HEMODILUTION: HOW LOW A MINIMUM HEMATOCRIT?

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Introduction: While the use of hemodilution in selected patients scheduled for major surgery involving large expected blood loss has grown, determination of an acceptable minimum hematocrit has been largely without experimental justification. We describe here the effects of a decreased red cell mass on oxygen transport and resultant serum lactate levels.

Methods: Approval for hemodilution had previously been given by our Human Research Committee in connection with other work. Six children aged 7 weeks to 9 years were submitted to hemodilution during major surgery after informed consent for the planned procedures and measurements. Each child was anesthetized with halothane in oxygen and given d-tubocurarine sufficient to abolish the twitch response. In 3 patients morphine sulfate 0.7 mg/kg IV was administered at induction and increments of 0.15 mg/kg were given to control increases in peripheral vascular resistance (PVR). Hemodilution was accomplished with 3 for 1 replacement of Lactated Ringer's for each ml of blood removed, and passive cooling to 30-32°C was allowed[1]. Mean blood pressure was lowered to 40-50 torr with increasing concentrations of halothane. Hematocrits of 15-20% after hemodilution dropped further with isovolemic replacement of ongoing blood loss, to 8-17% in our six patients. At the end of surgery, the patients blood was reinfused and Lasix 1 mg/kg IV was given to facilitate diuresis. Ventilation was supported for an average of four hours postoperatively, until the majority of infused crystalloid was excreted. In addition to routine clinical monitoring, measurements included cardiac output, systemic and pulmonary arterial pressures, arterial and venous blood gases, and serum lactates. Oxygen saturations were both measured directly and calculated. CPK isoenzymes were measured in two patients.

The mean and standard deviation of groups of lactate concentrations were calculated and individual values were evaluated for significance at greater than 1.5 standard deviations from the mean.

Results: No patient showed electrocardiographic evidence of ischemia or injury. No postoperative renal dysfunction was demonstrated. Serum lactate was significantly elevated in all patients after initial hemodilution, but returned to pre-infusion levels in 3 of 6 patients within 30 minutes. In three patients lactate remained elevated until infusion of blood and return to normotension. Post-induction cardiac index decreased an average of 25% from pre-induction values.

Oxygen Transport Index (O_2TI) was calculated combining two major factors affecting tissue oxygenation, that is, cardiac output and hemoglobin concentration by the formula O_2TI = Cardiac Index x CaO_2. An O_2TI below 300 ml/min/m^2 was associated with a significant increase in serum lactate, both in individual patients and when comparing peak and minimum values between patients (Table 1). In 2 of 3 patients in whom specific efforts were made to maximize forward flow by maintaining a low PVR, lactate was not significantly elevated above the mean pre-induction level. The third patient (#6) had the lowest PVR but a cardiac index as high as 4.1 was insufficient to prevent a rise in sequential serum lactates. CPK-MB was not elevated in the two patients screened.

Discussion: Earlier work by Bassenge et al in dogs showed a compensatory increase in cardiac output in response to normovolemic hemodilution[2]. Myocardial oxygenation was preserved in the face of a decreasing CaO_2 by increased flow and extraction until extraction capability was exceeded by O_2 requirements and hypoxic failure supervened. The addition of hypothermia and anesthesia enables one to decrease cardiac work while maintaining adequacy of oxygenation.

If one assumes a correlation between cellular hypoxia and the production of lactate, then our data supports the expected correlation between decreased oxygen transport and tissue hypoxia. The coincident high PVR with low $S\bar{v}O_2$ observed in patients 1 and 2 illustrates the basic relationship between flow and extraction. However, in the face of a decreased CaO_2 as a result of hemodilution, adequate cellular oxygenation can still be maintained by facilitation of forward flow with a low PVR. It remains to be demonstrated that regional hypoperfusion does not occur in the presence of mixed venous blood values consistent with adequate oxygenation. However, the lack of rise in CPK-MB isoenzyme, normal postoperative renal function, and preliminary work done with neurobehavioral testing all tend to support the lack of major-organ injury with our technique.

Conclusion: Acute normovolemic hemodilution can be a safe technique for major surgery involving large intraoperative blood loss. Maintenance of a low peripheral vascular resistance is associated with a preservation of oxygen transport and adequate cellular oxygenation in the face of a hematocrit as low as 14%. Lower hematocrits appear to require a compensatory increase in cardiac index.

References:

1. Furman, EB: Prevention of Acute Blood Loss: Hemodilution. ASA Annual Meeting 1978, Refresher Courses, p. 205B.
2. Bassenge, E, et al: Hemodilution: Theoretical Basis and Clinical Application. Basel, Switzerland, Karger, 1975, p. 44.

Table 1

Patient	Peak Lactate	Min. O_2TI	Peak PVR	Min. C.I.	Min. $S\bar{v}O_2$	Min. Hct.	Anesthetic
1	10.9*	59	7550	1.0	33%	08	Halo
2	9.6*	77	5930	1.3	54%	09	Halo
3	2.0*	129	3151	1.5	87%	17	Halo
4	1.7	310	1900	2.9	77%	17	Halo/MS
5	1.2	300	1702	3.9	73%	14	Halo/MS
6	3.3*	199	1042	2.5	70%	11	Halo/MS

* - greater than 1.5 SD from mean pre-induction value