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 anesthetised, 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 hemodilated 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 The blood volume withdrawn was 12 ml.kg-1 over 30 min.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

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Table 1

		Before Indu.	Ind. + 10 min	(H) Half hemo (C) Ind. + 20 min	(H) End hemo (C) Ind. + 30 min
HR b.min^{-1}	C	64 ± 06**	**60 ± 09	65 ± 7. 2	66 ± 07
	H	75 ± 15**	**61 ± 09	60 ± 13	59 ± 10
MAP mm Hg	C	92 ± 18**	**56 ± 10	62 ± 10	63 ± 12
	H	93 ± 14**	**58 ± 11	68 ± 20	74 ± 13
CI l.min^{-1}.m^{-2}	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
VO2 ml.m^{-2}	C	114 ± 27	94 ± 22	120 •• ± 49	124 ± 20
	H	127 ± 37**	**87 ± 20	81 ± 14	92 ± 18
DO2 ml.min^{-1}.m^{-2}	C	508 ± 111**	**513 ± 116	385 ± 122	400 ± 112
	H	536 ± 157**	**414 ± 113**	* 310 ± 88	345 ± 108
O2 Ext.	C	. 23 ± . 03	. 23 ± . 01	. 30 ± . 05	. 31 ± . 05
	H	. 24 ± . 07	. 22 ± . 05	. 27 ± . 05	. 28 ± . 06
Lact. Acid mM	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
H : hemodilution group
•• between groups p < . 05 (Student t test)
** within groups p < . 05 (Anova)

TITLE: **HYPOTHERMIA REDUCES THE CAPACITY FOR FIBRINOLYSIS IN HUMAN PLASMA**

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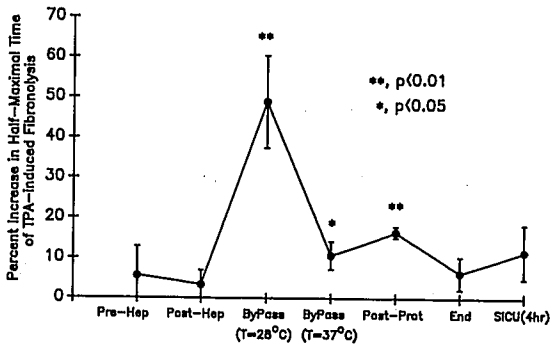
Platelet dysfunction is an unlikely cause of bleeding in clinically relevant hypothermia (1,2). We therefore examined the effect of hypothermia on the capacity for fibrinolysis in humans to determine if a defect in this pathway might explain the development of abnormal bleeding associated with hypothermic 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.



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