Title : NORMOVOLEMIC HEMODILUTION IN ANESTHETIZED PATIENTS WITH CORONARY ARTERY DISEASE : EFFECTS ON HEMODYNAMIC AND LV FUNCTION.

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INTRODUCTION. Among cardiovascular adaptations to normovolemic hemodilution (NVH), increased cardiac output plays a predominant role. In patients with coronary artery disease (CAD), this adaptation may also depend on the myocardial effects of NVH. Indeed, coronary artery stenosis may limit the required increase in coronary blood flow, thus impairing left ventricular (LV) function which in turn may limit systemic O2 transport (SO2T). On the other hand, it has been argued that NVH produces a more homogenous flow distribution (1) which could be beneficial even in patients with CAD (2). Additionally, the hemodynamic effects of anesthesia must be taken into account when evaluating the hemodynamic effects of NVH peroperatively. We conducted this study in anesthetized patients with CAD to investigate the effects of NVH on cardiac index (CI), LV function, SO2T and O2 consumption (VO2).

METHODS. Fifteen patients with CAD, aged 58±10 years (mean ± SD), scheduled for abdominal aortic surgery were included in this randomized study. All gave informed consent for the study after approval by our Ethics Committee. On preoperative myocardial thallium gammatomography, all had at least one defect on initial scans with a redistribution on delayed scans. Their left ventricular (LV) ejection fraction determined by gated radionuclide angiography was above 0.5 (mean 0.57±0.07). Under premedication (morphine 5 mg, scopolamine 0.5 mg), catheters were inserted in 2 peripheral veins and in a radial artery. A thermodilution Swan-Ganz catheter was positioned while EKG (lead CM5) was continuously monitored. A transoesophagal two-dimensionnal echocardiographic (TEE) probe was then positioned to obtain a LV cross-sectional view through the base of the papillary muscles. First measurements were performed after a resting period of 20 min (CONTROL). General anesthesia was induced using flunitrazepam (0.02 mg/kg), fentanyl (6 mcg/kg) and pancuronium bromide (0.1 mg/kg) and maintained with increments of fentanyl while under controlled ventilation (air/O2 : 50 %). Colloids (750 ml) were infused at the time of induction to limit the decrease in MAP. A second set of measurement was carried out 15 min after induction (T2). Patients were randomly assigned to a hemodiluted group (group H, n = 7), or to a non-hemodiluted group (group NH, n = 8). In group H, to achieve a final hematocrit (Hct) of 30 %, blood was withdrawn via a peripheral vein and simultaneously replaced by the same volume of colloids. A third set of measurements was performed 10 min after NVH in group H (T3), and 40 min after T2 in group NH (T3). Measurements included : hemodynamic parameters and arterial and mixed venous O2 contents (CaO2, CvO2). SO2T and VO2 were derived. The LV ejection fraction area (EFa) was calculated from end diastolic (EDa) and end systolic areas. Data were analysed using two way analysis of variance and expressed as mean ± SD.

RESULTS are expressed in Table 1. At CONTROL, there was no significant difference between groups for all parameters. In group H, no significant changes in MAP, PCWP, CI and EDa were noted after NVH (T3) when compared to anesthesia (T2) values. Lack of increase in CI thus decreased SO2T. However, VO2 was similar in both groups, since CvO2 was significantly lower in group H than in group NH. No ST-T segment depression was detected during NH and no new segmental wall motion abnormality was detected using TEE recordings.

DISCUSSION. The major finding in this study is the lack of increased CI in response to NVH in anesthetized patients with CAD. The absence of increase in both EDa and PCWP suggest that here, unexpectedly, venous return was not enhanced by NVH. Three mechanisms may explain this finding: (i) insufficient volume replacement during hemodilution is unlikely, since the same amount of colloids was infused ; (ii) impaired cardiac function due to NVH in these patients with CAD was discarded since no change in global and regional LV function was detected by TEE; (iii) and the most likely, increased venous compliance due to fentanyl benzodiazepine anesthesia, which may interfere with the effects of NVH on venous return. Furthermore, it should be noted that despite reduced SO2T, VO2 was similar in both groups. In conclusion, NVH achieving a Hct of 30 % appears to be a safe procedure in anesthetized patients with CAD and normal LV fonction, despite the lack of increased CI.

REFERENCES

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		CONTROL	T2	T3
Hct %	H	41±3	36±6**	●● 27±4**£££
	NH	43±5	38±4**	38±5**
MAP mmHg	H	100±16	75±12**	72±15**
	NH	106±13	75±17**	77±16**
PCWP mmHg	H	5.9±3.2	4.7±2.5	6.1±2.7
	NH	9.9±4.1	5.4±3.9**	5.4±3.7**
CI l/min/m^2	H	3.5±0.7	2.9±0.5*	2.9±0.6*
	NH	3.4±0.6	2.6±0.8**	2.5±0.6**
EDa cm2	H	15.0±3.7	14.0±3.0	13.8±3.4
	NH	17.8±3.7	15.1±3.4*	13.0±3.0**
EFa	H	0.55±0.08	0.52±0.09	0.52±0.09
	NH	0.53±0.07	0.56±0.07	0.53±0.07
SO2T ml/min/m^2	H	646±173	476±139	358±100**£
	NH	634±192	446±219**	418±141**
CvO2 ml/100ml	H	13.7±2.0	12.8±3.0	●● 8.6±2.0**£££
	NH	13.6±2.0	12.3±1.9**	12.3±1.7**
VO2 ml/min/m^2	H	164±33	107±24**	104±27**
	NH	157±35	112±33**	107±27**

* p < 0.05, ** p < 0.01 vs control
£ p < 0.05, £££ p < 0.01 T3 vs T2
●● p < 0.01 H vs NH