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OXYGEN CONSUMPTION, ASSESSED WITH THE OXYGEN ABSORPTION SPECTROPHOTOMETER, DECREASES INDEPENDENTLY OF VENOCONSTRICTION DURING HEPATIC ANAPHYLAXIS IN PERFUSED RAT LIVER

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ABSTRACT—Anaphylactic shock is accompanied by a decrease in oxygen consumption. However, it is not well known whether oxygen consumption decreases during local anaphylactic reaction in liver. We determined the effects of anaphylaxis and norepinephrine on oxygen consumption in isolated rat livers perfused portally and recirculatingly at constant flow with blood (hematocrit, 12%). Oxygen consumption was continuously measured by monitoring the portal–hepatic venous oxygen saturation differences using the absorption spectrophotometer, the probes of which were built in perfusion lines. Hepatic anaphylaxis was induced by an injection of ovalbumin (0.01 or 0.1 mg) into the perfusate of the isolated liver of the rat sensitized with subcutaneous ovalbumin (1 mg). Hepatic venoconstriction and liver weight loss were similarly observed in response to norepinephrine (0.01–10 μmol L^{-1}) and anaphylaxis. However, hepatic anaphylaxis reduced oxygen consumption, whereas norepinephrine increased it. There was a possibility that anaphylactic venoconstriction could reduce the perfused surface area, resulting in decreased oxygen consumption. However, pretreatment with a vasodilator of sodium nitroprusside substantially attenuated venoconstriction but not the decrease in oxygen consumption during anaphylaxis. Thus, we conclude that local hepatic anaphylaxis decreases oxygen consumption independently of venoconstriction in isolated blood-perfused rat livers.

KEYWORDS—Anaphylactic shock, hepatic circulation, norepinephrine, hepatic venoconstriction, portal venous pressure

INTRODUCTION

Circulatory shock is characterized by generalized cellular hypoxia and a decrease in oxygen consumption of organs and tissues (1–3). The severity and the mortality after shock is strongly associated with decreased oxygen consumption (2, 4). Indeed, anaphylactic shock is accompanied by decreased oxygen consumption (5, 6). There is a possibility that anaphylactic reaction itself reduces oxygen consumption. However, there are only a limited number of studies (7) to determine the effect of local anaphylaxis on oxygen consumption of organs such as the liver.

Therefore, for determination of the effect of local anaphylaxis on oxygen consumption of the liver, we used the isolated perfused liver, which was free from the influences of the other organs or many humoral mediators released from other organs during the time course of anaphylactic shock. We continuously measured the oxygen consumption of isolated blood-perfused livers during hepatic anaphylaxis by monitoring the oxygen (O_2) saturation differences between the inflow and outflow blood using a custom-made absorption spectrophotometer. To check the validity of this absorption spectrophotometer, we also measured the oxygen consumption during administration of norepinephrine because norepinephrine is known to increase oxygen consumption in the perfused rat liver (8–10). Another reason for investigating the effect of norepinephrine is that it induces hepatic venoconstriction. Oxygen consumption depends not only on metabolic factors but also on hemodynamic factors. From the hemodynamic point of view, isolated perfused rat livers showed similar predominant presinusoidal constrictive responses to anaphylaxis (11) and norepinephrine (12). In addition, to determine the effect of hepatic anaphylaxis on oxygen consumption without its hemodynamic influence, anaphylaxis-induced venoconstriction was suppressed by pretreatment with sodium nitroprusside (SNP) and then changes in oxygen consumption were determined during hepatic anaphylaxis.

MATERIALS AND METHODS

Animals

Seventy-four male Sprague-Dawley rats (Japan SLC, Shizuoka, Japan) weighing 270 to 370 g (mean ± SD, 325 ± 4 g) were used in this study. Rats were maintained at 23°C and under pathogen-free conditions on a 12-h dark/light cycle and received food and water *ad libitum*. The study protocol was approved by the Animal Research Committee of Kanazawa Medical University.

Sensitization

Twenty-one rats were sensitized by subcutaneous injection of an emulsion made by mixing complete Freund's adjuvant (0.5 mL) with 1 mg ovalbumin (grade V, Sigma, Saint Louis, MO) dissolved in physiological saline (0.5 mL) as previously described (11). Two weeks after injection, rats were used for the isolated perfused liver experiments.

Isolated liver preparation

The animals were anesthetized with pentobarbital sodium (50 mg kg^{-1} i.p.) and were mechanically ventilated with room air. The method for perfusion of isolated rat livers was previously described (11). In brief, after the right carotid artery

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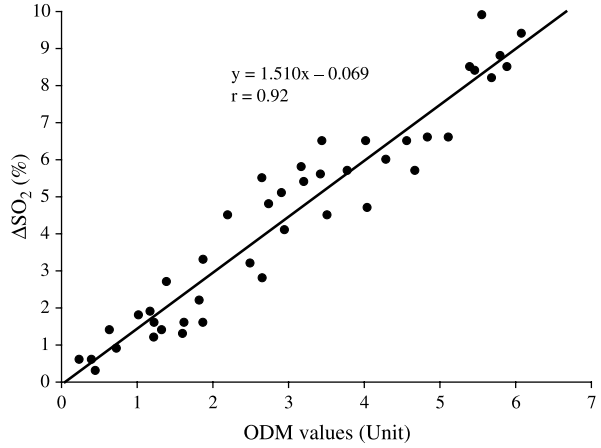

FIG. 1. **The relationship between the ODM values and the measured ΔSo_2 that were obtained in 10 intact livers.** The regression line equation is $\Delta So_2 = 1.5103 \times ODM - 0.0685$ ($r = 0.92$, $P < 0.05$).

cannulation and laparotomy, the hepatic artery was ligated, and the bile duct was cannulated with polyethylene tube (inner diameter [ID], 0.5 mm; outer diameter [OD], 0.8 mm). After intra-arterial heparinization (500 U kg^{-1}), 8 to 9 mL of blood was withdrawn through the carotid arterial catheter. The intra-abdominal inferior vena cava above the renal veins was ligated, and the portal vein was cannulated with a stainless cannula (ID, 1.3 mm; OD, 2.1 mm) for portal perfusion. After thoracotomy, the supradiaphragmatic inferior vena cava was cannulated through a right atrium incision with a larger stainless cannula (ID, 2.1 mm; OD, 3.0 mm), then portal perfusion was begun with the heparinized autologous blood diluted with 5% bovine albumin (Sigma-Aldrich, St. Louis, Mo) in Krebs solution (118 mmol L^{-1} NaCl, 5.9 mmol L^{-1} KCl, 1.2 mmol L^{-1} $MgSO_4$, 2.5 mmol L^{-1} $CaCl_2$, 1.2 mmol L^{-1} NaH_2PO_4, 25.5 mmol L^{-1} $NaHCO_3$, and 5.6 mmol L^{-1} glucose) at hematocrit of 12%. The liver was rapidly excised, suspended from an isometric transducer (TB-652T, Nihon-Kohden, Tokyo, Japan), and weighed continuously throughout the experimental period.

The livers were perfused at a constant flow rate in a recirculating manner *via* the portal vein with blood that was pumped using a Masterflex roller pump from the venous reservoir through a heat exchanger (37°C). The recirculating blood volume was 40 mL. The perfused blood was oxygenated in the venous reservoir by continuous bubbling with 95% O_2 and 5% carbon dioxide (perfusate Po_2 = 300 mmHg). The portal and hepatic venous pressures (Ppv and Phv, respectively) were measured using pressure transducers (TP-400T, Nihon-Kohden) attached by sidearm to the appropriate cannulas with the reference points at the hepatic hilus. Portal blood flow rate was measured with an electromagnetic flow meter (MFV 1200, Nihon-Kohden), and the flow probe was positioned in the inflow line. Bile was collected drop by drop in a small tube suspended from the force transducer (SB-1T, Nihon-Kohden). One bile drop yielded 0.018 g, and the time between drops was measured for determination of the bile flow rate. To determine the oxygen consumption, the blood oxygen saturation difference between the portal and hepatic venous blood was monitored continuously with a custom-made absorption spectrophotometer (ODM-1, Biomedical Science, Kanazawa, Japan), the probe of which consisted of a 660-nm wavelength light-emitting diode, a cuvette, and a photodiode detector. These 2 probes were built in the inflow and outflow perfusion lines. The blood oxygen saturation differences measured with this absorption spectrophotometer were expressed as the oxygen difference meter (ODM) values. The hepatic vascular pressures, ODM values, blood flow rate, liver weight, and bile weight were monitored continuously and displayed through a thermal physiograph (RMP-6008, Nihon-Kohden). All output were also digitized by the analog–digital converter at a sampling rate of 100 Hz. These digitized values were also displayed and recorded using a personal computer.

Measurement of oxygen consumption

Oxygen consumption was calculated according to the Fick principle by the following equation

$$\text{Portalbloodflow/liverweight(g)} \times 1.39\text{(mL/g)} \times \text{hemoglobin(g/mL)} \times \Delta S_{O_2} \quad (1)$$

where ΔSo_2 is the oxygen saturation difference between the inflow (portal) and outflow (hepatic vein) blood. To estimate ΔSo_2 by the ODM value of the absorption spectrophotometer, the validity of the ODM values was examined by comparing the ODM values with the ΔSo_2, which was determined by measuring the oxygen saturation of the inflow and outflow blood with the use of automatic blood gas analyzer. To determine the correlation of ODM value and ΔSo_2, the intact isolated rat livers (n = 10) were perfused at various blood flow rates (12 – 60 mL/min) to obtain various inflow-to-outflow oxygen saturation differences. Blood samples were collected simultaneously from the inflow (portal) and outflow (hepatic venous) lines at a given flow rate and the oxygen saturation of the inflow (So_2in) and outflow (So_2out) blood were measured by the blood gas analyzer (AVL OMNI Modular System, AVL Scientific Corp., Roswell, GA). The calculated oxygen saturation differences ($\Delta So_2 = So_2\text{in} - So_2\text{out}$) were compared with the corresponding ODM values. We obtained the regression line equation for So_2 measured and ODM values.

Experimental protocol

Hepatic hemodynamic parameters were observed for at least 20 min after the start of perfusion until an isogravimetric state (no weight gain or loss) was obtained by adjusting the portal blood flow rate and the height of the reservoir at a Phv of 0 to 1 cmH_2O and at a portal blood flow of 37 ± 1 mL min^{-1} per 10 g of liver. After the baseline measurements, the perfused livers excised from nonsensitized animals were randomly assigned to the control group or norepinephrine group and those from sensitized animals to the anaphylaxis group. In the norepinephrine group (n = 35), norepinephrine was administered into the perfusate to get the final concentration of 0.001, 0.01, 0.1, 1, and 10 μmol L^{-1}. The concentrations of norepinephrine were randomly assigned for individual preparations. In the anaphylaxis group, ovalbumin 0.01 (n = 7) or 0.1 mg (n = 7) was administered into the perfusate. In addition, to eliminate a possible effect of anaphylaxis-induced venoconstriction on oxygen consumption, a vasodilator of SNP at 100 μmol L^{-1} was administered 10 min before injection of 0.01 mg of ovalbumin (SNP group, n = 7). In the control group (n = 8), ovalbumin 0.1 mg was administered into the perfusate of the livers from nonsensitized rats.

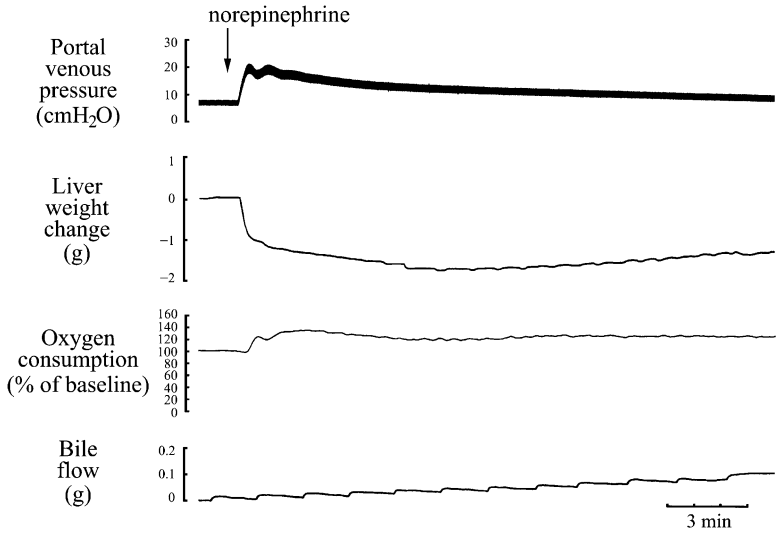

FIG. 2. **A representative recording of the response to norepinephrine at 1 μmol L^{-1}.**

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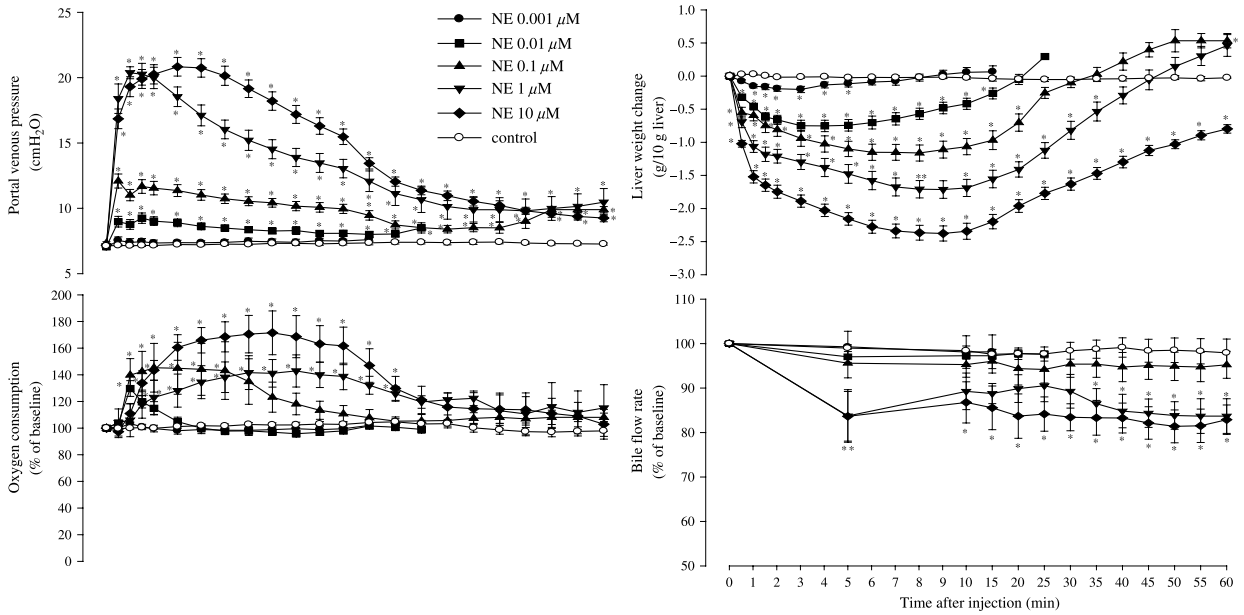

FIG. 3. **The time course changes in the Ppv, liver weight changes, oxygen consumption, and bile flow rate in the norepinephrine (n = 35) and control (n = 8) groups.** Values are given as means ± SE. *$P < 0.05$ versus the baseline. NE indicates norepinephrine.

Statistics

All results are expressed as means ± SE. Data were analyzed by one- and two-way analysis of variance, using repeated measures for 2-way comparison within groups. Comparisons of individual points between groups and within groups were made by Bonferroni test. The correlation between the ODM values and the measured oxygen saturation differences was analyzed by linear regression. Differences were considered as statistically significant at P values of <0.05.

RESULTS

Relationship between the ODM values and the measured oxygen saturations

Figure 1 shows the relationship between the ODM values and the measured ΔS_{O_2} that were obtained in 10 intact livers. The regression line equation is

$$\Delta S_{O_2} = 1.510 \times \mathrm{ODM} - 0.069 \; (r = 0.92, \; P < 0.05). \qquad (2)$$

This indicates that the ΔS_{O_2} can be estimated by ODM values in isolated perfused rat liver. The basal oxygen consumption calculated using the ODM values in the control, 0.1-mg ovalbumin, 0.01-mg ovalbumin, SNP, and 0.001-, 0.01-, 0.1-, 1-, and 10-μmol L^{-1} norepinephrine groups were 0.0072 ± 0.0005, 0.0078 ± 0.0002, 0.0054 ± 0.0005, 0.0055 ± 0.0003, 0.0065 ± 0.0008, 0.0069 ± 0.0009, 0.0066 ± 0.0008, 0.0070 ± 0.0008, and 0.0064 ± 0.0005 mL min^{-1} per gram of liver, respectively. In the present study, the changes in oxygen consumption in a given perfused liver were directly proportional to the ODM values because the other determinants of oxygen consumption such as perfusate hemoglobin and portal blood flow rate were not changed during the experimental period. Thus, oxygen consumption was expressed as the percentage of the baseline ODM value.

Effects of norepinephrine on hepatic oxygen consumption, Ppv, liver weight, and bile flow

Figure 2 shows a representative example of the hepatic response to administration of norepinephrine at 1 μmol L^{-1}. Figure 3 shows the summary data of the time-dependent changes in Ppv, liver weight, hepatic oxygen consumption, and bile flow rate of the norepinephrine groups. Significant changes in the variables were observed in response to norepinephrine at concentrations higher than 0.01 μmol L^{-1}. Hepatic oxygen consumption increased dose-dependently after norepinephrine, with the peak values being 129% ± 13% of the baseline at 0.01 μmol L^{-1}, 145% ± 42% at 0.1 μmol L^{-1}, 142% ± 27% at 1 μmol L^{-1}, and 171% ± 43% at 10 μmol L^{-1}. Norepinephrine at 0.01, 0.1, and 1 μmol L^{-1} dose-dependently increased Ppv to the peak, 9.2 ± 0.3, 12.1 ± 0.5, and 20.4 ± 0.8 cmH_2O, respectively, within 3 min. Maximal venoconstriction was observed at 1 μmol L^{-1}; the peak of Ppv after norepinephrine at 10 μmol L^{-1} (20.8 ± 0.7 cmH_2O) was comparable to that at 1 μmol L^{-1}. After administration of norepinephrine at 0.01, 0.1, 1, and 10 μmol L^{-1}, the liver weight decreased by 0.8 ± 0.1, 1.2 ± 0.2, 1.7 ± 0.2, and 2.4 ± 0.2 g per 10 g of liver, respectively. The bile flow rates decreased after norepinephrine administration at 1 and 10 μmol L^{-1} to 84% ± 6% and 84% ± 8% of the baseline of 8.6 ± 0.4 and 9.8 ± 0.5 mg min^{-1} per 10 g of liver, respectively.

Effects of anaphylaxis on hepatic oxygen consumption, Ppv, liver weight, and bile flow

Figure 4 shows a representative example of the response of the sensitized liver to the antigen (0.01 mg of ovalbumin). Figure 5 shows the summary data of the time course changes in Ppv, liver weight, hepatic oxygen consumption, and bile flow rate of the 0.01-mg ovalbumin group along with the data

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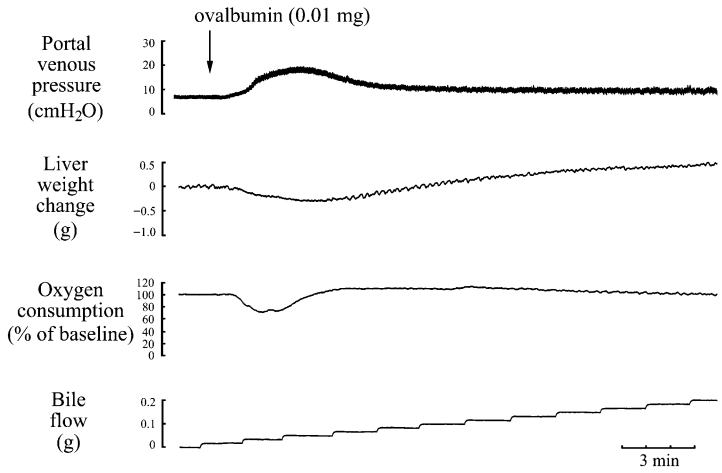

FIG. 4. **A representative recording of the responses to the antigen of ovalbumin (0.01 mg).**

of the 0.1-mg ovalbumin, 1- and 10-μmol L^{-1} norepinephrine, and control groups. After injection of 0.01 mg of ovalbumin, Ppv increased from the baseline of 6.9 ± 0.13 cmH_2O to the peak of 20.0 ± 1.4 cmH_2O at 4 min. Hepatic oxygen consumption decreased to the nadir of 75% ± 6% of the baseline at 2 min and then recovered to the baseline level at 4 min after administration of 0.01 mg of ovalbumin. The decrease in oxygen consumption after antigen administration contrasts with its increase after norepinephrine administration in that hepatic venoconstriction was similar in magnitude between the 0.01-mg ovalbumin group and the high dose of norepinephrine (1 or 10 μmol L^{-1}) group. The liver weight showed a rapid decrease, reaching the bottom of −0.4 ± 0.1 g per 10 g of liver at 4 min after 0.01 mg ovalbumin administration. The bile flow rate significantly decreased to 86% ± 1% of the baseline of 8.7 ± 0.5 mg min^{-1} per 10 g of liver at 5 min after antigen (0.01 mg of ovalbumin) administration and then gradually recovered. The responses of the variables to the high-dose ovalbumin (0.1 mg) were qualitatively similar to that of the low-dose ovalbumin (0.01 mg), but greater in magnitude, as shown in Figure 5.

Effects of SNP on Hepatic Anaphylactic Response

Figure 6 shows a representative example of the response of the sensitized liver to the antigen (0.01 mg of ovalbumin) after pretreatment with SNP at 100 μmol L^{-1}. Figure 7 shows the summary data of the time course changes of the 0.01-mg ovalbumin and SNP groups. The increase in Ppv in response to the 0.01-mg ovalbumin antigen was much smaller with SNP than without SNP. However, the decrease in hepatic oxygen consumption was similarly observed with or without pretreatment with SNP. At 2 min after antigen administration, hepatic oxygen consumption decreased to the nadir of 79% ± 3% of the baseline, which was not significantly different from that of the 0.01-mg ovalbumin group without administration of SNP. In contrast, Ppv increased only slightly to the peak of 9.9 ± 1.2 cmH_2O at 4 min from the baseline of 6.6 ± 0.15 cmH_2O. The liver weight showed a mild, but significant, decrease, reaching the nadir of −0.2 ± 0.1 g per 10 g of liver at 3 min after ovalbumin administration. The bile flow rate significantly decreased to 89% ± 1% of the baseline of 8.6 ± 0.5 mg min^{-1} per 10 g of liver at 5 min after antigen administration and did not return to the baseline.

DISCUSSION

In the present study, oxygen consumption was for the first time measured continuously by a custom-made absorption spectrophotometer, the probes of which were built in the perfusing circuit to monitor the portal–hepatic venous oxygen saturation difference of isolated perfused rat livers. We have shown that anaphylaxis decreased hepatic oxygen consumption, whereas norepinephrine increased it, although both caused similar hepatic venoconstriction and liver weight loss. Pretreatment with a vasodilator of SNP did not prevent the anaphylaxis-induced decrease in hepatic oxygen consumption, whereas it substantially attenuated the anaphylaxis-induced hepatic venoconstriction. These findings suggest that the decrease in hepatic oxygen consumption during anaphylactic shock could be caused by local hepatic anaphylaxis itself, and that it is independent of hepatic venoconstriction.

We demonstrated that there was a significant and good correlation between the ODM values of the absorption spectrophotometer and ΔS_{O_2} measured with the blood gas analyzer. This indicates that the ΔS_{O_2} and oxygen consumption can be indirectly measured by the absorption spectrophotometer in isolated perfused liver. Oxygen consumption of the perfused liver is usually measured with a Clark-type oxygen electrode (7, 13–15). However, this oxygen electrode monitors only the effluent, but not the influent, oxygen concentration and, thus, it alone cannot directly assess the portal– hepatic venous oxygen difference, which is the determinant of oxygen consumption. The application of the custom-made oxygen absorption spectrophotometer permits the continuous measurement of the portal–hepatic venous oxygen difference of the perfused livers.

An increase in oxygen consumption has been observed after norepinephrine administration in perfused rat livers (8, 16). In these studies, oxygen consumption was determined by measuring the effluent oxygen concentration with a Clark-type oxygen electrode. In the present study, we confirmed by using the oxygen absorption spectrophotometer that norepinephrine at concentrations higher than 0.01 μmol L^{-1}

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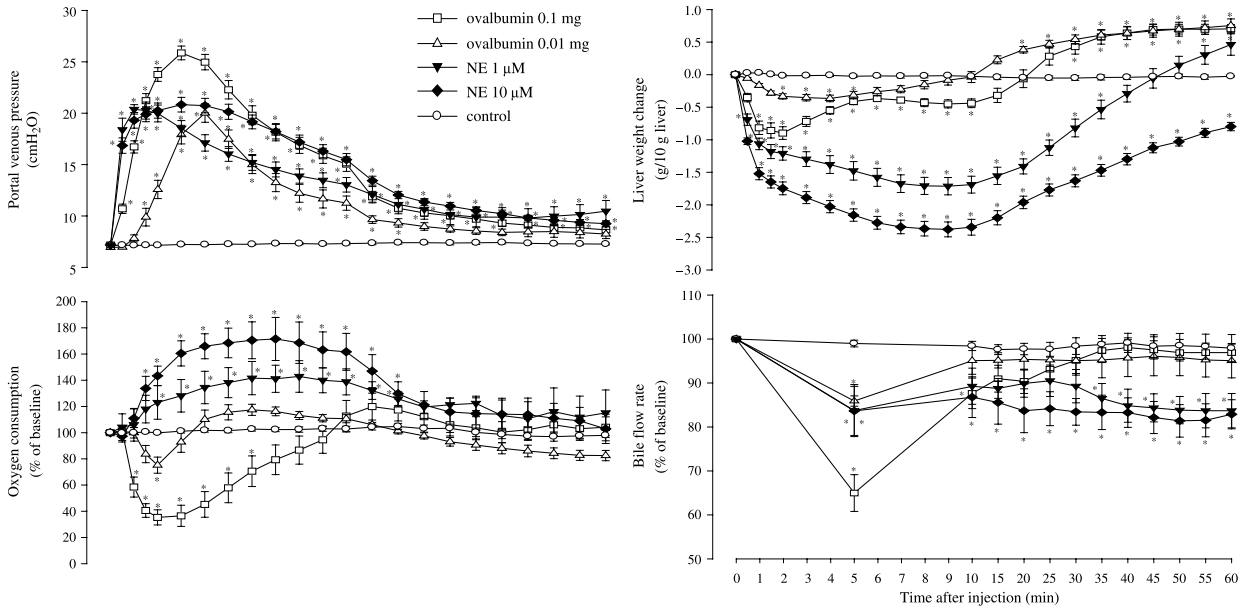

FIG. 5. **The time course changes in the Ppv, liver weight changes, oxygen consumption, and bile flow rate in the 0.1-mg ovalbumin (n = 7), 0.01-mg ovalbumin (n = 7), 1-μmol L^{-1} norepinephrine (n = 7), 10-μmol L^{-1} norepinephrine (n = 8), and control (n = 8) groups.** Values are given as means ± SE. *$P < 0.05$ versus the baseline. NE indicates norepinephrine.

increased hepatic oxygen consumption in isolated blood-perfused livers. This effect is ascribed to the metabolic effect of norepinephrine; norepinephrine is known to increase glycogenolysis using energy, which is reflected by an increase in oxygen consumption. Indeed, norepinephrine increases oxygen consumption of isolated cultured hepatocytes (17).

In the present study, in contrast to norepinephrine administration increasing hepatic oxygen consumption, anaphylaxis obviously reduced hepatic oxygen consumption (35% and 75% of baseline with ovalbumin at 0.1 and 0.01 mg, respectively). This finding is consistent with the investigation of Hines and Fisher (7), in which hepatic oxygen consumption initially increased and then largely decreased to about 24% during the antigen infusion. However, their experimental method was different from that of this study: In their study, the isolated rat livers were perfused with blood-free solutions in a nonrecirculating manner, whereas the livers in the present study were perfused more physiologically with blood. Another difference is that oxygen consumption was determined by measuring only the effluent oxygen concentration with an oxygen electrode in the previous study, whereas it was measured by directly determining the portal–hepatic venous oxygen saturation difference with the oxygen absorption spectrophotometer in the present study.

Hepatic oxygen consumption is regulated by the factors that affect metabolic and hemodynamic components of the liver. Thus, metabolic and hemodynamic factors may be involved in the anaphylaxis-induced decrease in oxygen consumption observed in the present study. With respect to the metabolic factors, pharmacologically active mediators, such as leukotrienes, thromboxane A_2 (TXA_2), platelet-activating factor, histamine, and serotonin are released during anaphylactic reaction. These mediators could directly inhibit oxygen consumption. Indeed, leukotriene C_4 and D_4 reduced oxygen consumption by 29% ± 3% and 30% ± 3% of baseline, respectively, and these inhibitory effects were independent of

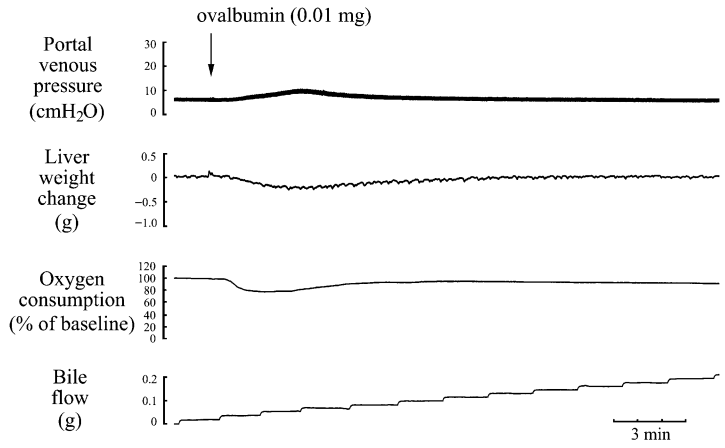

FIG. 6. **A representative recording of the responses to the antigen of ovalbumin (0.01 mg) after pretreatment with SNP at 100 μmol L^{-1}.**

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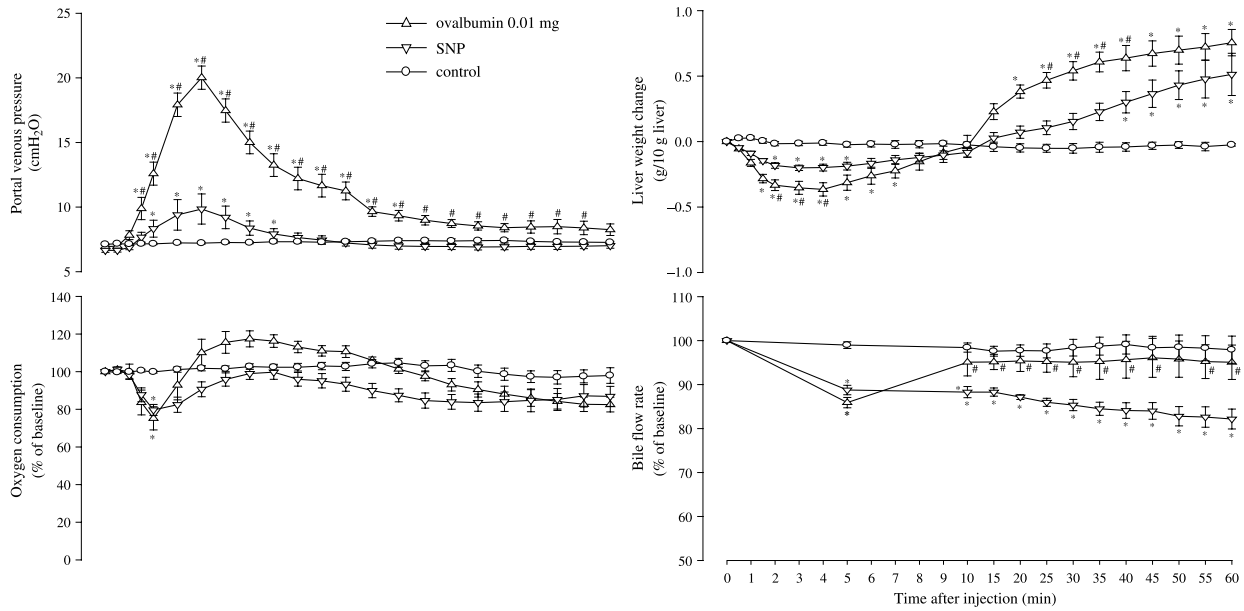

FIG. 7. **The time course changes in the Ppv, liver weight changes, oxygen consumption, and bile flow rate in the 0.01-mg ovalbumin (n = 7), SNP (n = 7), and control (n = 8) groups.** Values are given as means ± SE. $^{*}P < 0.05$ versus the baseline, $^{\#}P < 0.05$ versus SNP group.

changes in hemodynamic factors (13). U-46619, a TXA_2 analogue, also decreased oxygen consumption by 27% in perfused rat livers (13). Altin and Bygrave (14) also found that ONO-11113, another TXA_2 analogue, induced an initial small transient stimulation followed by a marked and sustained inhibition of oxygen uptake in perfused rat livers. Finally, platelet-activating factor increased glucose production concomitant with suppression of oxygen consumption in perfused rat livers (15). On the other hand, from the hemodynamic point of view, presinusoidal constriction observed during anaphylactic venoconstriction of the perfused sensitized rat livers (11) may account for decreased oxygen consumption—presinusoidal constriction caused a decrease in hepatic vascular surface area, which might result in a decrease in the number of active hepatocytes. However, even when anaphylactic hepatic venoconstriction to be induced by 0.01 mg of ovalbumin was substantially suppressed by pretreatment with SNP, a potent vasodilator, the anaphylaxis-induced decrease in oxygen consumption was still preserved (Fig. 7). Thus, although hemodynamic changes might partially contribute to the anaphylaxis-induced decrease in oxygen consumption, the contribution of the hemodynamic effect seems to be small.

To the best of our knowledge, this is the first study to measure oxygen consumption of perfused rat liver using absorption spectrophotometer, although this method has been well adopted for isolated perfused heart experiments (18, 19). Kameyama et al. (18) reported that the basal oxygen consumption of isolated perfused rat hearts measured with an absorption spectrophotometer (AVOX system) was 0.65 to 0.70 μL g^{-1} $beat^{-1}$ (about 0.16 mL min^{-1} per gram of heart weight). Oxygen consumption of isolated perfused rat hearts measured with an oxygen content difference analyzer (model PWA-200S, Shoe Technica, Chiba, Japan) was about 0.5 μL g^{-1} $beat^{-1}$ (0.15 mL min^{-1} per gram of heart weight) (19). The basal oxygen consumption of the present perfused rat livers calculated from the ODM values was 0.0054 to 0.0078 mL min^{-1} per gram of liver. Thus, these values are approximately one twentieth of those of perfused hearts (18, 19). This difference in oxygen consumption between heart and liver was also observed when a Clark-type oxygen electrode was used for the perfused rat heart (20–23) and perfused rat liver (8, 24–27): 28 to 49 μmol L^{-1} min^{-1} per gram of heart (20–23) and 1.78 to 3.0 μmol L^{-1} min^{-1} per gram of liver (8, 23–26), respectively. This indicates that the absorption spectrophotometer used in the present study can be used to measure oxygen consumption continuously in isolated perfused liver experiments.

In the present study, the bile flow rate decreased after norepinephrine at concentrations of 1 and 10 μmol L^{-1}. The reason for this decrease is not known, but it seems to be secondary to the hemodynamic effects (28). In hepatic anaphylaxis, the bile flow rate also reduced significantly at 5 min after antigen administration, even when the hemodynamic effect was eliminated by SNP. We therefore speculate that it is related to the reduction of hepatocyte activity, as reflected by a decrease in oxygen consumption during anaphylaxis.

In summary, we measured the changes in oxygen consumption during hepatic anaphylaxis and norepinephrine administration using a custom-made absorption spectrophotometer in isolated perfused rat livers. Hepatic venoconstriction and liver weight loss were similarly observed in response to norepinephrine and anaphylaxis. However, hepatic anaphylaxis caused a decrease in oxygen consumption, whereas norepinephrine increased oxygen consumption, as reported previously. Pretreatment with a vasodilator of SNP substantially attenuated anaphylaxis-induced venoconstriction, but not anaphylaxis-induced decrease in oxygen consumption. We conclude that local hepatic anaphylaxis decreases oxygen consumption independently of venoconstriction in isolated perfused rat livers.

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