Title: **CORONARY BYPASS GRAFT FLOW (CBGF) VASODILATION (VD) INDUCED BY ISOFLURANE HAS ONLY NEGLIGIBLE EFFECTS ON LEFT VENTRICULAR (LV) SYSTOLIC FUNCTION**

Authors: **LECLERC D, FRATACCI MD, DUPUY P, PAYEN D.**
Affiliation: Anes. Dept., Lariboisiere University Hospital, Paris, France.

The deleterious clinical impact of the coronary vasodilating properties of isoflurane (I) remains debated for at least two reasons: 1) is the VD partially mediated by I and/or by the adaptation of coronary resitances to driving pressure fall? 2) does this VD and potential coronary steal alter myocardial systolic function more than the direct I effect? Since coronary atherosclerosis is a diffuse disease of the all vascular bed, these questions should be important even after coronary revascularisation. This study was designed to assess both CBGF and its depending regional systolic LV thickening fraction, wich is an index of myocardial ischemia[1] (LVTF, %) 4 hrs after surgery in 6 pts (56 ± 10 yrs) with a preoperative LVEF over 45%. Following parameters were measured or calculated : heart rate (HR, bpm), arterial pressure (MAP, mmHg, radial catheter), cardiac output (CO, l/min, TDilution), pulmonary artery occluded pressure (PAoP), CBGF (8 MHz implantable pulsed Doppler microprobe[2]), LVTF (10 MHz epicardial pulsed Doppler microprobe[3] in the reperfused territory), velocity of regional contraction (dL/dt, arbitrary unit).

Protocol: Data were recorded before (C), 30 min after I inhalation titrated to induce a 20 % decrease in MAP (mean end-tidal I concentration (Datex) = 0.67% ± 0.3) and 30 min after a non pharmacologic MAP increase during I using lower body positive pressure (LBPP). This protocol gave the opportunity to study flow function at different MAP, with constant CO. Data were analysed by Wilcoxon test. $p < .05$

** vs C ; $ vs 1*

		C	1	2
MAP	mmHg	81 ± 13	63 ± 8 *	72 ± 10 *$
PAoP	mmHg	10.5 ± 2	10 ± 2.7	16 ± 5 *
SV	ml	80 ± 13	76 ± 13	75 ± 15 *NS*
CVR	mmHg/l/mn	2.31 ± 1	1.71 ± 1 *	1.45 ± .6 *
CBGF	ml/mn	74 ± 93	68 ± 63	92 ± 110 *
LVTF	%	25 ± 9	23 ± 10	24 ± 11 *NS*
dL/dt		2.2 ± .3	2 ± .4 *	1.9 ± .5 *

1 = ISO ; 2 = ISO + LBPP

Discussion: I-induced coronary VD was significant even when MAP was corrected, suggesting a direct coronary effect of I. Nevertheless systolic contraction depression during I was not further worsen during CBGF increase. This allows to conclude that coronary VD per se has little effect on contraction impairment.

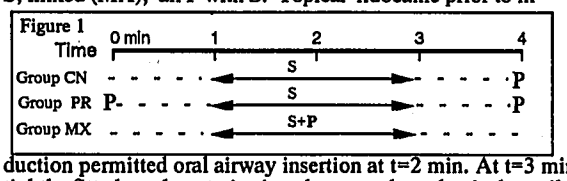
References: 1 Buffington, Anesthesiology 63, 651, 1985
2 Payen, Circulation 74: III-61, 1986
3 Hartley, Am J Physiol 245: H1066, 1983.

TITLE: PRIMING DOSE OF PANCURONIUM DOES NOT ATTENUATE CHEST WALL RIGIDITY FROM SUFENTANIL

AUTHORS: JT ABRAMS MD, J HORROW MD, RJ STORELLA PhD, DF VAN RIPER MD, DL LAMBSON MD, J RAWA BS
AFFILIATION: Department of Anesthesiology, Hahnemann University, Philadelphia, PA 19102

Chest wall rigidity, a predictable side effect of high dose opioid induction techniques,[1] is purportedly blunted by priming with muscle relaxant.[2,3] Previous studies lack an objective measure of the clinically relevant outcome variable: ventilatory compliance. This study assessed static ventilatory compliance during three different opioid induction techniques.

After institutional approval, informed consent, and premedication with morphine/scopolamine im, 30 patients for cardiac surgery received 3µg/kg sufentanil (S) over 2 min. Each of 3 groups received pancuronium (P), 100 µg/kg total, in the following randomized double-blinded fashion (Fig.1): control (CN), all P 1 min after S; primed (PR), 1 mg P 1 min before S, balance of P 1 min after S; mixed (MX), all P with S. Topical lidocaine prior to induction permitted oral airway insertion at t=2 min. At t=3 min, a tightly-fitted mask, anterior jaw thrust, and mechanical ventilator with RR=10, TV=10ml/kg permitted measurement of Δp (plateau airway pressure) and Δv (exhaled volume, Wright ventilometer at mask) in 5 replicates. Volume and pressure measurements were repeated at t=8 min. Median Δp divided into its associated Δv calculated static compliance. ANOVA and Sheffé's test analyzed between group data at each time; paired t-test compared t=3 vs t=8 data.



Results: Groups did not differ in demographics. No patient experienced early paralysis, heart rate<40, or systolic BP<80. 2 patients in group PR suffered S_aO_2<90%. MX was more compliant than CN and PR at t=3 but no different at t=8, at which time compliance was uniformly excellent. CN and PR were each less compliant at t=3 than t=8 (fig.2, mean±SEM).

Discussion: A previous study[3] indicated that priming decreased the severity but not the frequency of rigidity. That study lacked both an objective compliance measurement and a blinded, randomized design. The present data demonstrate a priming dose of P is clinically ineffective. However, concomitant infusion of S+P permits good ventilatory compliance without causing early paralysis in suitably pre-medicated patients.

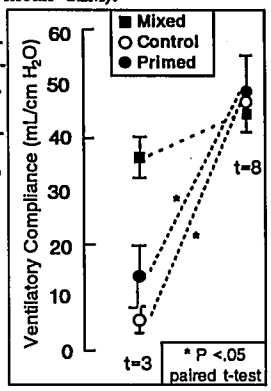


FIG.2 Compliance at t=3 and t=8 → (MX > PR = CN at 3 min)

References
1 HILL AB, ET AL.: ANESTHESIOLOGY 55:452-454, 1981.
2 BAILEY PL, ET AL.: ANESTH ANALG 64:48-53, 1985.
3 GRATZ I, ET AL.: ANESTH ANALG 68:S110, 1989.