# The Effects of Thoracic Aortic Cross-Clamping and Declamping on Visceral Organ Blood Flow

MORITSUGU OYAMA, M.D., J. JUDSON McNAMARA, M.D., GLENN T. SUEHIRO, B.S., ANTHONY SUEHIRO, B.S., KENDRICK SUE-AKO, B.S.

*From the Cardiovascular Research Laboratory and the Department of Surgery, John A. Burns School of Medicine, University of Hawaii, Queen's Medical Center, Honolulu, Hawaii*

**Blood flow was measured using radioactive microspheres in 11 macaque monkeys 1) before hemorrhagic shock, 2) after onset of shock, 3) after aortic cross-clamping and resuscitation, and 4) after release of the cross-clamp and stabilization. Hemodynamic parameters (cardiac output, arterial, right atrial and left atrial pressure) and blood gases were also monitored. Total abdominal organ flow fell with hemorrhage and fell further with aortic clamping. Reinfusion of shed volume did not restore abdominal organ flow (4.7% baselines) but increased LAP and cardiac output to the upper body. Release of the cross-clamp produced profound acidosis that was treated effectively with $NcHCO_3$. After stabilization of blood, flow to kidney remained low (49% baseline) although intestinal flow was increased threefold (320% of baseline). It is clear that thoracic aortic cross-clamping in shock further compromises already reduced visceral blood flow and may contribute to the problem of ischemic multiple organ failure after resuscitation from hemorrhagic shock.**

TEMPORARY THORACIC aortic cross-clamping is occasionally applied as an effective resuscitative procedure in profoundly injured hypotensive patients. This procedure aids in rapid restoration of vital signs, maintaining blood flow to brain and heart, and controlling of intra-abdominal bleeding, while the injuries and hypovolemia are corrected.[1-3] During aortic cross-clamping, the amount of blood flow to organs below the clamp depends on collateral flow reaching those organs. However, it is not known how thoracic aortic cross-clamping affects blood flow distribution to individual intra-abdominal organs and what happens to blood flow distribution after release of the clamp. Previous reported studies were limited by lack of adequate available means to measure organ blood flow.[4,5]

In 1967 Rudolf and Heymman introduced a method of measuring regional blood flow with radioactive microspheres.[6] The method utilized in this study collected peripheral arterial blood for a reference sample[8-11] which has the advantage of providing simultaneous measurement of cardiac output as well as regional blood flow.

Using this method, the authors studied the effects of thoracic aortic cross-clamping in hemorrhagic shock on visceral blood flow and effects of declamping after resuscitation.

## Materials and Methods

Eleven monkeys (macaca mulatto), weighing 3.5–8 kg, were anesthetized with pentothal and sernalyn, intubated, and ventilated with a Harvard animal respirator (Harvard Apparatus Co., Millis, MA). A lead II electrocardiogram was monitored. Catheters were placed in the left brachial and right femoral arteries and a venous cannula was advanced to the right atrium from the right femoral vein. A urinary catheter was inserted into the bladder. A left thoracotomy was performed, a catheter was inserted into left atrium through a purse-string suture, and the descending thoracic aorta isolated for later occlusion.

About 30 minutes after finishing all the surgical procedures, baseline studies were performed. Hemorrhagic shock was induced by withdrawal of blood from the femoral artery cannula until mean arterial pressure had fallen to 50 mmHg. Pressure was maintained at 50 mmHg for 15 minutes. Subsequently, a DeBakey vascular clamp was placed on the distal thoracic aorta (T7-T8) just above the diaphragm for 45 minutes. During the clamping period, the shed blood was reinfused through the venous catheter over 15 minutes. The clamp was slowly released and Ringer's lactate solution was infused rapidly through the venous line until brachial arterial pressure was restored to baseline level. Sodium bicarbonate was also administered to correct a consistent marked reduction of arterial pH.

Supported by U.S. Army Medical Research and Development Command Grant DADA17-73-C-3040.

Reprint requests: J. Judson McNamara, M.D., Department of Surgery, Queen's Medical Center, 1301 Punchbowl Street, Honolulu, Hawaii 96813.

Submitted for publication: November 16, 1982.

0003-4932/83/0400/0459 $01.05 © J. B. Lippincott Company

TABLE 1. *Hemodynamic and Physiologic Measurements During Four Periods*

| | | Baseline | Shock | Clamp | Declamp |
|---|---|---|---|---|---|
| | Mean BAP (mmHg) | 100.1 ± 6.1 | 50 ± 0† | 137.7 ± 7.9† | 98.0 ± 8.0 |
| | HR (beats/min) | 129.3 ± 10.1 | 153.9 ± 14.1* | 117.3 ± 12.1* | 128.8 ± 14.0 |
| | Mean LAP (mmHg) | 5.1 ± 0.9 | 1.5 ± 0.5† | 18.6 ± 2.6† | 6.6 ± 1.0 |
| | Mean CVP (mmHg) | 1.6 ± 1.5 | 0.3 ± 0.7 | 0.8 ± 1.1 | 2.6 ± 1.5 |
| | Hct (%) | 37.3 ± 1.0 | 32.8 ± 1.7† | 35.8 ± 1.7 | 32.2 ± 1.8** |
| | CO (ml/kg/min) | 179.4 ± 18.3 | 91.5 ± 13.6† | 76.2 ± 17.2† | 187.8 ± 34.5 |
| | BA | 7.39 ± 0.02 | 7.46 ± 0.02 | 7.43 ± 0.02 | 7.33 ± 0.02 (7.24 ± 0.02)* |
| pH | FA | 7.38 ± 0.02 | 7.46 ± 0.02 | 7.42 ± 0.02 | 7.33 ± 0.02 (7.26 ± 0.04)* |
| | CV | 7.39 ± 0.02 | 7.42 ± 0.02 | 7.33 ± 0.03 | 7.32 ± 0.02 (7.20 ± 0.02)* |

BAP: Brachial Arterial Pressure; CVP: Central Venous Pressure; BA: Brachial Artery; HR: Heart Rate; Hct: Hematocrit; FA: Femoral Artery; LAP: Left Atrial Pressure; CO: Cardiac Output; CV: Central Vein.

* Significant difference from baseline ($p < 0.05$ paired t test).
† Significant difference from baseline ($p < 0.01$ paired t test).
Value is mean ± SEM. Numbers in parenthesis are value immediately after declamping.

Data to be described were obtained at four different periods: 1) baseline, before hemorrhagic shock, 2) 15 minutes after hemorrhagic shock, 3) 45 minutes after application of the aortic cross-clamp, and 4) following release of clamp (15 minutes after mean brachial arterial pressure was restored to baseline). Heart rate and hemodynamic data were recorded with a Brush, Gould, Mark 260 recorder (Gould, Inc., Brush Instruments Div., Cleveland, OH). Mean brachial and femoral arterial pressure, mean left atrial pressure, and mean central venous pressure were measured via Statham P23 transducers. Both arterial pH and venous pH were measured with an I.L. blood gas machine (Instrumentation Laboratories, Lexington, MA). Hematocrit was also determined at each study period.

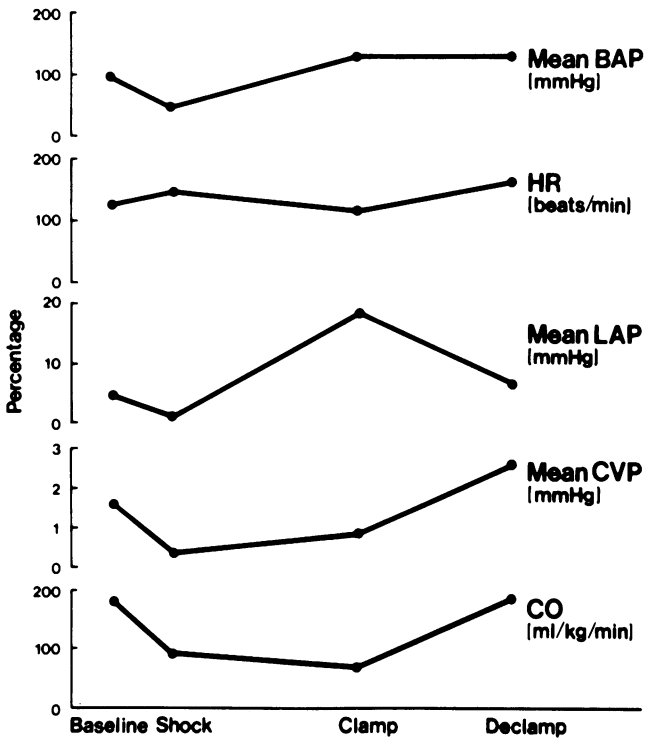


FIG. 1. Hemodynamic data during four periods.

After obtaining these data, cardiac output and regional blood flow were measured with four different labeled radioactive microspheres in seven of the 11 animals. Microspheres (Minnesota Mining and Manufacturing Co., St. Paul, MN) of 15 ± 5 $\mu$ diameter labeled with either $^{141}Ce$, $^{51}Cr$, $^{85}Sr$ or $^{46}Sc$ were suspended in a solution of 10% dextran and 0.5% Tween-80 by vigorous agitation in an ultrasonic bath. The suspended microspheres (800,000–2.4 × $10^6$) in 5 ml saline were injected with a plastic syringe into the left atrium over ten minutes. Reference blood samples were withdrawn by an infusion withdrawal pump (Model 600-900, Harvard Apparatus Co., Inc., Dover, MA) from the brachial artery at a constant rate (4.12 ml/min), starting 15 seconds before microsphere injections and continuing for one minute. The radioactivity of microspheres injected were determined by the activity in the syringe before and after injection. After the last microsphere injection, monkeys were sacrificed with intravenous infusion of saturated potassium chloride. Then visceral organs (liver, kidney, spleen, pancreas, stomach, small intestine, and colon) were removed. Organs were cut into small pieces and placed into weighed glass tubes and weighed. Individual organ radioactivity was determined by summing the radioactivity in all the sample tubes of organ. The radioactivity of these sample tubes were measured in a solid scintillation counter (Model 300, Beckman Instruments, Inc., Fullerton, CA). The resulting data were interpreted by an IMSAI 8080 microcomputer em-

ploying an equation to determine individual isotope concentration. Cardiac output and organ blood flow were calculated by the formulas:

$$\text{Cardiac Output} = \frac{\text{counts injected} \times \text{reference sample withdrawal rate}}{\text{reference sample counts}}$$

$$\text{Organ Blood Flow} = \frac{\text{organ counts} \times \text{reference sample withdrawal rate}}{\text{reference sample counts}}$$

Percentage of cardiac output was calculated by dividing the net radioactivity in each organ by a total injected radioactivity. Cardiac output was expressed in ml/kg/min and blood flow to the individual organ was expressed in ml/g/min. Statistical significance of difference between baseline and subsequent three periods were determined by paired t test.

## Results

The mean volume of blood withdrawn to induce hemorrhagic shock was 18.2 ml/kg. The mean duration of declamping period prior to the final study was 39.3 minutes and mean volume of Ringer's lactate solution infused during the clamping period was 3.6 ml/kg (145 ml). The hemodynamic data, arterial and venous pH, and hematocrit measured at each of the four periods are presented in Table 1 and Figure 1. After 15 minutes of shock, left atrial pressure and cardiac output were significantly lower and heart rate was higher compared with baseline. After application of the thoracic aortic cross-clamping, brachial arterial pressure rose promptly and femoral arterial pressure below the clamp dropped to a mean of 13 mmHg (6 to 24 mmHg) with loss of pulse pressure. Left atrial pressure rose significantly following reinfusion of the shed blood. Cardiac output decreased significantly during shock and remained low after clamping the aorta while the heart rate decreased significantly during clamping. Venous pH during clamping period fell significantly from 7.39 to 7.33. Immediately after release of the clamp, brachial arterial pressure dropped rapidly to shock levels with concomitant decrease in arterial and venous pH. Subsequently by the final study period after release of the clamp, all the data were restored to baseline value except hematocrit. Changes in CVP were not significant at any time period.

Absolute blood flows to each organ studied are shown in Table 2. The data are presented in Table 3 and Figure 2 as a percentage of baseline value and as a percentage of the cardiac output in Table 4. After 15 minutes of shock, visceral organ blood flow fell significantly in all organs except the liver. The component of liver blood flow measured directly with microspheres is only the hepatic arterial flow. Although hepatic arterial flow was not changed, the fraction of cardiac output to liver had to be significantly decreased during shock. Forty-five minutes after clamping, cardiac output remained low at 40.9% of baseline and blood flow to individual abdominal visceral organs was only an average of 4.7% of baseline. Fraction of cardiac output to total visceral organs also decreased significantly to 4.4%.

TABLE 2. *Absolute Blood Flow in Selected Organs (ml/g/min)*

| | Baseline | Shock | Clamp | Declamp |
|---|---|---|---|---|
| Liver | 0.71 ± 0.13 | 0.61 ± 0.10 | 0.03 ± 0.02† | 0.72 ± 0.33 |
| Kidney | 8.26 ± 1.16 | 4.04 ± 0.52† | 0.21 ± 0.02† | 4.16 ± 0.13† |
| Spleen | 3.07 ± 0.70 | 0.63 ± 0.22† | 0.04 ± 0.02* | 2.86 ± 1.18 |
| Pancreas | 1.95 ± 0.37 | 0.35 ± 0.07* | 0.10 ± 0.05* | 1.97 ± 0.52 |
| Stomach | 0.39 ± 0.06 | 0.14 ± 0.02* | 0.02 ± 0.01† | 0.66 ± 0.19 |
| Small intestine | 0.49 ± 0.09 | 0.34 ± 0.05* | 0.04 ± 0.01† | 1.71 ± 0.37* |
| Colon | 0.34 ± 0.05 | 0.22 ± 0.02* | 0.02 ± 0.01† | 0.60 ± 0.09 |

Value is mean ± SEM.
* Significant difference from baseline ($p < 0.05$ paired t test).
† Significant difference from baseline ($p < 0.01$ paired t test).

After release of the clamp, cardiac output had returned to baseline, however, blood flow to kidney was significantly reduced (49% of baseline, range 38.2% to 61%) and flow to small intestine was significantly increased (320%, range 172 to 471%). These changes were significant and consistent in all animals studied. Blood flow to stomach and colon after declamping appeared high compared with baseline values, but there was wide variability and these increases were not statistically significant. Blood flow as a fraction of cardiac output during declamping period fell in kidney and rose in pancreas, stomach, small intestine, and colon.

## Discussion

In this baseline study, performed after thoracotomy, changes in visceral organ blood flow and fraction of cardiac output were slightly different from the result from

TABLE 3. *Organ Blood Flow Expressed as a Per cent of Baseline Value*

| | Baseline | Shock | Clamp | Declamp |
|---|---|---|---|---|
| Cardiac output | 100 | 50.6 ± 5.4 | 40.9 ± 7.7 | 104.6 ± 13.3 |
| Liver | 100 | 102.8 ± 24.1 | 3.5 ± 1.9 | 77.5 ± 23.8 |
| Kidney | 100 | 47.5 ± 6.3 | 2.2 ± 1.1 | 49.4 ± 3.7 |
| Spleen | 100 | 19.5 ± 4.5 | 1.9 ± 0.7 | 82.5 ± 14.0 |
| Pancreas | 100 | 20.1 ± 4.7 | 5.8 ± 2.3 | 106.0 ± 19.4 |
| Stomach | 100 | 46.5 ± 9.2 | 4.6 ± 2.1 | 183.6 ± 31.3 |
| Small intestine | 100 | 78.9 ± 14.2 | 5.7 ± 2.4 | 326.4 ± 46.5 |
| Colon | 100 | 63.8 ± 8.2 | 8.3 ± 2.9 | 184.3 ± 32.9 |

Value is mean ± SEM.

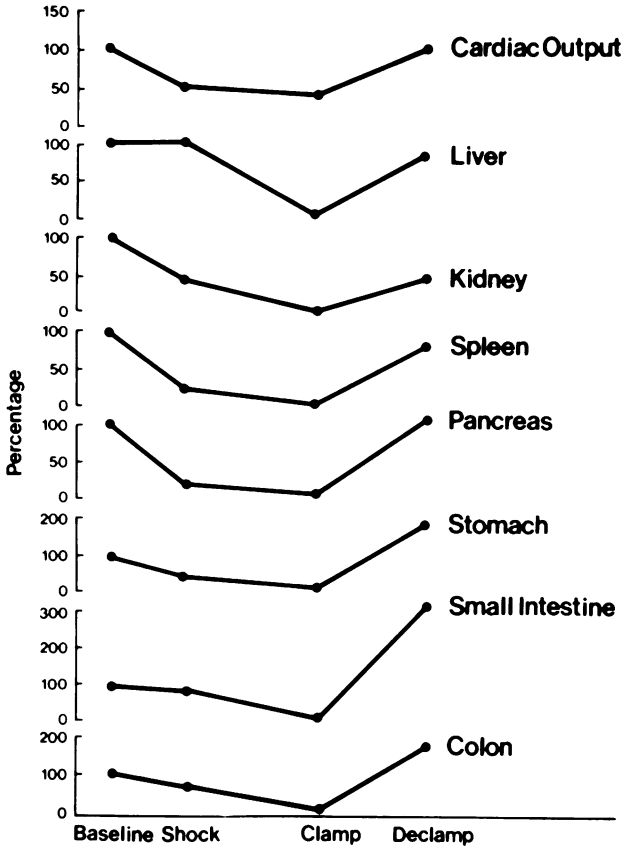


FIG. 2. Organ blood flow as a percentage of baseline.

those reported by others. Rudy et al.[12] found that cardiac output decreased significantly after thoracotomy and the fraction of systemic blood flow to kidney increased consistently to about 17%. Lees et al.[13] showed cardiac output decreased to less than half of awake monkeys after thoracotomy and percentage of cardiac output to liver and kidney were 12.3% and 11.8%, respectively. The percentage of cardiac output to kidney in this study was slightly higher than either of these previous studies.

After hemorrhagic shock, blood flow to each individual organ decreased significantly except to the liver, in fact, the percentage of cardiac output that was comprised of systemic liver blood flow to cardiac output was increased, yet decreased to spleen and pancreas. These findings are similar to the result studied by Forsyth et al.[14] It is still likely that total hepatic blood flow is substantially reduced since blood flow to all the organs in the portal circulation is significantly decreased and this remains the major source of hepatic blood flow.

TABLE 4. *Percentage of Cardiac Output to Selected Organs*

| | Baseline | Shock | Clamp | Declamp |
|---|---|---|---|---|
| Liver | 10.8 ± 2.2 | 17.0 ± 2.6† | 1.2 ± 0.8† | 10.7 ± 5.1 |
| Kidney | 23.6 ± 1.0 | 22.1 ± 0.9 | 1.3 ± 0.7† | 12.1 ± 1.3† |
| Spleen | 2.2 ± 0.6 | 0.9 ± 0.4* | 0.1 ± 0.0* | 2.1 ± 0.9 |
| Pancreas | 2.2 ± 0.6 | 0.7 ± 0.1* | 0.1 ± 0.0 | 2.4 ± 0.6* |
| Stomach | 1.8 ± 0.5 | 1.0 ± 0.0 | 0.3 ± 0.1 | 2.6 ± 0.8* |
| Small intestine | 4.0 ± 0.7 | 5.0 ± 0.3 | 0.9 ± 0.4* | 13.0 ± 2.7 |
| Colon | 3.2 ± 0.7 | 3.1 ± 0.4 | 0.1 ± 0.3* | 5.2 ± 0.9† |

Value is mean ± SEM.
* Significant difference from baseline ($p < 0.05$ paired t test).
† Significant difference from baseline ($p < 0.01$ paired t test).

Thoracic aortic cross-clamping applied during hemorrhagic shock produced a prompt rise in brachial arterial pressure that was maintained throughout the 45-minute clamping period. Cardiac output after clamping remained low in spite of the increased blood pressure and the reinfusion of the shed blood. Femoral arterial pressure below the clamp dropped to 13 mmHg, and this marked regional hypotension was associated with marked decrease of blood flow to visceral organs.

Little information is available on specific visceral organ blood flow during thoracic aortic cross-clamping. In previous studies, collateral blood flow was measured by timed collection of the blood from inferior vena cava. Bounous et al.[5] found that after one hour of aortic clamping above the diaphragm, blood flow from the lower part of the body below the clamp fell to about 15 ml/kg/min from average normal flow of 88 ml/kg/min. King et al.[4] also found that blood flow from the lower part of the body below the thoracic aortic clamping decreased to 8 to 18 ml/kg/min. In this present study, individual visceral organ blood flow during the clamping period was even more severely decreased to an average of 4.7% of baseline flow (from 1.9% in spleen to 8.3% in colon).

Cardiac output remained low (40.9% of baseline value) during the clamping period. The percentage of cardiac output to all the visceral organs was also decreased to 4.4% from 48% in baseline period. Although hind limb blood flow was not measured, these results indicated that less than 10% of cardiac output was distributed to the lower part of the body after clamping. Though cardiac output was decreased to 40% of baseline value after clamping, most of the cardiac output was distributed to the upper part of the body proximal to the clamp, so the blood flow to the upper body was probably not very different during the clamping period from the baseline period.

Reinfusion of the shed blood during the clamping period resulted in an increase in left atrial pressure to 18.6 mmHg. Acute left ventricular strain may result from thoracic aortic occlusion, especially when large volumes of blood are simultaneously infused, and this risk increases once blood pressure is restored to normal and rapid infusion of blood and fluid is continued.[2] Marked elevation of left atrial pressure following reinfusion of blood in this study suggests that overinfusion during clamping period may be a serious problem, especially in patients with associated cardiovascular disease.[1]

Metabolic and hemodynamic changes after temporary aortic occlusion has been studied by many investigators.[15–18] Aortic occlusion results in severe regional

shock distal to the clamp, which in turn results in poor tissue perfusion and accumulation of acid metabolites and regional acidosis. Upon releasing of the clamp, there is a rapid flushing of previously stagnant venous blood from the distal part of the clamp into the systemic circulation, providing relatively acute metabolic acidosis. Decreased vascular tone below the clamp results in a drop in systemic vascular resistance immediately after declamping and pooling of blood occurs in the previous excluded vascular bed. Increased blood flow to the previously excluded area after declamping results in hypotension followed by increasing vascular constriction and concomitant stabilization of blood flow to that area.

In this study, at an average of 40 minutes after declamping, cardiac output had returned to baseline value, however, flow to kidney was still decreased below 50% of baseline and flow to the small intestine was increased by 320% of baseline. These findings were consistent in all animals studied. Cardiac output and arterial pressure were not changed significantly from baseline, so these changes in blood flow were due to changes in organ vascular resistance. Morris et al.[19] also found that renal blood flow decreased to 46% of baseline value 40 minutes after declamping while it returned to normal within 24 hours. Urine output in this study after declamping was well maintained in spite of decreased renal blood flow, similar results were observed by Morris, et al.[19] Garci-Rinaldi et al.[1] concluded that there was a distinct pattern of renal function in clinical patients after declamping. The reasons for the changing pattern of blood flow to kidney and small intestine after declamping is not clear, however, these findings suggest that regional vascular resistance will not be uniform after declamping even though cardiac output, arterial blood pressure, and total systemic vascular resistance are restored to baseline.

Hematocrit fell significantly from baseline after declamping probably due to hemodilution with Ringer's. Whether anemia may result in the reduced renal flow and increased intestinal flow is not known, although Hoffbrand et al.[20] studied changes in organ blood flow distribution in moderate postoperative anemia (hematocrit 32%) and found no changes in renal and intestinal blood flow.

## Conclusion

Cardiac output remains low (40% of baseline) after thoracic aortic cross-clamping for hemorrhagic shock but is probably near normal for the upper part of the body. Blood flow to all the visceral organs is decreased after shock and further decreased after aortic clamping. This finding demonstrates that the cardiac output is redistributed to the upper part of the body proximal to clamp at the expense of visceral organs below the clamp.

After declamping, systemic arterial pressure and cardiac output were restored to baseline value, however, regional blood flow to kidney remained low and flow to the small intestine increased dramatically. These results indicate that regional vascular resistance will not be uniform for as long as 40 minutes after declamping even though cardiac output, arterial blood pressure, and total systemic vascular resistance are restored to baseline values by appropriate resuscitative measures.

## References


1. Garci-Rinaldi R, Defore WW, Mattox KL, Beall AC Jr. Unimpaired renal, myocardial and neurologic function after cross-clamping of the thoracic aorta. Surg Gynecol Obstet 1976; 143:249–252.
2. Sankaran S, Lucas C, Walt AJ. Thoracic aortic clamping for prophylaxis against sudden cardiac arrest during laparotomy for acute massive hemoperitoneum. J Trauma 1975; 15:290–296.
3. Ledgerwood AM, Kazmers M, Lucas CE. The role of thoracic aortic occlusion for massive hemoperitoneum. J Trauma 1976; 16:610–623.
4. King H, Shumacker HB. Mechanical by-pass during thoracic aortic cross-clamping. J Thoracic Surg 1957; 34:485–499.
5. Bounous G, King H. Cross-clamping of the descending thoracic aorta. Arch Surg 1959; 78:519–523.
6. Rudolph AM, Heymann MA. The circulation of the fetus in utero: methods for studying distribution of blood flow, cardiac output and organ blood flow. Circ Res 1967; 21:163–184.
7. Heymann MA, Payne BD, Hoffman JIE, Rudolph AM. Blood flow measurements with radionuclide-labeled particles. Prog Cardiovasc Dis 1977; 20:55–79.
8. Hoffbrand BI, Forsyth RP. Validity studies of the radioactive microspheres method for measuring the distribution of cardiac output, organ blood flow and resistance in the conscious rhesus monkey. Cardiovasc Res 1969; 3:426–432.
9. Buckberg GD, Luck JC, Payne DB, et al. Some sources of errors in measuring regional blood flow with radioactive microspheres. J Appl Physiol 1971; 31:598–604.
10. Archie JP Jr, Fixler DE, Ullyot DJ, et al. Measurement of cardiac output with organ trapping of radioactive microspheres. J Appl Physiol 1973; 35:148–154.
11. Bartrum RJ, Berkowitz DM, Hollenberg NK. A simple radioactive microsphere method for measuring regional flow and cardiac output. Invest Radiol 1974; 9:126–132.
12. Rudy LW Jr, Heymann MA, Edmunds LH Jr. Distribution of systemic blood flow during cardiopulmonary bypass. J Appl Physiol 1973; 34:194–200.
13. Lees MH, Herr RH, Hill JD, et al. Distribution of systemic blood flow of the rhesus monkeys during cardiopulmonary bypass. J Thorac Cardiovasc Surg 1971; 61:570–586.
14. Forsyth RP, Hoffbrand BI, Melmon KL. Redistribution of cardiac output during hemorrhage in the unanesthetized monkey. Circ Res 1970; 27:311–320.
15. Standness DE Jr, Parrish DG, Bell JW. Mechanism of declamping shock in operations on the abdominal aorta. Surgery 1961; 50:488–492.
16. Fry WJ, Keitzer WF, Kraft RO, DeWeese MS. Prevention of hypotension due to aortic release. Surg Gynecol Obstet 1963; 116:301–306.
17. Baue AE, McClerkin WW. A study of shock: acidosis and the declamping phenomenon. Ann Surg 1965; 161:41–45.
18. Schneiweiss R, Hadsall F, Gordon HE. Prevention of hypotension following release of aortic occlusion. Surgery 1966; 60:628–632.
19. Morris GC Jr, Moyer JH, Colley DA, Brockman HL. The renal hemodynamic response of hypothermia and to clamping of the thoracic aorta with and without hypothermia. Surg Forum 1954; 5:219–228.
20. Hoffbrand BI, Forsyth RP. The hemodynamic consequences of moderate postoperative anemia in monkeys. Surg Gynecol Obstet 1971; 132:61–66.