脊髄側副血行路を介した脊髄血流量と脊髄還流圧の同時測定：主に体血圧との相関について（実験的研究）

学位論文

Directly measuring spinal cord blood flow and spinal cord perfusion pressure via the collateral network: Correlations with changes in systemic blood pressure

琉球大学大学院医学研究科　医科学専攻

喜瀬　勇也

Directly measuring spinal cord blood flow and spinal cord perfusion pressure via the collateral network: Correlations with changes in systemic blood pressure

Yuya Kise, MD, Yukio Kuniyoshi, MD, PhD, Hitoshi Inafuku, MD, PhD,
Takaaki Nagano, MD, Tsuneo Hirayasu, MD, PhD, and Satoshi Yamashiro, MD, PhD

Department of Thoracic and Cardiovascular Surgery, Graduate School of Medicine, University of the Ryukyus, Okinawa, Japan

Abstract

Objective: During thoracoabdominal surgery in which segmental arteries are sacrificed over a large area, blood supply routes from collateral networks have received attention as a means of avoiding spinal cord injury. The aim of this study was to investigate spinal cord blood supply through a collateral network by directly measuring spinal cord blood flow (SCBF) and spinal cord perfusion pressure (SCPP) experimentally.

Method: Using beagle dogs (n=8), the thoracoabdominal aorta and segmental arteries (SAs) L1–L7 were exposed, and a temporary bypass was created for distal perfusion. Next, a laser blood flowmeter was placed on the spinal dura mater in the L5 region to measure the SCBF. The following were measured simultaneously when the direct blood supply from SAs L2–L7 to the spinal cord stopped: mean systemic blood pressure (mSBP); SCPP (blood pressure within the aortic clamp site); and SCBF supplied via the collateral network. These variables were then investigated for evidence of correlations.

Results: Positive correlations were observed between mSBP and SCBF during interruption of SAs flow both with ($r=0.844$, $p<.01$) and without distal aortic perfusion ($r=0.834$, $p<.01$). In addition, we observed significant correlations between SCPP and SCBF with and without distal perfusion ($r=0.803$, $p<.001$ and $r=0.832$, $p<.01$, respectively), and between mSBP and SCPP with and without distal perfusion ($r=0.898$, $p<.001$ and $r=0.837$, $p<.001$). The spinal cord was perfused from the collateral network from outside the interrupted segmental arteries, and high systemic blood pressure (about 1.33-fold higher) was needed to obtain the pre-clamping SCBF, while 1.68-fold higher systemic blood pressure was needed when distal perfusion was halted.

Conclusions: SCBF is positively correlated with mSBP and SCPP under spinal cord ischemia caused by clamping a wide range of segmental arteries. In open and endovascular thoracic and thoracoabdominal surgery, elevating mSBP is a simple and effective means of increasing SCBF, and measuring SCPP appears to be useful for monitoring perioperative SCBF.

Introduction

The complication of greatest concern in thoracoabdominal surgery is spinal cord injury from sacrifice of the segmental arteries (SAs), which serve a crucial role in supplying blood flow to the spinal cord. Various methods are used to avoid this (motor evoked potential monitoring, determining the origin and revascularization of the Adamkiewicz artery (AKA), distal aortic perfusion, cerebrospinal fluid drainage, spinal cord cooling, administration of certain drugs), but a definitive method has yet to be established [1].

Recent research on the blood supply to the spinal cord has reported that there is a rich network of spinal cord blood vessels and tissue around the spinal cord that plays an important role in spinal cord circulation [2]. In actual clinical practice, raising systemic blood pressure is useful as a means of increasing the blood supply from the collateral network. However, it is not possible to measure spinal cord blood flow (SCBF) in clinical settings, and there are few reports showing a correlation between systemic blood pressure and SCBF.

In this experiment, SCBF was measured directly via the collateral network while blood flow from several SAs, including the AKA, was interrupted, and the correlation between SCBF and mean systemic blood pressure (mSBP) was clarified.

Spinal cord perfusion pressure (SCPP), which is sometimes used to monitor SCBF perioperatively, was also measured, and its correlations with SCBF and mSBP were examined.

In addition, changes in SCBF with and without distal perfusion were measured, and the degree to which distal perfusion affects SCBF via the collateral network was investigated. Finally, the mSBP conditions to maintain SCBF prior to interruption of the SAs were investigated.

The aim of this study was to investigate spinal cord blood supply through a collateral network by directly measuring SCBF and SCPP experimentally.

1. Materials and methods

Animals

Animal care and all procedures were performed in compliance with the Guide for Care of Laboratory Animals. This study was approved by the Research Committee for Laboratory Animal Science at University of Ryukyus, Japan. Experiments were performed on 8 female beagle dogs weighing 7.5–10.0 kg.

Anesthesia

The dogs were sedated by intramuscular injection of 3 mg/kg ketamine hydrochloride and 0.2 mg atropine sulfate. They were then intubated endotracheally and ventilated mechanically. One intravenous line was inserted into an anterior limb vein. They were placed in the prone position on an operating table under intravenous anesthesia with propofol (0.3 mg/kg/min) and ketamine hydrochloride (0.05 mg/kg/min).

During the surgical procedure, the mean arterial blood pressure was maintained between 70 and 100 mmHg by controlling the propofol concentration and fluid infusion. Monitored with a rectal temperature probe, body temperature was maintained between 36°C and 37°C using a heating pad and blanket. Arterial blood gases were measured (i-STAT1®; FUSO Pharmaceutical, Ltd, Osaka, Japan) at 60-minute intervals. Metabolic and respiratory acid-base balance was confirmed to maintain pH between 7.35 and 7.45, PaO_2 was maintained above 100 mmHg, and $PaCO_2$ was maintained between 35 and 45 mmHg by adjusting respiratory volume and rate.

Surgical Procedure

Surgery was performed by a single cardiovascular surgeon to exclude effects on measurements related to surgeon expertise.

First, an L4 laminectomy was performed, and the dorsal aspect of the dura mater, measuring 1.5 cm×1.5 cm, over the spinal cord was exposed.

Second, the animals were placed in the right decubitus position. The chest was opened through a thoracotomy in the ninth left intercostal space. The abdominal aorta was exposed through a left flank incision and exfoliated from the descending aorta (Th11) to the trifurcation, with careful exposure of the L2-L7 SAs. After heparin was administered intravenously at 200 IU/kg, a temporary descending aorta to left external iliac artery bypass was created. At proximal sites, a 10-Fr aortic cannula (Duraflo II; Edwards Lifesciences, Irvine, CA) was inserted, and at distal sites, an 8-mm woven graft was anastomosed, and these devices were connected.

Then, 22-G cannulae were placed at the left common carotid artery, right femoral artery, and L5 level abdominal aorta to monitor the mean proximal arterial blood pressure (mPAP), mean distal arterial blood pressure (mDAP), and segmental arterial pressure (SAP), respectively. The SAP substituted for SCPP while the abdominal aorta was clamped (between L3 to L4 and L6 to L7) (Figure 1). Since the AKA branches from the L4 or L5 SA in dogs, this range of interruption included the AKA [3-5].

Measurement of spinal cord blood flow

Referring to Fujimaki et al.'s procedure [6], SCBF was measured using laser Doppler flowmetry (Omegaflow FLO-N1; Neuroscience, Tokyo, Japan) to assess real-time microcirculatory changes in the spinal cord. The laser probe was placed in contact with the intact dorsal dura mater at the L5 segmental level of the spinal cord, which was at the upper L4 vertebral level and connected to the laser Doppler flowmeter. The probe was affixed in a riding position over the spinal cord. Output signals were collected continuously throughout the experiment and averaged every 3 seconds.

Experimental protocol

Mean blood pressures (mPAP, mDAP, SAP(SCPP)) and SCBF were measured at the same time under the following three conditions (Figure 2): Condition 1, no aortic clamp and no SA clamp (control group); Condition 2, aortic clamp (between L3 to L4 and L6 to L7), and L2, L3, and L7 SA clamps (L2-L7 SAs flow halted) with distal perfusion; and Condition 3, aortic clamp (between L3 to L4 and L6 to L7) and L2, L3, and L7 SA clamps (L2-L7 SAs flow halted) with no distal perfusion.

The mean values of each measurement over 3 minutes from the start of measurements were taken as the baseline values. After completing the baseline measurements, Condition 1 was created, and continuous administration of 0.5 μg/kg/min norepinephrine was started. Measurement data for each site were sampled every 15 sec from the start of the rise in mSBP (the mPAP measurement value was taken to be mSBP). The continuous administration of norepinephrine was stopped after 5 minutes, and data sampling was done for about 10 minutes as blood pressure naturally decreased and stabilized. The same procedure was continued for Conditions 2 and 3.

A difference of less than 10 mmHg between mPAP and mDAP was taken to indicate that the distal bypass during the experiment was functioning effectively.

Data analysis

All data were recorded by the monitor (PowerLab, ADInstruments, ADInstruments Pty Ltd, Castle Hill, Australia), which allowed constant real-time recording of the arterial blood pressure (mPAP, mDAP, SAP(SCPP)) and SCBF. Data were entered into a database and analyzed using SPSS statistical software (version 21.0J; IBM Corp., Armonk, NY)

Scatter plots of mSBP and SCBF, SAP (SCPP) and SCBF, and mSBP and SAP (SCPP) were created, and levels of correlation were analyzed with Spearman's rank correlation coefficient and regression analysis. A probability of 5% was considered significant.

2. Results

Condition 1: When blood flow was maintained from the SAs, SCBF increased mildly relative to the increase in mSBP (Figure 3, *A*). The scattergram of percentage changes revealed that there was a weak positive correlation between mSBP and SCBF ($r=0.403$, $r^2=0.162$, $p<.01$) (Figure 3, *B*).

Condition 2: Under this condition, inflow from the SAs at L2-L7 was halted with distal perfusion. When inflow from the SAs was stopped, SCBF and SAP (SCPP) fell, but they rose as mSBP increased (Figure 4, *A*).

The scattergram of percentage changes showed that there were positive correlations between mSBP and SCBF ($r=0.844$, $r^2=0.711$, $p<.01$), between SAP(SCPP) and SCBF ($r=0.803$, $r^2=0.644$, $p<.001$), and between mSBP and SAP(SCPP) ($r=0.898$, $r^2=0.806$, $p<.001$) (Figure 4, *B*, *C*, and *D*).

Condition 3: Following condition 2, distal perfusion was halted. When the distal bypass was clamped, SCBF and SAP(SCPP) fell further, but they then rose as mSBP increased (Figure 5, *A*). The scattergram of percentage changes showed that there were positive correlations between mSBP and SCBF ($r=0.834$, $r^2=0.695$, $p<.01$), between SAP(SCPP) and SCBF ($r=0.832$, $r^2=0.692$, $p<.01$), and between mSBP and SAP(SCPP) ($r=0.837$, $r^2=0.700$, $p<.001$) (Figure 5, *B*, *C*, and *D*).

3. Discussion

Spinal cord injury (SCI) (paraparesis and paraplegia) caused by interruption of the blood supply to the spinal cord from SAs is one of the greatest concerns in thoracic and thoracoabdominal aortic aneurysm surgery. The AKA is a particularly important blood supply route to the anterior spinal artery (ASA), but in extensive aortic aneurysm surgeries, several pairs of SAs including the AKA are sacrificed. This makes preservation of perioperative SCBF an issue.

At such times, SCBF depends on flow from the collateral network, but experimental studies in recent years have shown that the collateral network to the spinal cord serves an important role in preserving SCBF [2, 7-10].

In actual clinical practice, SCBF decreases with sacrifice of the SAs, but SCBF from the collateral network is increased by augmenting the systemic blood pressure with the use of volume load and/or vasopressors such as phenylephrine and norepinephrine. This has been shown to result in effective reduction of SCI [11-14].

This suggests a positive correlation between SCBF and systemic blood pressure, but direct measurement of SCBF is difficult in actual clinical settings. Vascularization of the spinal cord of dogs is similar to that of human beings, and dogs have a rich anastomotic network of spinal cord vascularization, particularly at the conus medullaris, as do human beings [3, 5]. In this experimental study, six pairs of SAs including the AKA were interrupted, and the mSBP and SCBF were directly measured using a laser blood flow meter that can measure SCBF in real time [15, 16]. The correlation between them was then examined.

Reports showing a correlation between mSBP and SCBF in a simple aortic cross-clamping model in previous experimental research have been seen [17, 18], but in the present study, SAs were interrupted, and distal perfusion was maintained by a distal bypass. This has been shown to continuously maintain distal perfusion intraoperatively and postoperatively in endovascular aortic repair, while distal perfusion with left heart bypass or FF partial bypass for assisted circulation has been shown to be effective in protecting the spinal cord in open thoracoabdominal surgery [19-21]. An experimental model to simulate the above was devised. The clear decrease in SCBF with distal bypass interruption in this experiment showed that distal perfusion contributed significantly to SCBF via the collateral network.

The results of this experiment showed a highly positive correlation between the rates of increase in the mSBP and SCBF when six SAs including the AKA were interrupted. Under distal perfusion, a simple linear regression line showed that mSBP augmentation of 1.33-fold is needed after SA interruption to maintain the blood flow from before the interruption, while mSBP augmentation of 1.68-fold is needed with non-distal perfusion. This is the first report to experimentally measure and quantify the degree to which blood pressure needs to be increased to maintain SCBF. It is also significant in that it simultaneously shows the degree to which distal perfusion contributes to spinal cord circulation.

At the same time, a weak positive correlation was seen between the increases in mSBP and SCBF in the control group that did not have interrupted SAs in this experiment. SCBF did not change with mSBP of 50-135 mmHg in a monkey experiment [22], mSBP of 40-100 mmHg in a lamb experiment [23], or mSBP of 80-160 mmHg in a cat experiment [24]. These studies demonstrated the blood pressure range in which autoregulation acts in the SCBF experimentally. The paravertebral sympathetic ganglia have also been reported to have a specific role in autoregulation of the SCBF [24]. In this study, spinal cord circulation collapsed with extensive SA interruption, autoregulation was not seen, and a positive correlation was seen between the rise in mSBP and increased SCBF from the collateral network.

It is thought that SCI can be protected by maintaining the SCPP at 50-60 mmHg or greater [25]. Ets et al. placed a blood pressure monitoring catheter for several days in the distal end of the ligated SA at the time of thoracoabdominal surgery to measure SCPP both intraoperatively and postoperatively, and they stated that this was effective in managing spinal cord circulation [26-28]. Since it is difficult to place a catheter in the fine SAs of dogs, SCPP was substituted with placement of blood pressure monitoring catheters in the area of the aortic interruption (L4 to L6).

The results of this experiment showed a high positive correlation between SAP(SCPP) and SCBF, and monitoring of SAP(SCPP) after interruption of SAs is thought to be useful in appropriately maintaining spinal cord circulation. In addition, systemic blood pressure was found to be positively correlated with SAP(SCPP).

Blood supply from the collateral network was increased by increasing the mSBP to deal with the decrease in SCBF from sacrificing the SAs. In several experimental models, nerve damage after extensive SA sacrifice was shown to begin 1-5 hours postoperatively [26, 29], and maintaining a somewhat high blood pressure with hemodynamic manipulation for 24-72 hours until the collateral network develops [8, 14, 29] was demonstrated experimentally to be important as a means of preventing SCI.

4. Limitations

The erector spinae muscles are a source of some of the collateral network blood supply, but slight damage to these muscles could not be avoided during laminectomy and exposure of L5 dura mater. In addition, SCBF was measured on the dorsal side of the spinal cord, and due to the properties of the laser blood flow meter, tissue blood flow is only reflected to a depth of about 1 mm. Consequently, blood flow in the ASA region of the ventral spinal cord could not be measured directly. However, since there is communication between the posterospinal artery and the anterospinal artery via the epidural arcade and pial arterial plexus, increased SCBF on the dorsal side of the spinal cord is thought to reflect increased SCBF in the ASA region [1, 2, 8, 9].

SCPP is accurately expressed with SAP-cerebrospinal fluid pressure (CSFP). It would have been preferable to measure CSFP and show SAP-CSFP as SCPP in this experiment as well, but measuring CSFP was difficult because of the experimental procedures. Therefore, it is thought that the SCPP shown in this experiment should be used as an approximation of the actual SCPP.

Norepinephrine was used to raise the blood pressure because it is a drug generally used in cardiovascular surgery, but the pharmacological actions of norepinephrine on spinal cord vessels are not clear, and the effect of this will need to be clarified in future research.

This study did not show a relationship between changes in SCBF and neurological or histological changes. Appropriate simultaneous monitoring of transcranial motor evoked potentials is thought to also be useful in evaluating spinal cord function [30, 31].

Acknowledgments

The authors wish to thank Tetuji Chinen, Chisato Kamiya, Yuichi Totuka, Takahumi Kozaki, and Yuji Kaneshiro for their excellent technical assistance.

References

1. Martirosyan NL, Feuerstein JS, Theodore N, Cavalcanti DD, Spetzler RF, Preul MC. Blood supply and vascular reactivity of the spinal cord under normal and pathological conditions. J Neurosurg Spine 2011 Sep;15(3):238-51.

2. Etz CD, Kari FA, Mueller CS, et al. The collateral network concept: a reassessment of the anatomy of spinal cord perfusion. J Thorac Cardiovasc Surg 2011 Apr;141(4):1020-8.

3. Sindou M, Chignier E, Mazoyer JF, Pialat J, Fischer G, Descotes J. Revascularization of the spinal cord by micro-anastomoses in dogs. Surg Neurol 1979 Dec;12(6):492-5.

4. Kato S, Kawahara N, Tomita K, Murakami H, Demura S, Fujimaki Y. Effects on spinal cord blood flow and neurologic function secondary to interruption of bilateral segmental arteries which supply the artery of Adamkiewicz: an experimental study using a dog model. Spine 2008 Jun 15;33(14):1533-41.

5. Doppman JL, Ramsey R. Selective arteriography of the lumbar spinal cord in dogs. Neuroradiology 1971 Dec;3(2):64-7.

6. Fujimaki Y, Kawahara N, Tomita K, Murakami H, Ueda Y. How many ligations of bilateral segmental arteries cause ischemic spinal cord dysfunction? An experimental study using a dog model. Spine 2006 Oct 1;31(21):E781-9.

7. Uezu T, Koja K, Kuniyoshi Y, et al. Blood distribution to the anterior spinal artery from each segment of intercostal and lumbar arteries. J Cardiovasc Surg 2003 Oct;44(5):637-45.

8. Geisbusch S, Schray D, Bischoff MS, Lin HM, Griepp RB, Di Luozzo G. Imaging of vascular remodeling after simulated thoracoabdominal aneurysm repair. J Thorac Cardiovasc Surg 2012 Dec;144(6):1471-8.

9. Etz CD, Kari FA, Mueller CS, Brenner RM, Lin HM, Griepp RB. The collateral network concept: remodeling of the arterial collateral network after experimental segmental artery sacrifice. J Thorac Cardiovasc Surg 2011 Apr;141(4):1029-36.

10. Griepp RB, Griepp EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. Ann Thorac Surg 2007 Feb;83(2):S865-9; discussion S90-2.

11. Chiesa R, Melissano G, Marrocco-Trischitta MM, Civilini E, Setacci F. Spinal cord ischemia after elective stent-graft repair of the thoracic aorta. J Vasc Surg 2005 Jul;42(1):11-7.

12. Kawanishi Y, Okada K, Matsumori M, et al. Influence of perioperative hemodynamics on spinal cord ischemia in thoracoabdominal aortic repair. Ann Thorac Surg 2007 Aug;84(2):488-92.

13. Hnath JC, Mehta M, Taggert JB, et al. Strategies to improve spinal cord ischemia in endovascular thoracic aortic repair: Outcomes of a prospective cerebrospinal fluid drainage protocol. J Vasc Surg 2008 Oct;48(4):836-40.

14. Ullery BW, Cheung AT, Fairman RM, et al. Risk factors, outcomes, and clinical

manifestations of spinal cord ischemia following thoracic endovascular aortic repair. J Vasc Surg 2011 Sep;54(3):677-84.

15. Lindsberg PJ, O'Neill JT, Paakkari IA, Hallenbeck JM, Feuerstein G. Validation of laser-Doppler flowmetry in measurement of spinal cord blood flow. Amer J Physiol 1989 Aug;257(2 Pt 2):H674-80.

16. Lindsberg PJ, Jacobs TP, Frerichs KU, Hallenbeck JM, Feuerstein GZ. Laser-Doppler flowmetry in monitoring regulation of rapid microcirculatory changes in spinal cord. Amer J Physiol 1992 Jul;263(1 Pt 2):H285-92.

17. Taira Y, Marsala M. Effect of proximal arterial perfusion pressure on function, spinal cord blood flow, and histopathologic changes after increasing intervals of aortic occlusion in the rat. Stroke 1996 Oct;27(10):1850-8.

18. Izumi S, Okada K, Hasegawa T, et al. Augmentation of systemic blood pressure during spinal cord ischemia to prevent postoperative paraplegia after aortic surgery in a rabbit model. J Thorac Cardiovasc Surg 2010 May;139(5):1261-8.

19. Laschinger JC, Cunningham JN, Jr., Nathan IM, Knopp EA, Cooper MM, Spencer FC. 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 1983 Oct;36(4):417-26.

20. Strauch JT, Spielvogel D, Lauten A, et al. Importance of extrasegmental vessels for spinal cord blood supply in a chronic porcine model. Eur J Cardiothorac Surg 2003 Nov;24(5):817-24.

21. Safi HJ, Miller CC, 3rd, Huynh TT, et al. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aortic repair: ten years of organ protection. Ann Surg 2003 Sep;238(3):372-80; discussion 80-1.

22. Kobrine AI, Evans DE, Rizzoli HV. The role of the sympathetic nervous system in spinal cord autoregulation. Acta Neurol Scand Suppl 1977;64:54-5.

23. Hitchon PW, Lobosky JM, Yamada T, Johnson G, Girton RA. Effect of hemorrhagic shock upon spinal cord blood flow and evoked potentials. Neurosurgery 1987 Dec;21(6):849-57.

24. Young W, DeCrescito V, Tomasula JJ. Effect of sympathectomy on spinal blood flow autoregulation and posttraumatic ischemia. J Neurosurg 1982 May;56(5):706-10.

25. Kazama S, Masaki Y, Maruyama S, Ishihara A. Effect of altering cerebrospinal fluid pressure on spinal cord blood flow. Ann Thorac Surg 1994 Jul;58(1):112-5.

26. Etz CD, Homann TM, Plestis KA, et al. Spinal cord perfusion after extensive segmental artery sacrifice: can paraplegia be prevented? Eur J Cardiothorac Surg 2007 Apr;31(4):643-8.

27. Etz CD, Di Luozzo G, Zoli S, et al. Direct spinal cord perfusion pressure monitoring in extensive distal aortic aneurysm repair. Ann Thorac Surg 2009 Jun;87(6):1764-73; discussion 73-4.

28. Etz CD, Zoli S, Bischoff MS, Bodian C, Di Luozzo G, Griepp RB. Measuring the collateral network pressure to minimize paraplegia risk in thoracoabdominal aneurysm resection. J

Thorac Cardiovasc Surg 2010 Dec;140(6 Suppl):S125-30; discussion S42-S46.

29. Etz CD, Homann TM, Luehr M, et al. Spinal cord blood flow and ischemic injury after experimental sacrifice of thoracic and abdominal segmental arteries. Eur J Cardiothorac Surg 2008 Jun;33(6):1030-8.

30. Lips J, de Haan P, de Jager SW, Vanicky I, Jacobs MJ, Kalkman CJ. The role of transcranial motor evoked potentials in predicting neurologic and histopathologic outcome after experimental spinal cord ischemia. Anesthesiology 2002 Jul;97(1):183-91.

31. Weigang E, Hartert M, Siegenthaler MP, et al. Neurophysiological monitoring during thoracoabdominal aortic endovascular stent graft implantation. Eur J Cardiothorac Surg 2006 Mar;29(3):392-6.

Abbreviation and Acronyms

SCBF = spinal cord blood flow

SAs = segmental arteries

mSBP = mean systemic blood pressure

mPAP = mean proximal arterial blood pressure

mDAP = mean distal arterial blood pressure

SAP = segmental arterial blood pressure

SCPP = spinal cord perfusion pressure

SCI = spinal cord injury

AKA = Adamkiewicz artery

ASA = anterior spinal artery

CSFP = cerebrospinal fluid pressure

Figure legends

FIGURE 1. Experimental model

PAP, proximal arterial blood pressure; SAP, segmental arterial blood pressure; SCPP, spinal cord perfusion pressure; DAP, distal arterial blood pressure; SCBF, spinal cord blood flow; AKA, Adamkiewicz artery; ASA, anterior spinal artery

FIGURE 2. Experimental protocol

Condition 1, no aortic clamp and no segmental artery (SA) clamp (control group); Condition 2, L2–L7 SAs flow halted with distal perfusion; Condition 3, L2–L7 SAs flow halted with no distal perfusion.

FIGURE 3. Condition 1, no aortic clamp and no SA clamp (control group)

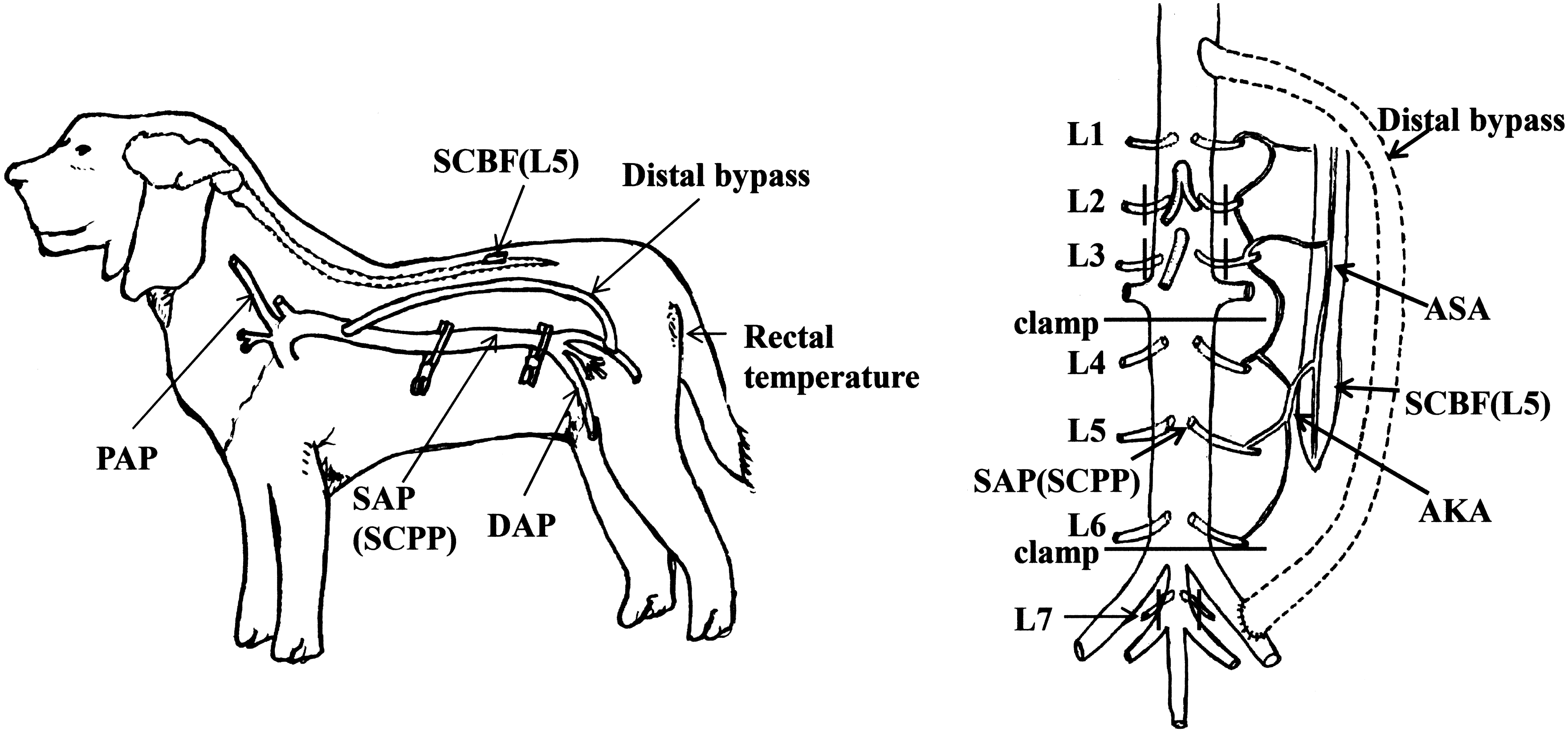
A, Laboratory chart shows the real-time record of systemic blood pressure (SBP), distal arterial blood pressure (DAP), and spinal cord blood flow (SCBF). SCBF increases mildly relative to the increase in mSBP. NOA, norepinephrine; B, The scattergram of percentage changes shows a weak positive correlation between mSBP and SCBF.

FIGURE 4. Condition 2, aortic clamp (between L3 to L4 and L6 to L7) and L2, L3, and L7 SA clamps (L2–L7 SAs flow halted) with distal perfusion

A, When inflow from the SAs is stopped, SCBF and SAP(SCPP) fall; however, they rise as systemic blood pressure increases; B, The scattergram of percentage changes shows a positive correlation between mSBP and SCBF; C, A positive correlation is seen between SAP(SCPP) and SCBF; D, A positive correlation is seen between mSBP and SAP(SCPP).

FIGURE 5. Condition 3, aortic clamp (between L3 to L4 and L6 to L7) and L2, L3, and L7 SA clamps (L2–L7 SAs flow halted) with no distal perfusion

A, When the distal bypass is clamped, SCBF and SAP(SCPP) fall further; however, they then rise as SBP increases; B, The scattergram of percentage changes shows a positive correlation between mSBP and SCBF; C, A positive correlation is seen between SAP(SCPP) and SCBF; D, A positive correlation is seen between mSBP and SAP(SCPP).



SCBF(L5)
Distal bypass
Rectal
temperature
PAP
SAP
(SCPP)
DAP
L1
L2
L3
clamp
L4
L5
SAP(SCPP)
L6
clamp
L7
Distal bypass
ASA
SCBF(L5)
AKA

FIGURE 1.

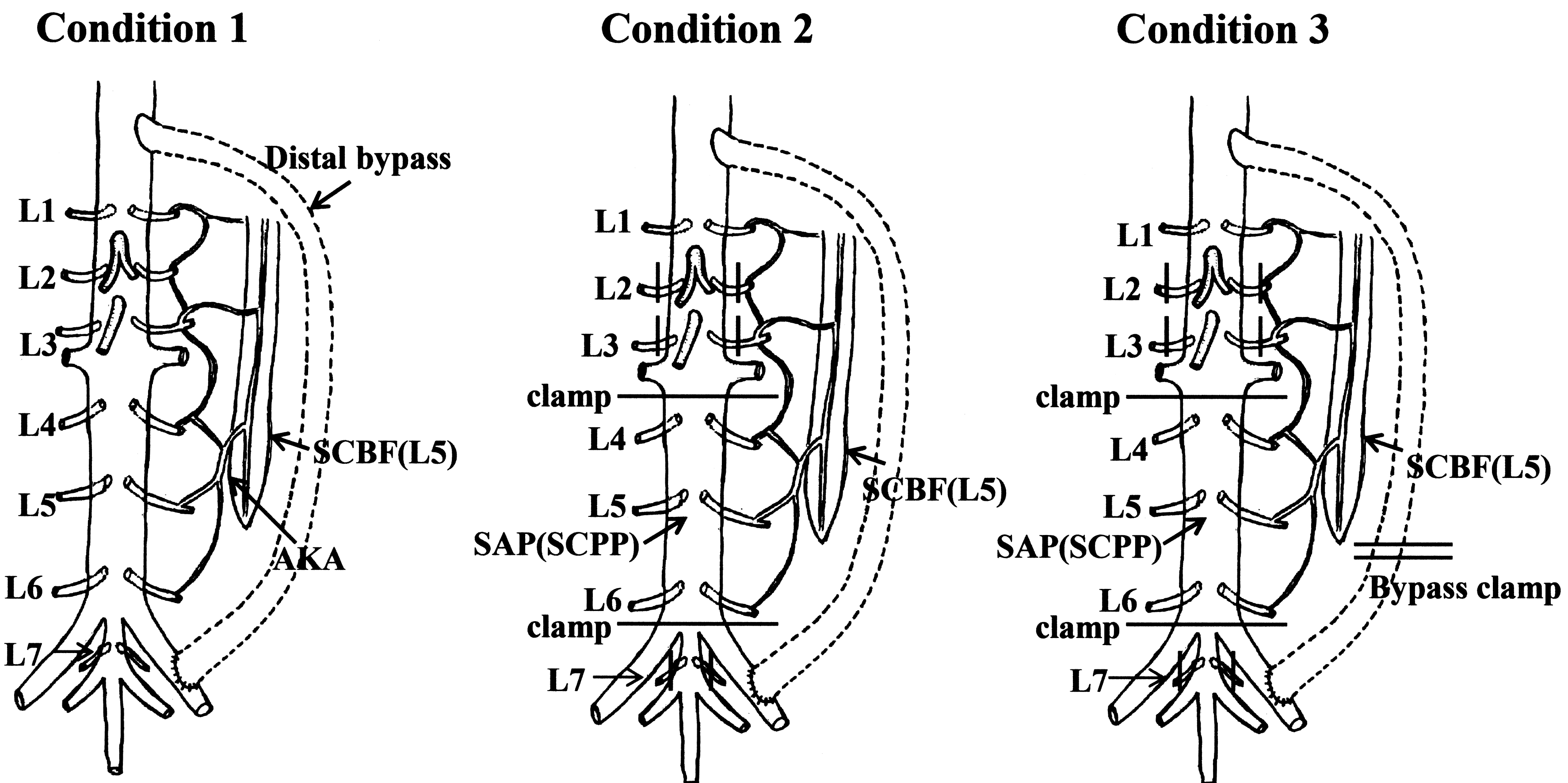

Condition 1
Condition 2
Condition 3
Distal bypass
L1
L2
L3
L4
L5
L6
L7
SCBF(L5)
AKA
clamp
SAP(SCPP)
Bypass clamp

FIGURE 2.

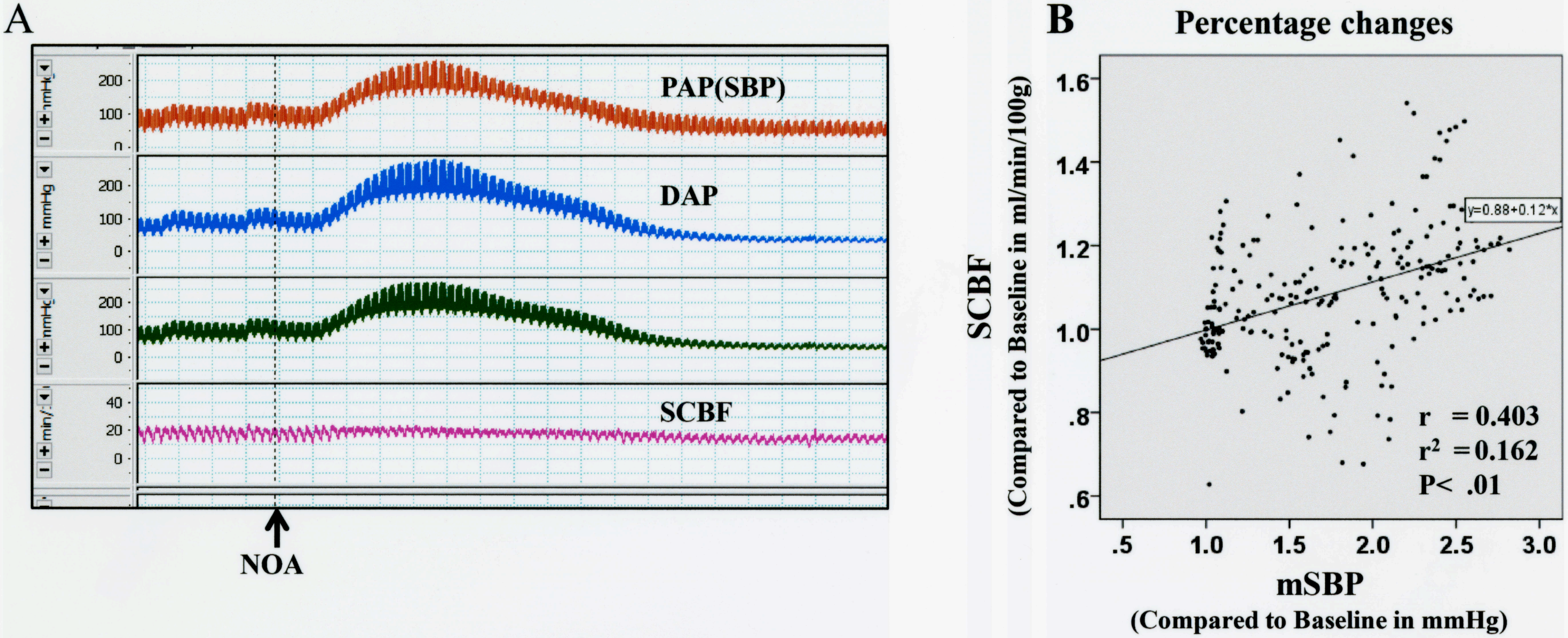

A
PAP(SBP)
DAP
SCBF
NOA
B
Percentage changes
SCBF
(Compared to Baseline in ml/min/100g)
y=0.88+0.12*x
r = 0.403
r2 = 0.162
P< .01
mSBP
(Compared to Baseline in mmHg)

FIGURE 3.

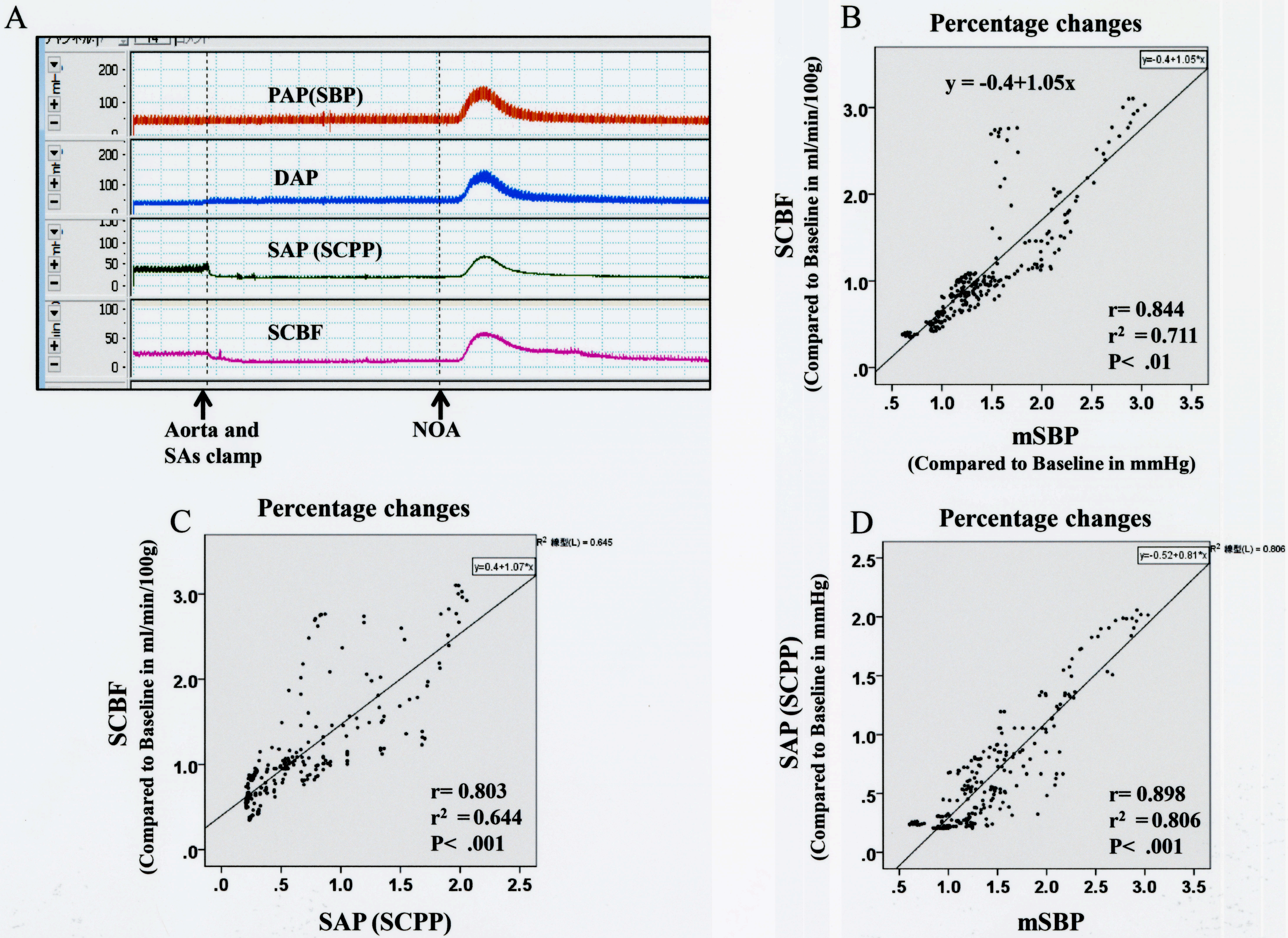

A
PAP(SBP)
DAP
SAP (SCPP)
SCBF
Aorta and SAs clamp
NOA
B
Percentage changes
y = -0.4+1.05x
r= 0.844
r2 =0.711
P< .01
SCBF
(Compared to Baseline in ml/min/100g)
mSBP
(Compared to Baseline in mmHg)
C
Percentage changes
y=0.4+1.07*x
r= 0.803
r2 =0.644
P< .001
SCBF
(Compared to Baseline in ml/min/100g)
SAP (SCPP)
(Compared to Baseline in mmHg)
D
Percentage changes
y=-0.52+0.81*x
r= 0.898
r2 =0.806
P< .001
SAP (SCPP)
(Compared to Baseline in mmHg)
mSBP
(Compared to Baseline in mmHg)

FIGURE 4.

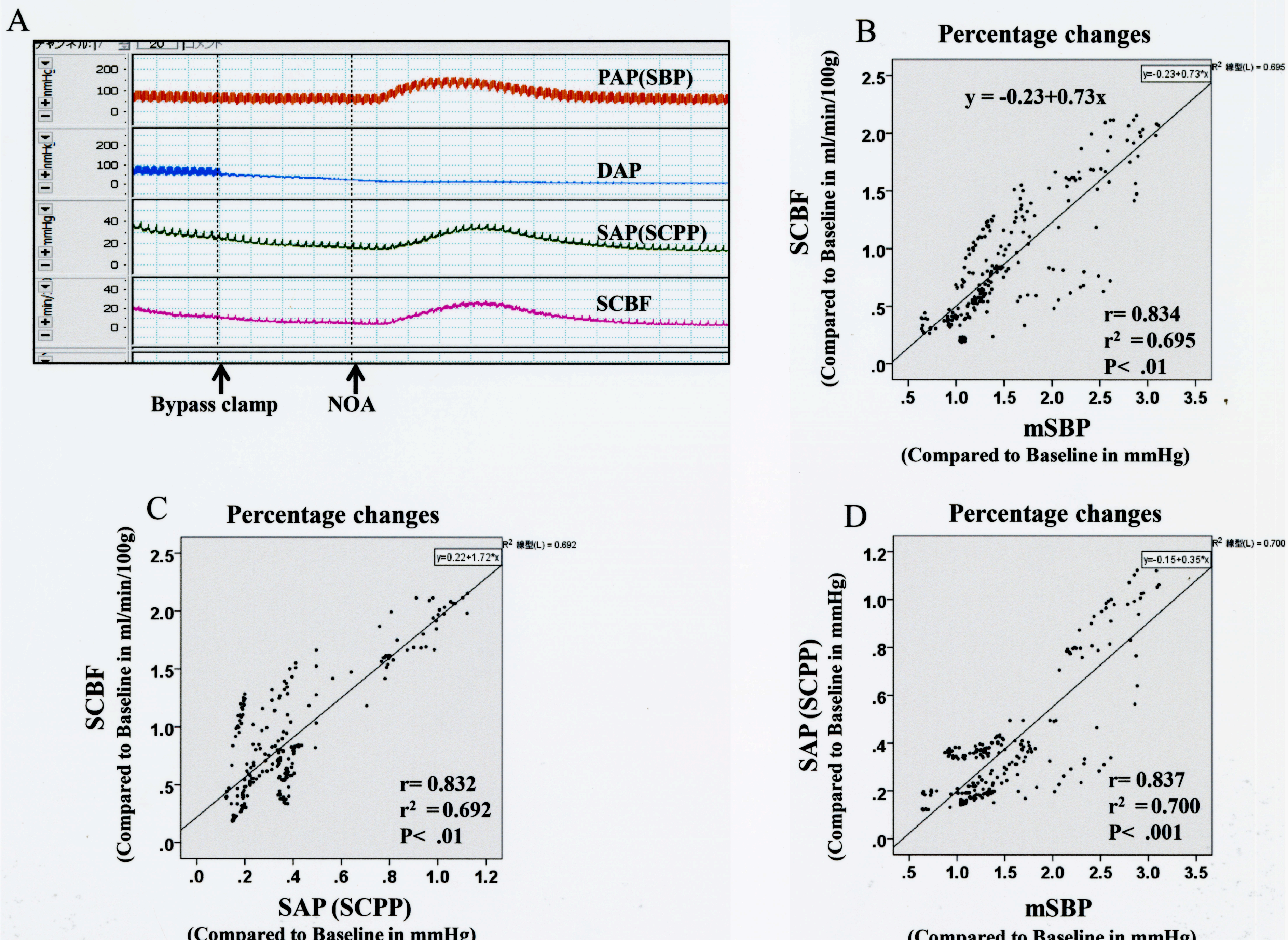

A
PAP(SBP)
DAP
SAP(SCPP)
SCBF
Bypass clamp
NOA
B
Percentage changes
y = -0.23+0.73x
y=-0.23+0.73*x
R² 線型(L) = 0.695
r= 0.834
r² =0.695
P< .01
SCBF
(Compared to Baseline in ml/min/100g)
mSBP
(Compared to Baseline in mmHg)
C
Percentage changes
y=0.22+1.72*x
R² 線型(L) = 0.692
r= 0.832
r² =0.692
P< .01
SCBF
(Compared to Baseline in ml/min/100g)
SAP (SCPP)
(Compared to Baseline in mmHg)
D
Percentage changes
y=-0.15+0.35*x
R² 線型(L) = 0.700
r= 0.837
r² =0.700
P< .001
SAP (SCPP)
(Compared to Baseline in mmHg)
mSBP
(Compared to Baseline in mmHg)

FIGURE 5.