Hemodilution Decreases Critical Closing Pressure But Does Not Increase The Aortic To Distal Pressure Gradient In Dogs

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Citation

J Urzúa, G Núñez, G Meneses, G Lema, R Canessa, C Sacco, J Medel, C Sáez. *Hemodilution Decreases Critical Closing Pressure But Does Not Increase The Aortic To Distal Pressure Gradient In Dogs*. The Internet Journal of Anesthesiology. 1998 Volume 3 Number 3.

Abstract

This work was financed by grant 1940584 from the National Research Fund (FONDECYT)

INTRODUCTION

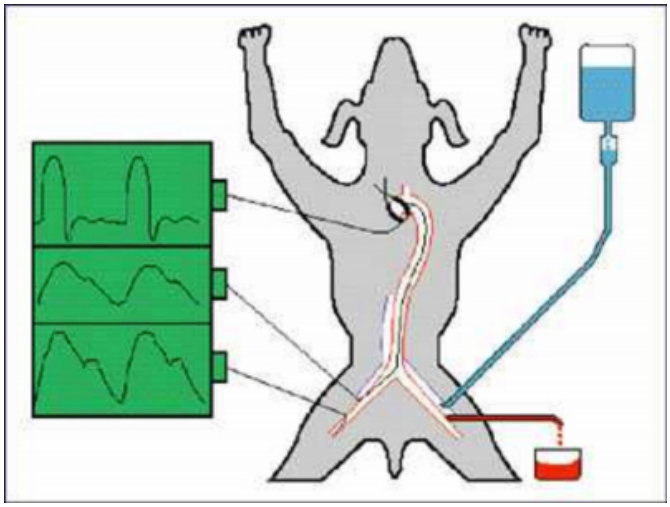
Hemodilution is used routinely during cardiac surgery and CPB. Its clinical and hemodynamic effects are well documented ($_1$); however, its effect on the arterial zero-flow («critical closing») pressure has not been established. Further, it has been proposed ($_2$) that hemodilution may be responsible for the aortic-to-distal arterial pressure gradient often seen after cardiopulmonary bypass. Aortic and radial arterial pressure waveforms have different waveforms. Normally, systolic pressure is higher in the radial artery than in the aorta; however, mean and diastolic pressures are similar. Resistance changes produced by vasodilators, by occlusion of the radial artery, or by thermoregulation, alter the aortic-to-distal pressure difference. Hemodilution decreases blood viscosity and thus resistance; therefore, the aortic-to-distal pressure difference may be altered as well.

METHODS

Animal handling and upkeep complied with institutional norms. Eleven mongrel dogs weighting 16.6±4.4 Kg were anesthetized with 35 mg/Kg of pentobarbital and 2 mcg/Kg of sufentanyl and mechanically ventilated with oxygen and air. Arterial blood gases and tympanic temperature were controlled. Millar catheter-tipped pressure transducers were located in the ascending aorta and in a distal femoral artery. A snare and an electromagnetic flowmeter were placed around the ascending aorta.

Figure 1

Figure 1: Schematically depicts the experimental model



Zero-flow pressure was measured by extrapolation of the arterial pressure decay obtained during a 4-second proximal aortic occlusion. Hemodilution was achieved removing 30 ml/Kg of blood over 5 to 10 minutes and replacing it with warm 0.9% NaCl as required to maintain constant systolic aortic pressure. Hematocrit decreased from 39 ± 11.2 % to 25.6 ± 4.95 %. The effects of 1 mcg/Kg phenyleprine and 4 mcg/Kg nitroprusside were recorded both before and after hemodilution. Effective downstream pressure was measured by extrapolation of the exponential pressure decay during occlusion of the aortic snare, before and after dilution. Statistics. Each dog was its own control. Paired student's «t» test and repeated- measurements analysis of variance were used. Significance was P=0.05. Results are mean ± SD.

RESULTS

Hemodilution decreased zero-flow pressure, from 44±9 to 36±7mmHg ($p<0.05$) in the aorta, and from 51±2 to 37±3.1mmHg ($p<0.05$) in the distal femoral artery. As expected, hemodilution increased cardiac output from 78±25 to 142±57 ml/min/Kg ($p<0.05$) and decreased vascular resistance from 1.3±0.5 to 0.8±0.4 {mmHg/ml/min}Kg, ($p<0.05$). Diastolic aortic pressure decreased from 86±17 to 79±15mmHg ($p<0.05$), but systolic and mean pressures were unchanged (from 108±20 to 104±21mmHg and from 97±20 to 93±17mmHg, respectively). Peak systolic femoral pressure was 13.5±7.2mmHg higher than aortic before, and 17.5± 8.7mmHg after, hemodilution ($p>0.1$). Mean arterial pressure in the aorta was not significantly different from the distal femoral artery, both before (97±20 vs. 95±19mmHg, respectively) and after hemodilution (93±17 vs. 91±17mmHg, respectively).

Figure 2

Figure 2: If blood flow is briefly stopped, the arterial blood pressure descends monoexponentially towards a value much higher than venous pressure (heavy interrupted line). This value is termed Â“effective downstream pressureÂ”. In the circulation remains stopped, then the pressure descends linearly towards a lower value termed zero-flow pressure (thin interrupted line) that is still higher than the venous pressure.PVC

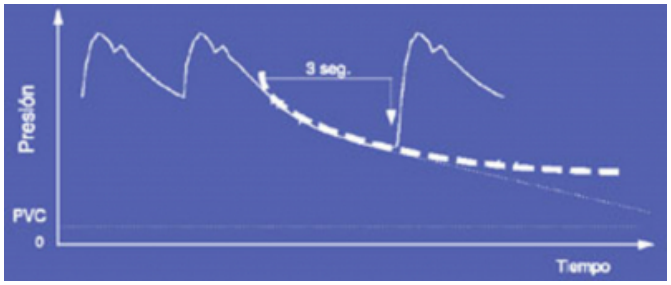


Figure 3

Figure 3: Depicts the effect of hemodilution on systolic, mean, diastolic and zero-flow arterial pressures. Systolic and mean pressures were unaffected but diastolic and zero-flow pressures significantly decreased

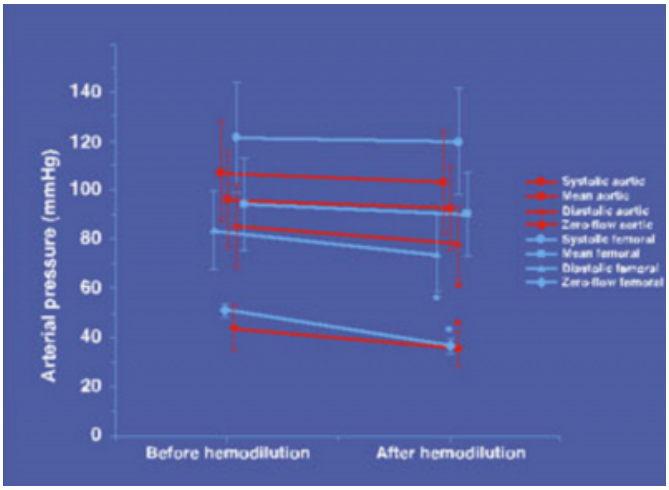


Figure 4

Figure 4: Depicts the relationship between cardiac output and mean arterial pressure before (red) and after hemodilution (yellow). Some zero-flow points are displaced towards negative flow values for illustration purposes, obviously there are no negative flows. Phenylephrine caused a clockwise shift and nitroprusside a counterclockwise shift, both before and after hemodilution

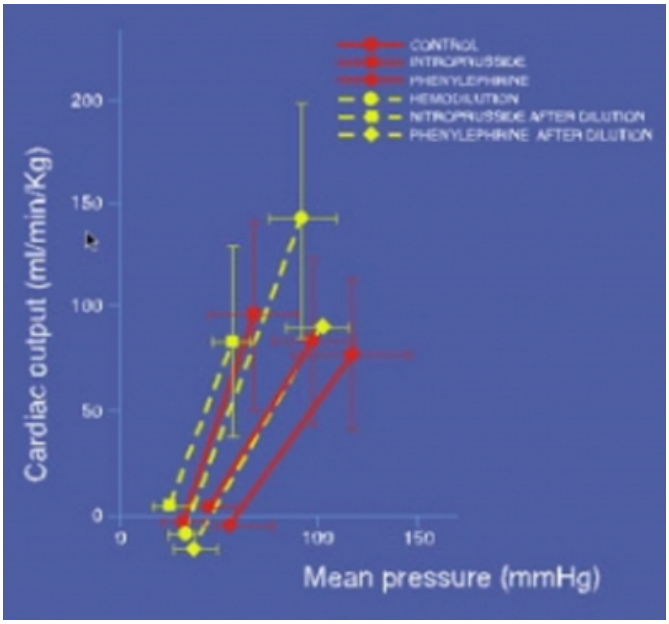


Figure 5

Figure 5: Against our hypothesis, hemodilution failed to alter the aortic (left) and femoral (right) arterial pressure waveforms

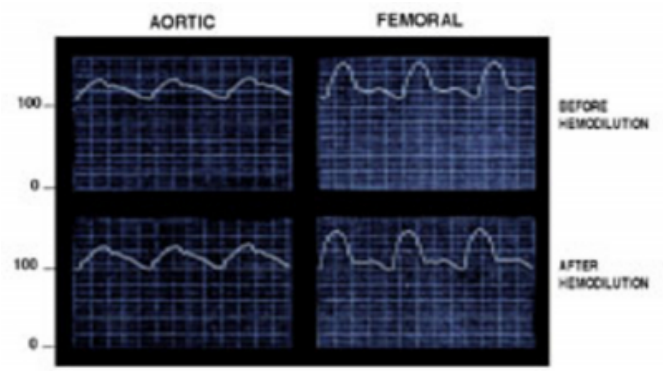


Figure 6

Table 1:

	Control	Phenylephrine	Control	Nipride	Control	Hemodilution	Control	Phenylephrine	Control	Nipride
Femoral systolic pressure mmHg	122.7	145.3*	119.3	85.1*	121.9	121.1	105.7	128.2*	110.2	71.2*
SD	24.3	27.4	25.9	26.4	22.7	21.8	13.8	14.0	15.8	11.7
Aortic systolic pressure mmHg	107.1	127.7*	105.0	77.4*	108.4	103.6	91.9	112.8*	94.5	68.5*
SD	23.9	28.6	23.4	20.2	20.2	20.9	11.0	11.9	13.9	12.8
Femoral less aortic peak systolic pressure difference mmHg	15.6	17.5	14.3	7.7**	13.5	15.9	12.5	14.0	14.3	2.5*
SD	6.7	6.6	6.3	16.3	7.2	8.7	8.2	9.6	8.8	10.8

* $p < 0.05$ ** $p > 0.05$

Figure 7

Table 2:

	Control	Phenylephrine	Control	Nipride	Control	Hemodilution	Control	Phenylephrine	Control	Nipride
FLOW ml/min/Kg	86.5	75.5	81.6	95.3	78.1	142.3*	95.0	88.7	75.7	83.0
SD	43.8	36.2	32.4	40.8	24.7	57.1	42.7	36.1	30.8	46.3
SVR resistance units	1.4	1.9*	1.3	0.8*	1.3	0.8*	1.1	1.4*	1.4	0.9*
SD	0.7	1.1	0.6	0.4	0.5	0.4	0.8	1.0	1.0	0.8
HEART RATE	119.2	112.3	123.4	135.8	123.0	138.0	147.6	136.6	156.1	164.7
SD	24.6	21.3	23.6	16.2	25.0	16.8	18.5	15.1	20.1	21.3

* $p < 0.05$

DISCUSSION

Hemodilution decreased significantly the zero-flow pressure. Causes underlying zero-flow pressure are not completely known. It is generally assumed that it results from microvascular collapse, due to vasoconstriction or higher surrounding pressure. However, the fact that hemodilution decreased zero-flow pressure suggests that physical characteristics of the blood, such as viscosity, are at least in part responsible. Contrary to our hypothesis, acute normovolemic hemodilution had no significant effect on the aortic-to-distal arterial pressure difference.

Unlike resistance decreases due to vasodilators or thermoregulation, resistance decrease due to hemodilution did not alter the normal difference between aortic and femoral systolic pressure. Similarly, the pressure waveforms were not affected. This was against our hypothesis and we have no firm explanation for this negative result.

The arterial pressure waveform is a very complex phenomenon. Differences in shape and peak systolic value between aortic and distal pressures depend on many factors, related to ventricular ejection and to the physics of the arterial tree. Ventricular factors include contractility, dP/dt and Vmax, pre- and afterload, pressure, stroke volume and velocity of ejection. Arterial factors include wall tension, wave propagation, characteristic frequencies, wall compliances, inertial effects, peripheral resistance, and pressure itself. We hypothesized that blood viscosity was one of the variables involved; unfortunately this was not the case.

The meaning of the arterial “critical closing pressure” is debatable. It has been attributed to dynamic compliance effects, to vascular collapse, to perivascular pressure, and to the particular rheological characteristics of blood. The fact that it decreased when viscosity decreased with hemodilution may suggest that rheological factors are possibly important.

References

1. J Thorac Cardiovasc Surg 87:175,1984;
2. ANESTHESIOLOGY 73:191,1990.

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