Acta Med. Nagasaki 35 : 25–28

ADH Responses to Thoracic and Abdominal Surgery under Epidural Anesthesia

Takayuki NOGUCHI, Shinichi YAMAMORI, Yoshio HAYANO
Shunsuke ODA, Kazuo TANIGUCHI and Natsuo HONDA

Department of Anesthesiology, Medical College of Oita,
1-1506, Idaigaoka, Hazama-cho, Oita-gun, Oita 879-56 JAPAN

Received for publication, January 4, 1990

ABSTRACT : Changes of plasma ADH concentration were studied during thoracic and abdominal operations under epidural anesthesia comnbimed with nitrous oxide inhalation. In abdominal surgeries, ADH level was elevated by operative invasion as usually reported, significantly by peritoneal irritation particularly. In thoracic surgeries, on the other hand, ADH level showed a rise by operative invasion, but little significant change was observed by irritation to the pleura. There was no significant difference in ADH level between the high frequency positive-pressure ventilation group and the intermittent positive-pressure ventilation group.

INTRODUCTION

It is well known that urinary output is reduced by anesthesia and operation, and antidiuretic hormone (ADH) participates in that besides alterations of renal hemodymamices.[1~9] However, the majority of reports on ADH, has been concerned with abdominal surgery,[4~9] but there has been few report concerning plasma ADH level during thoracic and abdominal surgeries under epidural anesthesia combined with nitrous oxide inhalation.

In this study, the author and associates are to present some interesting findings obtained by measurement of plasma ADH concentrations during thoracic and abdominal operations under epidural and nitrous oxide anesthesia.

1. SUBJECTS AND METHODS

Subjects (Table 1) were thirty-five patients aged ranging from 16 to 65, who underwent elective operations starting at 9 : 00 a. m. considerating of hormonal dianal variation. No patients had any abnormal findings in hepatic and renal functions, endocrine systems and cardiovascular disease, corresponding to Class I to II of physical status adopted by the American Society of Anesthesiologists.

All of patients, after 10 hours N. P. O. were given intramuscular injection of 0.5 mg of atropine sulfate and 10 mg of diazepam as premedication one hour before anesthesia. Anesthetic induction was carried out with 20 mg of diazepam and 6 mg of pancuronium bromide in all patients.

Twenty-two patients underwent abdominal surgeries. Anesthesia was maintained by epidural anesthesia punctured between Th_{12} to L_1 and supplemented with 60-70% nitrous oxide. According to the method of ventilation they were divided into two groups, abdominal I group (15 patients) and the other abdominal II group (7 patients). The former were ventilated with high frequency positive-pressure ventilation using VS600 jet ventilator (IDC Co. OHIO), and the latter intermittent positive-pressure ventilation.

In the thoracic group (13 patients), the

Table 1. Patients Characteistics

	No. of Patients	Body weight (Kg)	Age (yr)	Ane. time (min)	Ope. time (min)	Blood loss (cc)
Abdominal Group I	15	56.4± 7.6	51.1± 7.2	307± 55	264± 62	546±380
Abdominal Group II	7	58.4±12.4	49.7±14.0	339±113	285± 87	391±192
Thoracic Group	13	53.5± 7.8	54.0±17.0	286±144	227±135	442±213

Mean±SD

maintainance of anesthesia was performed by epidural anesthesia punctured between Th_7 and Th_8 in combination with nitrous oxide. The anesthetics used in these groups was 2% lidocaine with additional of half the initial dose every 45 minutes. All patients were intubated with endobroncheal double lumen tube (bronco cath) and they were ventilated by the method of differential lung ventiation, in which method high frequency positive-pressure ventilation was applied to the operated side and intermittent positive-pressure ventilation to the other side.

Following the induction, lactated Ringer solution was infused at the rate of 10 ml/kg/h. In the case of bleeding over 300 g, whole blood or concentrated Red cell were given. The systolic blood pressure was kept in the preanesthetic level with the aid of vasopressor and transient rapid fluid infusion.

Parameters measured were as follows; *i. e.,* plasma ADH concentration, plasma and urinary osmotic pressure, mean blood pressure, central, venous pressure and serum electrolytes concentration. Blood samplings were done through the catheter inserted into the radial artery. The time of measurements was settled in the following 7 points; *i. e.,* (1) as the control before anesthetic induction, (2) 30 minutes after the induction, (3) 30 minutes after the start of operation, (4) 60 minutes, (5) 120 minutes, (6) 180 minutes and (7) the postoperative period in the recovery room.

The ADH concentration was determined by the radioimmunoassay, plasma and urinary osmotic pressure by Osmometer (KNAUER Co. Ltd.) and electrolytes by Nova 5 (Nova biochemical Co. Ltd.) respectively.

The data obtaind were subjected to statistical analysis with Student-t-test, estimating $P<0.05$ as significance of change from control.

2. RESULTS

1) Backgrounds of subjects: As shown in Table 1, the average age, the operative duration and the amount of blood loss were; 56 ± 8.1 years old (mean ± standard deviation), 265 ± 62 minutes and 547 ± 383 g respectively in the sbdominal I groups, 49.7 ± 14.8 years old, 285 ± 87 minutes and 392 ± 193 g respectively in the abdominal II group and 52.4 ± 17 years old, 227 ± 136 minutes and 676 ± 988 g. respectively in the thoracic group. Among these three groups, no significant difference was notable, in all items.
2) Mean blood pressure, as illustrated in Fig. 1, showed preoperative rise, drop during operation and return to the control level in the postoperative period. No significant difference was observed among three groups,
3) Plasma ADH concentration: As displayed in Fig. 2, both the abdominal I and II group showed slight elevation in plasma ADH level by anesthesia alone, but marked elevation at the point of 30 minutes after the start of operation, indicating 60- to 100- fold values of the control. Thereafter, plasma ADH level declined and kept 20- to 30-fold values of the control.

In the thoracic group, slight elevation of plasma ADH level was noted 30 minutes after the start of operation, then 20- to 30-fold values of control were maintained till the postoperative period in the recovery room.

Comparing these three groups, there was little significant difference between the abdominal I and II group. In both abdominal

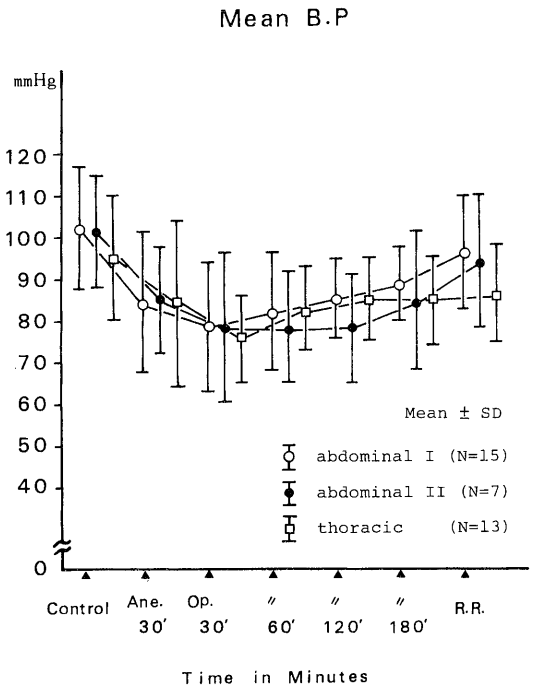

Fig. 1.

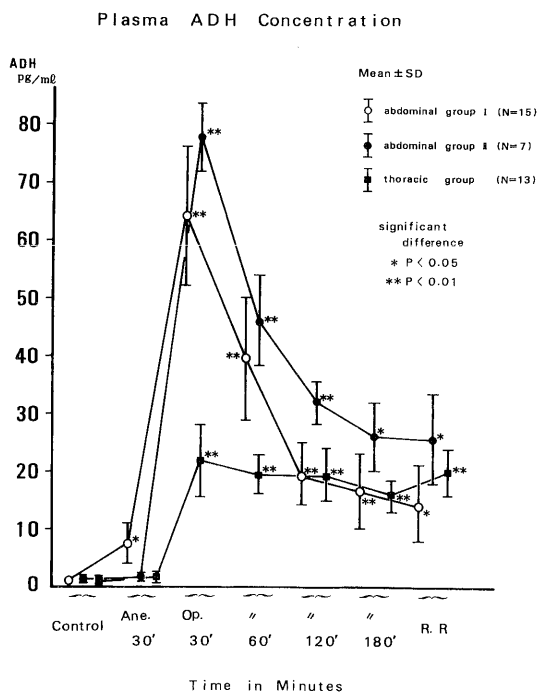

Fig. 2.

groups, however, plasma ADH level revealed three to four times higher than in the thoracic group at the time of 30 minutes after the start of operation, though having no significant change there after.

4) There was found no significant change in serum electrolytes concentration, and plasma, urinary osmotic pressure, and central venous pressure.

3. DISCUSSION

Usually, it is considered[2] that ADH secretion from pituitary gland in mediated by reactions mainly through baroreceptor, volumereceptor and osmorecepter.[6] Otherwise, painful stimulus and other factors may also have a certain influence during operation. The secretion rate is estimated to be rapid and the half-life to be as short as 5 to 10 mimutes.

In this study, a significant difference was ascertained in ADH level between the abdominal groups and thoracic group at the point of time of 30 minutes after the start of operation. In the light of this results, the difference in ADH level was hardly considered to be due to the baroreceptor- or volumereceptor-mediated reactions, because little significant difference was found in blood pressure and central venous pressure between two groups and there was noted a tendency to be rather lowered in the thoracic group. There was no significant difference in plasma osmotic pressure among three groups at every point of time, within no more than several mmHg, as Kono[5] and his collegues reported that ADH level increased by only 2 to 3 pg/ml.

As regards to the change od ADH level in the abdominal groups, almost the same results as in the case of GOF anesthesia reported by Sukenaga[4] and Kono[5] were obtained, attributing the cause to painful stimulus. In this study, however, it is difficult to explain the striking difference in ADH level 30 minutes after the start of operation between the abdominal groups and thoracic group. According to the reports by Ukai[2] and Oyama[1] operative stimulus is said to elevate ADH level, particularly by irritation to the peritoneum. Consequently, it may he considered that operative stimli may induce the elevation in ADH level particularly by irritation

to the peritoneum, while irritating stimulus to the pleura does not make ADH level rise up to considerable extent.

In our depaetment, the anesthetic method for thoracic surgeries is identical with that applied to the abdominal groups in this study, and then two types of ventilation technique were needed as control method in the abdominal groups, yet with little significant difference between intermittent positive-pressure ventilation and high frequency positive-pressure ventilation.

Since an increase in ADH secretion beyond the physiologic range has such harmful effects as to provoke coronary or visceral vasoconstriction and to promote reabsorption of water in the kindey,[8] it is desirable to suppress the accelerated secretion. Taking account of the results of this study along with the descriptions by Bormann[9] and his associates that epidural anesthesia has a benefit in respect with ADH secretiion, it would be considered that epidural anesthesia might be a useful technique for thoracic surgery.

REFERENCE

1. Oyama, T.: Anesthetic management of endocrine disease. Berlin, Springer-Verlag, 1973.
2. Ukai, M., Moran, W., Zimmerman, B.: The role of vischeral afferent pathway on vasopressine secration and urinary excretory patterns during surgical stress. *Ann. Surg.* **18**: 16-28, 1968.
3. Kehlet, H.: Epidural analgesia and the endocrine-metabolic response to surgery update and perspective. *Acta Anesthesiol. Scand.* **28**: 125-127, 1984.
4. Sukenaga, K., Kono, K., Tsuda, K.: Effect of preoperative hydration on intraoperative ADH response. *J. Jpn. Soc. Clin. Anesth.* **3**: 224-249, 1983. (Japanese)
5. Kono, K., Okutani, R., Tsuda, S.: Plesma ADH levels during hepatic resection. *J. Jpn. Soc. Clin. Anesth.* **3**: 236-243, 1983. (Japanese)
6. Robertson, G. L.: The osmoregulation of vasopressin. *Kidney international* **10**: 25-37, 1976.
7. Moran, W., Miltenberger, F. W., Shuayb, W. A.: The relationship of antidiuretic hormon secration to surgical stress. *Surgery* **56**: 99-108, 1964.
8. Philbin, D. M.: Anesthesia and antidiuretics. Weekly Anesthesiology, Up date Lesson 39, Vol. 2, 1979.
9. Bormann, B., Wiedler, B., Dehnhardt, R. *et al.*: Anaesthrsia Verfahren und postoperative ADH-sekretion. *Anaesthetist* **32**: 177-179, 1983.