THE EFFECT OF THE INTERRUPTION OF THE HEPATIC ARTERY ON THE PORTAL CIRCULATION AND ON THE OXYGEN CONTENT OF PORTAL BLOOD IN ASCITIC DOGS

By

Shigetoki Ito

2nd. Department of Surgery, Kanazawa University Medical School
(Director : Prof. Dr. Ichio Honjo)

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I. INTRODUCTION

Concerning the influence of the interruption of the hepatic artery upon the liver that receives both arterial and portal blood supply, Haberer[20] (1905) reported that the interruption of the hepatic artery always resulted in death of dog, cat and rabbit due to liver necrosis when the interruption of the hepatic artery was performed at a distal point from the bifurcation of the right gastric artery.

Huggins and Post[26] (1937) reported that the simultaneous interruption of the common hepatic artery, gastroduodenal artery and right gastric artery resulted in death of dogs due to liver necrosis within three days.

Whereas, Markowitz, Rappaport and Scott[32,33] (1949) successfully diminished the mortality in dogs after the interruption of the hepatic artery by an administ-

ration of penicillin as low a level as 35 per cent which had been 100 per cent previously. They thought the antibiotic action of penicillin prevented the growth of bacteria which in untreated animals was the supposed cause of death. This conception has been ascertained to be true by many investigators as GRINDLAY, MANN and BOLLMAN; FRASER, VUYLSTEKE and COLWELL[18]; TANTURI, SWIGART and CANEPA[49] and CHAU, GOLDBLOOM and GURD[12].

POPPER, JEFFERSON and NECHELS[38),39),40] reported that the animal could not survive despite the administration with antibiotic of any kind, when the interruption was performed upon all the arteries and arterial collateral vessels which streamed into the liver.

Furthermore, EZE[17] reported that the administration of penicillin is effective for protecting the liver from bacterial proliferation only for the period spent in forming arterial collateral vessels. GRINDLAY, MANN and BOLLMAN[10] reported that the development of liver necrosis has an intimate relationship to the collateral arterial vessels between the diaphragm and the liver.

Whereas, TANTURI, SWIGART and CANEPA[49]; CHAU, GOLDBLOOM and GURD[12] and ISHIGURO[27] reported that it was impossible to demonstrate the existence of any collateral vessels which adequately explain particularly the survival of short period directly after the interruption of the hepatic artery.

ELLIS and DRAGSTEDT[16] (1930) investigated histologically the cause of liver necrosis following the interruption of the hepatic artery, and observed a proliferation of anaerobic spore-bearing bacterium, commonly present in the liver of dog, in the hypoxic and necrotic areas. TANTURI and others (1950)[49] discovered an enzyme "Lecithinase" in the ascites of the dog died after the interruption of the hepatic artery. He identified thise nzyme with α-toxin of an anaerobic bacterium "Clostridium Perfringens" and assumed it to be an immediate cause of death after the interruption of the hepatic artery.

HONJO[22),23),24] and his associates demonstrated the cause of death following the interruption of the hepatic artery should be attributable to anoxic liver necrosis due to the disturbance of portal circulation after the interruption and consequent proliferation of anaerobic bacteria, and he reported that the disturbance of portal circulation greatly participated with the mobilization of liver ferritin into the blood.

Based on the early studies of HERRICK[21] and MCINDOE[35] concerning the intrahepatic circulation in the cirrhotic liver, RIENHOFF[42),43] and BERMAN[4),5),6),7] (1951), in experiments with portal hypertension in dogs, confirmed that ligation of the splenic and hepatic arteries produced a sustained fall of portal pressure and advocated an interruption of the hepatic artery as a treatment of liver cirrhosis. Thenceforth, CHENOWETH[13], KNEPPER, IDE, RIDDELL, MCDANIEL[31] and others reported favorable results of this procedure in human cirrhosis with ascites.

On the contrary, ROSENBAUM and EGBERT[44] reported a case in which the interruption of the hepatic artery resulted in death due to liver necrosis after two days. JAHNKE, SEELEY and PALMER[29] reported that this procedure seemed to be ineffective, judging from the observation by esophagoscope and the measurement of pressure

in esophageal varix. Taylor and Rosenbaum[50] reported that no descension of portal pressure could be observed by this procedure and noticed it clinically rather dangerous.

Furthermore, according to the clinical observation of their four cases, Desforges, Campbell and Robbins[15] reported that the interruption of the proper hepatic artery has but little significance. Generally speaking, this procedure of the interruption has been deemed to be dangerous because of fear for development of liver necrosis and has not yet obtained an established estimation.

On the other hand, it is widely noticed that the interruption of the hepatic artery in normal dogs results in extensive liver necrosis, while the procedure in ascitic dogs with cirrhotic change in the liver does not. In other words, there must be a difference of circulation between ascitic dogs and normal dogs, and the influence of the interruption of the hepatic artery upon ascitic dog should not be discussed similarly to that upon normal one. Therefore, it is seemed to be worth while to investigate thoroughly about efficiency of the interruption of the hepatic artery as a treatment of liver cirrhosis.

Adachi[1] reported that the oxygen content of portal blood in ascitic dog showed a continuous increase after the interruption of the hepatic artery reaching close to that of arterial blood, while in normal one it showed a temporary increase after the interruption, which shortly followed by a rapid and further gradual decrease.

In order to investigate the mechanism which causes the increase of oxygen content in portal blood and at the same time to ascertain the results reported by Adachi, the present author carried out the following experiments.

Ⅱ. PRODUCTION OF ASCITIC DOGS

Adult mongrel dogs weighing about 10kg were employed regardless of sexes. According to the method of McKee[31], under the anesthesia with intravenous injection of 'Pentobarbital' (20mg/kg). An endotracheal tube was inserted and the thorax was opened on the right VI intercostal space, when the artificial respiration with absolute oxygen was began. Then the inferior vena cava was separated from the right phrenic nerve and surrounding tissues at 3 cm distant point from the diaphragm under positive pressure respiration. The inferior vena cava was constricted with a cellophane tape of 1.5 cm in width. After the operation 100,000 units of aqueous penicillin was administered into the thoracic cavity. The attention was paid to prevent the pneumothorax by removing residual air in the thoracic cavity.

Ascites was observed about a week after the constriction and the dogs were not employed for the experiments until the amount of the ascites increased as much as 4,000 cc to 5,000 cc about a month after the constriction.

Ⅲ. THE INTERRUPTION OF THE HEPATIC ARTERY

Employing the method of Urabe[49] and Ishiguro[7], under the anesthesia with intravenous injection of Pentobarbital (20mg/kg), the laparotomy was performed in sterile conditions through median abdominal incision and the three arteries —— the

common hepatic, gastroduodenal and right gastric artery —— were simultaneously ligated and divided.

Ⅳ. MACROSCOPIC OBSERVATION OF THE LIVER AFTER THE LIGATION OF THE HEPATIC ARTERY

The liver of normal dogs showed a change of the color on its surface immediately after the ligation of the hepatic artery, which turned into dark red or indigo purple and showed a tendency to localize in certain areas 3 hours after surgery and this localized change was definitely accomplished 12 hours after the operation. This localizing change in color was supposed to be due to congestion and appeared in the areas where liver necrosis was apt to develop after ligature of the hepatic artery. In ascitic dogs produced by the constriction of the inferior vena cava, the change of the color and the localizing stasis as mentioned above could not be observed as was in the normal.

Ⅴ. DETERMINATION OF OXYGEN CONTENT OF VENOUS BLOOD IN THE SUPERIOR MESENTERIC AND SPLENIC VESSELS AND OF ARTERIAL BLOOD AFTER THE INTERRUPTION OF THE HEPATIC ARTERY.

1) Materials and Methods

The blood gas can be quantified either through the content, through the partial pressure or through the saturation. In the present experiment, however, the content, as the most elemental of these, was determined by the manometric method of van Slyke-Neil[55].

Blood samples were taken, by syringe, from the branches of the superior mesenteric vein or splenic vein periodically, i. e. before the interruption and 15 minutes, 60 minutes, 180 minutes and 360 minutes after the interruption. As an anticoagulant, a Heparin solution of 2 mg/cc was used. The syringe was coated its inside with fluid paraffin, lest the blood should have contact with oxygen. The blood was shut up in the tube that contained fluid paraffin more than 2 cm in depth.

The femoral arterial blood was substituted for the superior mesenteric arterial blood and splenic arterial blood.

The oxygen content was determined in each 10 dogs of the normal and the ascitic groups.

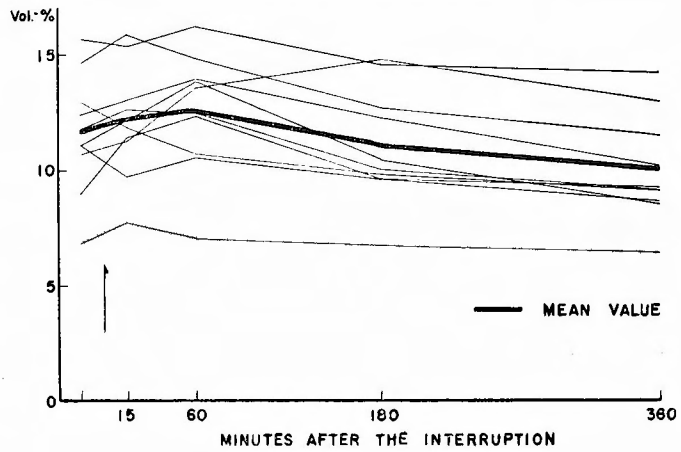
2) Results

i. Changes in oxygen content of superior mesenteric venous blood

a. The group of normal dogs (Fig. 1, Tab. 1)

The values were as follows: Before the interruption it ranged from 6.8 to 15.7, on the average 11.6 volume per cent. Fifteen minutes after the interruption it ranged from 7.7 to 15.8, on the average 12.1 volume per cent. Sixty minutes after the interruption it ranged from 7.0 to 16.2, on the average 12.5 volume per cent. One hundred and eighty minutes after the interruption it ranged from 6.7 to 14.7,

Fig. 1 Changes in oxygen content of superior mesenteric vein blood after interruption of hepatic artery in normal dogs.



Tab. 1 Changes in oxygen content of superior mesenteric vein blood after interruption of hepatic artery in normal dogs. (vol.%)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
1	F.	8.5	11.1	12.1	13.8	10.4	8.5
2	M.	10.5	15.7	15.3	16.2	14.5	14.2
3	F.	7.5	9.0	11.4	12.3	9.6	8.6
4	F.	9.0	11.8	12.6	12.4	10.0	9.1
5	M.	9.5	14.6	15.8	14.8	12.6	11.5
6	M.	7.0	6.8	7.7	7.0	6.7	6.4
7	M.	8.5	12.4	13.0	13.9	12.2	10.2
8	F	8.0	10.7	11.3	13.5	14.7	12.9
9	M.	9.0	13.0	11.9	10.7	9.8	9.1
10	M.	9.5	11.1	9.7	10.5	9.6	9.2
Means			11.6	12.1	12.5	11.0	10.0
S. D.			2.44	2.26	2.47	2.34	2.18

on the average 11.0 volume per cent. Three hundred and sixty minutes after the interruption it ranged from 6.4 to 14.2, on the average 10.0 volume per cent.

To summarize these results, a slight increase was observed at the 15th. minute by 0.5 volume per cent on the average, and at the 60th. minute by 0.9 volume per cent on the average as compared with the average before the interruption. As time went on, the value decreased by −0.6 volume per cent on the average 180 minutes after the interruption and 360 minutes after the interruption, by −1.6 volume per cent on the average compared with that before the interruption.

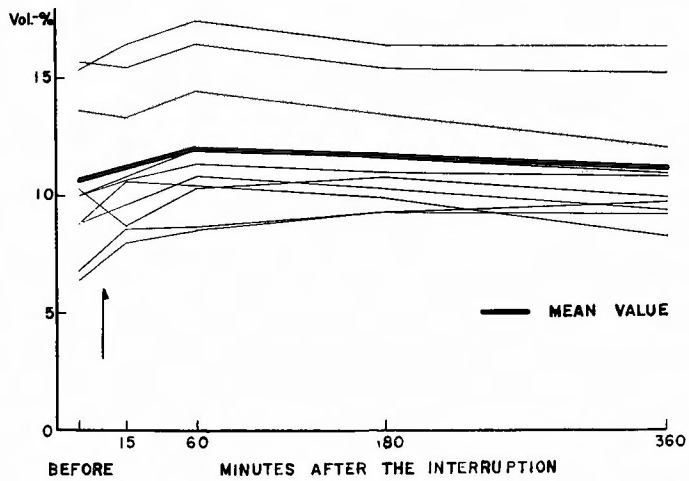
b. The group of ascitic dogs (Fig. 2, Tab. 2)

The values were as follows: Before the interruption it ranged from 6.4 to 15.7, on the average 10.6 volume per cent. Fifteen minutes after the interruption it

Tab 2 Changes in oxygen content of superior mesenteric vein blood after interruption of hepatic artery in ascitic dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
11	M.	8.5	10.0	10.8	12.0	11.6	10.9
12	M.	7.5	6.8	8.6	8.7	9.3	9.7
13	F.	8.0	8.8	10.6	10.4	9.9	8.2
14	M.	12.5	15.3	16.4	17.4	16.4	16.3
15	F.	9.0	10.3	8.7	10.3	10.8	9.9
16	M.	8.5	10.0	10.7	11.3	11.0	10.8
17	M.	12.0	15.7	15.4	16.4	15.4	15.2
18	F.	11.0	13.6	13.3	14.4	13.4	12.0
19	F	10.5	8.8	9.6	10.8	10.3	9.3
20	F	7.0	6.4	8.0	8.6	9.3	9.1
Means			10.6	11.2	12.0	11.7	11.1
S. D.			3.16	2.75	2.90	2.38	2.32

Fig. 2 Changes in oxygen content of superior mesenteric vein blood after interruption of hepatic artery in ascitic dogs.



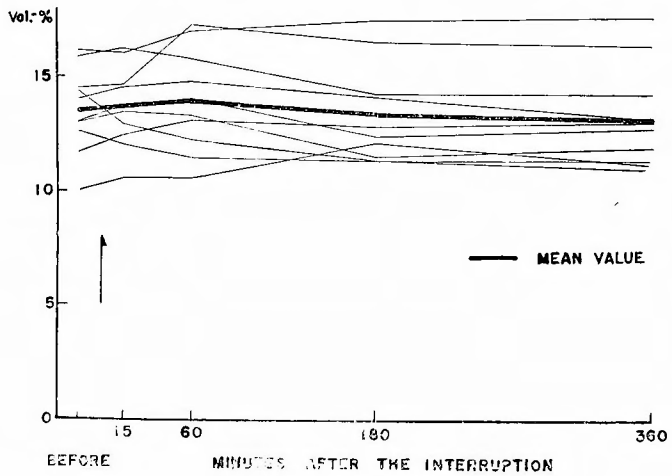
ranged from 8.0 to 16.4, on the average 11.2 volume per cent. Sixty minutes after the interruption it ranged from 8.6 to 17.4, on the average 12.0 volume per cent. One hundred and eighty minutes after the interruption it ranged from 9.3 to 16.4, on the average 11.7 volume per cent. Three hundred and sixty minutes after the interruption it ranged from 8.2 to 16.3, on the average 11.1 volume per cent.

To summarize these results, the value increased at the 15th. minute by 0.6 volume per cent and at the 60th. minute reaching the maximum by 1.4 volume per cent on the average as compared with the value before the interruption, and decreased so gradually thereafter that they were +1.2 volume per cent at the 180th. minute and +0.5 volume per cent on the average at the 360th. minute after the

Table 3 Changes in oxygen content of splenic vein blood after interruption of hepatic artery in normal dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
1	F	8.5	14.0	14.5	14.7	14.0	12.9
2	M.	10.5	16.1	16.0	17.0	17.3	17.4
3	F	7.5	11.7	12.4	13.0	12.7	12.8
4	F	9.0	13.0	13.4	13.2	11.4	11.7
5	M.	9.5	15.8	16.2	15.7	14.1	14.0
6	M.	7.0	10.0	10.5	10.5	12.0	11.0
7	M.	8.5	13.0	13.7	14.0	12.3	12.6
8	F.	8.0	14.5	14.6	17.3	16.4	16.1
9	M.	9.0	14.4	12.9	12.2	11.3	10.8
10	M.	9.5	12.6	12.0	11.4	11.2	11.2
Means			13.5	13.6	13.9	13.3	13.0
S. D.			1.76	1.69	2.17	2.04	2.13

Fig. 3 Changes in oxygen content of splenic vein blood after interruption of hepatic artery in normal dogs.



interruption still higher than that before the interruption. The value maintained an increased level or remained still at the level before the interruption at least 360 minutes after the interruption without showing a decrease of the value lower than that before the interruption as observed in normal dogs.

ii. The splenic venous blood oxygen content.

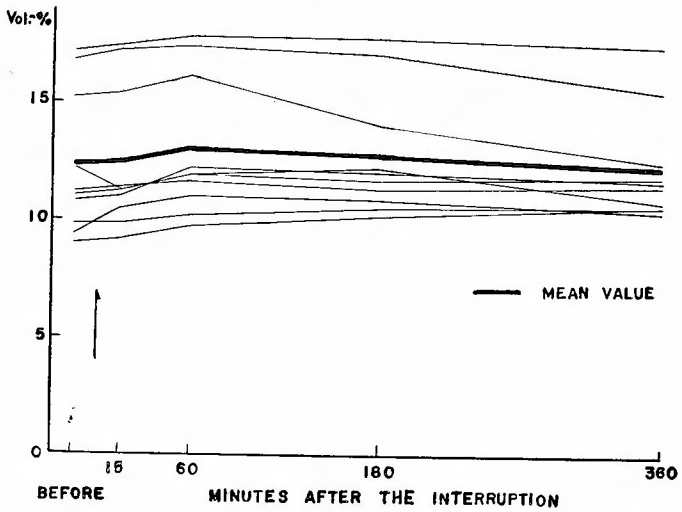
a. The group of normal dogs (Fig. 3, Tab. 3.)

The values were as follows: Before the interruption it ranged from 10 to 16.1, on the average 13.5 volume per cent. Fifteen minutes after the interruption it ranged from 10.5 to 16.2, on the average 13.6 volume per cent. Sixty minutes after the interruption it ranged from 10.5 to 17.3, on the average 13.9 volume per

Table 4 Changes in oxygen content of splenic vein blood after interruption of hepatic artery in ascitic dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
11	M.	8.5	10.8	11.0	12.2	12.0	11.6
12	M.	7.5	9.0	9.2	9.7	10.1	10.5
13	F	8.0	11.2	11.4	11.6	11.3	11.4
14	M.	12.5	17.2	17.4	17.8	17.7	17.3
15	F.	9.0	12.2	11.3	11.9	12.2	10.7
16	M.	8.5	11.0	11.2	11.9	11.7	11.8
17	M.	12.0	16.8	17.2	17.4	17.0	15.4
18	F.	11.0	15.2	15.4	16.1	14.0	12.4
19	F.	10.5	9.4	10.5	11.0	10.8	10.3
20	F.	7.0	9.8	9.8	10.2	10.5	10.5
Means			12.3	12.4	13.0	12.7	12.2
S. D.			2.89	2.89	2.82	2.53	2.22

Fig. 4 Changes in oxygen content of splenic vein blood after interruption of hepatic artery in ascitic dogs.



cent. One hundred and eighty minutes after the interruption it ranged from 11.2 to 17.3, on the average 13.3 volume per cent. Three hundred and sixty minutes after the interruption it ranged from 10.8 to 17.4, on the average 13.0 volume per cent.

That is to say, very slight decrease was observed as compared with that before the interruption at the 15th. minute by +0.1 volume per cent on the average and at the 60th. minute by +0.4 volume per cent on the average, and it decreased very slightly 180 minutes and 360 minutes after the interruption. A conspicuous change with time could not be observed as in the superior mesenteric vein blood.

b. The group of ascitic dogs (Fig. 4, Tab. 4)

Fig. 5 Changes in oxygen content of arterial blood after interruption of hepatic artery in normal dogs.

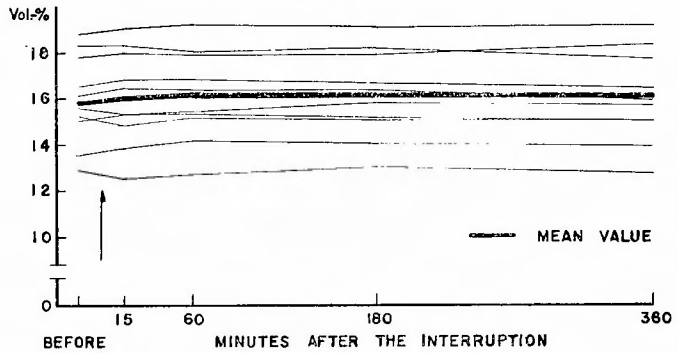


Fig. 6 Changes in oxygen content of arterial blood after interruption of hepatic artery in ascitic dogs.

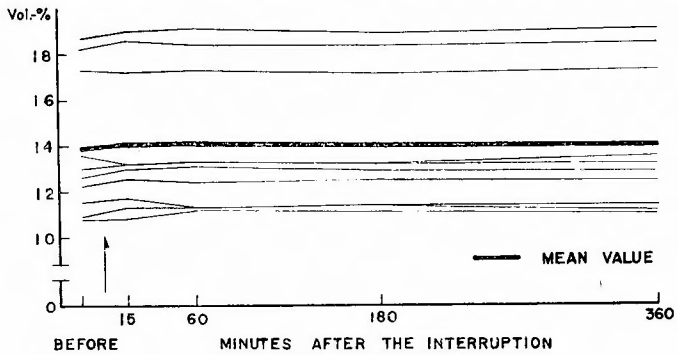


Table 5 Changes in oxygen content of arterial blood after interruption of hepatic artery in normal dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
1	F	8.5	15.0	15.3	15.3	15.1	15.0
2	M.	10.5	18.8	19.0	19.2	19.0	19.1
3	F	7.5	13.5	13.8	14.1	14.0	13.9
4	F.	9.0	16.1	16.4	16.3	16.3	15.9
5	M.	9.5	18.3	18.3	18.0	18.1	17.7
6	M.	7.0	12.6	12.5	12.7	13.0	12.7
7	M.	8.5	15.2	14.8	15.1	15.0	15.0
8	F.	8.0	17.8	18.0	17.9	17.9	18.3
9	M.	9.0	16.5	16.8	16.8	16.6	16.4
10	M.	9.5	15.6	15.3	15.4	15.8	15.7
Means			15.9	16.0	16.1	16.1	16.1
S. D.			1.90	1.95	1.86	1.80	1.88

Table 6 Changes in oxygen content of arterial blood after interruption of hepatic artery in ascitic dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
11	M.	8.5	12.2	12.6	12.4	12.5	12.4
12	M.	7.5	10.9	11.3	11.3	11.4	11.3
13	F.	8.0	13.0	13.2	13.3	13.2	13.1
14	M.	12.5	18.2	18.6	18.4	18.3	18.4
15	F.	9.0	13.6	13.2	13.3	13.2	13.5
16	M.	8.5	12.6	13.0	13.1	12.9	12.8
17	M.	12.0	18.7	19.0	19.1	18.9	19.0
18	F.	11.0	17.3	17.2	17.3	17.1	17.2
19	F.	10.5	11.5	11.7	11.3	12.4	11.2
20	F.	7.0	10.8	10.8	11.2	11.1	11.0
Means			13.9	14.1	14.1	14.0	14.0
S. D.			2.85	2.92	2.88	2.97	2.89

The values were as follows: Before the interruption it ranged from 9.0 to 17.2, on the average 12.3 volume per cent. Fifteen minutes after the interruption it ranged from 9.2 to 17.4, on the average 12.4 volume per cent. Sixty minutes after the interruption it ranged from 9.7 to 17.8, on the average 13.0 volume per cent. One hundred and eighty minutes after the interruption it ranged from 10.1 to 17.7, on the average 12.7 volume per cent. Three hundred and sixty minutes after the interruption it ranged from 10.3 to 17.3, on the average 12.2 volume per cent.

That is to say, the similar tendency as in the group of normal dogs was observed and no marked changes with time could be seen, either.

iii. The arterial blood oxygen content (Fig. 5, 6, Tab. 5, 6)

Oxygen content of the arterial blood before the interruption of the hepatic artery in the group of normal dogs and in the group of ascitic dogs were 15.9 volume per cent and 13.9 volume per cent on the average respectively, each of which had scarcely any marked changes with time.

iv. Difference of oxygen content between arterial and superior mesenteric venous blood

a. The group of normal dogs (Fig 7, Tab. 7)

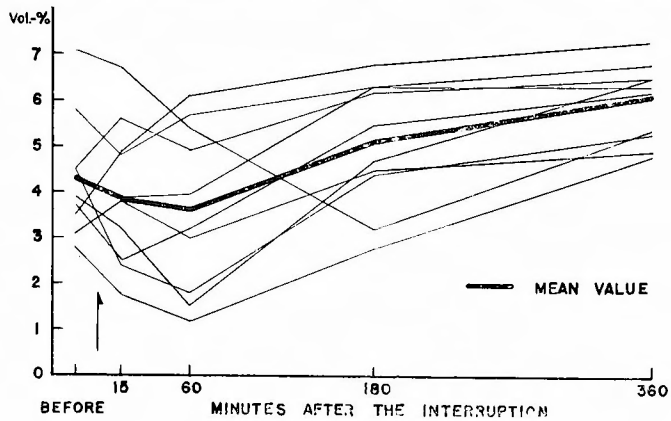
The values were as follows: Before the interruption it ranged from 7.1 to 2.8, on the average 4.3 volume per cent. Fifteen minutes after the interruption it ranged from 6.7 to 1.8, on the average 3.9 volume per cent. Sixty minutes after the interruption it ranged from 6.1 to 1.2, on the average 3.6 volume per cent. One hundred and eighty minutes after the interruption it ranged from 6.8 to 2.8, on the average 5.1 volume per cent. Three hundred and sixty minutes after the interruption it ranged from 7.3 to 4.8 on the average 6.1 volume per cent.

That is to say, as compared with the value before the interruption of 4.3 volume per cent, the values were 3.9 volume per cent at the 15th. minute and 3.6

Table 7 Changes in O_2-difference between arterial and superior mesenteric vein blood after interruption of hepatic artery in normal dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
1	F.	8.5	3.9	3.2	1.5	4.7	6.5
2	M.	10.5	3.1	3.7	3.0	4.5	4.9
3	F.	7.5	4.5	2.4	1.8	4.4	5.3
4	F.	9.9	4.3	3.8	3.9	6.3	6.8
5	M.	9.5	3.7	2.5	3.2	5.5	6.2
6	M.	7.0	5.8	4.8	5.7	6.3	6.3
7	M.	8.5	2.8	1.8	1.2	2.8	4.8
8	F.	8.0	7.1	6.7	5.4	3.2	5.4
9	M.	9.0	3.5	4.9	6.1	6.8	7.3
10	M.	9.5	4.5	5.6	4.9	6.2	6.5
Means			4.3	3.9	3.6	5.1	6.1
S. D.			1.26	1.47	1.72	1.30	0.81

Fig. 7 Changes in O_2-difference between arterial and superior mesenteric vein blood after interruption of hepatic artery in normal dogs.



volume per cent at the 60th. minute showing temporarily near level to that of arterial blood as 0.4 volume per cent and 0.7 volume per cent respectively. The difference, however, increased rapidly as 5.1 volume per cent (0.8 volume per cent) 180 minutes after the interruption, and as 6.1 volume per cent (1.8 volume per cent) 360 minutes after the interruption.

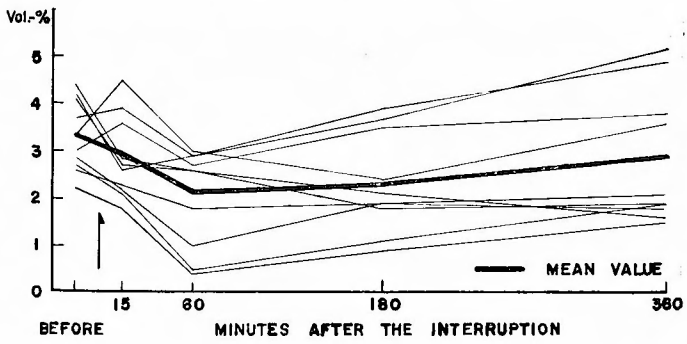
b. The group of ascitic dogs (Fig. 8, Tab. 8)

The values were as follows: Before the interruption it ranged from 4.4 to 2.2, on the average 3.3 volume per cent. Fifteen minutes after the interruption it ranged from 4.5 to 1.8, on the average 2.9 volume per cent. Sixty minutes after the interruption it ranged from 3.0 to 0.4, on the average 2.1 volume per cent. One hundred and eighty minutes after the interruption it ranged from 3.9 to 0.9, on the average 2.3 volume per cent. Three hundred and sixty minutes after the

Table 8 Changes in O_2-difference between arterial and superior mesenteric vein blood after interruption of hepatic artery in ascitic dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
11	M.	8.5	2.2	1.8	0.4	0.9	1.5
12	M.	7.5	4.1	2.7	2.6	2.1	1.6
13	F.	8.0	4.2	2.6	2.9	3.9	4.9
14	M.	12.5	2.9	2.2	1.0	1.9	2.1
15	F.	9.0	3.3	4.5	3.0	2.4	3.6
16	M.	8.5	2.6	2.3	1.8	1.9	1.8
17	M.	12.0	3.0	3.6	2.7	3.5	3.8
18	F.	11.0	3.7	3.9	2.9	3.7	5.2
19	F.	10.5	2.7	2.1	0.5	1.1	1.9
20	F.	7.0	4.4	2.8	2.6	1.8	1.9
Means			3.3	2.9	2.1	2.3	2.9
S. D.			0.72	0.83	0.98	0.99	1.33

Fig. 8 Changes in O_2-difference between arterial and superior mesenteric vein blood after interruption of hepatic artery in ascitic dogs.



interruption it ranged from 5.2 to 1.5, on the average 2.9 volume per cent.

That is to say, the difference of oxygen content between arterial and venous blood in the superior mesenteric vessels in the group of ascitic dogs was less than that in the group of normal dogs either before or after the interruption. The similar comment can be refered to the difference of oxygen content between arterial and venous blood in the splenic vessels.

In the group of ascitic dogs, as compared with the difference of oxygen content between arterial and venous blood in the superior mesenteric vessels before the interruption of 3.3 volume per cent, the difference decreased to 2.9 volume per cent (0.4 volume per cent) on the average 15 minutes after the interruption, to 2.1 volume per cent (1.2 volume per cent) on the average 60 minutes after the interruption, to 2.3 volume per cent (1.1 volume per cent) on the average 180 minutes after the interruption and to 2.9 volume per cent (0.4 volume per cent) on the average 360 minutes after the interruption respectively, being less than the value

Table 9 Changes in O_2-difference between arterial and splenic vein blood after interruption of hepatic artery in normal dogs. (vol. %)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
1	F.	8.5	1.0	0.8	0.4	1.1	2.1
2	M.	10.5	2.7	3.0	2.2	1.7	1.7
3	F.	7.5	1.8	1.4	1.1	1.3	1.1
4	F.	9.0	3.1	3.0	3.1	4.9	4.7
5	M.	9.5	2.5	2.1	2.3	4.0	3.7
6	M.	7.0	2.6	2.0	2.2	1.0	1.7
7	M.	8.5	2.2	1.1	1.1	2.7	2.4
8	F.	8.0	3.3	3.4	0.6	1.5	2.2
9	M.	9.0	2.1	3.9	1.6	5.3	5.6
10	M.	9.5	3.3	3.3	4.0	4.6	4.5
Means			2.4	2.4	2.2	2.8	3.1
S. D.			0.68	1.01	1.34	1.63	1.46

Table 10 Changes in O_2-difference between arterial and splenic vein blood after interruption of hepatic artery in ascitic dogs. (vol .%)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
11	M.	8.5	1.4	1.6	0.2	0.3	0.8
12	M.	7.5	1.9	2.1	1.6	1.3	0.8
13	F.	8.0	1.8	1.8	1.7	1.9	1.7
14	M.	12.5	1.0	1.2	0.6	0.6	1.1
15	F.	9.0	1.4	1.9	1.4	1.0	2.8
16	M.	8.5	1.6	1.8	1.2	1.2	1.0
17	M.	12.0	1.9	1.8	1.7	1.9	3.6
18	F.	11.0	2.1	1.8	0.8	3.1	4.8
19	F.	10.5	2.1	1.2	0.3	0.6	0.9
20	F.	7.0	1.0	1.0	1.0	0.6	0.5
Means			1.6	1.6	1.1	1.3	1.8
S. D.			0.39	0.35	0.52	0.78	1.37

before the interruption. A marked increase as in the group of normal dogs was not to be observed and it maintained a state approaching the level of arterial blood.

v. Difference of oxygen content between arterial and venous blood in the splenic vessels (Fig. 9, 10, Tab. 9, 10)

In the group of normal dogs, the difference of oxygen content between arterial and venous blood in the splenic vessels was 2.4 volume per cent on the average before the interruption, 15 minutes after the interruption 2.4 volume per cent on the average, 60 minutes after the interruption 2.2 volume per cent on the average, 180 minutes after the interruption 2.8 volume per cent on the average and 360

Fig. 9 Changes in O_2-difference between arterial and splenic vein blood after interruption of hepatic artery in normal dogs.

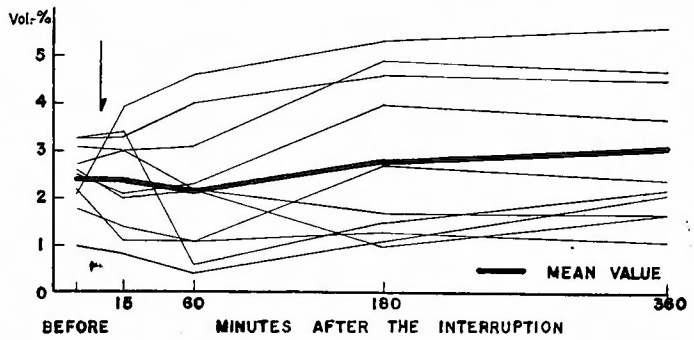
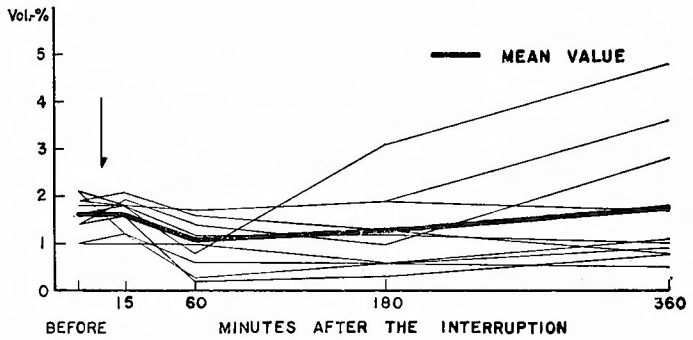


Fig. 10 Changes in O_2-difference between arterial and splenic vein blood after interrupiton of hepatic artery in ascitic dogs.



minutes after the interruption 3.1 volume per cent on the average.

In the group of ascitic dogs, the difference was 1.6 volume per cent on the average before the interruption, 15 minutes after the interruption 1.6 volume per cent on the average, 60 minutes after the interruption 1.1 volume per cent on the average, 180 minutes after the interruption 1.3 volume per cent on the average and 360 minutes after the interruption 1.8 volume per cent on the average.

The difference of oxygen content between splenic arterial and venous blood in ascitic dogs was smaller than in normal dogs. However, the fluctuation of the difference observed to be slight in both groups.

As shown evidently in the above mentioned results, there can be observed a difference, between ascitic dogs and normal ones, in the fluctuation of oxygen content of the superior mesenteric venous blood and in the superior mesenteric arterial-venous blood difference after the interruption of the hepatic artery. What mechanism should cause the difference between two groups? The following experiment was carried out in order to clarify the problem.

VI. DETERMINATION OF THE PORTAL PRESSURE

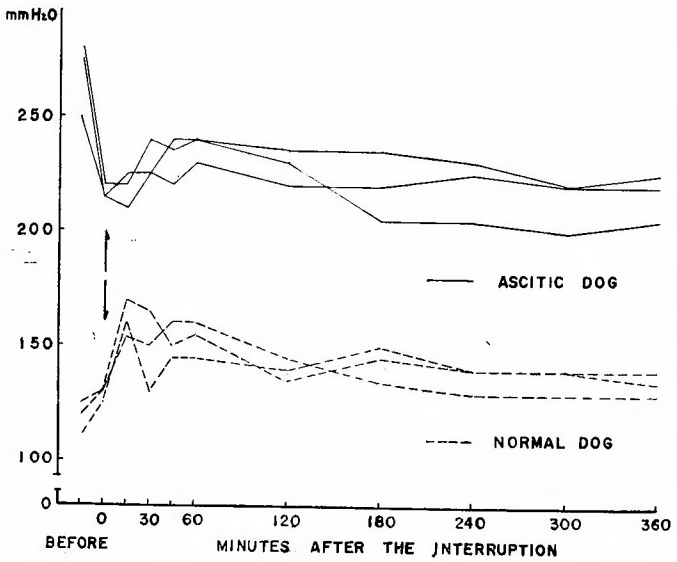
1) Method

The portal pressure was determined before and after the interruption with a

Table 11 Changes in portal pressure after interruption of hepatic artery. (mmH_2O)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)									
				0	15	30	45	60	120	180	240	300	360
NORMAL DOGS													
21	M.	8.0	110	125	160	130	145	145	140	150	140	140	140
22	F.	9.5	120	130	170	165	150	155	135	145	140	140	135
23	F.	7.0	125	130	155	150	160	160	145	135	130	130	130
ASCITIC DOGS													
24	F.	9.0	275	215	225	225	220	230	220	202	225	220	220
25	M.	10.5	250	215	210	225	240	240	230	205	205	200	205
26	M.	8.5	280	220	220	240	235	240	235	235	230	220	225

Fig. 11 Changes in portal pressure after interruption of hepatic artery.



vinyl tube inserted into the portal trunk from the gastro-splenic vein.

2) Results

a. The group of normal dogs (Fig. 11, Tab. 11)

The pressure showed a rapid increase at the 15th. minute after the interruption from 30 to 50 mmH$_2$O, 43 mmH$_2$O on the average and thereafter decreased so gradually that 360 minutes after the interruption it remained 5 to 30 mmH$_2$O higher, 17 mmH$_2$O on the average still 360 minutes after the interruption.

b. The group of ascitic dogs (Fig. 11, Tab. 11)

The pressure fell rapidly for 15 minutes following the interruption from 40 to 60 mmH$_2$O, 50 mmH$_2$O on the average, lower than the level before the interruption, and showed a slight and temporary increase, remaining still 45 to 55 mmH$_2$O, 51 mmH$_2$O on the average lower than the level before the interruption even 360 minutes after the interruption.

Ⅶ. DETERMINATION OF THE HEMATOCRIT RATIO

1) Method

The blood sample was taken from a branch of the superior mesenteric vein. Potassium oxalate of 2 to 3 mg/cc was used as an anticoagulant. The WINTROBE's tube was employed and for the determination it was centrifugated at a frequency of 3,000 r.p.m. for 30 minutes.

2) Results

a. The group of normal dogs (Fig. 12, Tab. 12)

As compared with the value before the interruption, Hematocrit (abbreviated to Ht.) ratios increased 1 per cent to 6 per cent, +4 per cent on the average at the 15th. minute, 5 to 12, +8.3 per cent on the average at the 60th. minute, 9 to 14, +12.0 per cent on the average at the 180th. minute, and 11 to 15, +13.3 per cent on the average at the 360th. minute after the interruption.

The ratio thus maintained its progressive increase after the interruption.

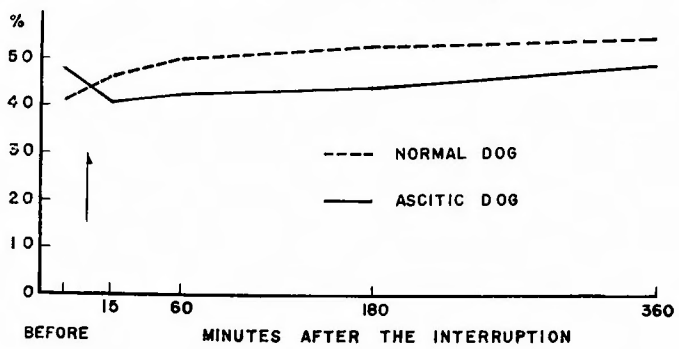
b. The group of ascitic dogs (Fig. 12, Tab. 12)

As compared with the levels before the interruption, the difference of Ht. ratio ranged 0 to −16, −6.7 per cent on the average at the 15th. minute, +4 to −12, −4.5 per cent on the average at the 60th. minute, +4 to −12, −4 per cent on

Table 12 Changes in Ht ratio after interruption of hepatic artery. (%)

Dog No.	Sex	Weight kg	Before Interruption	After Interruption (Minutes)			
				15	60	180	360
NORMAL DOGS							
27	F.	12.0	48	49	53	57	59
28	F.	9.5	40	46	48	53	55
29	M.	10.5	36	42	48	50	50
ASCITIC DOGS							
30	F.	13.0	48	32	36	36	49
31	F.	11.5	50	46	44	46	48
32	F.	10.0	45	45	49	49	50

Fig. 12 Changes in Ht ratio after interruption of hepatic artery.



the average at the 180th. minute and +5 to −2, +1 per cent on the average at the 360th. minute after the interruption.

A rapid decrease in the Ht. ratio was observed 15 minutes after the interruption, and the ratio increased thereafter gradually, and it still remained at the level near to the initial 360 minutes after the interruption, showing no progressive increase as observed in the group of normal dogs.

Ⅷ. HISTOLOGICAL EXAMINATION OF CIRCULATION IN PERIPHERAL VESSELS OF THE INTESTINE

1) The Submucous Plexus of the Intestine

i. Materials and Methods

A suspension of rice starch granules (20 gm/100cc) was injected into the superior mesenteric artery. The suspension was prepared according to the method of SCHARRER[45], suspending rice starch granules 4 to 5μ in diameter in basic perfusate of 5 per cent gelatin solution of body temperature. Immediately after the injection, the frozen section of intestinal wall was made which was cut approximately 10μ thick and stained with Lugol's solution, and examined by microscope.

The vessel is scarcely filled with this perfusate, when its diameter is less than 10μ.

ii. Results

The submucous plexus in the intestine of the ascitic dogs was observed to be dilated and tortuous (Fig. 11,) as compared with that of the normal (Fig. 13).

2) Circulation in the Intestinal Capillaries

i. Materials and Methods

In order to clarify the microscopic appearance of finer capillaries, physiological saline, added with a 25 per cent india ink, was injected into the superior mesenteric

Fig. 13 Normal Dog

Fig. 14 Ascitic Dog

Normal Dog

Fig. 15 Before Interruption.

Fig. 16 One Hour after Interruption.

Fig. 17 Six Hours after Interruption.

Ascitic Dog

Fig. 18 Before Interruption.

Fig. 19 One Hour after Interruption.

Fig. 20 Six Hours after Interruption.

artery, and the examination was performed following the lapse of time; before, 60 minutes, and 360 minutes after the interruption of the hepatic artery with the same procedure as was carried out after the injection of the starch gelatin perfusate.

ii. Results

a. Normal dogs (Figs. 15, 16, 17)

The intestinal capillary system presented an appearance of stagnation increasing with time after the interruption.

b. Ascitic dogs (Fig. 18, 19, 20)

The capillary system did not show an appearance of increasing stagnation after the interruption, but rather decreasing.

IX. DISCUSSION

The fact that the portal blood contains more plenty oxygen than the peripheral venous blood has already been reported by BRADLEY[10], SMYTH[11], KIMOTO[12] and others. Many reports have been accumulated as to the blood flow volume of the hepatic artery and portal vein, which supply the liver with oxygen.

According to UEDA[51)52)], 23 per cent of the total liver flow is supplied by the hepatic artery, while 77 per cent by the portal vein. BLALOCK[9] reported that 80.5 per cent of the total liver flow is supplied by the portal vein and Barcroft and Shore insisted that the hepatic artery supplies 34 per cent of the total liver flow.

On the other hand, as to the oxygen supply, SCHWIEGK[46] reported that the liver should receive 40 per cent of the total oxygen consumed in itself from the hepatic artery, while UEDA[51)52)] described that 72 per cent of its total oxygen consumption should depend upon the portal vein. It is generally thought that the hepatic artery chiefly supply the liver with oxygen and the portal vein participates in metabolism by supplying it with materials to be synthesized or decomposed.

The importance of the portal vein as a source of oxygen supply is also apparent. What should cause such characteristics of the portal blood concerning its oxygen content? It is noteworthy in the present experiment that the splenic venous blood shows a extremely high level of oxygen content. That is to say, in 10 of normal dogs the splenic arterial-venous blood oxygen difference being 2.4 volume per cent on the average was apparently less than the superior mesenteric arterial-venous blood oxygen difference of 4.3 volume per cent on the average. Considering that the mesenteric venous system and the splenic venous system mingle together into the portal trunk, it is obvious that the oxygen content of the portal trunk should show an intermediate level of those of the mesenteric venous system and the splenic venous system. The spleen consumes so little oxygen in the splenic arterial blood and send it into the splenic vein that the splenic vein might be assumed from a point of view of oxygen supply to play a role of an artery for the liver.

RIENHOFF[42)43)] successfully lowered the portal pressure by the interruption of the hepatic artery and applied this procedure to the treatment for ascites due to liver cirrhosis. Furthermore, he reported that the descension of portal pressure is promoted by adding the interruption of the splenic artery to the above mentioned procedure. This promoting effect can be clearly explained from the fact that approximately 30 per cent of the total blood flow carried through the portal system is depended upon the spleen. Solely from the point of view of oxygen supply of the portal venous blood to the liver, however, the interruption of the splenic artery together with the hepatic artery accompanies the fear that the interruption should cause a considerable decrease in oxygen content of the portal blood because of the high value of oxygen contet of the splenic venous blood. Thus the concurrent interruption of the

splenic vein involves an important problem to be solved further.

Many investigatotors have reported about oxygen content of portal blood and difference of oxygen content between arterial and portal blood. BRADLEY[10] reported the value of the difference of oxygen content to be 0.93 volume per cent, which was evaluated at operation of a liver cirrhosis and a portal hypertension. KIMOTO[30] reported it to be 1.4 volume per cent on the average of two cases of liver cirrhosis and a BANTI's syndrom. Here he pointed out that the difference of oxygen content between arterial and portal blood is less in portal hypertension than in the normal.

Also in the results of the present experiment, the difference of oxygen content of the mesenteric vessels was 4.3 volume per cent on the average in the group of normal dogs, while in the group of ascitic dogs it was 3.3 volume per cent on the average, and the difference of oxygen content of the splenic vessels was 2.4 volume per cent on the average in the group of normal dogs and in the group of ascitic dogs it was 1.6 volume per cent on the average, revealing a similar tendency.

As the cause of this fact, BRADLEY[11] has suggested the retrograde flow of hepatic arterial blood into the portal system in portal hypertension. UEDA[53] has assumed the opening of the arterio-venous shunt or an increase of its frequency judging from the fact that the difference of oxygen content between arterial and venous blood in upper extremities is less in liver cirrhosis than in the normal.

The author of the present experiment also observed, as shown in the figures (Figs. 13, 14), that in the intestine of the ascitic dog, compared with the normal, the submucous plexus was more dilated and more tortuous. This finding enables a presumption that in the ascitic dogs the portal pressure is increased by the constriction of the inferior vena cava and for its comparatively long duration the intestinal arterio-venous shunt would be caused to increase the frequency to open and dilate, and thus the difference of oxygen content between arterial and portal blood is less in the ascitic dogs than in the normal.

Fujo and his associates made a series of experimental studies in order to clarify the reason why the liver necrosis seldom develops by the interruption of hepatic artery in the ascitic dogs and obtained the results as follows: NAKASE[37], one of his associates reported that the amount of liver ferritin markedly decreased in the ascitic dogs, which showed very slight decrease by the interruption of the hepatic artery and the localizing stagnation of the portal circulation scarcely occurred after the interruption. YAMABE reported of 7 ascitic dogs similar to cirrhosis that did not show development of liver necrosis regardless of the administration of penicillin, and activity of lecithinase C was also proved to be negative. They insisted that ascitic dogs have a resistance against the interruption of the hepatic artery without the administration of penicillin, revealing the possibility to survive well.

MIYAZAKI[36] reported that the blood ammonia level in the ascitic dogs stood around the value before the interruption except the temporary increase during the periods from 3 to 6 and 9 to 21 hours after the interruption and concluded that hepatic parenchymal damage due to the disturbed circulation of the portal vein after the interruption of the hepatic artery did not occur easily in ascitic dogs.

Hosono[25] reported that the velocity of the blood flow through the portal system increases in normal dogs immediatey after the interruption of the hepatic artery and comes back to the level before the interruption 5 to 20 minutes later, then decreases gradually thereafter. On the contrary, in the ascitic dogs the velocity of blood flow through the portal system that shows an increase immediately after the interruption does not decrease to the level before the interruption, but remains at an increased level.

Adachi[1] reported that in the ascitic dogs oxygen content of the portal venous blood increases remarkably and continuously after the interruption of the hepatic artery and consequently it comes near to the level of arterial blood. Whereas, in normal dogs the oxygen content of the portal blood shows a temporary increase after the interruption which is followed shortly by a decrease with further tendency of descension.

In the results of the present experiment, in normal dogs the oxygen content of the superior mesenteric venous blood showed a temporary increase until 60 minutes after the interruption then decreased lower than the level before the interruption 360 minutes after the interruption. On the contrary, in the ascitic dogs it showed the similar increase, as mentioned above, after the interruption which was followed by a extremely gradual decrease, remaining higher than the level before the interruption even 360 minutes after the interruption, without showing a marked decrease lower than before the interruption as observed in normal dogs (Fig. 21.)

The observation of the difference of oxygen content of superior mesenteric vessels also revealed the similar tendency. Namely, in normal dogs the difference temporarily decreased after the interruption which was followed by a rapid increase. On the contrary in the ascitic dogs the difference was less still 360 minutes after the interruption than that before the interruption (Fig. 22.)

Furthermore, the results obtained from the determination of portal pressure also showed a rapid increase in normal dogs followed by tendency of gradual decrease,

Fig. 21 Changes in oxygen content of superior mesenteric vein blood after interruption of hepatic artery.

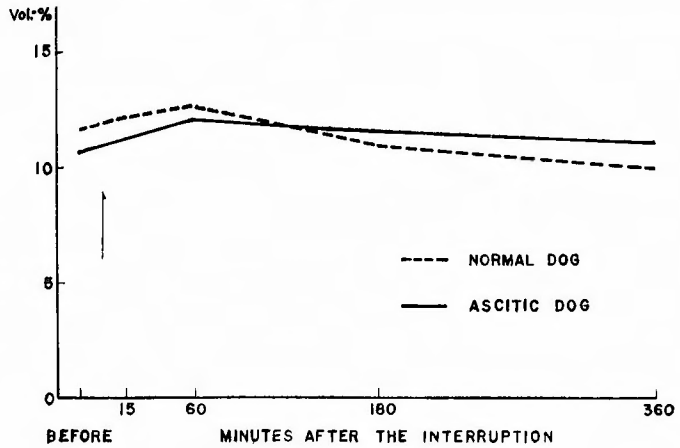
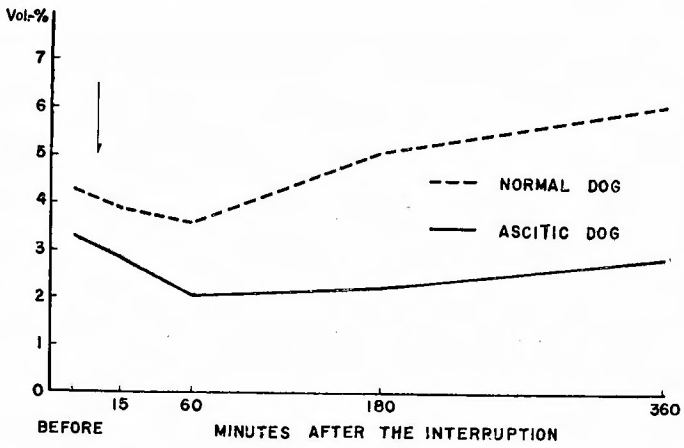


Fig. 22 Changes in O_2-difference between arterial and superior mesenteric vein blood after interruption of hepatic artery.



and it remained in a higher level than before the interruption. On the contrary, in ascitic dogs the pressure rapidly decreased after the interruption, maintaining a lower level than before the interruption.

As to Ht. ratio, it gradually increased on in normal dogs after the interruption, while the ratio decreased after the interruption in ascitic dogs, then it gradually increased up to the level before the interruption.

The injection of india ink revealed the fact that the stagnation increased in the submucous plexus in normal dogs, while in ascitic dogs the stagnation decreased after the interruption.

That is to say, the portal circulatory disturbance strongly develops in normal dogs after the interruption of the hepatic artery, which incurs stasis of portal system, decrease of circulating blood volume and rapid increase of portal pressure. At first, the oxygen content of portal venous blood temporarily increases with the above mentioned increase of Ht. ratio. Here the circulation insufficiency of the whole body develops, which advances to the shock-like condition. Then, oxygen content conspicuously decreases by the continuously existing portal circulatory disturbance. This finding also accords with that of increasing stasis in the submucous plexus of the intestine. On the contrary, in the ascitic dogs the increase in circulating blood volume, decrease in portal pressure, decrease in Ht. ratio and decrease in stasis in the capillaries were observed after the interruption of the hepatic artery. This finding suggests the lessening in portal circulatory disturbance in the ascitic dogs, and owing to the recovery of portal circulation the oxygen content of the superior mesenteric vessels and further that of the portal trunk comes to increase, at least, without showing a conspicuous decrease as observed in normal dogs. In both normal and ascitic dogs, the fluctuation of oxygen content in the splenic vein after the interruption of the hepatic artery is much less than that in the mesenteric vein. While there can be observed a definite difference of the attitude of the oxygen content of the superior mesenteric vein after the ligation between normal and ascitic

Fig. 23 Changes in oxygen content of splenic vein blood after interruption of hepatic artery.

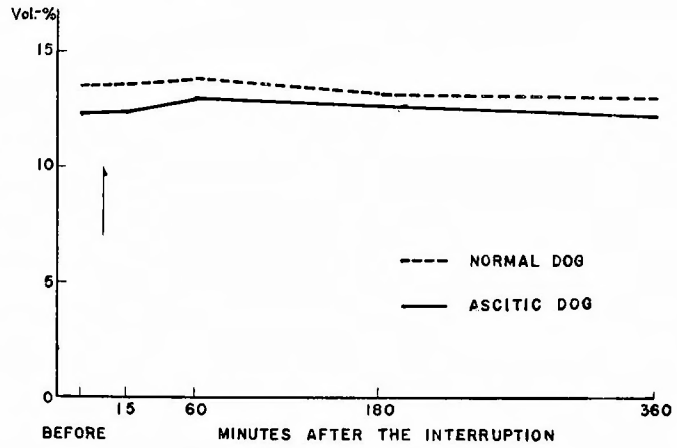
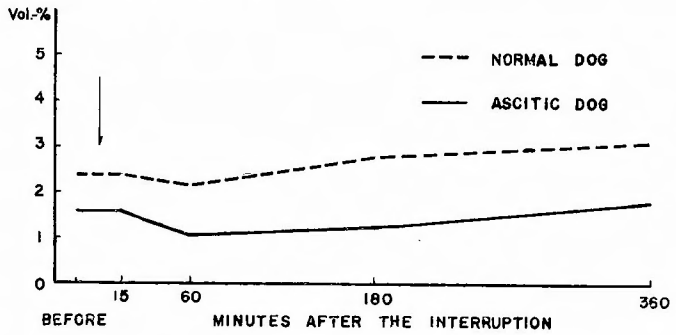


Fig. 24 Changes in O_2-difference between arterial and splenic vein blood after interruption of hepatic artery.



dogs, any difference is not observed to be mentioned in the oxygen content of the splenic vein between the two groups. This fact suggests the adaptability of the spleen in shock (Fig. 23, 24).

From the results of the present experiment presenting that the oxygen content of portal blood increases and the portal pressure decreases after the interruption of the hepatic artery in ascitic dogs, it is naturally assumed that the hypoxic state of the liver tissue, which usually occurs in normal dogs, does not easily develop in ascitic dogs. This fact also comes to accordance with the fact that in the ascitic dogs liver necrosis searcely occurs by the interruption of the hepatic artery.

X. CONCLUSION

1) The difference of oxygen content between the superior mesenteric artery and vein and that between the splenic artery and vein in ascitic dogs were smaller than in normal dogs.

2) Oxygen content of the superior mesenteric venous blood shows a temporary increase in normal dogs after the interruption of the hepatic artery which is followed

by a tendency of decrease. On the contrary, in ascitic dogs the content increases after the interruption then it decreases gradually which still remains above the level before the interruption until 6 hours after the interruption and maintains the increased level without showing a rapid decrease as observed in normal dogs.

3) Oxygen content of the splenic venous blood after the interruption of the hepatic artery fluctuates less in normal dogs than that of the superior mesenteric venous blood. There could not be observed such a marked difference between oxygen content of the splenic venous blood in normal dogs and that in ascitic dogs as compared with the difference of those of the superior mesenteric venous blood.

4) The portal pressure rapidly increases after the interruption of the hepatic artery in normal dogs which is followed by extremely gradual decrease and maintains a higher value than that before the interruption even 6 hours after the interruption. On the contrary, the pressure showed a rapid decrease after the interruption and maintains a lower level than that before the interruption with a slight fluctuation in ascitic dogs.

5) The Ht. ratio of the superior mesenteric venous blood shows a gradual increase after the interruption in normal dogs, while the ratio shows rather a decrease which is followed by a slight increase maintaining an approximate level to that before the interruption even 6 hours after the interruption in ascitic dogs.

6) In ascitic dogs, enlargement and tortuousity of the submucous plexus in the intestine were observed and it is presumed that there might exist the opening or dilatation of arterio-venous shunts.

7) An increasing stasis is observed in the capillaries of the intestine in normal dogs after the interruption, while it decreases contrariwise in ascitic dogs.

8) From these results, it comes to be possible, to some extent, to explain the mechanism of an increase in the oxygen content of portal blood, the difficulty of development of hypoxic state in the liver tissue and the difficulty of occurrence of liver necrosis in ascitic dogs after the interruption of the hepapatic artery. Furthermore, the possibility has been ascertained that the portal circulation would be promoted in ascitic dogs after the interruption.

I am deeply indebted to Prof. Dr. Ichio Honjo for his continuous and eager guidance throughout the present experiment.

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和文抄録

腹水犬の門脈血行と酸素含量に対する肝動脈遮断の影響

金沢大学医学部第二外科学教室（指導：本庄一夫教授）

伊藤戊辰

Rienhoff, Berman 等が肝硬変症の治療に肝動脈遮断が有効であることを提唱して以来，本法は諸家により，種々追試検討されて来たが，この術式は肝壊死発生の危懼の故に一般に危険視され，本法の是非に就ては未だ定説を得ない現状である．腹水の貯溜せる肝硬変類似の犬では，肝動脈遮断により，門脈血酸素含有量の増加を来し，肝壊死を発生し難いことは既に知られているが，その増加の機序に就ては未だ明らかでない．

著者は足立の実験成績を追試すると共に，その増加の機序に就ても検討を加えんと，以下の実験を行つた．

実験動物は10kg前後の雑種成犬を雌雄の別なく使用し，正常犬群，腹水犬群の2群に分ち，肝動脈遮断後の上腸間膜動静脈血酸素含有量，脾動静脈血酸素含有量を Van slyke Neil の大型検圧器を用いて経時的に測定し，併せて遮断による門脈圧，上腸間膜静脈血ヘマトクリット値の影響を窺い，腸管末梢血管の血行動態の面より検討を加えた．

尚，腹水犬の作成には胸部下大静脈狭窄の法を用い肝動脈遮断には総肝動脈，胃十二指腸動脈，右胃動脈の3動脈を結紮切離した．

実験成績

1) 腹水犬の上腸間膜動静脈血酸素較差並びに脾動静脈血酸素較差は正常犬のそれに比し，小であつた．

2) 正常犬の肝動脈遮断後の上腸間膜静脈血酸素含有量は，遮断前に比し，遮断後15分では平均 0.5Vol.-%,遮断後60分では平均 0.9Vol.-%と軽度の増加を示すが，遮断後180分では遮断前に比し，平均0.6Vol-%,360分では1.6Vol-%と逆に急激なる減少を示した．

これに対し，腹水犬では，遮断前に比し，遮断後15分では平均0.6Vol-%,遮断後60分では平均1.4Vol-% の増加を示して最高となり，以後極めて徐々に低下するが，遮断後180分，遮断後360分に於ても尚遮断前に比し，平均1.2Vol-%,0.5Vol-% の増加を示しており，正常犬群の如き急激なる低下は認められない．

3) 正常犬の肝動脈遮断後の脾静脈血酸素含有量は上腸間膜静脈血のそれに比して経時的変動が少なく，正常犬群と腹水犬群との間にも上腸間膜静脈にみられるような著名な差は認められなかつた．

4) 門脈圧は正常犬に於ては，肝動脈遮断後15分で遮断前圧に比し平均43mmH_2Oの急激なる上昇を示し，以後極めて徐々に下降するも，遮断後6時間に於ても遮断前より17mmH_2O高い圧を維持する．

これに対し，腹水犬では逆に，遮断直後，平均 50 mmH_2Oの急激なる低下を示し，以後多少の増減はあるが，遮断後6時間に於ても尚，遮断前に比し 平均 51 mmH_2O低い圧にて推移する．

5) 上腸間静脈血のヘマトクリット値は，正常犬に於ては遮断後逐次増加するが，腹水犬に於ては寧ろ遮断により，低下し，その後やゝ増加の傾向を示すが遮断後6時間に於ても尚略々，遮断前値に近い値を示している．

6) 腹水犬の腸管には，粘膜下血管叢の拡張並びに蛇行が認められ，動静脈間短絡の開放乃至は拡張が推定される．

7) 正常犬の腸管毛細管系には，肝動脈遮断後，経時的鬱滞増強の像を認めるが，腹水犬に於ては肝動脈遮断により，寧ろ軽減を思わせる像を見る．

8) 以上のことより，腹水犬に於ける肝動脈遮断後の門脈血酸素含有量の増加の説明が或る程度可能となり，他方肝の乏酸素状態を生じ難く，肝壊死の発生し難い理由の一端を解明するとともに，寧ろ門脈血行動態の改善され得る可能性を実証し得たものと信ずる．