Hemodynamic consequences of hemodilution

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The hemodynamic alterations following hemodilution for extracorporeal circulation are almost entirely the result of changes in blood viscosity. Although these changes unequivocally improve quality of perfusion, they also pose special problems after perfusion. For present purposes hemodilution describes acute normovolemic anemia at a hematocrit not less than 20%.

The well known relationship between mean arterial blood pressure, systemic vascular resistance and cardiac output (MAP = SVR × CO) assumes blood viscosity to remain constant. The true relationship is expressed as MAP = viscosity × vascular resistance × cardiac output. Among the major determinants of blood viscosity are hematocrit and temperature. Unfortunately, simple quantitative relationships cannot be drawn between these since blood viscosity is highly dependent on blood flow velocity (shear rate) which rises with vessel size. The smaller the vessel, the slower the blood velocity and the higher the viscosity. A reasonable approximation of the direct relationship between viscosity and hematocrit is that they change proportionately. A 50% decrease in hematocrit is associated with approximately 50% decrease in viscosity.

On the other hand, viscosity is inversely related to tempertature and the proportionality is not so uniform. As an approximation, a 10-C decrease in temperature increases blood viscosity by 20% to 25%.

Among the clinical implications of these physical phenomena, one particularly applies to induced hypothermia. To maintain viscosity constant during induced hypothermia to 20 C, hematocrit would have to be reduced from 45% to approximately 25%. Failure to hemodilute leads to increased resistance to flow by increased viscosity with an approximate 30% decrease in tissue blood flow at the same arterial pressure and vascular resistance. The decrease in flow is most marked in small vessels (microcirculation) and would be even more severely reduced if the patient were also polycythemic. Under the same circumstances at normothermia, a reduction of hematocrit to 25% almost doubles tissue blood flow by virtue of decreased viscosity. During clinical perfusion with hemodilution, none of these determinants of flow remains constant. Temperature tends to fall, vascular resistance tends to increase and hematocrit tends to rise, all leading to increased resistance to flow. Deliberate hemodilution by decreasing viscosity tends to correct for these changes and improve tissue perfusion.

From this consideration of viscosity, it should be apparent that an arbitrary perfusion pressure cannot serve as an important criterion of perfusion adequacy. With constant flow and vascular resistance, perfusion pressure varies directly with viscosity. It is therefore possible, with a sufficiently low hematocrit and normothermia, for pressure to remain between 20–30 torr during perfusion at completely acceptable flow rates. This is commonly observed at the onset of perfusion when using entirely cystalloid prime and before complete mixing with blood has occurred. A requirement for an arbitrary perfusion pressure ignores the physical role of viscosity and leads to needless administration of blood or drugs to increase pressure for unphysiologic reasons. Reports of brain damage at perfusion pressures below arbitrary levels ignore the role of viscosity in arriving at these pressures.

A final clinical consideration applies to the period immediately after cardiopulmonary bypass and the ability of a diseased heart to function in the presence of normovolemic or even hypervolemic anemia. A number of ingenious experiments clearly demonstrated that the increase in cardiac output associated with normovolemic anemia is not a compensatory response to inadequate oxygen delivery to tissues. The increased cardiac output is also a consequence of decreased viscosity and reflects the increased venous return which increases proportionately to the decrease in hematocrit. In man, deliberate hemodilution to 20% hematocrit increases cardiac output only about 50% with no increase in heart rate or A-V O_2 difference, with no adrenergic stimulation, and with little increase in myocardial work or myocardial oxygen uptake. At this hematocrit, the relative distribution of blood flow to the major organs remains unchanged from normal. The hemodynamic response to normovolemic anemia is largely passive and accountable almost entirely on the basis of decreased viscosity.

When hematocrit is reduced to less than 20%, hemodynamic changes are no longer entirely passive. Data available suggest that normovolemic anemia at hematocrit of 10% to 15% is associated with relative ischemia of the subendocardium and renal cortex, with adrenergic stimulation and an increase in heart rate.

In the absence of stresses which require increased myocardial work, anemia represented by a hematocrit greater than 20% is well tolerated even by those with severe coronary or valvular heart disease, since myocardial oxygen demand is little changed. However, in the presence of stresses, such as hemorrhage, fever, hypotension or myocardial infarction, cardiac reserve is clearly diminished. The degree to which cardiac output and oxygen extraction can increase to supply tissue oxygen in the presence of low flow or increased demand is unequivocally compromised. Thus hazards from hemodilution exist when reserve circulatory functions are required to compensate for imposed abnormal stresses.