An Experimental Study on Transplantation of the Aortic Arch*

Part. I. An Experimental Study on Interruption of the Ascending Aorta

By

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(Received for publication, April 22, 1957)

INTRODUCTION

Remarkable advances have been made in cardiovascular surgery in recent years. Especially, in the field of vascular surgery, not only the peripheral blood vessels and abdominal aorta, the thoracic aorta is the subject of surgery. The latter which once considered to be difficult to interrupt for a long period under normal body temperature is now possible to interrupt its circulation by applying hypothermia and various shunts. Restoration of the vascular continuity after resection of the abdominal aneurysm has also become feasible. In case aneurysm involves the ascending aorta and aortic arch, and when malignant tumor in the chest extends to this region, the surgical approach, however, has been considered to be difficult. Since Hardin *et al.*¹⁾ in 1952 reported an experimental study on transplantation of the aortic arch, sporadic clinical cases on this subject have appeared in the literatures. As one of the application of severe hypothermia which is studied by Watanabe *et al.*²⁾ at this clinic, the author attempted to transplant the aortic arch after the cessation of circulation for a long time under hypothermia without aid of shunts. As a preliminary experiment, cessation and reanastomosis following division of the ascending aorta under various rectal temperatures were performed to study its applicability.

EXPERIMENTAL

Method

Anesthesia to obtain severe hypothermia

* Read in part at the 9th Annual Meeting of the Japan Thoracic Society, Tokyo, October 22, 1956.

After subcutaneous injection with 0.5–0.8 cc. atropine, the dog was anesthetized with injection of Ravonal 15–20 mg./kg. body weight. Then, endotracheal tube was inserted, which was connected to a closed circuit anesthesia machine. When the 3rd phase of 3rd plane anesthesia was obtained, the whole body including the head was immersed into a ice water tub in a supine position. In case shivering was noticed during cooling, ether was added to the machine till it stopped. 100% oxygen was used for respiration. Though the dogs were allowed to breath by its own will, assisted respiration was started when it became weak at rectal temperature 20°–23°C and controlled respiration after it completely stopped. Rectal temperature was all kept below 20°C (19.6°–14°C). When ECG showed cardiac arrest, the animal was immediately removed from the ice water, wiped dry and operation started. The time for cooling was 50–140 minutes.

Operation

The chest was opened at the left 4th intercostal space without cutting the sternum. The ascending aorta was freed from its origin till branching of the right brachiocephalic artery distally. In case complete dissection was impossible, a longitudinal incision was placed in the pericardium and the fat tissue at the reflecting point of the pericardium was excised.

Interruption of the circulation to and from the heart

The ascending aorta was interrupted with a Satinsky's clamp covered with rubber which was placed just above the aortic valve in 15 of 19 dogs. 4 were interrupted under severe hypothermia with simultaneous interruption of the intrapericardial superior and inferior vena cavae by constricting the Nelaton's tube and the ascending aorta just as before. Release of the interruption of inflow tract and the outflow tract in the order given was carried out.

Division of the ascending aorta and its reanastomosis

After the cessation of blood flow of the heart, a Satinsky's rubber shod clamp was placed on the right brachiocephalic artery adjacent to the point where it came off the aortic arch and the ascending aorta was divided in between. 10% sodium citrate solution was frequently injected into the inside to prevent blood clot formation. The posterior row was first anastomosed by twisting 2 supporting stitches on both corners 180 degrees, which was followed with anterior anastomosis. The sutures used were black silk No. 3. Anastomosis was placed intimately with an interval 1–1.5 mm.

Cardiac resuscitation and rewarming

After the interruption was released, the clamp on the central side was removed and the heart was massaged several times, which was followed by removing the distal placed clamp. The dog was transferred from the

operating table to a tub filled with warm water kept at about 40°C. With its left side upwards, it was placed on a vinyl cloth, which was covered over the water. Placing the dog in a slight Trendelenburg's position, cardiac massage was instituted, while the dog was hyperventilated via artificial intermittent respiration during warming process. Cardiac massage was effected by placing both palms of hands intrapericardially, with a rate of 24–30 times a minute, which was increased to 60 times a minute in accordance to the elevation of body temperature. Starting cardiac massage, cardiac standstill was converted to ventricular fibrillation or rhythmic contraction of the right atrium within 4–5 minutes. Counter shock was, however, not used till the rectal temperature rose over 28°C, when 0.2–0.8 cc. noradrenaline was injected into the left ventricle. Counter shock was applied to the fibrillating heart which became active by injection of drug. After defibrillating the heart, in case the cardiac rhythm was weak, 2% calcium chloride 10–15 cc. was injected slowly through the sublingual vein. Confirming that the cardiac contraction became strong, the dog was removed from the tub and chest closed with a draining tube inserted. Rewarming was continued till the rectal temperature returned to normal. The total time required for warming the body was 100–140 minutes.

Results

Group with interruption of the ascending aorta

1) Under normal body temperature. All 3 dogs died by massive bleeding at the clamped site of the ascending aorta within 2–4 minutes. Cardiac standstill and ventricular fibrillation were, however, not observed.

2) Under hypothermia (rectal temperature 28°–24.5°C). 4 dogs were used, of which 3 died from rupture at the clamped site 11, 14 and 18 minutes, respectively. Macroscopically, all cases but one didn't show arrhythmia. Another was interrupted for 19 minutes at rectal temperature 14.5°C. 3 minutes after the release of clamps, bradycardia and arrhythmia occurred, which was followed with ventricular fibrillation. Cardiac massage and warming were both started immediately. 0.6 cc. noradrenaline was injected into the left ventricle at rectal temperature 28°C and counter shock applied, which brought temporary normal rhythm to the heart. Eventually the dog died an hour later.

3) Under severe hypothermia (rectal temperature 17°C, 14.5°C) (Table I). In dog No. 24 cardiac resuscitation was successful. The dog died, however, an hour later with central respiratory paralysis. The circulation of No. 25 was interrupted for 70 minutes at rectal temperature

TABLE I
Cessation of the Ascending Aorta at Severe Hypothermia

Dog No.	Body weight (kg)	Rect. temp. (°C)	Time of interruption (min.)	Cardiac resuscitation	Survival time	Cause of death
24	8.0	17.0	34	Success	one hour	Paralysis of respiration
25	6.5	14.5	70	Success	one year	Living

Fig. 1. Electrocardiographic tracing in dog No. 25. *a*: body temperature 36°C. (control ECG, 2nd lead). *b*: rectal temperature 22°C (during cooling). *c*: 16.5°C (during cooling). *d*: 14.5°C (during cooling). *e*: 25 minutes after interruption of blood flow. *f*: 50 minutes after interruption of blood flow. *g*: 70 minutes after interruption of blood flow (release of clamp). *h*: right after counter shock (heart out of pericardium). *i*: rectal temperature 33°C (during rewarming). *j*: 8 days after operation.

14.5°C, and it is still living one year later. No postoperative sequelae has been observed. The ECG are reproduced in Fig. 1. With fall of rectal temperature, tendency of sinus bradycardia is observed. At 22°C, negative T, notching of ST, prolongation of PR interval and QRS; at 14.5°C, wide P, remarkable decrease of ST, reversed T, notching in QRS were observed. 15 minutes after cessation of blood flow, prolongation of P, and amplitudes of QRS were noticed. Extrasystoles were observed 25 minutes later, complete A-V block 50 minutes later, and transition to ventricular fibrillation with release of obstruction 70 minutes later. After successful defibrillation after rewarming to rectal temperature 28°C, tachycardia and elevation of QRS were noticed, and return to normal tracing was noticed on the 8th postoperative day.

Group with division and reanastomosis of the ascending aorta under severe hypothermia (rectal temperature 18°–12°C)

1) Interruption of the ascending aorta alone (Table II). 6 dogs were used. No permanent survival was obtained. The time for anastomosis was 20–57 minutes at rectal temperature 18° – 14.5° C, while the time

TABLE II
Division and Reanastomosis of the Ascending
Aorta under Severe Hypothermia

Dog No.	Body weight (kg)	Rect. temp. ($^{\circ}$ C)	Time of interruption (min.)	Cardiac resuscitation	Survival time (hrs.)	Cause of death
21	12	16	32	Success	2	Bleeding from anastomosis.
22	13	18	48	Success	0	Bleeding from clamped site.
23	8	17	30	Failure		Mishappening of injecting citrate.
26	14	13	38	Success	0	Bleeding from anastomosis.
27	12	15	65	Success	2	"
28	14	15	60	Success	1	"

for cessation of blood flow was 32–65 minutes. All cases but No. 23 were successfully resuscitated. The cause of death in Nos. 21, 26, 27 and 28 is due to rupture at the anastomosis. Bleeding was not observed during cardiac massage when blood pressure was low, whereas massive bleeding occurred when normal heart beat returned after electric shock was applied. Rupture at the clamped site was the cause of death in No. 22, which occurred by twisting the clamp during posterior anastomosis. The injury was overlooked, and the death was not due to overburden on the heart (Fig. 2).

Fig. 2. Injury of intima at the left lower corner (↑) is observed in dog No. 22.

2) Cessation of inflow and outflow tract of the heart (Table III). 4 dogs were used. The time for interruption was 49–75 minutes. All but No. 31 were resuscitated, but no permanent survival was obtained. The cause of death was obstruction due to thrombus at the descending thoracic aorta and the ascending aorta, respectively. Bilateral pneumothorax occurred in No. 34 during operation, which showed atelectasis of lung at autopsy (Fig. 3).

TABLE III
Anastomosis of the Ascending Aorta with Simultaneous
Cessation of the Inflow and Outflow Tract of Heart

Dog No.	Body weight (kg)	Rect. temp. (°C)	Time of interruption (min.)	Cardiac resuscitation	Survival time (hrs.)	Cause of death
31	15.0	15.0	65	Failure		
32	11.5	13.0	75	Success	4	Shock due to obstruction of the descending thoracic aorta.
33	11.0	12.0	49	Success	4	Thrombus at anastomosis of the ascending aorta.
34	9.0	14.0	53	Success	48	Collapse of lung.

Fig. 3. Removed specimen in dog No. 34.

DISCUSSION

When the ascending aorta is interrupted, the blood flow to the brain is completely shut off. Cessation of cerebral circulation using such method is reported only by Nakayama³⁾ and Cooley *et al.*⁴⁾ The dog in their experiments died in cardiac arrest which occurred after a few heart beats

or 4 minutes later in an over distended ventricle. The author experienced that rupture at the clamped site occurred within 2–4 minutes before cardiac arrest. Kimoto *et al.*⁵⁾ observed that in case the inflow tract of the heart and the aorta were both obstructed at the same time over 5 minutes, all dogs died in cardiac arrest or ventricular fibrillation with loss of cerebral function after 3 minutes. The survival limit of interrupting cerebral blood flow depends on the condition which it was carried out. It is believed that 3 minutes is the limit at normal body temperature.

After advances in hypothermia, it has become possible to prolong the cessation of blood flow of the heart. Marshall *et al.*⁶⁾ found out that when dog was cooled to 26°C, the tissue oxygen consumption would diminish about 1/3. They hypothesized that it would be possible to prolong the cessation of circulation 3 times longer in a dog at this temperature, which was substantiated by performing various method of cessation. In our hands, as the dog died from rupture at the clamped site, nothing can be said about damage of cerebral functions. According to Toyoshima⁷⁾ at this clinic, the oxygen consumption decreased 50% at 26°C, 80% at 20°C and 90% at 16°C. In our experiments, when the ascending aorta was interrupted under severe hypothermia, the cardiac muscles became bright red due to maintenance of the coronary circulation. After several heart beats a minute lasting for 20–30 minutes (the longest continued 70 minutes), it converted to ventricular fibrillation. In case the inflow tract was also interrupted, the cardiac muscles became immediately bluish, converted to ventricular fibrillation and stopped in cardiac standstill. All hearts were, however, successfully resuscitated.

In those cases with reanastomosis of the ascending aorta, permanent survival could not be obtained. The main causes are bleeding from the anastomosis in face of enough mobilization of the aorta. Especially, it is difficult to perform posterior anastomosis by twisting the aorta. In dogs the ascending aorta is very fragile and difficult to suture which is shown by the wide range of time for anastomosis (20–50 minutes). As anastomosis was securely made, no bleeding was observed during cardiac massage. Massive bleeding was, however, observed after defibrillation by electric shock. There was no way to prevent bleeding from the posterior wall.

SUMMARY

In case the ascending aorta is interrupted, the clamped site will rupture prior to heart failure due to rapid overload on the heart regardless under normo or hypothermia.

Under severe hypothermia, it will, however, tolerate long cessation due to remarkable decrease of burden on the heart. Most of the cases

could be successfully resuscitated after the aorta alone is interrupted or the inflow and outflow tract are both obstructed at the same time. In case the ascending aorta is divided and reanastomosed, performance of operation is difficult and the main fatal cause is massive bleeding from the anastomosis.

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