



Original contribution

Comparing the effect of arginine vasopressin on ear and finger photoplethysmography

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Received 3 July 2006; revised 7 June 2007; accepted 5 September 2007

Keywords:

Arginine vasopressin;
Noninvasive monitoring;
Plethysmograph;
Waveform analysis

Abstract

Study Objective: To test whether the relative insensitivity of craniofacial vessels to catecholamines differs in response to arginine vasopressin.

Design: Prospective, observational human study.

Setting: University hospital.

Patients: 8 ASA physical status I and II women scheduled for elective myomectomy.

Interventions: Patients underwent elective myomectomy surgery with intrauterine injection of arginine vasopressin.

Measurements: Finger, ear, and forehead photoplethysmographs were monitored. Changes in the plethysmographic amplitudes were recorded before and after arginine vasopressin injection.

Main Results: In all subjects, ear photoplethysmographic amplitude (but not oxygen saturation) decreased precipitously ($62\% \pm 10\%$; $P < 0.001$) after arginine vasopressin injection. In contrast, there was no significant decline in the finger signal ($4.5\% \pm 27\%$; $P = 0.19$). The forehead plethysmograph decreased in amplitude, but this finding did not achieve significance ($33\% \pm 18\%$; $P = 0.18$).

Conclusion: In contrast to prior observations during adrenergic activation, arginine vasopressin induced relatively greater vasoconstriction at the ear and forehead than at the finger. This finding has potential implications with respect to arginine vasopressin's effect on blood flow and indicates that monitoring the ear plethysmographic signal may provide useful information during arginine vasopressin administration.

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1. Introduction

Arginine vasopressin (AVP), also known as antidiuretic hormone, is an endogenous peptide secreted by the

hypothalamus and released from the posterior pituitary. In recent years, the synthetic form of AVP has been gaining acceptance in a number of clinical settings, including septic shock, and cardiopulmonary resuscitation [1-3]. In contrast to those of catecholamines, AVP's vasoconstrictive actions are mediated via specific AVP receptors (V1). It thus may be preferable in selected settings to induce regional and/or systemic vasoconstriction. Sympathetic activation such as cold pressor testing and pharmacological agents (ie,

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phenylephrine) cause disproportionate vasoconstriction of an adrenergically rich region such as the finger, as opposed to the ear or forehead [4-6].

To date, only a limited number of studies in human subjects have addressed the effect of AVP on different vascular beds. Arginine vasopressin is known to have distinct responses in different organs and systems. This fact is, in part, related to the distribution of its receptors throughout the body. There is evidence that isolated human cerebral arteries constrict when exposed to varying concentrations of AVP [7]. In addition, patients who have had a recent ischemic stroke show high plasma levels of endogenous AVP when compared with controls [8]. Because an increasing number of cardiac arrest victims receive AVP as part of resuscitation algorithms, it becomes important to develop ways to detect physiological changes related to AVP administration.

Arginine vasopressin has been used extensively in gynecology practice to control bleeding [9,10]. Some gynecologists inject AVP directly into fibroid tumors (or myomas) intraoperatively so as to minimize blood loss. This practice becomes an opportunity to study the effects of AVP on plethysmographic signals from various body sites of anesthetized patients. We hypothesized that AVP would elicit not only a vasoconstrictive response but also substantial changes in the plethysmographic waveform over different, more central body sites as compared with a stress response or to the administration of catecholamines (more peripheral).

2. Materials and methods

With approval of the Yale–New Haven Hospital Human Investigations Committee, we enrolled in the study 8 ASA physical status I and II female patients who were scheduled for elective myomectomy. None of the study patients had a history of neurological disease, congestive heart failure, or valvular heart disease. Clinical monitoring included standard oscillometric blood pressure (BP), 5-lead electrocardiography, pulse oximetry, airway pressure, and end-tidal carbon dioxide (ETCO₂). In addition, infrared reflectance photoplethysmographs¹ (model MLT1020; ADInstruments, Colorado Springs, CO) were applied to a finger, an ear, and the forehead, then interfaced to a quad bridge amplifier (ML112, ADI), which was, in turn, interfaced to a computer with a customized acquisition program (Chart, ADInstruments).

¹ It should be noted that the use of this setup in a subsequent study resulted in a burn on the forehead of a research subject. It occurred with the probe attached with a transparent dressing (Tegaderm; 3M, St. Paul, MN) without application of external pressure. The mechanism of injury appeared to be due to a low-voltage current leak from the probe, as described in Leeming MN, Jacobs RG, Howland WS. Low voltage, direct current plethysmograph burns. *Med Res Eng* 1971;10:19-21. At this time, use of the ADInstruments IR plethysmographic transducer cannot be recommended.

All patients received intravenous (IV) midazolam 1 to 3 mg for premedication. Anesthesia consisted of an IV induction (propofol 2.0 to 3.0 mg/kg IV), and tracheal intubation was facilitated with vecuronium 0.1 mg/kg. Sevoflurane one to three percent, in combination with nitrous oxide 60% in oxygen, was administered for maintenance of anesthesia. Additional medications (fentanyl, morphine, and ondansetron) were given at the discretion of the anesthesia care team. An upper body warming unit (Bair Hugger Model 505; Augustine Medical Inc, Eden Prairie, MN) with plastic face sheet was applied at the start of the procedure and used throughout the case.

Before intraoperative intrauterine injection of AVP (1-17 units, depending on the surgeon's preference), two minutes' worth of predrug data were collected. Immediately after injection, two minutes' worth of postdrug data were collected. The waveforms at each site were analyzed with respect to beat-to-beat measurement of amplitude of the plethysmographic waveform (Tech Note 20B; Igor Pro, Wave Metrics, Inc, Lake Oswego, OR). The amplitudes were compared with paired student *t* tests; $P < 0.05$ was considered to be significant; results are reported as means \pm SD.

3. Results

In all 8 subjects, ear blood flow (ear photoplethysmographic amplitude) decreased precipitously, with an overall decline of $62\% \pm 10\%$ ($P < 0.001$). These data are summarized for all subjects (Fig. 1) and also detailed in a

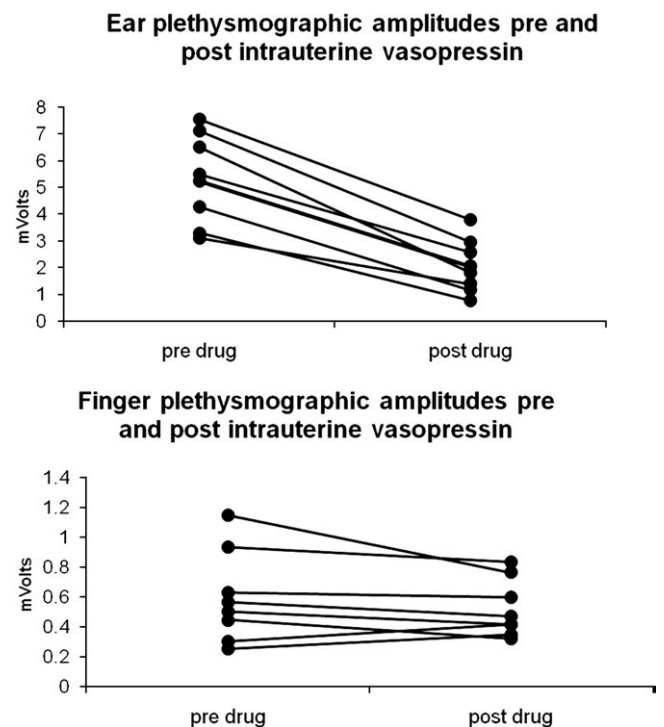


Fig. 1 Effect of vasopressin injection on the amplitude of ear (upper view) and finger (lower view) plethysmography.

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