

TITLE: EFFECT OF PROSTAGLANDIN E₁ (PGE₁) ON SKIN BLOOD FLOW AND THERMOREGULATION DURING ENFLURANE ANESTHESIA

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Suppression of thermoregulatory mechanisms during anesthesia sometimes causes post-anesthetic shivering. To examine the effects of PGE₁ on thermoregulation, we measured cardiac output (CO), skin blood flow (SBF) and core-surface temperature gradients during enflurane anesthesia in man.

After institutional approval and written informed consent, thirty-nine ASA physical status I or 2 patients, scheduled for elective surgeries, were studied. Anesthesia was induced with thiamylal and maintained with 67% N₂O in oxygen and enflurane (0.5-2.0%). PGE₁ was administered to thirty-one patients to control hypertension and to produce controlled hypotension (group P), and not to eight patients (group C). SBF was measured by laser Doppler flowmetry (ALF 2100, ADVANCE)¹. The probe was positioned at the palmar side of the thumb base. CO was measured continuously by ultrasound Doppler cardiac output monitor (L3000, LAWRENCE)². The probe was placed in the esophagus after induction of anesthesia. Rectal and palmar skin surface temperatures were also monitored to calculate temperature gradients. We intended to define the relationship between temperature gradients and post-anesthetic shivering observed.

The two groups were similar in age, height,

weight, duration of anesthesia and volume of infusion. Continuous infusion of PGE₁ (0.05-0.3 µg·kg⁻¹·min⁻¹) reduced mean arterial pressure to about 80% of the pre-treatment values. There was no significant difference in heart rate. CO increased significantly to 5.9 ± 1.4 l/min from 4.1 ± 1.1 l/min. SBF also increased to 27.2 ± 12.2 ml/min/100g tissue from 15.9 ± 9.2 ml/min/100g tissue. After discontinuation of infusion CO decreased to the pre-treatment value in 15 minutes and SBF decreased significantly to 9.5 ± 7.2 ml/min/100g tissue at the end of anesthesia. In group P, temperature gradients were decreased from 1.4 ± 1.4 to 1.0 ± 0.8 ° during infusion, but gradually increased after discontinuation of infusion to be 2.0 ± 1.5 ° at the end of anesthesia. In group C, SBF continued to decrease during anesthesia and fell down to 5.1 ± 3.4 ml/min/100g tissue at the end of anesthesia. Temperature gradients increased to 4.3 ± 3.0 ° at the end of anesthesia which were significantly greater than those in group P. In group C, post-anesthetic shivering was observed in 3/8 patients, significantly higher compared to group P (2/31 patients).

Continuous infusion of PGE₁ increased SBF by 71%. This value is larger than that of CO which was increased by 44%. Although CO decreased to be control value after discontinuation of infusion, SBF in group P was kept higher than that in group C and temperature gradients were smaller in group P compared to group C. We speculated that well-maintained skin circulation produced by PGE₁ infusion might inhibit the increment of temperature gradients and diminish the frequency of post-anesthetic shivering.

References

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TITLE: DO RADIAL ARTERY PRESSURE WAVEFORMS REFLECT MYOCARDIAL PERFORMANCE?

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INTRODUCTION: Some clinicians equate a rapid upstroke of the radial blood pressure (BP) with good myocardial contractility, pulse pressure (PP) with stroke volume, and a steep downstroke with low peripheral vascular resistance (PVR).¹ However, radial systolic arterial pressure (SAP) can be 40 mmHg higher than the aortic in man² and after cardiopulmonary bypass (CPB), wrist compression distal to the cannulated radial artery increases radial SAP³ without aortic SAP changes.

METHODS: Twenty-two IRB approved, consenting patients undergoing CPB had their radial artery BP measured directly. The measuring system's natural frequency and damping coefficient were determined. After CPB, radial BP was measured with and without compression. Simultaneously, central BP (measured from the aorta or femoral artery), CVP, heart rate (HR) and cardiac output (CO) were obtained. Unchanged HR, central BP, and CVP were required for valid comparisons. dP/dt, rate of pressure decline (-dP/dt) of the radial BP, PVR, and SVR were obtained. Stroke volume equated CO/HR. Changes were evaluated by the Wilcoxon signed-rank test; appropriated correlations were sought.

RESULTS: Radial SAP, DAP, mean arterial pressure, PP, dP/dt increased significantly (P<0.001) in all

patients. PVR and -dP/dt increased in 9. SVR and stroke volume did not change. Neither the dP/dt nor the PP correlated with stroke volume. PVR and -dP/dt changed in the same direction. The figure illustrates the changes.

DISCUSSION: Failure of dP/dt and PP to correlate with stroke volume confirms previous findings,⁴ in which the PP at the root of the aorta did not correlate with stroke volume, but impedance spectra analysis indicated peripheral wave reflection. We conclude that radial artery dP/dt and PP are not valid monitors of cardiac function and that the steepness of arterial pressure downstroke might indicate increases in PVR.

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