Title: SYSTEMIC VASCULAR RESISTANCE NEEDS CORRECTION FOR HEMATOCRIT AND TEMPERATURE VALUES

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Introduction: Systemic vascular resistance (SVR) is a critical determinant of intraoperative hemodynamic changes during coronary artery bypass graft (CABG) surgery.[1] SVR consistes of a vascular componet and a viscosity componet. Interpretation of SVR can be misleading if proper correction is not made for blood viscosity changes where marked changes of hematocrit (Hct) and temperature occur. We defined the vascular component of SVR as the Corrected Systemic Vascular Resistance (C-SVR), using the Vand viscosity-hematocrit relation[2] and relation of temperature blood viscosity. The purpose of this study was to determine whether the C-SVR is a clinically valid index of changes in vascular caliber.

Methods: Sixty-three patients undergoing CABG surgery were retrospectively studied with institutional approval. Anesthesia consisted of diazepam, fentanyl, and pancuronium. Cardiopulmonary bypass (CPB) was initiated with a clear prime of 2 liter crystalloid under moderate systemic hypothermia (25°C). Multiple hemodynamic variables were determined at pre-induction (C), post-intubation (S1), post-sternotomy (S2), post-cannulation (S3), during aortic cross clamp (S4), immediate post-pump (S5), post-protamine (S6), the end of operation (S7) and arrival to ICU (S8). The C-SVR is defined by the following equation: The SVR may be separated into a viscosity factor (μ) and C-SVR which characterizes vascular caliber: C-SVR=μ ($1/\mu$)xSVR. The viscosity may in turn be expressed in terms of the hematocrit H and plasma viscosity μp, by means of the Vand equation: $\mu=\mu p(1+0.025H+0.000735H^2)$. As an approximation, blood viscosity increases by 5% for each 1°C decrease in temperature (t°C) below 37°C. The Vand equation may be modified: $\mu=\mu p \times 1.05^{(37-t)} \times (1 + 0.025H + 0.000735H^2)$. Thus, μp is constant if protein is constant. Then, if the normal value for blood viscosity at 45% Hct value and 37°C temperature is taken as 3.8 cp, $1/\mu p=0.95$. The C-SVR becomes: $C\text{-}SVR=0.95 \times 1.05^{(t-37)} \times SVR/(1+0,025H+0.000735H^2)$. Paired t-test were performed. All data are mean ±SEM.

Results: The changes of SVR and C-SVR were expressed as % of control (C) (Fig. 1). Following induction, SVR decreased while C-SVR remained unchanged. Hct and temperature decreased to 37±4% and 35.5±0.2°C from 40±0.4% and 36.2±0.1°C, respectively. At (S2), both SVR and C-SVR and C-SVR increased while Hct values were unchanged (37±0.5%) and temperature decreased (35.2±0.1°C). During pump both SVR and C-SVR were greater than those of control, although C-SVR did not increase significantly. Hct and temperature reached the lowest value (23±0.1%, and 25.2±0.3°C). At the postbypass period (S5, S6), SVR decreased significantly from on pump values at S4 and was lower than control, while C-SVR remained unaltered and did not differ from the contol. Temperature rose to 36.3±0.1°C and Hct was 26±0.4% at S5. At the end of operations (S7) and arrival to ICU (S8), C-SVR was greater than the control, while SVR values were not significantly different from the control. During this period, Hct increased gradually to 29±0.6% and temperature decreased to 34.3°±0.1°C at (S8).

Discussion: The importance of SVR has been emphasized for the treatment of hypotension or hypertension during CABG surgery. But SVR is usually used synonymous to total peripheral resistance which consists of both a vascular component and a viscosity component. The wide variations in Hct and temperature cause enormous effects on the rheological properties of blood. When SVR is reduced due to the viscosity change, a reduction of SVR should not be interpreted as an index of peripheral vasodilation. Indeed, in our patients at the immediate postbypass period, SVR was lower than control value yet C-SVR was within normal range, suggesting the presence of normal vascular caliber rather than vasodilatation. At the end of operation, SVR did not differ from the control whereas C-SVR was higher than control, suggesting the presence of peripheral vasoconstriction rather than normal vasculature. Thus, misinterpretation of SVR may lead to the mismanagement of the patients.

References:

1. Estafanous HG, et al: Pattern of hemodynamic alterations during coronary artery operations. J Thorac Cardiovasc Surg 87:175, 1984
2. Merrill EW: Rheology of Blood. Physiol Rev 49:863, 1969

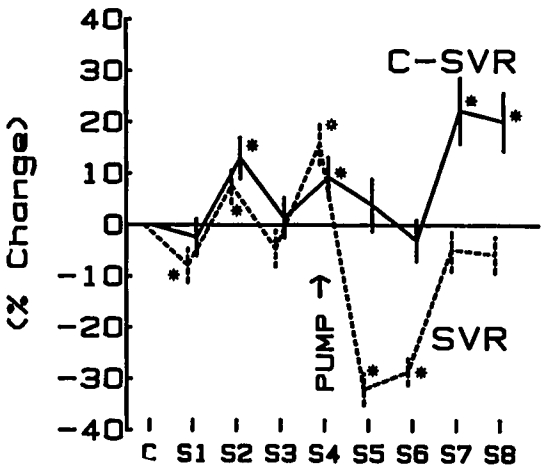

Fig. 1 Changes of SVR and C-SVR expressed as % of control value. Mean ±SEM. *P<0.05 vs (C).