCHEMIA DUE TO REDUCED TISSUE PERFUSION.

TABLE 1: HEMODYNAMIC/METABOLIC CHANGES DURING AAA (MEAN VALUES, N=18; \* P < .05)

|             |      | CLAMPING |      | DECLAMPING |       |
|-------------|------|----------|------|------------|-------|
|             | C    | 5'       | 30'  | 5'         | 20'   |
| SBP (MM HG) | 113  | 125*     | 128* | 129        | 114   |
| CO (L/MIN)  | 7.4  | 6.8*     | 5.3* | 6.7        | 7.2   |
| SW (NM)     | 1.4  | 1.8*     | 1.6  | 1.8        | 1.6   |
| ADOA (NM)   | 185  | 494*     | 367  | 392        | 230   |
| ADOPA (NM)  | 172  | 388      | 339  | 391        | 347   |
| HYPOA (UM)  | 0.95 | 1.00     | 0.98 | 2.26*      | 1.71* |
| HYPOPA (UM) | 1.01 | 0.88     | 1.36 | 2.02*      | 2.09* |
| LACA (MM)   | 0.58 | 0.58     | 0.66 | 1.24*      | 0.99* |
| LACPA (MM)  | 0.56 | 0.54     | 0.66 | 1.16*      | 0.96* |
| PHA         | 7.38 | 7.38     | 7.38 | 7.31*      | 7.35  |

TABLE 2: HEMODYNAMIC/METABOLIC CHANGES DURING TAA (MEAN VALUES, N=7; \* P < .05)

|             |     | CLAMPING |      | DECLAMPING |      |
|-------------|-----|----------|------|------------|------|
|             | C   | 5'       | 30'  | 5'         | 20'  |
| ICP (MM HG) | 8   | 19*      | 20*  | 18*        | 5    |
| ADOCs (NM)  | 147 | 258      | 333* | 357*       | 394* |
| HYPOCs (UM) | 1.9 | 2.5      | 2.8* | 5.0*       | 9.5* |