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Author(s)	FUJITA, NOBUHIRO; SAKAGUCHI, TAKEO; OHTAKE, MASAHIRO; AONO, TAKASHI; ISHIZUKA, DAI; MURATA, TOMOAKI; MAKINO, SHIGETO; TSUKADA, KAZUHIRO; HATAKEYAMA, KATSUYOSHI
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Suppression of Hepatic Portal Blood Flow Caused by Carbon Dioxide Pneumoperitoneum Can Be Restored After Dopamine Administration in Pigs

Nobuhiro Fujita[1], Takeo Sakaguchi[2], Masahiro Ohtake[1], Takashi Aono[1], Dai Ishizuka[1], Tomoaki Murata[1], Shigeto Makino[1], Kazuhiro Tsukada[1] and Katsuyoshi Hatakeyama[1]

The Departments of [1]Surgery and [2]Physiology, Niigata University School of Medicine, Niigata 951, Japan

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Abstract

Portal venous blood flow (PVF), hepatic arterial blood flow (HAF) and systemic arterial pressure (SAP) were examined after dopamine (DA) injection into the jugular vein under carbon dioxide pneumoperitoneum in pigs. When intraabdominal pressure (IAP) was increased by 12 mmHg, PVF and HAF were reduced, but SAP was unchanged. When IAP was kept at 12 mmHg, the injection of DA at 10 μg/kg/min for 2 min produced an increase in PVF without causing any change in HAF or SAP. The response in PVF was dose-dependent. When IAP was increased to 16 mmHg, PVF response was diminished, and no change in HAF or SAP was seen at the same dose of DA.

These observations suggest that DA is effective in increasing PVF under enhanced IAP conditions, but such circulatory improvement due to the agent would be prominent when IAP is below 12 mmHg.

Introduction

Various types of laparoscopic surgery, including cholecystectomy, appendectomy, bowel resection, and selective vagotomy have been conducted [1-5]. These procedures appear to be associated with a low operative mortality, but cardiovascular collapse or suppression of intraabdominal organ blood flow after carbon dioxide (CO_2) pneumoperitoneum has also been documented[6-9]. Recently, the threshold pressure obtained with pneumoperitoneum that has a minimal influence on systemic and intraabdominal hemodynamics was estimated to be less than 12 mmHg[10,11]. Exogenously administered dopamine (DA) has been shown to improve systemic and hepatic circulatory deterioration in animals and humans in the absence of pneumoperitoneum[12-16].

This experiment was designed to investigate whether DA influences systemic and hepatic circula-

Correspondence: N. Fujita, M.D. The Department of Surgery, Niigata University School of Medicine, Niigata 951, Japan.

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Key words: Liver, Catecholamine, Intraabdominal pressure, Portal vein, Hepatic artery

tion associated with CO_2 pneumoperitoneum.

Materials and Methods

Animals

Six Landrace-White-Duroc pigs weighing about 25–28 kg were used. They were housed individually (12 : 12h light dark cycle) with lights on at 6:00 h. They were fed on a standard diet (Spurt G, Nihon Nosan, Yokohama, Japan) with free access to tap water.

Anesthesia and maintenance

Through an endotracheal tube, the animals were anesthetized with a mixture of 1.0% halothane and 1.0 L/min oxygen, and they were allowed to breathe mechanically on a closed air circuit (Acoma Med Products, Tokyo). The anal temperature was maintained at 35.0±1.5C° with a heating pad (UH-CHW-II, Junkan, Saitama). Analysis of gas in the blood was necessary in order to maintain adequate ventilation with an analyzer (ABL-2, Radiometer, Copenhagen)[17]. The blood gas conditions during the experiment are summarized in Table 1.

Measure blood flow

An ultrasonic blood flow meter (Transonic T201, Advance, NY) was utilized to measure the blood flow[18,19]. After laparotomy, the probes for blood flow estimation were placed around the portal vein and the hepatic artery at a position 1–2 cm caudal to the intrahepatic portion. The portal venous blood flow (PVF) and hepatic arterial blood flow (HAF) were recorded on a graph with a pen (Biocolor Graph 2G82, Nihon Denki San Ei, Tokyo). The systemic arterial pressure (SAP) was recorded from the carotid artery, and it was recorded on the same recorder.

Control of intraabdiminal pressure

The two layers of the abdominal wall were closed. Purse-stringsutures were tied securely around each catheter and line to prevent loss of gas and subsequent loss of intraabdominal pressuure (IAP). A veress needle was inserted into the peritoneal cavity. This needle was attached to a surgical CO_2 insufflator (Olympus Winter & Ibe, Hamburg) which could be adjusted to increase IAP and then to keep the pressure constant at the required level.

Table 1 Blood gas parameters after pneumoperitoneum

		IAP (mmHg)		
		0	12	16
Temp	(C°)	37.0±0.1	37.0±0.1	37.0±0.1
pH	(mmHg)	7.342±0.029[a]	7.231±0.006[b]	7.159±0.017
Pco_2	(mmHg)	51.5±9.9	68.1±2.6	84.9±3.4[c]
Po_2	(mmol/L)	196.9±6.5	189.7±6.1	193.5±8.5
HCO_3	(mmol/L)	26.9±0.7	27.7±0.9	28.9±0.7
TCO_2	(mmmol/L)	28.5±0.8	29.8±1.0	31.5±0.8
BE		10.4±4.3	11.5±5.4	5.3±3.6

Values are the mean±SEM (n=6). [a]P<0.01 vs 12 and 16 mmHg. [b]P<0.05 vs 16 mmHg. [c]P<0.01 vs 0 mmHg

Chemical analysis

The collected blood samples were cooled immediately with ice and centrifuged at 2,200 rpm for 20 min. Then the separated plasma was stored at −20C° until measurement of the following parameters of liver and renal functions with an autoanalyzer (Hitachi- 736, Hitachi, Tokyo)[18]: total protein (TP, Biuret method), albumin (Alb, Bromcresol green method), glucose (Glc, glucose oxidase method), total cholesterol (Tch, cholesterol oxidase colorimetric method), total bilirubin (TB, azobilirubin method), glutamic pyruvic transaminase (GPT, Ultraviolet method), alkaline phosphatase (Alp, Bessey-Lowry method), blood urea nitrogen (BUN, urease ultraviolet method), creatinine (Cre, Jaffe method), and Na^+-K^+-Cl^- (Selective electrode method).

Test solution

DA (Shionogi Pharmaceutical Co., Ltd., Tokyo) dissolved in saline was injected into the left side of the jugular vein. The amount used in each test injection was 1.0 ml and it was completed in 2 min with an infusion pump. Saline was injected as the control. It was preliminarily observed that the same amount of saline produced no measurable effect on either flow or pressure, and portal flow response due to DA was saturated 2 min after injection, and the magnitude of the response remained almost the same even when the injection was continued for more than 2 min. Test injections were given at approximately 12–15 min intervals as in previous experiments[17–19].

Data analysis

The results were calculated as a percentage of the control value, with each animal serving as its own control. The control value was the value immediately before drug administration. Samples were collected following the 1st and 2nd responses by each animal to a specific solution. The data obtained were ANOVA analyzed, and specific values were evaluated by Duncan's multiple range test.

Results

The blood pH and gas tension showed adequate ventilation of the animals when IAP was 0 mmHg (Table 1). It is possible that considerable factors modulating DA action on the vasculature could be fixed[17–19]. It was noted that pH was reduced and pressure of CO_2 increased 5–10 min after IAP was enhanced. Basal levels of SAP, HAF and PVF before and 5–10 min after enhancing IAP are shown in Table 2. Circulatory parameters associated with enhanced IAP application remained almost the same during DA injection test.

When IAP was kept at 12 mmHg, PVF was increased by the injection of DA (10 μg/kg/min)

Table 2 Hepatic circulatory parameters after pneumoperitoneum

		IAP (mmHg)			
		0	8	12	16
SAP	(mmHg)	96.6±4.2[a]	96.0±7.4	102.0±4.0	82.6±5.4
PVF	(ml/min)	265.5±13.5[b]	233.0±15.5[c]	166.5±14.2	187.5±12.0
HAF	(ml/min)	168.0±13.4[d]	180.3±9.7	104.0±12.1[e]	124.5±12.6

Values are the mean±SEM (n=6). [a]$P<0.05$ vs 16 mmHg. [b]$P<0.01$ vs 12 and 16 mmHg. [c]$P<0.05$ vs 12 mmHg. [d]$P<0.01$ vs 16 mmHg. [e]$P<0.05$ vs 0 and 8 mmHg.

into the jugular vein (Fig. 1). The increase in PVF reached its maximum 1–2 min after the injection, then returned to the control level within another 2 min (Fig. 2). ANOVA revealed that the differences among groups and among times were significant: $F_{1,83}=63.461$, $p<0.002$ and $F_{6.83}$

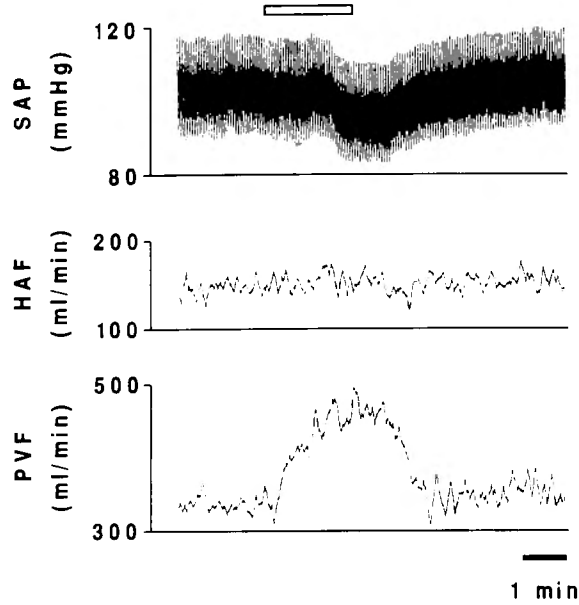

Fig. 1 Effects of jugular injection of DA on SAP, HAF and PVF under IAP at 12 mmHg. DA (10 μg/kg/min) was given for 2 min. A bar shows the time of injection.

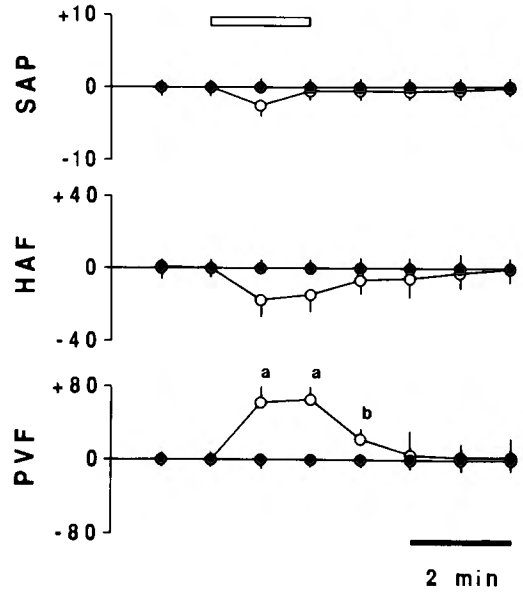

Fig. 2 Time courses for SAP, HAF and PVF following DA administration. Jugular injection of DA (○, 10 μg/kg/min) or saline (●) was done with IAP and 12 mmHg. A bar indicates the time of injection. Values are the mean±SEM (n=6). $^{a}P<0.01$ vs ●. $^{b}P<0.05$ vs ●.

$=14.552$, $p<0.002$, respectively. Based on this finding the PVF response due to DA was compared 2 min after DA administration. It was found that the response due to DA was dose-dependent (Fig. 3).

HAF was unchanged after DA (10 μg/kg/min) injection into the jugular vein under IAP at 12 mmHg (Figs. 1 and 2). The differences among groups and among times were not reliable: $F_{1,83}=1.942$, $p>0.05$ and $F_{6.83}=2.816$, $p>0.05$, reapectively. Significant reduction in HAF was seen when DA 20 μg/kg/min was injected (Fig. 3).

SAP was unchanged when DA (10 μg/kg/min) was injected into the jugular vein after enhancing IAP at 12 mmHg (Figs. 1 and 2). The differences among groups and among times were reliable: $F_{1,83}=1.4872$, $p>0.05$ and $F\ 6.83=1.095$, $p>0.05$, respectively. Significant decrease in SAP was seen when DA 20 μg/kg/min was given (Fig. 3).

PVF responses due to DA 10.0 μg/kg/min were compared under different levels of IAP, and the response was diminished when IAP was increased to 16 mmHg (Fig. 4).

Blood chemical parameters indicating liver and kidney functions are presented in Table 3. It is shown that BUN was increased after 16 mmHg IAP was applied.

Discussion

Although enhanced IAP has been shown to suppress the blood supply to the abdominal organs by reducing effective perfusion pressure[10,11], it was found that DA administration is capable of increasing PVF under an enhanced IAP condition (Figs. 1, 2 and 3). This is consistent with the view that systemic administration of DA enhances hepatic blood flow in animals and humans[12–16]. The action of DA on the vascular wall seemed to be specific to DA, because the PVF response due to DA was dose-dependent (Fig. 3).

The action site of DA has been considered to be localized in the superior mesenteric arterial vascular bed[13,15], but a vasodilative action of DA on the hepatic vascular bed has also been suggested based on the finding that DA injected into the portal vein reduces portal vein resistance[20]. In the present PVF response, the mesenteric artery or the portal vein or both would therefore be involved.

The mode of DA action on the vascular wall has been presumed from the flow response. Several investigators noted a biphasic change in the superior mesenteric arterial blood flow after DA administration[21,22], and they interpreted this to mean that the initial decrease may be caused by a contractile effect, and that any subsequent rise may be due to a direct vasodilator effect on the mesenteric vasculature. In this study, DA induced PVF response with an inhibitory monophasic pattern. Considering this finding together with the report that the portal vasculature is responsive to DA[20], it is possible that the PVF response observed was a result of action primarily determined by dopamine receptor in the portal vascular walls.

PVF effects following DA injection were of short duration (Figs. 1 and 2), possibly owing to rapid removal from the circulation[23], and it has been shown that the biological activity of DA is reduced by 80% after a single hepatic circulation[24]. DA exogenously injected at 10 μg/kg/min caused an increase in PVF without a change in SAP (Fig. 2). This indicates that DA is substantially metabolized and inactivated by the liver [23,24]. Moreover, enhanced IAP seemed to have a effect on the DA metabolism because PVF responses due to DA were not similar for different levels of IAP application (Fig. 4).

The concentration of DA which had an effect on PVF was unchanged during IAP application

between 0–12 mmHg, but the effective concentration was increased when IAP was increased to 16 mmHg (Figs. 3 and 4). Since the hepatic vascular bed is regarded as a powerful blood reservior[25] and enhanced IAP has been shown to compress the vascular walls, decreasing the circulation to the viscera and causing stasis[26], the shift in the concentration may indicate a functional property of the

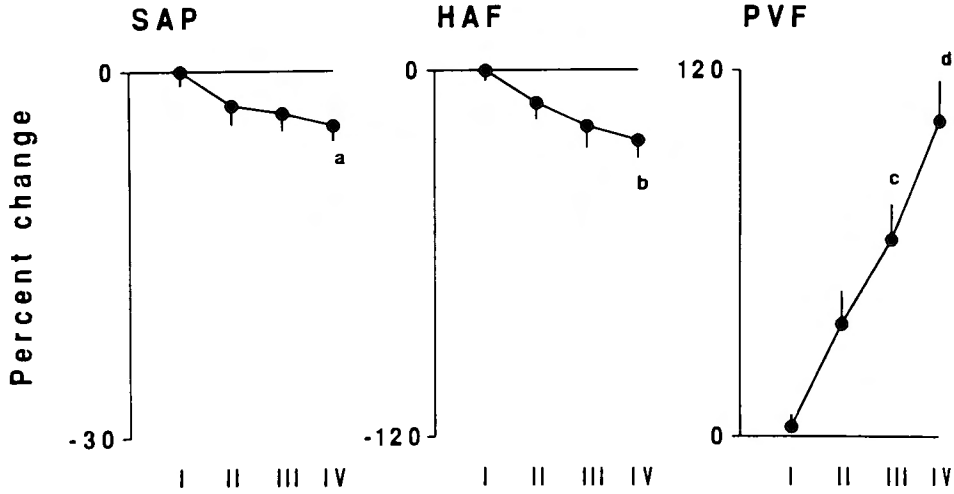

Fig. 3 Percent changes in SAP, HAF and PVF after DA injection. Three different doses of DA (II, 5 mg/kg/min: III, 10 μg/kg/min: IV, 20 μg/kg/min) were injected into the jugular vein under IAP at 12 mmHg, and parameters 2 min after injection are compared. Saline was injected as the control (I). Values are the mean±SEM (n=6). $^{a}P<0.01$ vs I. $^{b}P<0.05$ vs I. $^{c}P<0.01$ vs I. $^{d}P<0.01$ vs II.

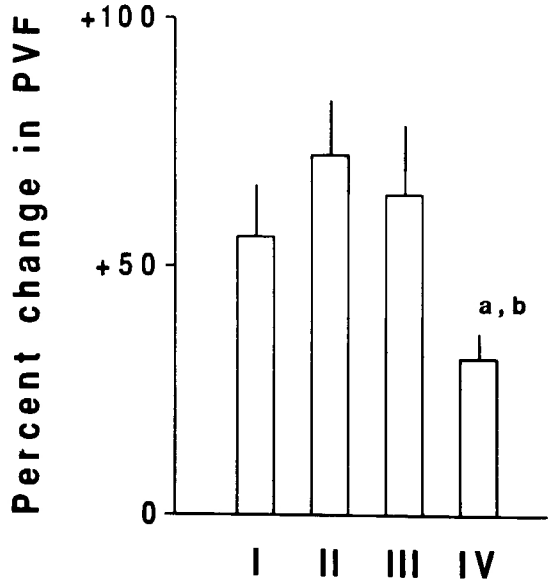

Fig. 4 Percent changes in PVF associated with DA under different levels of IAP. Four different levels of IAP (I, 0 mmHg; II, 8 mmHg; III, 12 mmHg; IV, 16 mmHg) were applied after DA 10 μg/kg/min was given. PVF values 2 min after DA injections were compared. Values are the mean±SEM (n=6). $^{a}P<0.05$ vs I. $^{b}P<0.01$ vs II and III.

hepatic blood reservior.

Of particular interest is the response in HAF (Figs. 2 and 3). Although responses in PVF due to DA were relatively great when the IAP was enhanced, the response in HAF was small (Figs. 1, 2 and 3). This could mean that IAP impact on the vascular walls is prominent in the veins rather than the arteries. The sympathetic nerve innervating the liver is a considerable factor in regulating HAF, and stimulation of this nerve has been shown to decrease HAF[27]. The finding mentioned above therefore also suggests that neural participation is not involved in the HAF response.

There is a mechanical interaction between the peritoneal cavity and the thoracic cavity through the diaphragm, and heart function can be depressed when the diaphragm is moved upward by increased IAP[26]. But an increase in IAP to 12 mmHg had a minimal effect on the heart, because SAP response to DA administration was small (Figs. 1, 2 and 3). It is considered that cardiac function in response to DA cannot be modulated by IAP when it is below 12 mmHg.

A slight fall in SAP was seen 2 min after DA 20 μg/kg/min injection. It is not easy to explain, but, as was observed in dogs, DA reduces vascular resistance, and arterial blood pressure is reduced 2 min after injection[28]. This may hold true for the SAP response observed. Another explanation is that DA induces a shift in circulating blood volume from the systemic circulation to splanchnic circulation because a fall in arterial blood pressure follows a marked increase in mesenteric blood flow[28].

The appearance of DA action on SAP changes according to the concentration; at lower concentration α-action is dominant, and β-action can be presented at higher concentration[29,30]. And a lower dosage of DA has been presumed to increase hepatic blood flow without changes in cardiac output[20,31]. In this study, unfortunately, no record of cardiac output has been made, but SAP was un changed after DA injection. It can therefore the deduced that the PVF response observed is derived from the α-action.

Although the pressure of CO_2 in the blood (Pco_2) is increased during CO_2 pneumoperitoneum,

Table 3 Blood chemical parameters after pneumoperitoneum

		IAP (mmHg)		
		0	12	16
TP	(g/dl)	5.6±0.1	5.5±0.1	5.5±0.2
Alb	(g/dl)	3.3±0.1	3.3±0.1	3.3±0.1
Glc	(mg/dl)	133±32	128±14	108±18
Tch	(mg/dl)	68±3	66±3	66±4
TB	(mg/dl)	0.2±0.0	0.2±0.0	0.2±0.0
GPT	(U)	30±2	30±3	31±2
Alp	(U/l)	298±32	312±34	340±42
BUN	(mg/dl)	11.8±0.6	13.6±0.5	15.7±1.4[a]
Cre	(mg/dl)	1.2±0.1	1.1±0.1	1.2±0.1
Na^+	(mEq/l)	138±1	139±1	138±1
K^+	(mEq/l)	5.3±0.2	5.5±0.2	5.8±0.4
Cl^-	(mEq/l)	103±1	103±1	104±1

Values are the mean±SEM (n=6). [a]$P<0.05$ vs 0 mmHg.

this is a result of the combined effects of absorpotion of CO_2 across the peritoneum[32-35], but with the mechanical ventilation used in this study, the change in Pco_2 is not so large. It appears that the cardiostimulatory effects of hypercapnia are minimal in this situation.

Finding that DA effectively increased PVF even when IAP was increased (Fig. 3) and DA 10 μg/kg/min administration for 1 h under IAP at 12 mmHg (n=3) reproduced an hepatic circulatory effect without any change in hepatic functional scores (unpublished data, Fujita & Sakaguchi) suggests that improvement in hepatic circulation induced by DA can be expected during laparoscopic surgery in clinical practice, especially in cases where hepatic circulation deteriorates during a long operation with CO_2 pneumoperitoneum[6,10].

The body is divided into four circulatory compartments: the peritoneal cavity, the thoracic cavity, the forebody and the hindquarters[26]. The obseved response is only a chain of mechanical events that influence the circulatory dynamics within each of the four compartments, but the physical impact on the viscera induced kidney parameters which indicate dysfunction (Table 3). There is much room for reserach into this aspect.

These observations lead us to conclude that the administration of DA is useful in improving hepatic circulatory efficiency when laparoscopic surgery is done with CO_2 pneumoperitoneum.

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和文抄録

二酸化炭素気腹で抑制される豚の門脈血流量はドパミン投与によって回復する

新潟大学第一外科
藤田 亘浩，大竹 雅広，青野 高志，石塚 大，
丸田 智章，牧野 茂人，塚田 一博，畠山 勝義

第一生理
坂口 武夫

豚を用いて二酸化炭素気腹下でドパミンの頸静脈投与が門脈血流量，肝動脈血流量，体血圧に対する影響を調べた．腹腔内圧を 12 mmHg まで高めると，門脈と肝動脈血流量は減少したが，体血圧には変化を認めなかった．腹腔内圧を 12 mmHg で維持した状態で，ドパミン 10 μg/kg/min で2分間の投与は肝動脈血流量と体血圧に変化を与えることなく，門脈血流量を増加させた．こうしたドパミンによる門脈血流量反応は用量依存性を示した．腹腔内圧 16 mmHg では同用量のドパミンの投与で，門脈血流量反応は低下したが，肝動脈血流量と体血圧には変化を認めなかった．

これらの観察から，ドパミンは腹腔内圧上昇時に肝門脈血流量を増加させること，しかしそうしたドパミンの肝循環改善効果は腹腔内圧 12 mmHg 以下で顕著であることを導いた．