

TITLE: O₂ DELIVERY (DO₂) AND O₂ CONSUMPTION (VO₂) UNDER GENERAL ANESTHESIA AND HEMODILUTION.
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General anesthesia modifies the DO₂-VO₂ relationship compared to conscious state (1). It reduces the DO₂ and participates in an increase in cellular O₂ Ext. It appears to react by reducing general metabolism, which decreases VO₂. Consequently VO₂ is maintained at low level of DO₂. Are these relationships perturbed by other parameters which act on DO₂ ?

We have studied the role of intraoperative normal acute hemodilution on the VO₂-DO₂ relationship. We have anesthetized, paralyzed and ventilated 15 patients undergoing abdominal aortic surgery (informed consent and approval by the ethic committee of Pellegrin Hosp., Bordeaux, France). Right heart catheterism allowed us to measure pressures (MPAP, RAP, PcwP) and CO by thermodilution. The MAP was continuously measured through a radial catheter. Hemodynamic measures were coupled with arterial and venous blood withdrawals for O₂ Sat., O₂ content, O₂ capacity, lactic acid and Hte. The calculation of the VO₂ and DO₂ were corrected by the Moreno Statistical Method in order to limit any effect on common variables by errors.

Out of the 15 patients, 7 controls underwent a series of measurements (1 before anesthesia and 4 after induction of anesthesia at 10 min. intervals). Surgical procedure began 40 min. after induction. The other 8 were hemodiluted 10 min. after induction. The blood volume withdrawn was 12 ml.kg⁻¹ over 30 min. Volume substitution was composed of 4 % albumin. The pre and postanesthesia induction measurements were followed by 2 series of measurements (half hemodilution and half end hemodilution). The operation began after hemodilution.

The results are presented as the mean ± SD and the means are compared using a Student (t) test and an Anova (one way, repeated measures associated with Scheffe F-test). A p value < 0.05 is considered statistically significant. The results are summarised in table 1.

Under general anesthesia normovolemic acute hemodilution was not accompanied by an increase in CO and the VO₂-DO₂ relationships was independent of a non variable level.

Hemodilution induced a sharp drop in DO₂, associated with a decrease in VO₂ and a rise in O₂ Ext. without an increase in lactacidemy. Everything took place as if hemodilution made VO₂ dependent on DO₂ without any metabolic consequences and with a partial compensation by O₂ Ext. increase.

This state posed no problem for the patient but may be destabilised by metabolic factors or by an organic limitation of an increase in cellular O₂ ext. (2).

References

1. Crit Care Med 12 : 540-646, 1983.
2. Chest 13 : 223-229, 1984.

Table 1

		Before Indu.		Ind. + 10 ml		(H) Half hemo		(H) End hemo	
		C	H	C	H	(C) Ind. + 20 min	(C) Ind. + 30 min	(C) Ind. + 30 min	(C) Ind. + 30 min
HR	C	64 ± 06	66 ± 09	65 ± 7.2	66 ± 07				
	H	75 ± 15*	61 ± 09	60 ± 13	59 ± 10				
MAP	C	92 ± 18*	86 ± 10	62 ± 10	63 ± 12				
	H	93 ± 14*	88 ± 11	68 ± 20	74 ± 13				
CI	C	3.1 ± .6*	2.6 ± .6	2.4 ± .6	2.5 ± .6				
	H	3.2 ± .8*	2.5 ± .6	2.0 ± .4	2.4 ± .5				
VO ₂	C	114 ± 27	94 ± 22	120 ± 49	124 ± 20				
	H	127 ± 37*	87 ± 20	81 ± 14	92 ± 18				
DO ₂	C	508 ± 114*	413 ± 116	385 ± 122	400 ± 112				
	H	536 ± 157*	414 ± 113*	310 ± 88	345 ± 108				
O ₂ Ext.	C	.23 ± .03	.23 ± .01	.30 ± .05	.31 ± .05				
	H	.24 ± .07	.22 ± .05	.27 ± .05	.28 ± .06				
Lact. AC	C	2.6 ± 1.0	2.5 ± .6	2.5 ± 0.5	2.4 ± 0.5				
	H	2.9 ± 1.1	3.2 ± 1.1	3.2 ± 1.4	3.2 ± 1.4				

C : control group * * between groups p < .05 (Student t test)
H : hemodilution group * * within groups p < .05 (Anova)

TITLE: HYPOTHERMIA REDUCES THE CAPACITY FOR FIBRINOLYSIS IN HUMAN PLASMA
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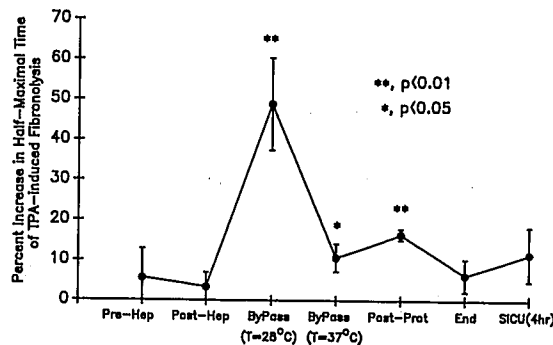
Platelet dysfunction is an unlikely cause of bleeding in clinically relevant hypothermia (1,2). We therefore examined the effect of hypothermia on the capacity for fibrinolysis in humans to determine if a defect in this pathway might explain the development of abnormal bleeding associated with hypothermic patients.

Methods: After obtaining institutional approval and patient consents, citrated blood samples were collected at eight time points during cardiac surgery procedures in 16 patients. Fibrin polymer formation in non-heparinized and heparinized samples was induced by adding atroxin (2.5µg/ml) to cuvettes containing 2ml of plasma. Tissue plasminogen activator (0.2µg/ml) then was added to samples that were continuously monitored spectrophotometrically at 633nm and the time for lysis of 50% of the clot was measured (LT50). The temperature of each sample was maintained throughout this analysis at the same temperature of the patient at the time of sampling.

Results: Hypothermia during extracorporeal reduced the capacity for fibrinolysis in human plasma by 50% (p<0.01, Figure 1). When patients were rewarmed to 37°C, the LT50 was prolonged by only

11% (p<0.05) and after protamine reversal of heparin, it remained prolonged by 16% (p<0.01). At the end of surgery and after 4 hours in the ICU, the capacity for fibrinolysis in patients had returned to normal.

Discussion: These results show that hypothermia inhibits the fibrinolytic capacity of plasmin in human plasma and that this property will not immediately normalize with rewarming. For clinical bleeding to be attributed to increased fibrinolysis in hypothermic patients, circulating levels of plasmin would have to be elevated and/or those of plasmin inhibitors would have to be reduced.



References:

- 1) Ann Surg. 205:175,1987
- 2) Throm. Res. 37:503, 1985