Title: ATP-INDUCED HYPOTENSIVE ANESTHESIA DURING SURGERY

Authors: A. F. Fukunaga, M.D., Ph.D., K. Ikeda, M.D., Ph.D., and I. Matsuda, M.D., Ph.D.

Affiliation: Department of Anesthesiology, Harbor-UCLA Medical Center, 1000 W. Carson St., Torrance, CA. 90509, and Department of Anesthesiology, Hamamatsu University School of Medicine, Hamamatsu, Japan.

INTRODUCTION. Although deliberate hypotension with adenosine triphosphate (ATP) has long been employed during clinical anesthesia in Japan, the detailed pharmacological and circulatory effects have not been well documented. Thus, the present study was undertaken to supply such information obtained during anesthesia in man.

METHODS. Twenty-seven patients for whom hypotensive anesthesia with ATP was chosen as part of their anesthetic management were studied with their informed consent and institutional approval. Premedication consisted of demerol (50mg) and atropine (0.5mg). Anesthesia was induced with thiopental, followed by succinylcholine for intubation. The maintenance anesthesia consisted of halothane (1% inspired)-N_2O(60%) in oxygen, and the lungs were mechanically ventilated. Arterial pressure was measured from indwelling radial artery canula. In order to determine clinical doses of ATP necessary to reduce blood pressure by 40%, a 10% solution was administered by a calibrated infusion pump into an IV line independent from other drugs. In all subjects, mean arterial pressure (MAP), heart rate (HR) and EKG were recorded, and in 4 subjects, a Swan-Ganz thermodilution catheter was inserted and pulmonary hemodynamic variables were measured. Arterial blood samples were taken for blood gas analysis every 30 minutes.

RESULTS. Hemodynamic and blood gas data are summarized in Figs 1-3 and Table 1. IV infusion of ATP in doses of 0.2-0.6 mg/kg/min (Mean value 0.41 ± 0.04 SEM mg/kg/min) effectively reduced blood pressure to a level of 57.0 ± 1.8mmHg. As shown in Fig. 1, the onset was immediate and the BP recovered quickly (3 to 5 min). The duration of hypotension lasted 97 ± 19 min, and BP remained remarkably stable with no tachyphylaxis. HR remained unchanged during and after hypotension (Fig. 2). Measurement of cardiac output (CO) revealed well maintained values (Fig. 2) with significantly decreased systemic vascular resistance ($p<0.05$).

DISCUSSION. In the past years, various drugs have been evaluated for controlled hypotension during anesthesia. These have included peripheral vasodilators, ganglionic blockades, calcium channel inhibitors and halogenated inhalation anesthetics. However, each drug has been criticized as having major drawbacks such as ineffectiveness, tissue toxicity, tachycardia, tachyphylaxis or excessive myocardial depression with severely disturbed tissue perfusion. ATP is a physiological intracellular substance, which relaxes and dilates vascular smooth muscles including coronary and cerebral arteries. During hypotensive anesthesia, maintenance of tissue perfusion is vital. ATP-induced hypotension maintains CO without tachycardia, tachyphylaxis and with no sign of acute toxicity. Therefore, ATP should potentially be considered for use among the vasoactive hypotensive drugs.

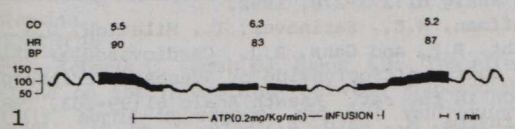

Fig 1

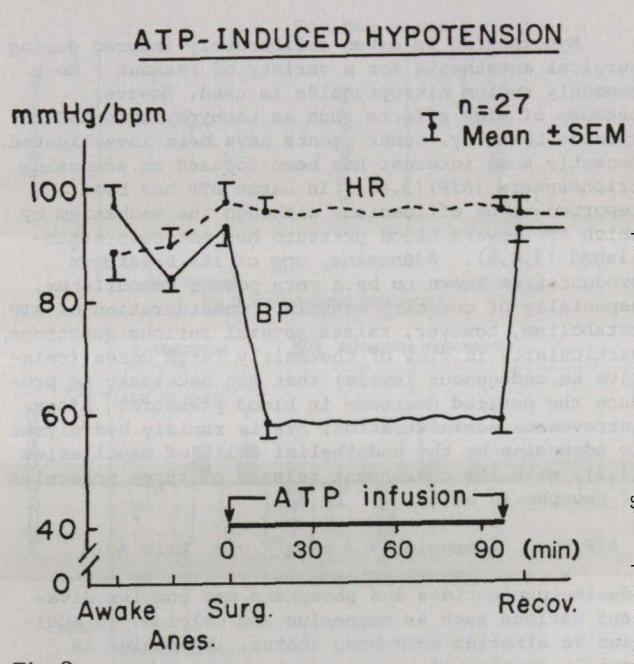

Fig 2

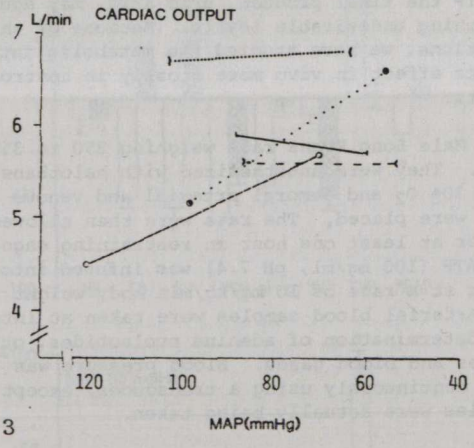

Fig 3

Table. ARTERIAL BLOOD GAS VALUES (mean ± SEM)

	Pre-ATP	ATP 60 min.	Recovery
pH	7.44 ± 0.01	7.43 ± 0.01	7.39 ± 0.01*
$PaCO_2$(mmHg)	34.6 ± 1.2	31.2 ± 1.0*	35.4 ± 1.3
PaO_2(mmHg)	194 ± 13	182 ± 11	194 ± 13
BE(mEg/l)	-1.0 ± 0.6	-2.4 ± 0.6*	-2.5 ± 0.6*

*$P<0.05$ vs Pre-ATP values.

Downloaded from http://asa2.silverchair.com/anesthesiology/article-pdf/57/3/A65/630452/0000542-198209001-00065.pdf by guest on 19 April 2024