Effectiveness of Assistance Circulations for Distal Circulatory Support during Cross-clamping of the Descending Thoracic Aorta

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ABSTRACT

In order to find the ideal distal circulatory support during cross-clamping of the descending thoracic aorta, the author compared the effects of simple shunting (SS) and centrifugal pump (CP) on hemodynamics and metabolisms in mongrel dogs. In group I (control), the aorta was cross-clamped for two hours without SS or CP; in group II (SS), a temporary shunt was placed between the left common carotid and left femoral arteries during cross-clamping; in group III (CP), with left heart bypass, the flow was maintained about the same as that in group II; in group IV (CP), proximal pressure was maintained unchanged. Hemodynamic and metabolic parameters were recorded prior to cross-clamping and every 30 min for four hours during and after cross-clamping. All animals in group I suffered from hemodynamic instability, metabolic abnormalities and neurologic injury and died within 12 hours. Hemodynamic changes were more unstable in group III than in groups II and IV. Three dogs in group III and also in group IV but none in group II, suffered from neurologic injury. Metabolic changes in groups II, III and IV were not significant. The author conclude that hemodynamic and metabolic abnormalities can be minimized through the efficient use of a shunt in cross-clamping of the descending thoracic aorta and postoperative complications such as paraplegia, renal failure and hepatic dysfunction can be prevented. The centrifugal pump as a distal circulatory support device is able to maintain stable hemodynamics and normal distal organic metabolisms if aortic pressure proximal to the clamp is maintained essentially unchanged through regulation of pump flow. However, it is unable to prevent paraplegia during cross-clamping of the descending thoracic aorta for two hours. Shortening the time of cross-clamping of the descending thoracic aorta is necessary to prevent paraplegia.

Key words: *Aorta, Clamp, Shunt.*

Cross-clamping of the proximal descending thoracic aorta causes a severe and sudden increase in peripheral resistance and left ventricular afterload. These changes frequently lead to sufficient heart strain to induce ventricular fibrillation or heart failure and cardiac arrest. Moreover, cross-clamping the distal descending thoracic aorta may cause temporary or permanent ischemic injury to distal organs. Moreover, after declamping, reperfusion injury resulting in metabolic disorder and shock can occur, particularly when the clamp-time is excessive and collateral circulation is cut off. For these reasons, surgeons have developed shunting procedures using a simple catheter or a centrifugal pump to decompress the proximal compartments and perfuse the distal aortic segment. In 1910, Carrel[4] performed thoracic aortic occlusion and recommended temporary diversion of the aortic blood flow to prevent paraplegia. In 1984, Olivier et al[28] reported on the use of the Biomedicus centrifugal pump in the surgical treatment of traumatic tears of the descending thoracic aorta. They showed more reliable decompression of the left ventricle and better control of distal aortic flow than with simple passive shunts. These techniques have been studied extensively in laboratory and in clinical work[3,5,6,13,16,19,25,30,35]. Despite the introduction of these techniques however, operations involving cross-clamping of the proximal descending thoracic aorta are still associated with an incidence of paraplegia of 7% to 16%[1,2,7,11,17,20–22,26,35,38], of renal failure of 5% to 10%[22,32,33], and a mortality of about 13%[22]. Thus, the usefulness of distal circulatory shunt during cross-clamping of the descending thoracic aorta remains controversial[5,8,24,25,28].

A number of clinical problems remain to be solved. For example, the shunt flow in the passive bypass (SS) is variable because it is a function of the inner diameter of the cannulation sites. This parameter can not be assessed precise-

ly preoperatively. Active bypass (CP) allows more control over flow, but the question remains as to the optimum flow while the aorta is clamped. In clinical practice, it is difficult to draw conclusions because mortality and morbidity are related to various uncontrolled factors, such as the status of patients, the duration of aortic occlusion, the use of different drugs, individual technique, and complications involving the circulatory support methods. Criteria for selecting an appropriate technique of distal circulatory support under a given set of clinical conditions remain to be established. The present study examines hemodynamic and metabolic changes associated with two techniques of distal circulatory support. The arterial blood ketone body ratio (the ratio of acetoacetic acid to β-hydroxybutyric acid) is used as an index of organ perfusion and hepatic energy metabolism[15,29,34,39,40].

MATERIALS AND METHODS

Mongrel dogs weighing 12 to 18.5 kg were divided into four groups of six animals each: Group I, the control group, underwent cross-clamping of the descending thoracic aorta for two hours without any distal circulatory support. At the end of two hours, the cross-clamp was released, and the animals were observed for an additional two hours. In group II, during cross-clamping, a 800-mm long shunt with the proximal and distal inner diameters (ID) of 3 mm and with the diameter of 8.0 mm in its middle was interposed between a left common carotid artery and a left femoral artery. In group III or IV, a centrifugal pump was connected with cannulas measuring 5.0 mm ID at the proximal tip, 8.0 mm ID in the middle, and 3.0 mm ID at the distal tip between the left atrium and the left femoral artery. Bypass flow in group III was maintained about the same as that in group II, and in group IV, proximal pressure was maintained unchanged by regulating the bypass flow.

The dogs were anesthetized with pentobarbital (25 mg/kg) and connected to a mechanical ventilator. Thoracotomy was performed in the fourth left intercostal space. The shunt was opened when the descending thoracic aorta was clamped distal to the first four pairs of intercostal arteries, distal to the ligamentum arteriosum. The pressure monitoring lines were placed, one in the ascending aorta via the right carotid artery and another in the right femoral artery. An electromagnetic blood flowmeter probe (Model FF-100 T, Nihon Kohden Corp., Tokyo, Japan) was connected between the shunting cannulas in groups II, III or IV. A Swan-Ganz catheter was inserted through the right jugular vein and floated in the pulmonary artery. Cardiac output was determined by the thermal dilution technique using a cardiac output computer (Edward Laboratories,

Santa Ana, CA). The arterial blood ketone body ratio was measured using a commercially available KETO-II kit (Sanwa Kagakukenkyusho Co., Ltd., Nagoya, Japan). The descending thoracic aorta was cross-clamped for two hours. Hemodynamic data and blood samples were obtained prior to aortic cross-clamping and thereafter every 30 minutes until the end of the experiment. Animals in groups II, III and IV were given heparin 200 units/kg intravenously to prevent thrombosis in the shunt and pump. Crystalloid and 10% glucose solutions 12–14 mL/kg/h were administered intravenously to all animals to maintain hemodynamic stability. Vasoactive drugs, blood, or blood products were not used.

The hemodynamics parameters measured were heart rate, cardiac output, ascending aortic pressure, femoral arterial pressure, shunt flow, and pump flow. The metabolic parameters measured were: SGPT, blood urea nitrogen (BUN), arterial blood pH, and the arterial blood ketone body ratio.

All data are presented as means $\pm$ SD. Student's two tailed t test for unpaired data was used to determine statistically significant differences between groups, and Student's two tailed t test for paired data was used to determine differences within a group. Differences were considered significant for p values less than 0.05.

Animals used in this study received humane care, in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals," prepared by the National Academy of Science and published by the National Institutes of Health (NIH publication No. 86–23, revised 1985).

RESULTS

Baseline values showed no significant intergroup difference in any hemodynamic or metabolic parameter prior to cross-clamping the descending thoracic aorta (Table 1).

Experimental outcome. All six animals in group I died within 12 hours following the termination of the experiment. Only three animals of them awoke from anesthesia but suffered from gastrointestinal hemorrhage. Two of the three had paraplegia and the third one had paraparesis. All animals in group II recovered from anesthesia without neurologic or gastrointestinal complication. Two of the animals died within 12 hours, one of pulmonary edema secondary to excessive fluid replacement and the other of pneumothorax. Five animals in group III died within 12 hours of hemothorax from hemorrhage in the intercostal muscle after operation. All animals in this group awoke from anesthesia, and three of them were able to stand and walk normally, while the remaining three dogs showed paraparesis: they

Table 1. Base line Hemodynamic and Metabolism Parameters Prior to Cross-clamping the Descending Thoracic Aorta

Variables	Group I	Group II	Group III	Group IV	p Value
AP (mm Hg)	164±5	173±16	176±31	152±7	NS
FAP (mm Hg)	172±7	180±21	171±32	164±14	NS
CO (mL/kg/min)	288±94	207±49	197±34	236±13	NS
HR (beats/min)	214±22	197±27	212±16	211±22	NS
KBR	0.62±0.2	0.83±0.25	0.86±0.31	0.54±0.10	NS
GPT (IU/L)	63±23	78±55	60±31	69±53	NS
ABpH	7.57±0.08	7.47±0.08	7.5±0.07	7.46±0.07	NS
BUN (mg/dL)	18.4±2.6	23.9±6.6	16.6±7.3	13.3±0.77	NS

AP: Aortic pressure. FAP: Femoral arterial pressure. CO: Cardiac output. HR: Heart rate. KBR: Ketone body ratio. AB pH: Arterial blood pH. GPT: Glutamic-pyruvic transaminase. BUN: Blood urea nitrogen. Values represent mean±SD.
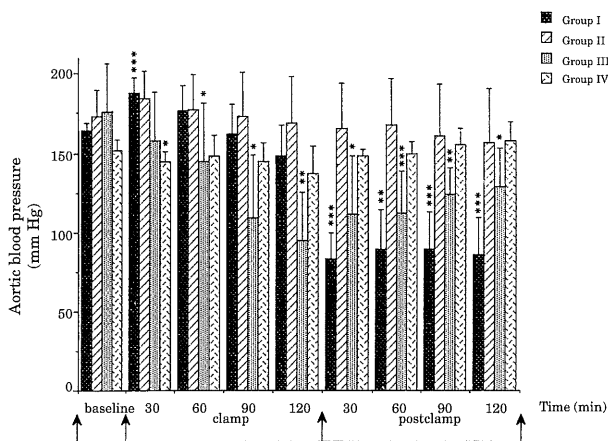

Fig. 1. Serial changes in ascending aortic pressure in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.
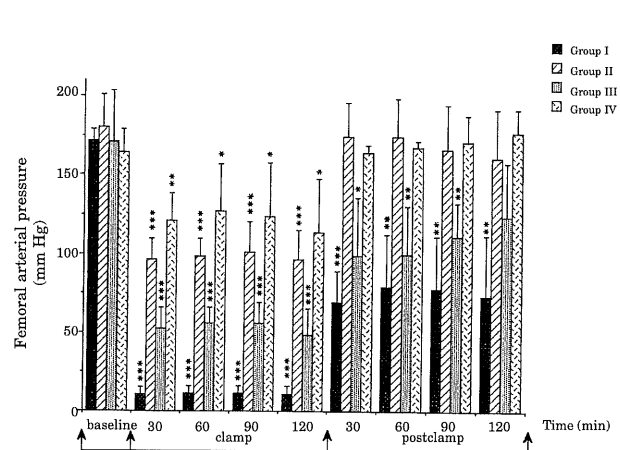

Fig. 2. Serial changes in femoral arterial pressure in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.

were able to move their hind legs but unable to stand or walk normally. No animal in the group suffered from gastrointestinal hemorrhage. All six dogs in group IV awoke from anesthesia immediately and lived for over 24 hours. Three of them were able to stand and walk normally, while the remaining three dogs showed paraparesis.

Ascending aortic pressure. Cross-clamping of the descending thoracic aorta elevated the proximal aortic pressure during the initial period. The pressure in group I rose from 164±5 (control) to 188±10 mm Hg at minutes 30 (p<0.01), and returned to 148±19 mm Hg at minutes 120. In group III, the pressure fell from 176±31 to 145±37 mm Hg at minutes 60 (p<0.05) and to 95±30 mm Hg at minutes 120 (p<0.01). Following the release of the aortic clamp, the ascending

aortic pressure fell below the baseline level in group I (p<0.001). The pressure in group III fell below baseline at minutes 60 and 90 (p<0.01), but then rose. The ascending aortic pressure in groups II and IV were maintained essentially unchanged during the entire period of observation (Fig. 1).

Femoral arterial pressure. Following aortic cross-clamping, the femoral arterial pressure dropped dramatically in group I from 172±7 to 11±5 mm Hg (p<0.001) and remained depressed. After release of the aortic clamp, the pressure rose to 69±20 mm Hg at minutes 30 and to 73±38 mm Hg at minutes 120 but remained significantly lower than the baseline value (p<0.01). In groups II and IV, the pressure fell during cross-clamping, but was higher than in group I (p<0.001) and group III (p<0.01), being between

96 ± 13 and 101 ± 20 mm Hg in group II, and between 113 ± 34 and 127 ± 30 mm Hg in group IV. The pressure returned to baseline immediately after the release of the cross-clamp. In group III, the pressure also dropped following cross-clamping and was between 48 ± 17 and 55 ± 13 mm Hg. The pressure increased to 111 ± 22 mm Hg at minutes 90 following declamping and returned to baseline at minutes 120 (Fig. 2).

Cardiac output. The cardiac output decreased significantly in group III and was $57 \pm 10\%$, $46 \pm 15\%$, $35 \pm 7\%$, and $35 \pm 8\%$ of control after minutes 30, 60, 90, and 120 of cross-clamping and $56 \pm 10\%$, $51 \pm 11\%$, $48 \pm 9\%$, and $51 \pm 22\%$ of control after minutes 30, 60, 90, and 120 of declamping (p<0.01), respectively. Group II also showed a decrease in cardiac output, being $76 \pm 17\%$, $71 \pm 13\%$ and $65 \pm 11\%$ of control after minutes 60, 90, and 120 of cross-clamping (p<0.01) and $71 \pm 17\%$ and $73 \pm 20\%$ of control after minutes 60 and 90 of declamping (p<0.05), respectively. Cardiac output was higher in group II than in group III (p<0.05). Cardiac output decreased significantly in Group I from 90 minutes in cross-clamping to the end of the experiment, $33 \pm 7\%$ of control at minutes 120 after release of the cross-clamp (p<0.01). In group IV, cardiac output also tended to decrease gradually, being about 83%, 68% and 58% of control by minutes 60, 90, and 120 of cross-clamping, respectively. After the release of the cross-clamping, cardiac output recovered partially but remained significantly lower than baseline. The change was similar to that in group II (Fig. 3).

Shunt and pump flow. Bypass flow in group II was 50 ± 15, 47 ± 13, 44 ± 12 and 41 ± 10 mL/kg/min at minutes 30, 60, 90, and 120 of cross-clamping, respectively. Flow was significantly less at minutes 120 than 30 (p<0.05). The shunt carried 28, 31, 31, and 31% of cardiac output at minutes 30, 60, 90, and 120 of cross-clamping (mean, 30%), being 43 ± 9, 41 ± 8, 40 ± 11, and 39 ± 13 mL/kg/min in bypass flow in group III, respectively. Flow was not different between group II and group III, but the ratio of bypass flow to cardiac output was larger in group III than group II. This difference resulted from a decrease in cardiac output in group III. Thus group III showed signs of hemodynamics instability. Bypass flow in group IV also tended to decrease gradually following cross-clamping, from 83 ± 13 at minutes 30 to 71 ± 13, 57 ± 16, and 53 ± 22 mL/kg/min at minutes 60, 90, and 120, respectively. It was approximately 37%, 36%, 36% and 39% of the cardiac output at minutes 30, 60, 90, and 120,
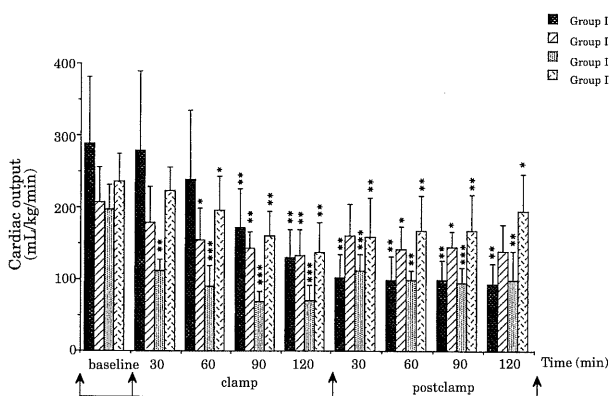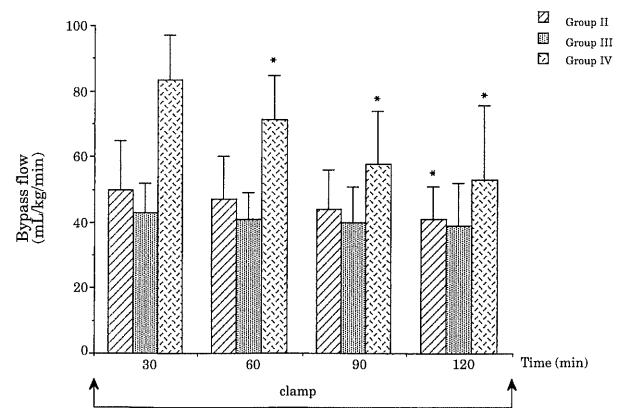

Fig. 4. Serial changes in shunting and bypass flow in group II, III and IV during cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the value at 30 minutes in the same group. n=6 in each group.

Fig. 3. Serial changes in cardiac output in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.
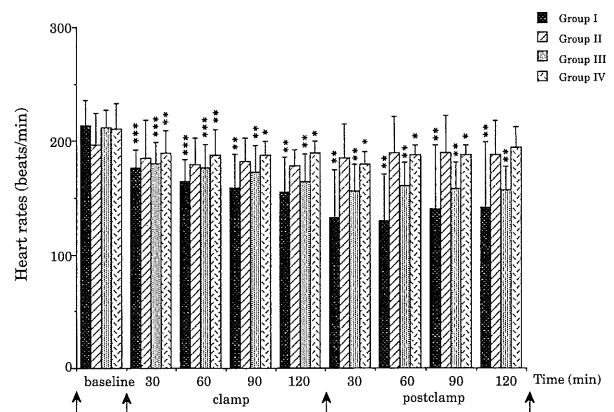

Fig. 5. Serial changes in heart rate in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.
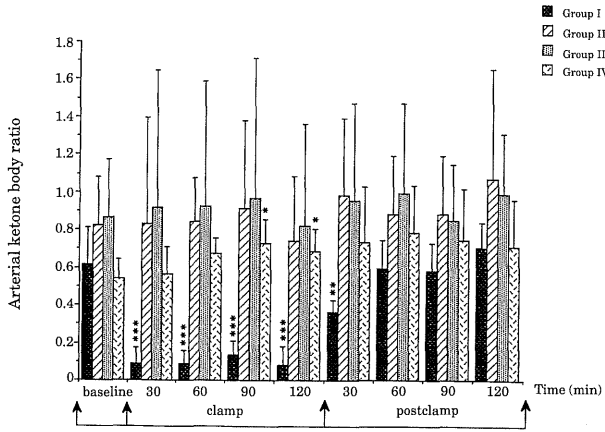
Fig. 6. Serial changes in arterial blood ketone body ratio in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.
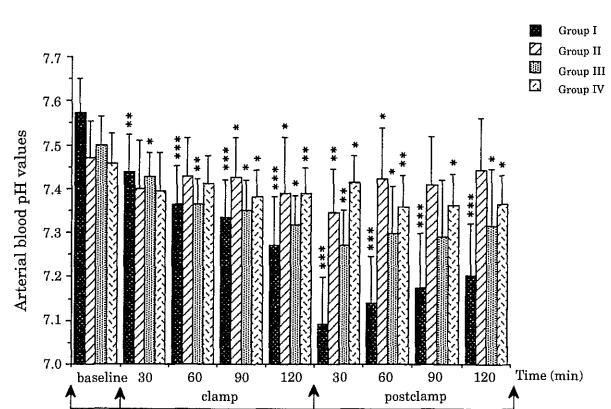

Fig. 8. Serial changes in arterial blood pH in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.
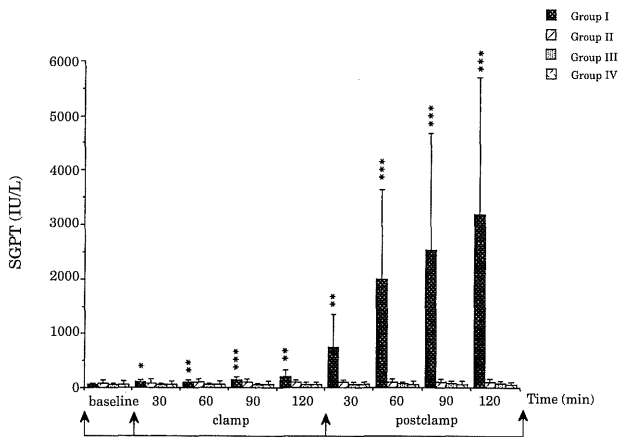

Fig. 7. Serial changes in SGPT concentration in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.
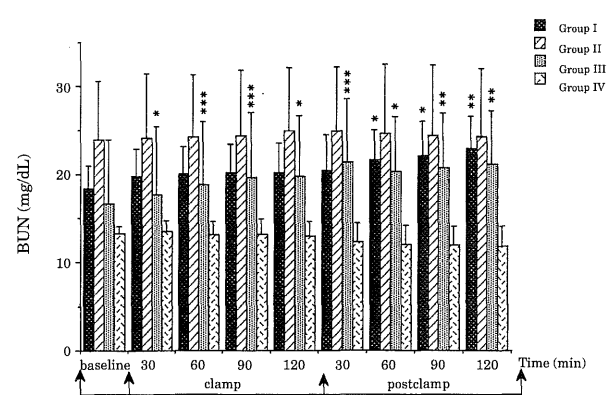

Fig. 9. Serial changes in BUN in group I, II, III and IV before, during and after cross-clamping of the descending thoracic aorta. *p<0.05, **p<0.01, ***p<0.001 versus the baseline in the same group. n=6 in each group.

respectively, during occlusion (mean 37%). The ratio of pump flow to cardiac output in group IV was essentially unchanged, despite the decrease in cardiac output throughout the experiment (Fig. 4).

Heart rate. The heart rate in group II was unchanged during the experiment. Groups I, III and IV showed a significant and persistant reduction in heart rate in the experimental period (p<0.01) (Fig. 5).

Ketone body ratio. The arterial ketone body ratios were essentially unchanged during the entire period of observation in groups II, III, and IV except for a slight increase at minutes 90 and 120 after aortic occlusion in group IV. Thus, pump

flow in group III was adequate to meet hepatic metabolic needs, despite hemodynamic instability. It also demonstrates that stability of hepatic energy metabolism is needed for hemodynamic recovery. On the other hand, an increase in the ketone body ratio at minutes 90 and 120 during aortic occlusion in group IV reflected a hepatic steal phenomenon because bypass flow in this period tended to decrease. There was a dramatic fall in the ketone body ratio in group I to 0.09±0.09 (p<0.001) after cross-clamping, which persisted for the entire period of the cross-clamping, showing that energy metabolism was profoundly disturbed during cross-clamping. Although the ketone body ratio returned to base-

line at minutes 60 after release of the clamp, ir-reversible hemodynanic changes had occurred (Fig. 6).

SGPT. The SGPT concentration in group I increased from 63 ± 23 to 92 ± 44 IU/L at minutes 30 (p<0.05) and to 202 ± 120 IU/L at minutes 120 of cross-clamping (p<0.01), and to 746 ± 597 IU/L at minutes 30 (p<0.01) and to 3186 ± 2518 IU/L at minutes 120 following release of the clamp (p<0.001). The SGPTs did not change significantly in groups II, III and IV (Fig. 7).

Arterial blood pH. Following the application of the aortic cross-clamp, arterial blood pH decreased immediately in group I and remained lower during the entire study period (p<0.01). The most severe acidosis occurred at minutes 30 following clamp release (7.09 ± 0.11, p<0.001). A significant drop also occurred in group III (p<0.05) and was most profound at minutes 30 after declamping (p<0.01). Arterial blood pH in group IV decreased significantly, beginning at minutes 90 after cross-clamping and continuing until the end of the experiment. These changes in pH reflect tissue hypoxia. The arterial blood pH in group II was lower only at minutes 90 and 120 during cross-clamping, and at minutes 30 and 60 after declamping (p<0.05) (Fig. 8).

BUN. The BUN concentrations did not change significantly in groups II and IV. In group I, the BUN was elevated at minutes 60, 90, and 120 following declamping (p<0.05). In group III, the BUN was elevated during the entire period of observation (p<0.01). This showed that not only sufficient bypass flow ($>41 \pm 10$ ml/kg/min) but also higher pressure ($>96 \pm 13$ mm Hg) is needed to maintain renal function (Fig. 9).

DISCUSSION

Repair of thoracic aortic aneurysms and traumatic disruption of the thoracic aorta require that the vessel be clamped. Clamping results in profound hemodynamic changes and metabolic abnormalities[5,19]. Postoperative renal failure and neurologic complications also occur in some patients who undergo cross-clamping of the descending thoracic aorta[5,16,24]. The severity of these changes is related to the site and duration of aortic occlusion and to the degree of distal organic ischemia. A number of techniques to solve these problems have been devised. In 1988, Verdant and his colleagues[37] reported that they prevented neurologic injury in 173 patients with descending thoracic aortic aneurysms using a 9-mm Gott aneurysm shunt placed between the ascending aorta or the proximal descending aorta and the abdominal aorta or the left femoral artery. The mean flow in their patients was 2475 mL/min (range 1100 to 4000 mL/min). The author's study also showed the effectiveness of a simple shunt for preservation of spinal cord per-

fusion during cross-clamping. However, other authors[3,5,28] have reported an incidence of paraplegia of 7%, of renal failure of 3.7% and of renal dysfunction of 46% in patients treated with a Gott shunt. These investigators believed partial cardiopulmonary bypass is preferable for reducing afterload and maintaining distal perfusion during occlusion of the descending aorta, simultaneously allowing better control of flow, and of proximal and distal arterial pressure. In the author's study, however, animals in group III receiving left heart bypass showed an unstable hemodynamic pattern. This outcome may be related to the fact that neither vasoactive drugs nor blood or blood products were used and that the pump flow was limited to the rate of the shunting flow in group II. Cartier et al[5] did not refer to ideal pump flow or distal pressure. Crawford et al[8] compared patients treated with circulatory support with those without and showed that the incidence of paraplegia and the survival rate in patients without shunt or bypass were 0.9% and 91%, respectively. These investigators concluded that descending thoracic aortic aneurysms can be replaced more safely without shunts or bypass because operation is simpler and complications associated with bypass are avoided. In 1985, Molina and colleagues[25] performed a study in mongrel dogs weighing 25 to 35 kg, in which they used three different sized shunts to assess the effectiveness of flow for the prevention of spinal cord injury during occlusion of the thoracic aorta for one hour. They felt that even the largest shunt available for clinical use (9 mm inner diameter) was inadequate to maintain the distal perfusion sufficient for the prevention of spinal cord injury. The author were able to maintain a distal pressure between 96 ± 13 and 101 ± 20 mm Hg, with a shunt flow of 41 ± 10 to 50 ± 15 mL/kg/min. These findings support Molina's contention that the distal pressure must be higher than 80 mm Hg to prevent neurological complication.

The usefulness of circulatory support during cross-clamping of the thoracic aorta remains controversial. Complications such as spinal cord injury, renal dysfunction and heart failure occur but by avoiding injury to the intercostal and mediastinal collateral circulation, reducing of intro and postoperative hemorrhage, the use of vasodilator therapy, appropriate fluid replacement and reducing clamp time, these complication can be minimized[5,8,19]. Circulatory support decompresses the proximal aorta and improves distal blood flow, but its application prolongs surgery and increases the complexity of the operation. The situation is rendered more complex by the necessity of choosing active (using pump) or passive (using simple shunting) circulatory support in the absence of clearly defined criteria under a given set of clinical conditions.

The author found that passive bypass (simple shunting) was more efficient than active bypass (pump) for hemodynamic regulation, maintenance of homeostasis and prevention of paraplegia if the fittest shunting cannula and sites of the recipient blood vessels were selected. Shunt flow is not only a function of the inner diameter of the cannula but also of cardiac function, aortic pressure and distal resistance. However, distal resistance is regulated in part by neuro-endocrine hormones produced by the aminal itself. During cross-clamping of the descending thoracic aorta, the body tends to minimize hemodynamic and metabolic abnormalities through neuro-endocrine hormone regulation. An external pump is not controlled by the body necessity. Experimental results in groups II and III proved this point that they showed different hemodynamic changes despite that they had the same bypass flow. If initial pump flow in group III is increased to balance with body necessity, perhaps hemodynamics will be improved[23]. On the other hand, this experiment also indicated that even though the active shunting flow was the same as the passive one, the physiological efficiencies were different to some degree. For this reason, we must not reproduce this passive shunting flow which provides ideal circulatory support only when centrifugal pump flow is regulated. Experimental results in group IV demonstrated that the centrifugal pump played an important role as a distal circulatory support technique during cross-clamping of the descending thoracic aorta. It maintained normal upper extremity pressure and metabolism, provided an increased distal pressure, and protected hepatic and renal function in this period. In clinical practice, however, the use of vasoactive drugs, blood, and blood products, and patient variability make the benefits of the pump difficult to prove. If bypass flow provided by the pump does not balance the body with circulatory support, the condition in group III may occur in these patients.

Olivier, et al[28] indicated that flows through the pump should be kept at a minimum of 1 liter per minute to prevent stasis within the pump circuit and to eliminate the possibility of systemic embolization. A mean arterial pressure of 50 to 70 mm Hg was maintained in the upper extremities. The pump flow rates are from 1.0 to 4.0 L/min. Hess[14] noted that pump flow was initiated at 1.0 to 1.5 L/min and increased to as high as 3 L/min during clamping. Mean pressures in the lower extremities were kept at 50 to 70 mm Hg. Upper extremity pressure was maintained at normal levels by increasing or decreasing pump flow. Diehl[10] found that flows between 2 and 4 L/min are to be used to achieve optimal afterload reduction (mean pressures between 50 and 75 mm Hg in the upper extremities) and to provide distal perfusion (pressure >60 mm Hg).

It is difficult to avoid the occurrence of some pump-related complications. However, it is also difficult to monitor exactly the use of vasoactive drugs, blood and blood products. The author believe that an ideal circulatory support method minimizes pathologic changes from cross-clamping the descending thoracic aorta. In addition, it must be easy to apply with few complications. The result of the experiment in group II showed that the passive shunt demonstrated these features. Adequate flow can be maintained through a cannula inserted into the carotid and femoral arteries, inner diameter being similar to the lumen of the recipient blood vessels. In the results of the experiment in group IV the active shunt also demonstrated these features to a certain degree. Namely, if the aortic pressure proximal to the clamp was maintained essentially unchanged through regulation of pump flow during the cross-clamping, stable hemodynamics and normal organ metabolisms were maintained. Though bypass flow tended to decrease (from 83 ± 13 to 53 ± 22 ml/kg/min), and cardiac function (cardiac output and heart rate) and arterial blood pH also tended to decreased following prolonged clamping, distal pressure was still maintained between 119 ± 15 to 99 ± 39 mmHg (mean $\pm$ SD), the ratio of pump flow to cardiac output was essentially maintained unchanged and hepatic and renal function were also protected well. With respect to protective action of centrifugal pump on the spinal cord during cross-clamping of the descending thoracic aorta for two hours, the result of the author's experiment demonstrated that even if femoral arterial pressure is maintained at a high level, this method is not able to avoid the occurrence of postoperative paraplegia. It may be due to a nonpulsatile bypass perfusion and spinal cord ischemia secondary to hepatic steal. DeBakey, Najafi, and co-workers[9,26] found that shunting or bypass does not prevent paraplegia and that paraplegia depends on anatomical variability.

The spinal cord is supplied by the anterior spinal artery, which supplies the anterior four-fifths of the cord, and posteriorly by two posterolateral arteries supplying the less functionally important one-fifth. The thoracolumbar portion of the anterior spinal artery is formed by the anastomosis between ascending and descending divisions of the radicular spinal arteries which arise from intercostal arteries. The cervical portion of the anterior spinal artery is formed from branches of the subclavian and vertebral arteries. These radicular spinal arteries are few in number, consisting of one or two cervical, one thoracic, and a large thoracolumbar artery, the artery of Adamkiewicz.

Because of these few radicular arteries and the

poor anastomosis of the anterior spinal arteries between regions of the cord, interruption of any one of the radicular spinal arteries may lead to cord ischemia. Occlusion of the artery of Adamkiewicz is especially hazardous because of poor collateralization. The artery of Adamkiewicz usually arises from the aorta between the levels of T-5 and L-3. This regional blood supply to the spinal cord and its functional implications make the maintenance of blood flow in the distal aorta and avoidance of ischemia in occlusion of the anterior spinal artery important.

Reuter and associates[31] reported an experimental study in which they developed a model of spinal cord ischemia, based on the amplitude reduction of the motor-evoked potential (MEP), which produced approximately a 90% incidence of paraplegia. They believe that spinal cord MEP may contain valuable information for predicting paraplegia. Although the return of the spinal cord MEP is not a predictor of motor function recovery, MEP amplitude reduction and wave morphology correlate well with regional blood flow, histopathologic damage, and neurologic outcome. Histopathologic analysis of spinal cord sections revealed a positive correlation between MEP amplitude reduction, decreased perfusion, and increased severity of histologic injury. However, the correlation between the histopathologic damage and the neurologic outcome is not clear. For example, in their study, analysis of the spinal cord at the level of L-4 in one dog with minimal motor deficits revealed extensive spongiosis affecting 40% to 60% of the gray matter, a diffuse microgliosis, and many swollen and pyknotic neurons, even though this dog had normal muscle tone and a brisk withdrawal reflex. One possible explanation for the intact motor function is that, despite the damage to many neurons, critical neuronal networks may not have been significantly affected by the ischemia, and thus motor control was preserved. Another question that arises is this: why did some dogs have severe spasticity and yet maintain a slow withdrawal to a pinch of the interdigital web, whereas other dogs exhibited only a slight hypertonicity, yet were unable to withdraw their legs in response to the painful stimulus?

Because of left atrial drainage and non-pulsatile femoral arterial perfusion during the use of the centrifugal pump, the normal neurological reflex maintaining physiological pressure may be disturbed. Increase in the arterial blood ketone body ratio, reflecting hepatic flow and energy metabolism[27,29], suggested an abnormal increase in hepatic flow. This "steal phenomenon" of the liver may be responsible for the spinal cord ischemia. On the other hand, it has also been shown that the physiological efficiency of pulsatile arterial pressure is different from that of non-pulsatile pressure despite similar mean values[12,36]. The effects of ischemia and ischemia-reperfusion on the spinal cord are very complex. At present, no known method in clinical practice can reliably prevent the occurrence of paraplegia during cross-clamping of the descending thoracic aorta for an extended period. If paraplegia does occur in postoperative patients, vigorous treatment should be adopted. Kobayashi, et al[18] reported a clinical experience using hyperbaric oxygenation for paralytic lower extremities after abdominal aortic surgery with some success. The author believes that shortening the time of cross-clamping of the descending thoracic aorta is necessary to prevent paraplegia. The author thinks that simultaneous recording of motor-evoked potential may be helpful to evaluate the time and extent of spinal cord ischemia when the centrifugal pump is used as a distal circulatory support technique.

ACKNOWLEDGEMENTS

REFERNCES

1. **Blaisdell, F.W. and Cooley, D.A.** 1962. The mechanism of paraplegia after temporary thoracic aortic occlusion and its relationship to spinal fluid pressure. Surgery **51:** 351–355.
2. **Brewer, L.A., Fosburg, R.G., Mulder, G.A. and Verska, J.J.** 1972. Spinal cord complications following surgery for coarctation of the aorta. A study of 66 cases. J. THORAC. CARDIOVASC. SURG. **64:** 368–381.
3. **Carlson, D.E., Karp, R.B. and Kouchkos, N.T.** 1983. Surgical treatment of aneurysms of the descending thoracic aorta: An analysis of 85 patients. Ann. Thorac. Surg. **35:** 58–69.
4. **Carrel, A.** 1910. On the experimental surgery of the thoracic aorta and the heart. Ann. Surg. **52:** 83–95.
5. **Cartier, R., Orszulak, T.A., Pairolero, P.C. and Schaff, H.V.** 1990. Circulatory support during cross-clamping of the descending thoracic aorta. J. THORAC. CARDIOVASC. SURG. **99:** 1038–1047.
6. **Connolly, J.E., Wakabayashi, A., German, J.C., Stemmer, E.A. and Serres, E.J.** 1971. Clinical experience with pulsatile left heart bypass without anticoagulation for thoracic aneurysms. J.

THORAC. CARDIOVASC. SURG. **62:** 568–576.

7. **Crawford, E.S. and Rubio, P.A.** 1973. Reappraisal of adjuncts to avoid ischemia in the treatment of aneurysms of the descending thoracic aorta. J. THORAC. CARDIOVASC. SURG. **66:** 693–704.

8. **Crawford, E.S., Walker, H.S., Saleh, S.A. and Normann, N.A.** 1981. Graft replacement of aneurysm in descending thoracic aorta: results without bypass or shunting. Surgery **89:** 73–85.

9. **DeBakey, M.E., McCollum, C.H. and Graham, J.M.** 1978. Surgical treatment of aneurysms of the descending aorta. J. Cardiovasc. Surg. **19:** 571–576.

10. **Diehl, J.T., Payne, D.D. and Rastegar, H.** 1987. Arterial bypass of the descending thoracic aorta with the Bio-Medicus centrifugal pump. Ann. Thorac. Surg. **44:** 422–423.

11. **Donahoo, J.S., Brawley, R. and Gott, V.L.** 1962. The hepatic-coated vascular shunt for thoracic aortic and great vessel procedures: a ten-year experience. Ann. Thorac. Surg. **23:** 507–513.

12. **Dunn, J., Kirsch, M., Harness, J., Carroll, M., Straker, J. and Sloan, H.** 1974. Hemodynamic, metabolic and hematologic effects of pulsatile cardiopulmonary bypass. J. Thorac. Cardiovasc. Surg. **68:** 138–142.

13. **Frantz, P.T., Murray, G.F., Shallal, J.A. and Lucas, C.L.** 1981. Clinical and experimental evaluation of left ventriculoiliac shunt bypass during repair of lesions of the descending thoracic aorta. Ann. Thorac. Surg. **3:** 551–557.

14. **Hess, P.J., Howe, H.R., Robicsek, F., Daugherty, H.K., Cook, J.W., Selle, J.G. and Stiegel, R.M.** 1989. Traumatic tears of the thoracic aorta: improved results using the Bio-Medicus pump. Ann. Thorac. Surg. **48:** 6–9.

15. **Hirai, F., Aoyama, H., Ohtoshi, M., Ukikusa, M., Shimabara, Y., Asano, M., Ozawa, K. and Tobe, T.** 1981. Hepatic energy metabolism of sepsis in pigs and its relationship to hemodynamic changes. Eur. Surg. Res. **13:** 72–73.

16. **Jex, R.K., Schaff, H.V., Piehler, J.M., King, R.M., Orszulak, T.A., Danielson, G.K., Pairolero, P.C., Pluth, J.R. and Ilstrup, D.** 1986. Early and late results following repair of dissections of the descending thoracic aorta. J. Vasc. Surg. **3:** 226–237.

17. **Katz, N.M., Blackstone, E.H., Kirklin, J.W. and Karp, R.B.** 1981. Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transection. J. THORAC. CARDIOVASC. SURG. **81:** 669–674.

18. **Kobayashi, S., Takahashi, H., Yano, T., Ikezawa, T. and Sakurai, T.** 1993. Clinical experience in hyperbaric oxygenation for paralytic lower extremities after abdominal aortic surgery. Jpn. J. Cardiovasc Surg. **22:** 1–6.

19. **Kouchoukos, N.T., Lell, W.A., Karp, R.B. and Samuelson, P.N.** 1979. Hemodynamic effect of aortic clamping and decompression with a temporary shunt for resection of the descending thoracic aorta. Surgery **85:** 25–30.

20. **Krouse, A.H., Ferguson, T.B. and Weldon, C.S.** 1972. Thoracic aneurysmectomy utilizing the TDMAC-heparin shunt. Ann. Thorac. Surg. **14:** 123–132.

21. **Laschinger, J.C., Cunningham, J.N., Nathan, I.M., Knopp, E.A., Cooper, M.M. and Spencer, F.C.** 1983. Experimental and clinical assessment of the adequacy of partial bypass in maintenance of spinal cord blood flow during operations on the thoracic aorta. Ann. Thorac. Surg. **36:** 417–426.

22. **Lawrence, G.H., Hessel, E.A., Sauvage, L.R. and Krause, A.H.** 1977. Results of the use of TDMAC-heparin shunt in the surgery of aneurysms of the descending thoracic aorta. J. THORAC. CARDIOVASC. SURG. **73:** 393–398.

23. **Liu, G., Fukunaga, S., Sueda, T. and Matsuura, Y.** 1994. Efficiency of a centrifugal pump for distal circulatory support during cross-clamping of the descending thoracic aorta. Vasc. Surg. **28:** 167–176.

24. **Livesay, J.J, Cooley, D.A. and Rogelio, A.V.** 1985. Surgical experience in descending thoracic aneurysmectomy without adjuncts to avoid ischemia. Ann. Thorac. Surg. **39:** 37–46.

25. **Molina, J.E., Cogordan, J., Einzig, S., Bianco, R.W., Rasmussen, T., Clark, R.M. and Borgwardt, B.** 1985. Adequacy of ascending aorta-descending aorta shunt during cross-clamping of the thoracic aorta for prevention of spinal cord injury. J. THORAC. CARDIVASC. SURG. **90:** 126–136.

26. **Najafi, H., Javid, H., Hunter, J., Serry, C. and Monson, D.** 1980. Descending aortic aneurysmectomy without adjuncts to avoid ischemia. Ann. Thorac. Surg. **30:** 326–335.

27. **Ohno, M., Yamasaki, M., Saibara, T., Izumi, S. and Tamiya, T.** 1992. Clinical and experimental study of arterial blood ketone body ratio during cardiopulmonary bypass for open heart surgery. Jpn. J. Artif. Organs **21:** 755–759.

28. **Olivier, H.F., Maher, T.D., Geoge, A.L., Park, S.B., Burkholder, J.A. and Magovern, G.J.** 1984. The use of the Biomedicus centrifugal pump in traumatic tears of the thoracic aorta. Ann. Thorac. Surg. **38:** 586–591.

29. **Ozawa, K., Aoyama, H., Yasuda, K., Shimahara, Y., Nakatani, T., Tanaka, J., Yamamoto, M., Kamiyama, Y. and Tobe, T.** 1983. Metabolic abnormalities associated with postoperative organ failure: a redox theory. Arch. Surg. **118:** 1245–1251.

30. **Pett, S.B., Wernly, J.A. and Akl, B.F.** 1987. Observations on flow characteristics of passive external aortic shunts. J. THORAC. CARDIOVASC. SURG. **93:** 447–453.

31. **Reuter, D.G., Tacker, W.A., Badylak, S.F., Voorhees, W.D. and Konrad, P.K.** 1992. Correlation of motor-evoked potential response to ischemic spinal cord damage. J. Thorac. Cardiovsc. Surg. **104:** 262–272.

32. **Rittenhouse, E.A., Dillard, D.H., Winterscheid, L.C. and Merendino, K.A.** 1969. Traumatic rupture of the thoracic aorta. Ann Surg. **170:** 87–100.

33. **Roberts, A.J., Nora, J.D. Hughes, W.A., Quintanilla, A.P., Ganote, C.E., Sanders, J.H., Moran, J.M. and Michaelis, L.L.** 1983. Cardiac and renal responses to cross-clamping of the descending thoracic aorta. J. THORAC.

CARDIOVASC. SURG. **86:** 732–741.

34. **Shimahara, Y., Ozawa, K., Ida, T., Ukikusa, M. and Tobe, T.** 1981. Four stages of mitochondrial deterioration in hemorrhagic shock. Res. Exp. Med. **179:** 23–33.

35. **Stallone, R.J.** 1978. Management of ruptured thoracic aortic aneurysms. Am. J. Surg. **136:** 162–164.

36. **Taylor, K.M., Bain, W.H., Russel, M., Brannan, J.J. and Morton, I.J.** 1979. Peripheral vascular resistance and angiotensin II levels during pulsatile and non-pulsatile cardiopulmonary bypass. Thorax **34:** 594–598.

37. **Verdant, A., Page, A., Cossette, R., Dontigny, L., Page, P. and Baillot, R.** 1988. Surgery of the descending thoracic aorta: spinal cord protection with Gott shunt. Ann. Thorac. Surg. **46:** 147–154.

38. **Wakabayashi, A. and Connolly, J.E.** 1976. Prevention of paraplegia associated with resection of extensive thoracic aneurysms. Arch. Surg. **111:** 1186–1189.

39. **Yamamoto, M., Shimahara, Y., Ozawa, K., Tobe, T. and Isselhard, W.** 1984. Relationship of deranged energy metabolism in liver and kidney to arterial ketone body ratio following liver ischemia in rats. Jpn. J. Surg. **14:** 52–60.

40. **Yamamoto, M., Tanaka, T., Ozawa, K. and Tobe, T.** 1980. Significance of acetoacetate/β-hydroxybutyrate ratio in arterial blood as an indicator of the severity of hemorrhagic shock. J. Surg. Res. **28:** 124–131.