No Decrease in Central Venous Pressure When Epinephrine-Induced Hypotension Occurs

Jian-jun Yang, Xiao-li Xu, Jin-chun Shen, Wei-yan Li and Zhi-qiang Zhou

Department of Anesthesiology, Jinling Hospital, School of Medicine, Nanjing University, Nanjing, P.R. China.

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Previously (1, 2) we have shown that local infiltration with epinephrine-containing solutions on nasal field or scalp may produce hypotension because of the possible activation of β_2 receptors. However, whether central venous pressure (CVP) will change when epinephrine-induced hypotension occurs remains unknown. We hypothesized that CVP should also decrease when hypotension happened.

The present study was approved by the Ethics Committee of Jinling Hospital and conducted according to the Helsinki Declaration, and the prior written informed consents were obtained from all patients. Twenty patients without premedication (male/female: 13/7, age: 36 ± 12 years, weight: 65 ± 10 kg, American Society of Anesthesiologists physical status: I or II) undergoing elective endoscopic sinus surgery with general anesthesia were included. After anesthesia induction with midazolam 0.03 mg/kg, rocuronium 0.8 mg/kg, and target controlled infusion with propofol 4 μg/ml and remifentanil 5 ng/ml, anesthesia was maintained with propofol 3.5-4 μg/ml and remifentanil 3.5-4 ng/ml by target controlled infusion. All the patients were mechanically ventilated to keep the end tidal carbon-dioxide tension around 35 mmHg. About 20 min after the mechanical ventilation, the surgeons decongested the nose with four cotton swabs containing oxymetazoline and lidocaine for 3 min. Thereafter, they locally infiltrated epinephrine 20 μg with normal saline 4 ml on nasal septum, middle nasal meatus, and inferior nasal concha before surgery and all the infiltration was accomplished within 25-30 s. The previous studies have shown that the lowest and the highest arterial blood usually occur at 1.5 and 3 min after local infiltration, respectively[1,2]. Therefore, mean arterial pressure (MAP), heart rate (HR), cardiac output (CO), and systemic vascular resistance (SVR) monitored by impedance cardiography and CVP monitoring via central venous catheter were recorded at 4 time points: before local infiltration (baseline) and 1.5, 3, and 4.5 min after the beginning of local infiltration.

Statistical analysis was performed by statistics package for social science of 13.0-version. Comparisons were made by analysis of variance for repeated measurements followed by the least significant difference test for multiple comparisons. Difference was considered to be significant at $P < 0.05$. Compared with the baseline, MAP and SVR decreased and HR and CO increased significantly ($P < 0.01$) at 1.5 min, then recovered at 3 and 4.5 min. But CVP did not change significantly during this period. (Table 1). We presumed that the decrease in MAP was due to the decrease in SVR (1-5) induced by the activation of β_2 receptors (1, 3); the increase in CO was due to the activation of β_1 receptors[1] and the reflex to the decrease in SVR, and the increase in HR was due to the activation of β_1 receptors and the baroreflex to the decrease in MAP (1, 2, 4, 5).

Corresponding Author: Jian-jun Yang, Department of Anesthesiology, Jinling Hospital, School of Medicine, Nanjing University, Nanjing, P.R. China. yjyangjj@126.com

Table. 1 Changes in MAP, HR, CO, SVR, and CVP after the beginning of local infiltration (n = 20)

	Baseline	1.5 min	3 min	4.5 min
MAP (mmHg)	77 ± 8	59 ± 11*	78 ± 10	75 ± 8
HR (beat/min)	62 ± 6	77 ± 10*	63 ± 7	65 ± 7
CO (l/min)	4.6 ± 0.5	5.3 ± 0.8*	4.9 ± 0.6	4.8 ± 0.6
SVR ($dyn \cdot s \cdot cm^{-5}$)	1225 ± 203	805 ±269*	1188 ± 216	1168 ± 197
CVP (cmH_2O)	7.3 ± 1.3	7.0 ± 1.5	7.3 ± 1.5	7.2± 1.4

Values are mean ± SD. MAP, HR, CO, SVR, and CVP are recorded at 4 time points; *, significant drop from baseline. $^{**}P < 0.01$, compared with the baseline.

In the present study, no decrease in CVP was observed. This is a good explanation for that the fluid expansion is not a good measure to prevent epinephrine-induced hypotension (4), which remains unknown in our previous study (5).

The decreased SVR indicated that systemic arteries and arterioles, as resistance vessels, dilated significantly after epinephrine infiltration on nasal field; however, the stable CVP implied that systemic venules and veins, as capacitance vessels, dilated indistinctively. Further studies are needed to explore the underlying mechanisms for the differences of epinephrine on resistance and capacitance vessels.

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