EXPERIMENTAL STUDIES ON THE TRANSIENT OCCLUSION OF THE PORTAL VEIN IN HYPOTHERMIA

by

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Contents

I. INTRODUCTION

Pancreatoduodenectomy was once accomplished as early as in 1935 by WHIPPLE[51] as a radical operative treatment for malignant tumors in the pancreas and neighbouring organs of its head and the duodenum. However, many inoperable cases can be seen still today, because of the anatomical construction of this region. As the head of the pancreas encircles the stem of the portal vein, an invasion or adhesion of malignant tumors in the head of the pancreas spread readily to the portal stem frequently, even in cases of early stage. Transient occlusion of the portal vein, regardless of its duration, is a problem of utmost importance surgeons are forced to confront, in order to avoid the eventual injury of the portal stem, at surgery of these cases.

It has been well known that the interruption of the portal vein frequently results in fatality, since the experiment made by ORE[33] in 1856 in rabbits. NEUHOF[31], SCHIFF[41],

ELMAN & COLE[15][16] and BOYCE[1] also reported thereafter constant fatality within an hour after the interruption of the portal trunk. Although the cause of the death has been discussed as toxemia, exsanguination, neurogenic disorder or disturbance of liver function, conclusive results are not yet acquired. In 1950, CHILD[11] reported many cases of survival in monkeys that had undergone the ligation of the portal vein, and he concluded that the fatality of the procedure in animals of other species is due to the absence of portal collaterals and postulated a difference between mortality due to the ligation of the portal vein in primates and that in other animals. COLP[10] and BRUNSCHWIG[3] clinically reported, on the other hand, the danger of acute ligation of the portal vein. Many attempts to lessen the danger of this procedure have been carried out[3][3], that is MALLET GUY et al[28]. and ELMAN & COLE[15] applied blood transfusion, and THÖLE[17] cut off the splanchnic nerve in this aim. Both of these, however, had mere temporary effect.

Concerning the method of general hypothermia, SMITH & FAY[13] adopted this method for the first time in 1939, as a treatment of cancer bearing patient. ALLEN[2], BLALOCK & MANSON[5] reported beneficence of this method in preventing shock. In 1951, LABORIT[20] insisted the effectiveness of pharmacological hibernation for treatment of shock. Moreover, many reports of experiments on the hypothermia have been published chiefly in the field of surgery and physiology, following the application of hypothermia for cardio-surgery by McQUISTON[27], Bigelow[7][3][9] and others, in which an interruption of the circulation is required. Reflecting upon the application of hypothermia to surgery of the portal system, GOODALL and others[21] reported that the application of the method was profitable to some operations which require resection of the liver or ligation of the portal vein. In 1953, CSILLAG et al.[12], for the first time, aquired favorable results in prolongation of survival time in transient ligation of the portal vein by the application of hypothermia in experiment. Thus the applicability of hypothermia has been extended in portal surgery. However, only a few reports have been published on the pathophysiology of transient interruption of the portal vein under hypothermia.

The author of the present experiment made a series of experiments with special reference to permissible duration of the occlusion of portal flow under hypothermia and influence of that at the transient interruption of the portal vein chiefly upon respiration, circulation and gas metabolism.

Ⅱ. MATERIAL AND METHODS

(Ⅰ) Material

Healthy adult mongrel dogs weighing 6.5 to 16.0 kg were employed being divided into normothermic control and hypothermic group.

(Ⅱ) Methods

(1) Anesthesia, cooling and rewarming.

Atropin of 0.3 to 0.4 mg was injected intramuscularly 60 minutes before the introduction of anesthesia. Anesthesia was commenced with intravenous injection of Isozol of 10 to 15mg/kg. Tracheal tube was inserted and anesthesia was maintained with ether and oxygen at the depth of 2nd. phase of 3rd. stadium. The dog was cooled to 30°C through the method of so-called indirect immersion, that is essentially consisted in dipping

dogs wrapped up with vinyl sheet into ice-water. Degree of after drop of body temperature was observed to be 0.3 to 2.0°C. Body temperature was measured by the use of electric thermometer in the rectum. After operation, rewarming was performed in room temperature or in hot water of 45°C. In both occasions rewarming was carried out until body temperature arose to 35.0 to 35.5°C and awakening was intended thereafter.

(2) Occlusion of the portal vein and survival rate.

The abdomen was opened with upper median incision and the portal vein was occluded with Blalock's clamp between the empting of the pyloric vein and the bifurcation of the portal vein. The interruption was released after an interval. The influence of the interruption was finally evaluated from 48 hours' survival after the release of the interruption.

(3) Histological examinations.

Experimental animals were autopsied immediately after death or slaughter with intravenous rapid injection of Isozol and histological appearance of heart, liver, spleen and intestinal canal was studied by hematoxylin-eosin double staining.

(4) Determination of arterial and portal pressure.

Blood pressure was determined by mercurial manometer connected to a glass canula inserted into the femoral artery. Portal pressure was determined from a vinyl tube which was inserted into the gastrosplenic vein.

(5) Observation on electrocardiography.

Electrocardiograph equipped with heated stylus (Fukuda Co. Ltd.) was used for this purpose. Electrocardiogram was taken from the limb and precordial leads both before and after the interruption.

(6) Circulating blood volume and circulation time.

Two milliliters of 0.5 per cent Evans Blue solution was injected from the right femoral vein following the method of GREGERSON[9]. Blood sample was taken from the right femoral artery 5 minutes after the injection. Density of Evans Blue in the plasma was estimated by electrophotometer (Shimazu Co. Ltd.). Circulating blood volume and circulation time were calculated from following formulas,

$$\text{Circulating plasma volume (cc)} = \frac{Es \times 500 \times 2}{Em}$$

$$\text{Circulating blood volume (cc)} = \frac{\text{C.P.V (cc)} \times 100}{100 - \text{Ht (\%)}}$$

where Es is electrophotometric absorption of the plasma estimated through filter of 624 Å with a control of plasma before the injection of the solution, and Em is electrophotometric absorption of Evans Blue solution in 500 times dilution estimated in the same way with a control of 10 times diluted plasma before the injection.

Circulation time was observed following lobeline method, that is, 0.3 cc of 1 per cent lobeline was injected into the femoral vein and the interval required for manifestation of tachypnoe (femoral-broncheal time) was determined.

The determination was carried out in room temperature as well as in hypothermia and 30 minutes after the occlusion and release respectively.

(7) Estimation of oxygen consumption.

After insertion of the tracheal tube, it was connected to the Knipping's apparatus and oxygen consumption was estimated following the method of Knipping.

(8) Gas analysis.

Mixed venous blood sample was taken through 8-F heart catheter inserted from the right jugular vein to the right atrium under fluoroscopy and arterial blood was taken at the same time from the left femoral artery, both of which were immediately enclosed with fluid paraffin in test tubes. Blood samples were provided rapidly for analysis, at latest within 6 hours. Blood gas analysis was performed following VAN-SLYKE-NEIL's[49] method, volume percentage of oxygen being estimated after FUJII's[17] simultaneous estimation method, and that of carbon dioxide being estimated following AUSTIN's method[1].

(9) Calculation of cardiac output, stroke volume and peripheral resistance.

These were calculated from following formulas based on the results of right heart catheterization,

$$\text{Cardiac output} = \frac{O_2}{O_A - O_V} \quad (cc)$$

$$\text{Stroke volume} = \frac{\text{Cardiac output}}{\text{Pulsation number}} \quad (cc)$$

where O_2 is oxygen consumption due to lung respiration in a minute, O_A is oxygen content of 1.0 cc of arterial blood and O_V is oxygen content of 1.0 cc of mixed venous blood in the right atrium.

Total peripheral resistance was calculated as follows employing C. G. S. units of WIGGERS[50],

$$\text{Total peripheral resistance} = \frac{\text{Average arterial pressure (mm Hg)} \times 1333}{\text{Cardiac output (cc/sec)}}$$

$$= \text{dynes. sec. /cm}^5$$

(10) Density of Evans Blue injected into the portal vein in the systemic circulation.

In order to observe the appearance of flow of the portal blood into systemic circulation, following experiments were carried out. Fifteen milliliters of 76 per cent urografin was injected into the gastrosplenic vein for portalography and the absence of the direct path to the liver was assured, i. e. the occlusion was complete. Then, 4.0 cc of 0.5 per cent Evans Blue was similarly injected into the portal vein 10 minutes after the occlusion. Blood was taken after certain intervals of time from the right heart catheter and the plasma was provided for electrophotometry. The change in density of Evans Blue in the blood and interval between the injection and appearance was observed.

Ⅲ. Results

(1) Survival rate and permissible duration of the occlusion.

Occlusion of the portal vein was performed in 17 dogs, whose rectal temperature being kept to be 28.5 to 30.0°C.

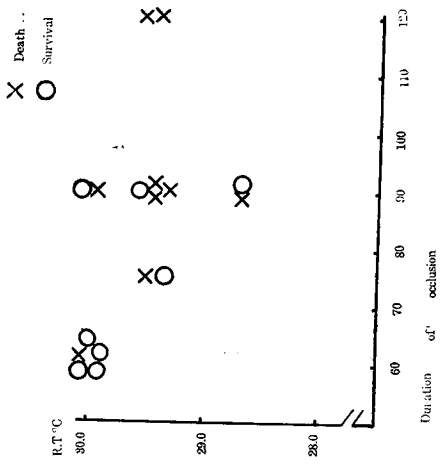
Four dogs out of five (80%) undergone 60 minutes's interruption, one out of two (50%) undergone 75 minutes' interruption and three out of eight (38%) undergone 90 minutes' interruption survived. Death resulted in all the dogs undergone 120 minutes' interruption. No ventricular fibrillation, as Swan reported, could be observed under the

condition of 26.0 to 30.0°C of rectal temperature. Hence, it is assumed that hypothermia of 26.0 to 30.0°C has little influence in itself upon survival rate and the upper limit of permissible duration of the interruption under the hypothermia of this temperature is 60 minutes (Fig. 1).

(2) Macroscopic and pathologic findings.

Intestinal surface in hypothermic group showed macroscopically fresh red tincture even 60 minutes after the interruption and no bleeding in the intestinal canal and mesentery, although accompanied merely by a slight edema in the mesentery, in brief, far slight macroscopic changes were observed compared to that of normothermic group. No changes were observed in the liver, while the spleen swelled approximately two times bigger conceivably due to conspicuous congestion of the blood.

Fig. 1 Survival rate



Autopsy of six dogs out of nine revealed accumulation of considerable amount of bloody exudation in the abdominal cavity, slight bleeding in the canal of the jejunum and ileum and mesenterial ecchymoses. Cause of the death could not be clarified in three other dogs on autopsy. Particular change of any kind could not be observed in all the dogs survived the interruption.

Histological examination also revealed approximately normal appearance except congestion and bleeding in the liver and spleen of the survivors (Plate. 2, 3.), showing no remarkable change as was observed in normothermic group such as bleeding in the intestinal wall and canal (Plate. 4, 5), and fragmentation, isolation and bleeding in fibre of the heart muscle. It is suggested from these findings that hypothermia has a protective effect against irreversible changes of organs caused by the interruption of the portal vein.

(3) Arterial and portal pressure.

Arterial pressure in normothermic group began rapidly to fall down after the interruption and about 10 minutes later it came near its minimum, while portal pressure arose contrariwise showing the maximal level (540 to 870 mmH_2O) several minutes after the interruption. At that time, portal pressure showed slightly lower level than arterial pressure with a difference of a few millimeters in mercurial manometer (Fig. 2). This finding is accepted to suggest that a plenty amount of blood is pooled in the splanchnic area. On the contrary, in hypothermic group, descension of arterial pressure was mild and portal pressure, although arose (480 to 670 mmH_2O), was observed to be lower than that of the former group with a difference of 20mmHg between arterial and portal pressure (Fig. 3).

(4) Changes in electrocardiogram.

Fig. 2 Arterial and portal pressure, 6 dogs averaged. (Normothermic group)

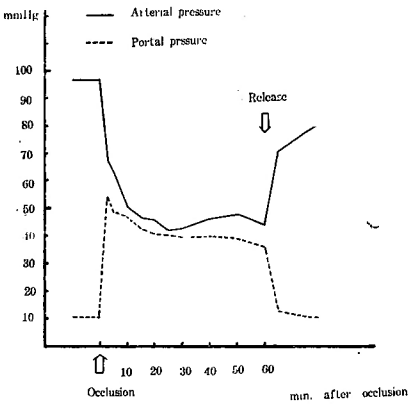
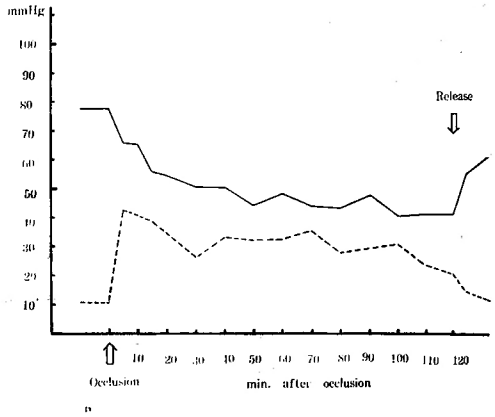


Fig. 3 Arterial and portal pressure, 7 dogs averaged. (Hypothermic group)



Fifteen minutes after the interruption in normothermic group, lowering of ST and T was observed, and negative T and low voltage in some cases. In hypothermic group, changes in electrocardiogram appeared first more than 30 minutes after the interruption, that is, slight lowering of ST, prolongation of QRS-time, negative T or flattening of T. It is assumed that in this group also there occurred impairment, though slight, of heart muscle due to anemia presumably, as mentioned later, together with decrease in circulating blood volume and cardiac output.

(5) Measurement of circulating blood volume.

Circulating blood volume was measured in normothermic group to be 86.3 cc/kg before the interruption, 51.5 cc/kg 30 minutes after the interruption showing a decrease of 40.3 per cent and 54.8 cc/kg after the release showing a decrease of 36.5 per cent (Tab. 1, Fig. 4). On the other hand in hypothermic group, it decreased from 78.7 cc/kg before the occlusion to 62.0 cc/kg after that showing a decrease of 21.2 per cent and 72.9 cc/kg after the release with a decrease of 7.4 per cent, thus suggesting little influence of the occlusion (Tab. 1, Fig. 4).

Circulating plasma volume in normothermic group also showed a decrease of 37.9 per cent after the interruption and 30.6 per cent after the release respectively compared to those before the interruption, while in hypothermic group the degree of the decrease was more slight, being 32.9 per cent and 14.6 per cent respectively (Tab. 2).

Circulation time was prolonged by the occlusion in both group, i. e. twice as long as in normothermic group and thrice as long as in hypothermic group. This suggests that the response of circulatory system becomes dull and tardy under hypothermia (Tab. 3, Fig. 5).

(6) Changes in cardiac output and peripheral resistance.

Cardiac output decreased by 90.1 per cent from 307.2 cc/kg before the interruption to 30.3 cc/kg after it, which still maintained lowered value of 69.8 cc/kg even after the release of the interruption, i. e. decrease of 77.3 per cent compared to that before the interruption (Tab. 4, Fig. 6). The value was also markedly decreased in hypothermic group showing decrease of 80.6 per cent from 292.3 cc/kg before the interruption to

Tab. 1 Circulating blood volume. (cc/kg)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
27	77.0		50.0	48.5
30	84.2		47.3	49.7
32	88.7		51.2	57.0
44	95.3		59.0	63.2
mean	86.3		51.5	54.8

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
15	79.1	75.8	65.2	57.2
16	87.0	80.0	73.1	76.2
17	94.3	83.4	70.4	—
18	95.2	90.5	66.5	91.1
19	72.0	60.9	45.5	57.4
20	74.7	65.5	40.0	92.9
21	89.0	76.3	—	—
22	87.4	74.0	60.2	—
28	—	89.1	65.1	54.5
31	—	91.5	72.4	80.8
mean	84.8	78.7	62.0	72.9

Fig. 4 Circulating blood volume.

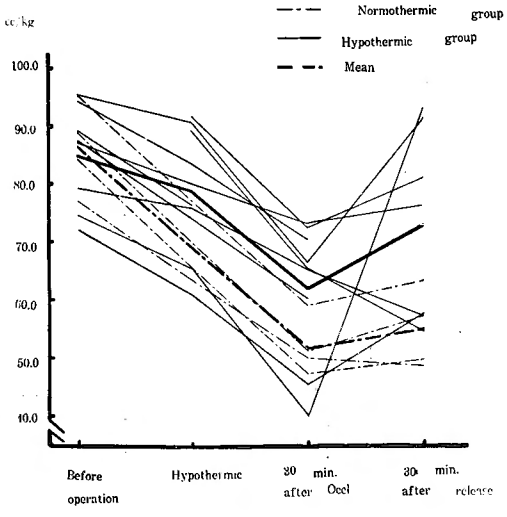
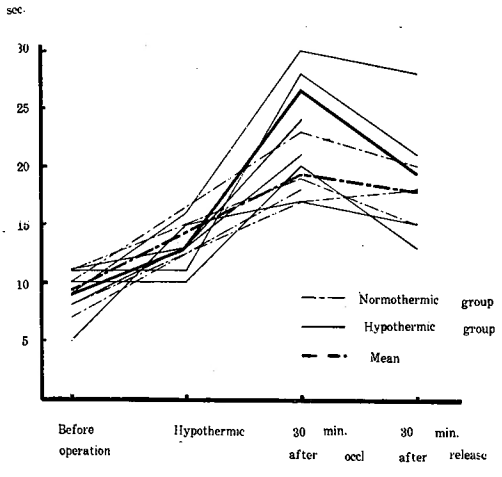


Fig. 5 Circulation time.



53.7cc/kg after it, the degree of which is less than in normothermic group. But the value showed no increase after the release remaining within the lowered level after the interruption (Tab. 4, Fig. 6.)

Tab. 2 Circulating plasma volume. (cc/kg)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
27	60.0		38.5	40.5
30	57.0		34.2	30.1
32	51.4		28.5	38.4
44	48.3		30.8	41.3
mean	54.2		34.0	37.6

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30min. after occl.	30min. after release
15	50.0	30.2	35.2	37.2
16	47.3	38.5	40.0	30.8
17	59.5	45.1	21.3	—
18	62.5	70.3	41.4	54.1
19	46.4	32.0	20.2	38.1
20	44.0	40.0	23.2	—
21	57.0	54.3	—	—
22	56.3	49.2	40.2	—
28	—	52.0	35.1	31.8
31	—	53.0	24.5	46.0
mean	52.9	46.5	31.2	39.7

Tab. 3 Circulation time. (sec)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
27	10		23	20
30	8		17	18
32	11		19	15
44	7		18	—
mean	9		19.3	17.8

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
16	8	13	21	—
17	11	13	24	—
19	9	16	30	28
20	11	11	28	21
28	10	10	20	13
31	5	15	17	15
mean	9	13	26.7	19.3

Tab. 4 Cardiac output. (cc/kg/min)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
33	370.6		31.6	11.9
35	187.0		23.2	106.5
37	151.4		—	—
39	350.4		41.5	66.7
42	476.4		24.9	94.1
mean	307.2		30.3	69.8

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	160.0	170.0	37.1	60.0
38	272.0	262.0	60.0	50.5
40	426.0	466.7	70.7	62.5
41	333.0	263.0	—	—
43	323.6	300.0	50.5	41.5
mean	302.9	292.3	53.7	53.6

Tab. 5 Stroke volume. (cc/kg)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
33	2.2		0.2	0.2
35	1.8		0.3	0.6
37	1.3		—	—
39	2.4		0.4	0.6
42	3.7		0.2	0.5
mean	2.3		0.3	0.5

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	2.1	1.9	0.8	1.0
38	2.0	1.7	0.8	0.6
40	3.6	3.0	0.7	0.8
41	2.1	1.9	—	—
43	2.2	2.5	0.6	0.8
mean	2.4	2.2	0.7	0.8

As to stroke volume, it showed the same tendency as described above in normothermic group, while in hypothermic group the decrease was observed to be slight less by the interruption from 2.2 cc/kg to 0.7 cc/kg, that is decrease of two thirds (Tab. 5).

Tab. 6 Peripheral resistance. (dynes. sec/cm^5)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. afterrelease
33	6798.3		11997.0	15196.2
35	9331.0		9331.0	15996.2
37	10397.4		—	—
39	6665.0		7998.0	10397.4
42	4932.1		13330.0	7998.0
mean	7598.1		10664.0	12396.9

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. afterr elease
34	6798.3	8664.5	8797.8	7998.0
38	9331.0	6931.6	10664.0	16662.5
40	5332.0	5998.5	14263.1	10130.8
41	7331.5	9064.4	—	—
43	5998.5	6798.3	12663.5	10930.6
mean	6931.6	7731.4	11597.1	11463.8

Fig. 6 Cardiac output.

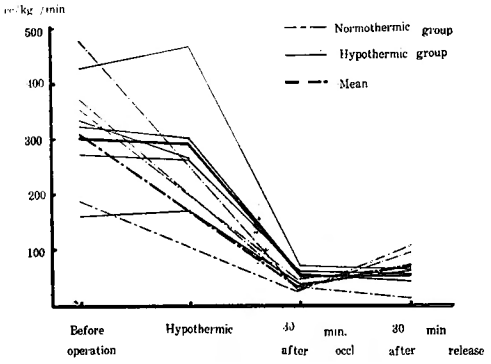
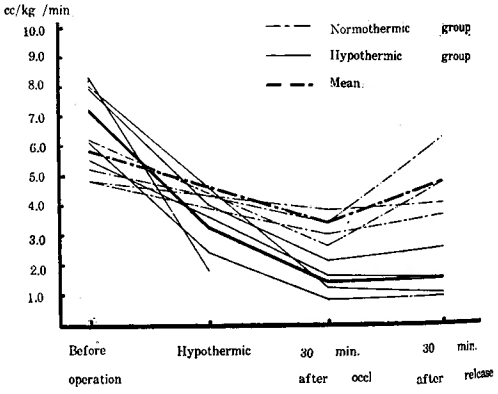


Fig. 7 Oxygen consumption.



Peripheral resistance increased after the interruption by 40 per cent in normothermic group and 50 per cent in hypothermic group. After the release of the interruption, it showed a tendency of decrease in hypothermic group, what was not observed in normothermic group, maintaining higher level. It is suggested from this finding that insufficiency of peripheral circulation is not readily restored in normothermic group after the interruption released (Tab. 6).

(7) Changes in oxygen consumption.

A remarkable difference between oxygen consumption in normothermic group and that in hypothermic group was observed after the interruption, it being 3.4 cc/kg/min. in the former (decrease of 41.4%) and 1.4 cc/kg/min (decrease of 80.6%) in the

Tab. 7 Oxygen consumption. (cc/kg)

(Normothermic group)

Dog. No.	Before operation	30 min. after occl.	30 min. after release
33	4.8	3.8	4.0
35	5.2	3.4	6.2
37	4.8	3.0	3.6
39	8.2	4.0	5.0
42	6.2	2.6	4.7
mean	5.8	3.4	4.7

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	6.1	2.4	0.8	0.9
38	8.0	4.6	1.2	1.0
40	7.9	4.0	2.1	2.5
41	8.3	1.8	—	—
43	5.5	3.6	1.6	1.5
mean	7.2	3.3	1.4	1.5

latter which is less than one half of the former. After the interruption was released, it was estimated to be 4.7cc/kg/min. in normothermic group and 1.5 cc/kg/min. in hypothermic group suggesting less requirement of oxygen in the latter (Tab. 7, Fig. 7.).

(8) Changes in gas metabolism.

(A) Oxygen content of arterial and venous blood and its difference.

Conspicuous decrease in oxygen content of venous blood was observed in normothermic group after the interruption, that is decrease by 11.2 vol per cent, while in hypothermic group it showed slight decrease by 4.7 vol. per cent which is less than a half

Tab. 8 Oxygen content of arterial and venous blood. (vol. %)

(Normothermic group)

Dog. No.		Before operation	30 min. after occl.	30 min. after release
33	arterial venous	23.1 21.8	18.5 9.5	14.8 9.8
35	arterial venous	24.8 22.0	21.6 10.8	23.7 17.9
37	arterial venous	25.2 22.2	—	—
39	arterial venous	19.2 17.0	19.5 7.9	21.2 16.2
42	arterial venous	17.1 15.8	13.2 5.8	18.1 11.6
mean	arterial venous	21.8 19.7	18.2 8.5	19.5 13.9

(Hypothermic group)

Dog. No.		Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	arterial venous	17.7 13.9	20.9 19.5	18.3 16.2	15.7 14.2
38	arterial venous	19.8 16.9	19.1 17.4	17.7 15.6	20.8 18.8
40	arterial venous	20.6 19.1	21.8 20.6	23.1 20.1	24.2 20.2
41	arterial venous	24.1 21.6	26.3 25.5	—	—
43	arterial venous	18.5 16.8	15.9 14.9	10.9 7.7	12.2 8.6
mean	arterial venous	20.1 17.6	20.8 19.6	17.5 14.9	18.2 15.5

Tab. 9 Difference of oxygen content between arterial and venous blood. (vol. %)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
33	1.3		9.0	5.0
35	2.8		10.8	5.8
37	3.2		—	—
39	2.2		11.6	5.0
42	1.3		7.4	6.5
mean	2.2		9.7	5.6

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	3.8	1.4	2.1	1.5
38	2.9	1.7	2.1	2.0
40	1.5	1.2	3.0	4.0
41	2.5	0.7	—	—
43	1.7	1.0	3.2	3.6
mean	2.5	1.2	2.6	2.8

compared to that in the other group. Moreover, oxygen content of arterial and venous blood in hypothermic group decreased in parallel with each other (Tab. 8).

Accordingly difference of oxygen content between arterial blood and venous one increased markedly from 2.2 vol. per cent to 9.7 vol. per cent after the interruption in normothermic group, which remained in as high a level as 5 6 vol. per cent after the release. On the contrary, the difference in hypothermic group showed only a slight fluctuation from 1.2 vol. per cent to 2 8 vol. per cent throughout the whole course (Tab. 9, Fig. 8).

Tab. 10 Carbon dioxide content of arterial and venous blood. (vol. %)

(Normothermic group)

Dog. No.		Before operation		30 min. after occl.	30 min. after release
33	arterial venous	33.6 36.7		36.6 49.0	34.5 36.5
35	arterial venous	36.2 37.7		39.8 49.3	39.8 49.3
37	arterial venous	34.3 42.4		—	—
39	arterial venous	37.7 39.6		36.2 42.1	29.3 35.1
42	arterial venous	32.2 33.8		28.8 34.5	26.5 29.9
mean	arterial venous	34.8 38.0		35.4 43.9	32.5 37.7

(Hypothermic group)

Dog. No.		Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	arterial venous	36.0 37.6	29.3 34.4	36.0 42.7	44.8 48.0
38	arterial venous	35.3 46.2	41.6 42.1	38.7 42.4	37.1 39.5
40	arterial venous	40.3 42.3	37.6 37.9	31.5 37.5	32.0 33.0
41	arterial venous	25.7 29.2	31.6 32.8	—	—
43	arterial venous	43.6 44.7	33.0 37.5	44.0 45.7	28.1 37.4
mean	arterial venous	36.2 40.0	34.6 36.9	37.5 42.0	35.5 39.5

Tab. 11 Difference of carbon dioxide content between arterial and venous blood. (vol. %)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
33	3.1		12.4	2.0
35	1.5		9.5	9.5
37	8.1		—	—
39	1.9		5.9	5.8
42	1.6		5.7	3.4
mean	3.2		8.4	5.1

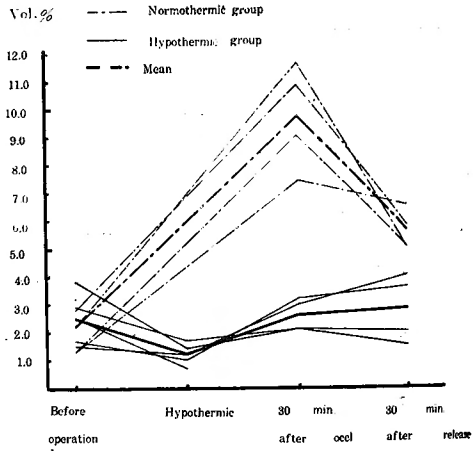
(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after occl.	30 min. after release
34	1.6	5.1	6.7	3.2
38	10.9	0.5	3.7	2.4
40	2.0	0.3	6.0	1.0
41	3.5	1.2	—	—
43	1.1	4.5	1.7	9.3
mean	3.8	2.3	4.5	4.0

(B) Carbon dioxide content of arterial and venous blood and its difference.

Carbon dioxide content fluctuated in a resembling attitude as that of oxygen. In normothermic group carbon dioxide content showed relatively high level when the portal vein was interrupted (Tab. 10). While the difference of carbon dioxide content increased by 5.2 vol. per cent from 3.2 vol. per cent to 8.4 vol. per cent by the interruption in normothermic group, it remained in a slight increase of 2.2 vol. per cent from 2.3 vol. per cent to 4.5 vol. per cent in hypothermic group. Although the difference decreased after the release in both groups, the degree of decrease being prominent in normothermic group (Tab. 11).

Fig. 8 Difference of oxygen content between arterial and venous blood.



(C) Oxygen and carbon dioxide content of the portal blood.

Oxygen content showed marked decrease as early as 15 minutes after the occlusion in normothermic group and 60 minutes after the occlusion oxygen in the portal blood deemed to have almost vanished away. On the other hand, in hypothermic group, a tendency of marked decrease of oxygen content was observed first more than 30 minutes after the interruption, suggesting that animals can endure longer the interruption of the portal vein under hypothermia, owing to the reduced oxygen requirement of the tissues (Tab. 12, Fig. 9). Carbon dioxide content showed a similar tendency of gradual decrease after the interruption in both groups (Tab. 12, Fig. 9).

(D) Oxygen saturation.

Oxygen saturation decreased more remarkably in normothermic group by 9.1 per cent, whereas the decrease being as slight as 4.1 per cent in hypothermic group. After the release of the interruption, oxygen saturation showed a tendency to restore to the value before the interruption (Tab. 13, Fig. 10). It is assumed, when observed at least from a point of oxygen-combining power of the blood, that the animals of both groups still possess a reversibility even 60 minutes after the interruption.

(9) Appearance of flow of the portal blood into systemic circulation after the

Tab. 12 Oxygen and carbon dioxide content of the portal blood. (vol. %)

(Normothermic group)

Dog. No.		Before occl.	After occlusion (min.) 5	15	30	60
37	CO_2	48.2	46.3	40.7	39.8	32.1
	O_2	13.1	12.5	10.3	2.4	1.9
44	CO_2	40.2	39.0	37.1	—	30.5
	O_2	16.1	15.7	8.4		3.1
mean	CO_2	44.2	42.7	38.9	—	31.3
	O_2	14.6	14.1	9.7		2.5

(Hypothermic group)

Dog. No.		Before occl.	After occlusion (min.) 5	15	30	60
41	CO_2	43.4	40.2	—	38.2	38.4
	O_2	18.2	16.3		7.5	4.5
46	CO_2	39.2	39.3	—	37.3	30.5
	O_2	25.1	20.4		15.1	13.2
47	CO_2	41.2	34.2	32.2	30.9	33.9
	O_2	14.5	13.2	12.7	11.1	8.4
mean	CO_2	41.3	37.9	—	35.5	34.3
	O_2	19.3	16.6		11.2	8.7

Tab. 13 Oxygen saturation. (%)

(Normothermic group)

Dog. No.	Before operation		30 min. after occl.	30 min. after release
33	93		85	90
35	97		85	90
37	90		—	—
39	90		73	87
42	92		90	95
mean	92.4		83.3	90.5

(Hypothermic group)

Dog. No.	Before operation	Hypothermic	30 min. after ocol.	30 min. after release
34	90	90	90	93
38	86	89	84	86
40	95	95	93	95
41	97	94	—	—
43	97	100	91	100
mean	93	93.6	89.5	93.5

Fig. 9 Oxygen and carbon dioxide content of theportal blood.

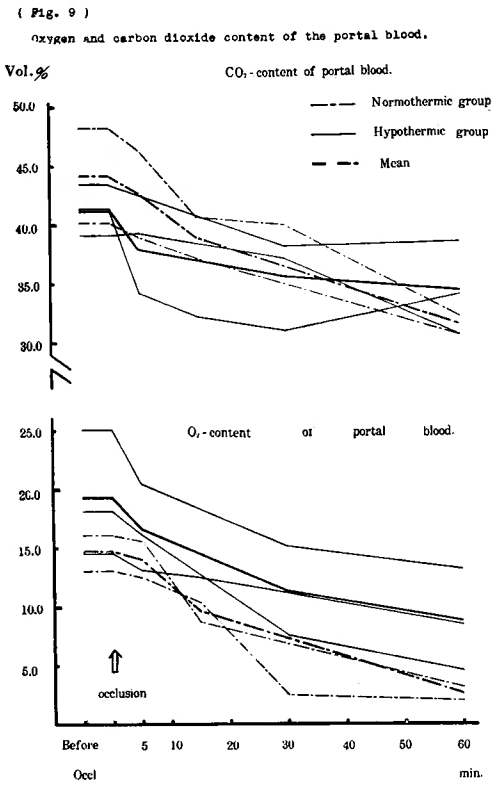


Fig. 10 Oxygen saturation.

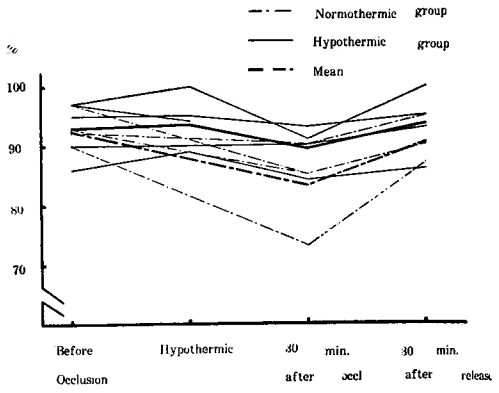
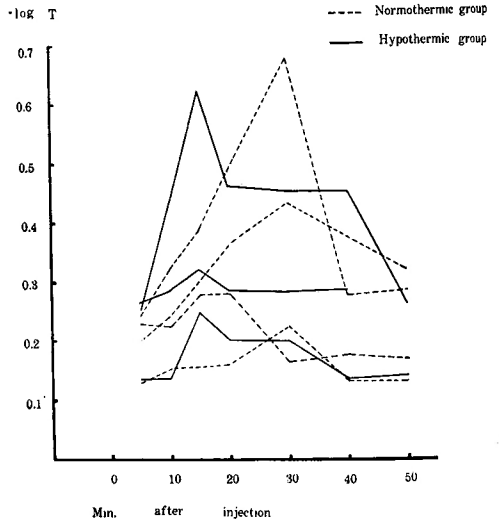


Fig. 11 Photometric concentration of Evans Blue injected into the portal vein determinated in the blood of systemic circulation after the occlusion of the portal vein.



interruption.

Although there have been many reports on the existence of portal collaterals at portal interruption in dogs, it was impossible to clarify this problem in the present experiment. It was, however, at least presumed from the above mentioned observation on prominent difference of the relationship between arterial and portal pressure in normo- and hypothermic groups that there might exist some difference in appearance of flow of congested portal blood into systemic circulation in these two groups. Here the appearance of Evans Blue, injected into the portal vein, into systemic circulation was pursued, taking blood samples at certain intervals of time after the injection through the right heart catheter in dogs of both groups in which the complete interruption of the portal vein had been assured by portalography after the interruption.

On portalography, no collateral was noticed in both groups (Plate 5, 6).

Fluctuation of Evans Blue density in the right atrium showed its peak 15 minutes after the injection in hypothermic group, while the peak appeared 20 to 30 minutes after the injection in normothermic group, what enables the presumption that the flow of the

Tab. 14 Photometric concentration of EVANS-BLUE injected into the portal vein determinated in the blood of systemic circulation after the occlusion of the portal vein. (-log T)

(Normothermic group)

Dog. No.	Minutes after injection						
	5	10	15	20	30	40	50
44	0.229	0.224	0.276	0.278	0.163	0.175	0.168
46	0.242	0.321	0.386	0.485	0.675	0.275	0.283
47	0.130	0.151	0.156	0.160	0.225	0.157	0.156
51	0.200	0.241	0.252	0.360	0.431	—	0.320

(Hypothermic group)

Dog. No.	Minutes after injection						
	5	10	15	20	30	40	50
48	0.135	0.136	0.248	0.202	0.200	0.135	0.140
49	0.265	0.283	0.320	0.285	0.282	0.285	—
50	0.252	0.433	0.620	0.460	0.452	0.450	0.260

blood from portal system to systemic circulation is delayed in the latter group, namely in the former group the congested portal blood rapidly flows into systemic circulation (Tab. 14, Fig. 11).

Ⅶ. DISCUSSION

Concerning the cause of fatality of acute portal occlusion, CLAUDE BERNARD[3], in 1887 put emphasis on the exsanguination which is caused by the congestion in the intestine, and impediment of the liver function was pointed out later by SCHIFF[11]. SOLOWIEFF[12] reported, on the other hand, favorable results of survival of his experiment in which ligation of the portal branches was performed in several stages with intervals of days and the ligation of the portal trunk was carried out finally. ITŌ and OMI[33] reported the same results as SOLOWIEFF's ones, with a warning that the ligation of those branches and the trunk all at once would result in fatality. NEUHOF[11] sought an explanation of the advantage of the ligation in several stages in the establishment of collaterals. THÖLE[17] and BOYCE[4] made experiments from different aspect, that is, the former demonstrated that animals undergone the ligation of the portal vein can survive longer without showing haemorrhagic infarction or subserous bleeding in the intestine when the splanchnic nerve is previously cut off, and the latter attached great importance to the participation of neurogenic factor in maintenance of blood pressure after the interruption. PECK and GROVER[37] sought the cause of death in toxemia.

ELMAN & COLE[15][16] asserted, thereafter, in 1934, based on their estimation of circulating blood volume, that the cause of fatality of the ligation of the portal vein is attributable to decrease in circulating blood volume which is caused by acute congestion of the blood in splanchnic area, and that this is closely related to bleeding shock. This assertion became gradually to be accepted widely. They reported the decrease in circulat-

ing blood volume to be 5.2 per cent of body weight, when determined from weights of extirpated abdominal organs, which is 9.92 per cent according to BOYCE[4], and they both asserted the decrease of this extent to be reasonable amount to cause haemorrhagic death. Thanks to the recent advance of the method of circulating blood volume determination, it has become possible to perform the determination with more accuracy. MALLET-GUY[28] reported the decrease to be 49.3 per cent according to his experiment using Chicago blue, JOHNSTONE[24] reported it to be 57.9 per cent using radioactive phosphorus and DATE[14] noticed a decrease of 44.4 per cent using Evans Blue. The author of the present experiment obtained slightly lower value of 40.3 per cent, it is presumably due to the fact that the determination was carried out 30 minutes after the interruption of the portal vein when the circulation had already been stabilized.

As to the change of circulating blood volume under hypothermia, there are very few reports. The author obtained in hypothermic group as little decrease as 21.2 per cent, which is a decrease of one half compared to that of normothermic group, what makes it possible to assume that the congestion in splanchnic area is much lessened by hypothermic treatment.

Concerning the permissible duration of portal ligation, DUCHINOVA[13] reported in 1926, the constant fatality of the interruption over 35 minutes, when the hepatic artery and portal vein are interrupted temporarily at the hilum of the liver. His study was followed by successive experiments on permissible duration of sole interruption of the portal vein. NEUHOF[31], JOHNSTONE[24], HATAGOSHI[22] and YABUKI[53] noticed it to be 20 minutes, and RAFFUCI[39] to be 30 minutes. OYANAGI[35] in our clinic also reported it to be 30 minutes. There are several reports also on permissible duration of the interruption under hypothermia. CSILLAG et al[12]. reported that the dogs survived the portal interruption of 45 to 51 minutes under hypothermia, SHIMIZU[43] reported 80 per cent survival of 60 minutes' interruption and MIKAMI[29] asserted that the permissible duration can be prolonged as long as 60 minutes. OKAMURA[34] reported a case of his clinical experience in which he observed a possibility of portal interruption for one hour and one third without life threatening symptoms by employing hypothermia when he unexpectedly injured the portal vein at the operation of cancer infiltrating into the vein. The author of the present experiment also obtained survival of 80 per cent of the portal interruption for 60 minutes in hypothermic group which abruptly diminished when the interruption was prolonged over 60 minutes. Judging from these results, permissible duration of the portal interruption under hypothermia might be justifiably assumed to be 60 minutes, twice longer than in normothermic group.

In 1950, CHILD[11] reported the results of his experiments of portal ligation in monkeys, that is, although 17 monkeys out of 76 died of inadequate return of the portal blood into systemic circulation, the remainders survived for long without revealing unfavorable changes of any kind. Thus he postulated a difference of the mortality between monkeys and other mammalians, which is because of the existence of adequate portal collaterals in monkeys, enabling the animals to survive the acute and complete occlusion of the portal flow even in usual temperature.

It is obviously assumed from the results of the present experiment that hypothermia

has less influence upon organism in respect of permissible duration of the portal interruption as well as of circulating blood volume, and furthermore it was ascertained that in a hypothermic state cardiac function is less influenced by portal occlusion.

On electrocardiographic observation, neither lowering of ST and T nor ventricular fibrillation was observed under hypothermia, as were observed by TANTURI[38] and RAÚL[40] in usual temperature. CSILLAG et al[12] reported that no marked change of electrocardiogram was observed following the portal ligation under hypothermia, whereas change of electrocardiographic pattern, although slight, was observed 30 minutes after the interruption in the present experiment, suggesting an existence of slight impairment in myocardium. Concerning cardiac output, FISCHER[13], NIEDER[32] and others conceived a decrease of approximately two thirds following the portal interruption. According to the results of the present experiment, the decrease in hypothermic group was observed to be about nine tenths, remaining in a slight degree of one half of that in normothermic group, what is accepted to be beneficient in preventing the animals from circulatory collaps together with more slight decrease in circulating blood volume.

RANSOHOF[35] first directed his attention to the descension of blood pressure after the portal ligation. PECK and GROVER[37] explained the rapidly descending curve of the blood pressure after the ligation to be due to the neurogenic reaction and that of gradual descension to be due to decrease in circulating blood volume. They further postulated that the initial fall could be prevented by administration of ACTH and the second fall by blood transfusion. KRYMHOLZ[25] attributed the cause of the descension to decline of cardiac activity, and NIEDER et al[32]. to shortage of returning blood volume to the heart.

In the present experiment, it was obviously observed that the fall of blood pressure develops in hypothermic group more gradually and that difference between arterial and portal pressure is larger, mechanism of which was, however, not to be clarified. Nevertheless, it might be assumed that the portal blood is situated, under hypothermia, in a condition in which it is inclined to return directly to systemic circulation without passing through the liver, judging from slight congestion in the splanchnic area, slight decrease in circulating blood volume in hypothermic group and delayed reaction of circulatory system, and furthermore based on the fact that early appearance of Evans Blue injected into the portal vein in systemic circulation and conspicuous difference observed in the histological findings of intestinal canal between normothermic group and hypothermic one.

It has been ascertained by BIGELOW[5][39] that in hypothermia oxygen consumption is decreased. He observed linear relationship between rectal temperature and oxygen consumption in dog under hypothermia, oxygen consumption being reduced almost to zero, when rectal temperature is kept to be 10 to 12°C. WATANABE[52] also observed decrease of 30 per cent in oxygen consumption with rectal temperature of 30°C, decrease of 50 per cent with 27°C and that of 94 per cent with 16°C, and he drew an interesting conclusion that the decrease in oxygen consumption depends solely upon the change of temperature if the depth of anesthesia is maintained invariably. In the present experiment, the decrease in oxygen consumption was observed in hypothermia to be 46 per cent, nearly being similar to those of their reports. Furthermore, the decrease was 80.6 per cent at portal interruption in hypothermia, while it being 41.4 per cent at the same

procedure in usual temperature, what leads to a presumption that the requirement of oxygen is reduced in hypothermia and the animal can endure the portal interruption for longer duration under this condition.

SWAN[44)45)] made an experiment of circulatory occlusion in order to perform cardiosurgery under hypothermia and observed no increase in carbon dioxide in the venous blood after circulatory occlusion for 15 minutes with hypothermia of 23°C. In the present experiment dealing with portal interruption for 60 minutes, only a slight increase in carbon dioxide in both arterial and venous blood was noticed.

On the other side, despite the tissue respiration of the organs in the splanchnic area, a decrease in carbon dioxide in the portal blood was observed suggesting no release of that into the portal blood. Accordingly, it is readily presumed that carbon dioxide might be accumulated within the tissues, which would be represented by metabolic disturbance of the organs in this area.

The difference of oxygen content between arterial and venous blood in hypothermic group showed slight change from 1.2 vol. per cent throughout the experimental course, while in normothermic group it leapt up from 2.2 vol. per cent to a peak of 9.7 vol. per cent and maintained as high a level as 5.7 vol. per cent even after the release. FUJIWARA[18)] observed the difference of oxygen content of 2.85 vol. per cent 30 minutes after the interruption in hypothermia, and SHIMIZU[16)] reported it to be 6.3 vol. per cent, which are somewhat higher than in the present experiment. This seems to be attributable to the fact that oxygen content of mixed venous blood is higher than peripheral venous blood as in their experiments.

Since NEUHOF[31)] made studies on histological findings in acute portal ligation, there have been published detailed reports on this subject by CSILLAG et al[12)]. and MORINO[10)]. The similar findings were observed also in the present experiment, animals in the hypothermic group showing slight change and those of long survival showing no different findings from intact animals.

Judging from above mentioned results, it is assumed that circulatory system shows relatively mild response in hypothermia partly owing to the suppression of metabolism and the portal interruption might be carried out without danger, if performed within the permissible duration. Moreover, if cooling be carried out safely without causing dangerous complications as ventricular fibrillation or after-bleeding, it would be made possible to prolong the duration of the interruption by lowering temperature further, being supported by BIGELOW's[3)] results that oxygen consumption draws near zero as body temperature falls to 10 to 12°C.

V. SUMMARY AND CONCLUSION

The influence of acute complete portal interruption were studied under hypothermia, employing dogs, and results obtained are as follows ;

(1) Survival rate was 80 per cent when the portal interruption was performed for 60 minutes under rectal temperature of 28.5 to 30.0°C, and the rate rapidly decreased when the interruption was carried out longer. Hence, permissible duration of portal interruption is assumed to be around 60 minutes.

(2) In hypothermic group, both descension of arterial pressure and elevation of portal pressure are gradual after the interruption, showing a difference of approximately more than 20 mmHg during the interruption. Evans Blue injected into the portal vein appears more rapidly into the systemic circulation in hypothermic group. These findings suggest the fact that less congestion of the blood in splanchnic area is caused by the portal interruption under hypothermia.

(3) Circulating blood volume showed a decrease of 21.2 per cent in hypothermic group 30 minutes after the interruption which is a decrease of one half compared to normothermic group, what also enables an assumption that animals are less influenced by the interruption under hypothermia.

Circulation time is prolonged 30 minutes after the occlusion in normothermic group and hypothermic one as long as twice and thrice, respectively.

(4) Electrocardiographic changes were slight in hypothermic group. Decrease in cardiac output was conspicuous in the both groups, but its degree was more slight in hypothermic group compared to normothermic group.

(5) Oxygen consumption decreased by 80.6 per cent in hypothermic group by the interruption, while 41.4 per cent in normothermic group.

(6) Observing from an aspect of gas metabolism, the difference of oxygen content between arterial and venous blood increased from 2.2 vol. per cent to 9.7 vol. per cent by the interruption in normothermic group, on the contrary, it showed only a slight increase from 1.2 vol. per cent to 2.6 vol. per cent in hypothermic group.

Oxygen saturation in arterial blood showed decrease of 9.1 per cent by the interruption in normothermic group, whereas it remained within as low a level as 4.1 per cent in hypothermic group.

Accomplishing the experiment, I express my deepest gratitude to Prof. Dr. Ichio Honjo for his enthusiastic guide and benevolent advises. I am indebted at the same time to the members of our clinic for their kind assistances throughout the work.

VI. References

1) Austin, J. H. : A note on the estimation of carbon dioxide in serum in the presence of ether by the Van Slyke method. J. Biol. Chem., **61**, 345, 1924.
2) Allen, F. M. : Theory and therapy of shock reduced temperatures in shock treatment. Amer. J. Surg., **60**, 335, 1943.
3) Bernard, M. C. : Critique experimentale sur la fonction glycogénésique du foie. Compt. rend. Acad. sc., Paris. **84**, 1201, 1877.
4) Boyce, F. F., Lampert, R., Mc Fetridge, E. M. : Occlusion of the portal vein, an experimental study with its clinical application. J. Laborat. Clin. Med., **20**, 935, 1935.
5) Blalock, A., Manson, M. F. : A comparison of the effects of heat and those of cold in the prevention and treatment of shock. Arch. Surg., **42**, 1054, 1941.
6) Brunschwig, A. : The surgical treatment of carcinoma of the body of the pancreas. Ann. Surg., **120**, 406, 1944.
7) Bigelow, W, G,. Callaghan, J. C., Hopps, J. A. : General hypothermia for experimental intracardiac surgery. Ann. Surg., **132**, 531, 1950.
8) Bigelow, W. G., Lindsay, W. K., Greenwood, W. F. : "Hypothermia" its possible role in cardiac surgery. An investigation of factors govering survival in dogs at low body temperature. Ann. Surg., **132**, 849, 1950.
9) Bigelow, W. G. et al. : Oxygen transport and utilization in dogs at low body temperatures. Amer. J.

Physiol., **160**, 125, 1950.
10) Colp, R. : The treatment of pylephlebitis of appendicular origin. With a report of three cases of ligation of the portal vein, Surg. Gyn. Obstetr., **43**, 627, 1926.
11) Child, C. G., et al. . Sudden and complete occlusion of the portal vein in the Macaca Mulatta monkey. Ann. Surg., **132**, 475, 1950.
12) Csillag, I., Jellinek, H., Egedy, E. : Prevention of portal death by means of hypothermia. Acta Morphol., **4**, 259, 1954.
13) Duchinova, S. I. : Über temporäre Abklemmung des Lig. Hepatoduodenale für blutlose Operationen an der Leber. Z. Org. ges. Chir., **35**, 581, 1926.
*14) Date, M. : Experimental studies on reestablishment of the portal vein. Arch. Jap. Chir., **29**, 1667, 1960.
15) Elman, R., Cole, W. H. : Loss of blood as a factor in death from acute portal obstruction. Proc. Soc. Exper. Biol. Med., **29**, 1122, 1932.
16) Elman, R., Cole, W. H. . Hemorrhage and shock as causes of death following acute portal obstruction. Arch. Surg,. **28**, 1166, 1934.
*17) Fujii, Y. Biochemical experimental methods. Nanzando. 1946.
*18) Fujiwara, K. : Studies on the general hypothermia and the induction of the ventricular fibrillation. J. J. S. S., **57**, 1129, 1955.
19) Fisher, B. et al. . Observation on liver blood flow. Its relationship to cardiac output in anesthetized and unanesthetized animals. Arch. Surg., **72**, 600, 1956.
20) Gregerson, M. I. : A practical method for the determination of blood volume with dye T-1824. J. Laborat. Clin. Med., **29**, 1266, 1944.
21) Goodall, G. W., Burton Hydman, W. W., Gurd, F. N. : Studies on hypothermia in abdominal surgery. II. Occlusion of vascular inflow to the liver. Arch. Surg., **75**, 1011, 1957.
*22) Hatagoshi, M. . An experimental studies on the occlusion of the portal vein. J. J. S. S., **17**, 150, 1913.
23) Itō, H., Omi, K. : Klinische und Experimentelle Beiträge zur Chirurgischen Behandlung des Ascites. Dtsch. Zschr. Chir., **62**, 141, 1902.
24) Johnstone, F. R. C. : Acute ligation of the portal vein. Surg., **41**, 958, 1957.
25) Krymholz, H. L. : Experimenteller Beitrag zur Frage der Unterbindung der Pfortader (Einwirkung auf Druck und Gerinnungsvermögen des Blutes). Zsch. Exper. Med., **67**, 319, 1929.
26) Laborit, H., Huguenard, P. L'hibernation artificielle par moyens pharmacodynamiques et physique. Presse Méd., 1329, 1951.
27) Mc Quiston, W. O. Anesthetic problems in cardiac surgery. Anesthesiology., **10**, 590, 1949.
28) Mallet-Guy, P. et al. : Étude expérimentale de l'interruption brusque du courant portal. Lyon Chir., **48**, 852, 1950.
*29) Mikami, J. et al. : An experimental studies on the occlusion of afferent hepatic flow in hypothermia. J. J. S. S., **60**, 28, 1960.
*30) Morino, M. . Experimental studies on reestablishment of the portal vein. pathological findings of dogs with portal occlusion, portal transplantation and Eck's fistula. Arch. Jap. Chir., **30**, 148, 1961.
31) Neuhof, H. . Experimental ligation of the portal vein ; its application to the treatment of suppurative pylephlebitis. Surg. Gyn. Obstetr., **16**, 481, 1913.
32) Nieder, F. F., Mattes, W. : Über die Unterbindung der Vena Portae. Dtsch. Med. Wschr., **81**, 458, 1956.
33) Oré, M. . Influence de L'oblitération de la veine porte sur la sécrétion de la bile et sur la fonction glycogénique du foie. Compt. rend. Acad. sc., Paris. **43**, 463, 1935.
*34) Okamura, H. et al. . Studies on the application of hypothermia to the portal vein injury. Rinshō Gekka., **12**, 715, 1957.
*35) Oyanagi, T. : in press.
36) Popper, H. L., Jefferson, N. C. . A simple method of gradually producing permanent occlusion of portal vein in the dog. Proc. Soc. Exper. Biol. Med., **85**, 67, 1954.
37) Peck, M. E., Grover, R. F. . Cardiovarcular responses to acute ligation of portal vein. Arch. Surg., **64**, 665, 1952.
38) Ransohof, J. L. . Cause of sudden fall in blood pressure while exploring the common bile-duct. Ann. Surg., **48**, 550, 1908.
39) Raffucci, F. L. : The effect of temporary occlusion of the afferent hepatic circulation in dogs. Surg.

133, 342, 1953.

40) Raúl, H., Mejíia, M. D. . Myocardial necrosis in experimental occlusion of the portal vein. Circulat. Res., **8**, 495, 1960.

41) Schiff, M. : Über das Verhältniss der Lebercirculation zur Gallenbildung. Zbl. med. Wiss., **1**, 115, 1863.

42) Solowief, A. : Veränderung in der Leber unter dem Einflusse künstlicher Verstopfung der Pfortader. Virchows Arch. Path. Anat., **62**, 195, 1875.

43) Smith, L. W., Fay, T. : Temperature factors in cancer and embryonal cell growth. J. Amer. Med. Asso., **113**, 653, 1939.

44) Swan, H., Zeavin, I., Virtue, R. W. : Cessation of circulation in general hypothermia. II. Anesthetic management. Anesthesiology., **15**, 113, 1954.

45) Swan, H. et al. Hypothermia in surgery, analysis of 100 clinical cases. Ann. Surg., **142**, 382, 1955.

*46) Shimizu, Y. . A fundamental study on utilization of hypothermia in hepatic surgery. Fukuoka Igaku Zassi., **50**, 2140, 1959.

47) Thöle, F. . Verletzung der Vena Portae. Neue Dtsch. Chir., **4**, 131, 1912.

48) Tanturi, C. et al. Electrocardiographic and humoral changes in transient occlusion of the portal vein in dog. Surg. Gyn. Obstetr., **110**, 537, 1960.

49) Van Slyke, D. D., Neil, J. M. : The determination of gases in blood and other solutions by vaccum extraction and manometric measurement. J. Biol. Chem., **61**, 523, 1924.

50) Wiggers, C. J. . Peripheral circulation. Annual Rev. Physiol., **9**, 255, 1947.

51) Whipple, A. O., Parsons, W, B., Mullins, C. R. . Treatment of carcinoma of the Ampulla of Vater. Ann. Surg., **102**, 763, 1935.

*52) Watanabe, A. : Fundamental and clinical studies on hypothermia in general and cerebral surgery. J. J. S. S., **59**, 743, 1957.

*53) Yabuki, K. . Circulatory disturbances of the portal trunk and changes in the liver. J. J. S. S., **20**, 81, 1917.

(* Written in Japanese)

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低体温下，門脈暫定的遮断に関する実験的研究

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著者は犬を用い，低体温下門脈急激完全遮断の許容時間を検討し，門脈一時遮断に際しての低体温の主として呼吸，循環動態ならびにガス代謝に及ぼす影響を実験的に追求し，次の結果を得た．

(1) 直腸温 28.5乃至 30.0℃の間では60分遮断にて80%の生存率を得，これを越えると生存率の激減することより，門脈遮断の安全許容時間は60分前後とみなし得る．

(2) 低温群では遮断後，動脈圧の下降，門脈圧の上昇ともに緩除であり，遮断中に大略 20mmHg 以上の圧差を示す．また門脈へ注入した Evans-Blue は低温下では速やかに大循環系へ現われる．これらの事実は低温下では，門脈遮断による門脈領域への血液貯溜のすくないことを意味する．

(3) 循環血液量は遮断後30分にて，低温群は 21.2%の減少と常温群の約 1/2 の値を示し，遮断による影響はすくない．循環時間は遮断後30分で常温群では2倍，低温群では3倍の延長を示す．

(4) 低温群では心電図上の変化も軽微であり，分時搏出量は常温群低温群共に著減するが，低温群は常温群に較べ，その度は軽い．

(5) 酸素要求量も遮断時，常温群の 41.4%の減少に比し，低温群は 80.6%の減少を示した．

(6) ガス代謝の面からみると，動静脈酸素較差は結紮時，常温群は2.2vol.%より9.7vol.%と上昇を示すのに比し，低温群は1.2vol.%より2.6vol.%と遮断による増減はすくなかつた．また動脈血酸素飽和度は遮断後，常温群は 9.1% の減少を示すに対し，低温群では4.1%の減少にとゞまる．

Plate 1 : Liver
Hypothermic group. H. E. × 100

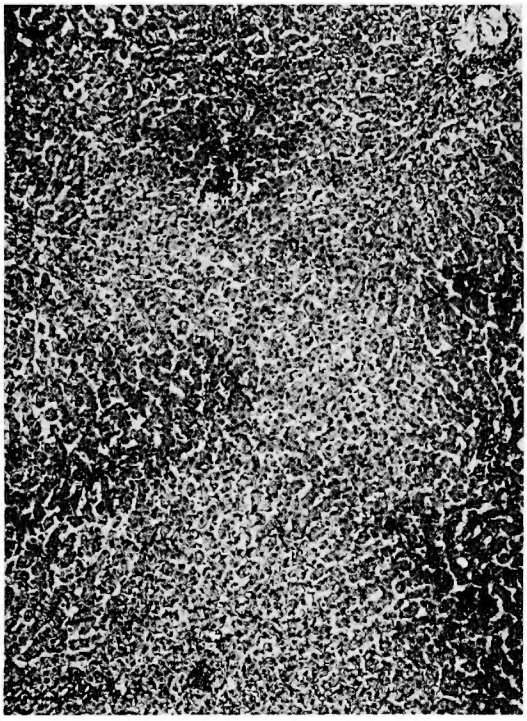


Plate 2 : Liver
Normothermic group. H. E. × 100

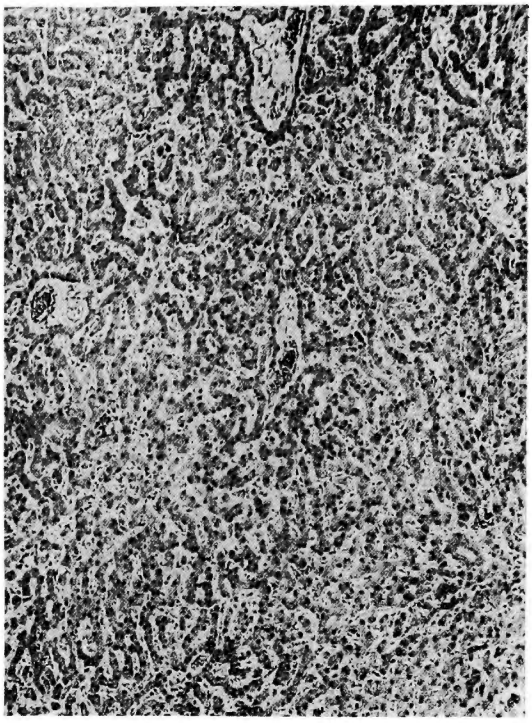


Plate 3 : Small intestine
Hypothermic group. H. E. × 100

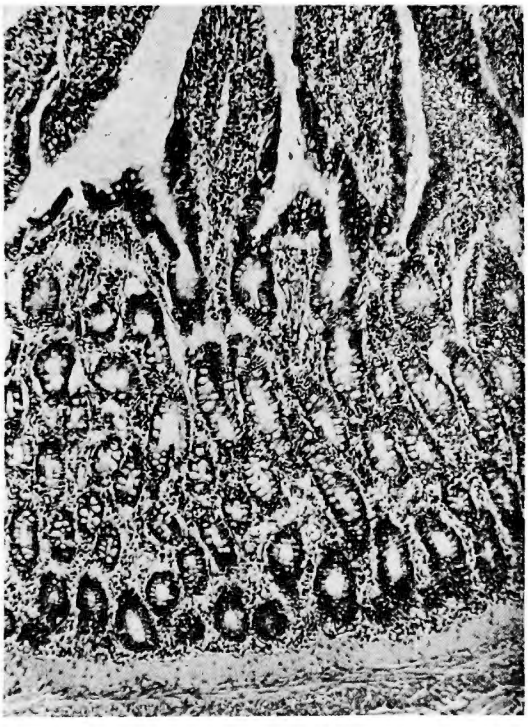
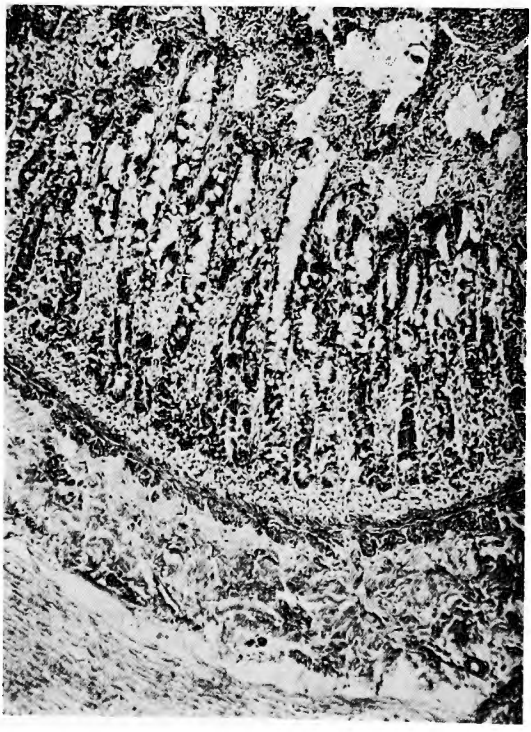


Plate 4 : Small intestine
Normothermic group. H. E. × 100



Portalograph

Plate 5 : Hypothermic group

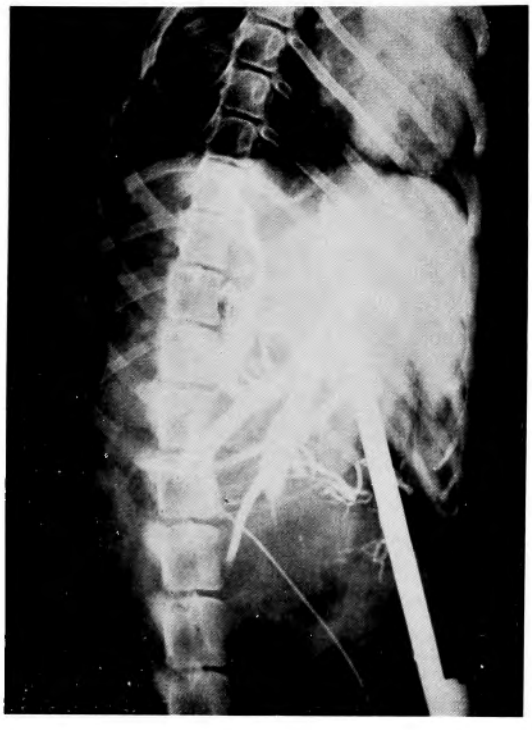


Plate 6 : Normothermic group