ASPECTS OF THE SPINAL CORD CIRCULATION AS ASSESSED BY INTRATHECAL OXYGEN TENSION MONITORING DURING VARIOUS ARTERIAL INTERRUPTIONS IN THE PIG

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Objective: We sought to study the effect of various modes of interruption of the spinal cord blood supply on intrathecal oxygenation.

Methods: In 24 pigs intrathecal PO_2 , PCO_2 , and pH were continuously monitored with a multiparameter catheter (Paratrend 7, Biomedical Sensors; Diametrics Medical, Inc, St Paul, Minn) during and after aortic crossclamping or selective interruption of segmental arteries and proximal collateral circulation.

Results: Proximal aortic clamping ($n = 6$) produced complete ischemia, whereas a second clamp close to the celiac trunk ($n = 4$) partly protected against spinal cord ischemia. This is explained by prevention of the *steal phenomenon* in the excluded part of the aorta. Adding clamps to the subclavian arteries ($n = 6$) created complete spinal ischemia as the collateral circulation was interrupted. In another group ($n = 4$) all segmental arteries below T5 were occluded with no reaction in the intrathecal variables. Additional selective clamping of supreme intercostal arteries ($n = 4$) showed the relative importance of the subclavian and vertebral collateral pathways.

Conclusions: Continuous intrathecal PO_2 was monitored during various modes of interruption of the spinal cord blood supply. This provided insight into the ischemia mechanisms and relative importance of the segmental contribution and proximal collateral pathways of the spinal cord circulation in pigs. A short literature review is given, and aspects of comparative anatomy are discussed. (J Thorac Cardiovasc Surg 2001;121:762-72)

Spinal cord ischemia during thoracoabdominal aortic operations remains a clinical concern because the incidence of neurologic sequelae is still substantial. A great number of studies exist, but further experimental studies and monitoring techniques are needed to increase our knowledge of the spinal cord circulation. We have recently described and validated¹⁻³ the application of a multiparameter catheter for online intrathecal PO_2 , PCO_2 , and pH monitoring. It is important to study how the intrathecal variables react to manipulations of the spinal cord circulation and to establish critical levels of PO_2 (ie, where the aerobic threshold is reached and dysoxia threatens). For this reason, a model

for the study of selective spinal cord ischemia and reperfusion without confounding changes in the systemic circulation is needed. The aim of the present study was to investigate the functional vascular anatomy of the spinal cord by using intrathecal PO_2 monitoring during various modes of aortic clamping and selective interruption of segmental arteries or collateral pathways.

Methods

Twenty-four domestic pigs (14 male and 10 female animals) with a mean weight of 24.5 ± 3.5 kg were used. The anesthesia was induced with 3 mg/kg intramuscular tiletamin and 3 mg/kg zolazepam (Zoletil 100, Reading, Pa), 2.2 mg/kg xylazine (Rompun vet, Bayer AG, Leverkusen, Germany), and 0.04 mg/kg atropine sulphate. The animals were placed in a dorsal recumbent position, an intravenous line was inserted, and 20 mg of morphine was administered intravenously before tracheostomy. After muscular relaxation with 8 mg of pancuronium bromide (Pavulon, Organon Teknika, Eppelheim, Germany), ventilation was maintained (Servo ventilator, 900C, Siemens-Elema AB, Solna, Sweden), with a tidal volume of approximately 10 mL/kg and with an oxygen-air mix-

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ture of 40%. This resulted in good systemic oxygenation during all phases of the experiments ($\text{PaO}_2 > 18 \text{ kPa}$; oxygen saturation as measured by pulse oximetry, 96%-99%). Arterial blood gas analysis was used to adjust the respiratory rate (23-27 breaths/min) to normocapnia (PCO_2 5.0-5.8 kPa) at baseline conditions. A moderate positive end-expiratory pressure of 4 cm H_2O was used.

Anesthesia was continued with an infusion consisting of 1000 mL of glucose (25 mg/mL), 5000 mg of ketamine, 120 mg of morphine, and 60 mg of pancuronium bromide at a rate of $4 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ intravenously. A continuous infusion of isotonic saline solution (mean total, 1490 mL) was given for basal fluid requirements and intraoperative losses at $10 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$. Blood loss was moderate ($< 20\%$ of blood volume) and replaced by 250 to 750 mL of dextran 70 (Macrodex, Pharmalink, Copenhagen, Denmark). Hemoglobin levels were $75 \pm 15 \text{ g/L}$ during the experiments. Normothermia was maintained with prewarmed infusions and by the use of a thermostatically controlled heating pad.

Catheterization of the right carotid artery, the right femoral artery (18 gauge, Ohmeda Medical, Laurel, Md), and the right internal jugular vein (7F thermodilution catheter, Ohmeda) was performed, allowing monitoring of central hemodynamics, arterial pressure distal to aortic clamping, mixed venous PO_2 , and core temperature. For all hemodynamic measurements, Ohmeda transducers (Medical Devices Division) were used, and data were displayed with a Siemens Sirecust 1280/1281 surveillance monitor and printer (Siemens Medical Electronics Inc). Electrocardiography was continuously recorded. A suprapubic catheter was inserted for urinary output monitoring.

After a median sternotomy, the subclavian arteries, the aortic arch, and the entire descending thoracic aorta were exposed. Through a paramedian retroperitoneal approach, the abdominal aorta and the iliac artery bifurcation were dissected. The left diaphragm was circumferentially divided, and anterior mobilization of the left kidney was performed. The abdominal visceral, intercostal (below T5), and lumbar arteries were also identified.

A limited laminectomy was performed at the lower thoracic level, and through an arterial needle introducer, a multiparameter PO_2 , PCO_2 , pH, and temperature sensor (Paratrend 7, Biomedical Sensors; Diametrics Medical, Inc, St Paul, Minn) was introduced into the intrathecal space for continuous monitoring of the cerebrospinal fluid (CSF). Only a minimal amount of CSF was drained during this procedure. The principles and the use of this thin and flexible sensor device for detection of spinal cord ischemia during experimental and preliminary clinical aortic crossclamping have previously been reported.^{1,3} Spinal application of polarographic oxygen measurements was earlier described by Wadough and Svensson.¹ Blood gas samples were examined in the automated blood gas analyzer ABL 300 (Radiometer A/S, Copenhagen, Denmark), with the hemoximeter calibrated for pig hemoglobin.

At the end of a stabilization period of about 30 minutes after preparation, baseline hemodynamic measurements,

blood gases, and intrathecal recordings were obtained. The animals were divided into 5 groups according to different manipulations of the spinal cord circulation (Fig 1): group P, proximal aortic crossclamping just below the left subclavian artery; group D, double aortic crossclamping (proximal plus distal clamping just above the celiac trunk); group DS, double aortic crossclamping combined with clamping of both subclavian arteries; group I, interruption of all intercostal (below T5) and lumbar segmental arteries; and group IS, interruption of all segmental arteries combined with selective clamping of supreme intercostals from the subclavian arteries.

Complete recordings for all groups were made at baseline and after 30, 60, and 90 minutes. The clamping time was 30 minutes, followed by 60 minutes of reperfusion in groups P, D, and DS. In groups I and IS segmental arteries were clamped throughout the 90-minute experiments because removal of clamps from the small-diameter vessels would not guarantee reperfusion because of the risk of vasospasm or permanent occlusion.

To control proximal hypertension (mean arterial pressure, $> 130 \text{ mm Hg}$) after crossclamping (groups P, D, and DS), a sodium nitroprusside infusion (mean total dose, 53 mg) was given for cardiac unloading during clamping. Just before declamping in the same groups, this infusion was replaced by a dopamine infusion (mean total dose, 78 mg) for vasoconstriction and inotropic support to achieve a mean pressure of more than 70 mm Hg. During the first minutes after declamping of the aorta, the pigs in groups P, D, and DS were given 200 mL of tromethamine (INN: trometamol)/bicarbonate buffer (Tribonat, Pharmacia-Upjohn, Stockholm, Sweden).

Supplementary experiments were performed to study the *steal phenomenon* during proximal aortic crossclamping and to evaluate the role of the proximal collateral pathways for the spinal cord circulation. Duplex ultrasonography (Diasonics Synergy, GE Medical Systems, Milwaukee, Wis) and spectral Doppler registration (10-MHz linear probe) of blood flow in the lower intercostal arteries were performed after application of the proximal aortic crossclamping (group P). This was done to demonstrate flow reversal in intercostal arteries in relation to aortic clamping. To demonstrate the important role of the distal thoracic intercostal arteries for the steal phenomenon, additional experiments were made in group D. By moving the distal aortic clamp from 2 segments above the celiac trunk to a position close to the trunk, we could study the effect of clamp position on intrathecal oxygenation and intercostal blood flow. In group IS the contribution of vertebral artery blood supply to the spinal cord was studied by moving the clamp from selective supreme intercostal arteries to proximal subclavian arteries.

After the last measurements, the pigs were given a lethal injection of 10 to 20 mL of potassium chloride while still anesthetized. Surgical dissections were extended for photo documentation of the vascular anatomy of segmental and collateral arteries. All animals were treated in compliance with the "Guiding Principles in the Care and Use of Animals," which was approved by the Council of The American

Table I. Hemodynamic and arterial blood gas data

Measuring point	Parameter								
	Cardiac output	Pulse	MAP*	PCWP*	MPAP*	CVP*	a-pH	a-PCo ₂ †	a-PO ₂ †
Baseline									
Group P, n = 6	3.1 ± 0.9	100 ± 5	90 ± 12	10 ± 2	20 ± 1	6 ± 2	7.42 ± 0.01	5.5 ± 0.2	27.5 ± 3.9
Group D, n = 4	4.2 ± 1.3	121 ± 37	96 ± 22	7 ± 2	23 ± 4	5 ± 1	7.43 ± 0.04	5.4 ± 0.3	25.6 ± 4.3
Group DS, n = 6	4.1 ± 0.9	144 ± 34	86 ± 8	10 ± 1	22 ± 2	8 ± 2	7.42 ± 0.03	5.8 ± 0.4	22.6 ± 0.8
Group I, n = 4	4.0 ± 0.4	109 ± 16	89 ± 21	12 ± 2	24 ± 1	7 ± 1	7.38 ± 0.08	5.2 ± 0.5	25.3 ± 5.5
Group IS, n = 4	3.4 ± 0.7	84 ± 12	79 ± 14	8 ± 2	17 ± 2	6 ± 2	7.45 ± 0.04	5.3 ± 0.2	30.2 ± 2.8
30 min									
Group P	3.9 ± 0.6	218 ± 13	109 ± 12	9 ± 2	22 ± 1	8 ± 1	7.37 ± 0.01	4.3 ± 0.2	30.6 ± 1.6
Group D	5.3 ± 0.2	192 ± 20	129 ± 5	12 ± 6	26 ± 4	8 ± 1	7.49 ± 0.04	3.6 ± 0.4	30.2 ± 0.4
Group DS	3.6 ± 1.7	179 ± 15	136 ± 15	12 ± 4	23 ± 2	8 ± 1	7.55 ± 0.16	3.4 ± 1.2	25.1 ± 6.2
Group I	3.5 ± 0.7	106 ± 16	91 ± 13	11 ± 2	23 ± 2	7 ± 1	7.37 ± 0.07	5.3 ± 0.5	24.5 ± 5.3
Group IS	3.4 ± 0.3	88 ± 19	85 ± 7	7 ± 2	15 ± 1	7 ± 3	7.42 ± 0.02	5.5 ± 0.3	26.1 ± 3.9
60 min									
Group P	6.5 ± 2.1	188 ± 26	74 ± 6	10 ± 3	29 ± 2	8 ± 2	7.27 ± 0.09	6.4 ± 1.3	27.9 ± 0.9
Group D	6.0 ± 1.4	187 ± 6	70 ± 7	8 ± 2	27 ± 4	6 ± 2	7.29 ± 0.04	5.4 ± 0.4	27.5 ± 2.3
Group DS, n = 4	4.7 ± 1.8	152 ± 14	85 ± 31	16 ± 7	33 ± 5	10 ± 2	7.21 ± 0.12	6.7 ± 0.9	18.9 ± 2.6
Group I	3.4 ± 0.6	102 ± 12	92 ± 10	14 ± 2	24 ± 1	7 ± 0	7.38 ± 0.07	5.2 ± 0.5	25.9 ± 4.8
Group IS	3.2 ± 0.5	83 ± 18	73 ± 6	9 ± 3	15 ± 1	7 ± 2	7.44 ± 0.03	4.9 ± 0.2	31.5 ± 1.4
90 min									
Group P	6.3 ± 2.4	183 ± 22	73 ± 6	10 ± 2	25 ± 2	8 ± 1	7.33 ± 0.05	5.6 ± 0.7	28.6 ± 0.9
Group D	5.0 ± 1.2	173 ± 17	80 ± 14	9 ± 2	26 ± 3	6 ± 2	7.32 ± 0.04	5.2 ± 0.5	25.1 ± 3.9
Group DS, n = 4	4.8 ± 1.0	144 ± 14	69 ± 10	15 ± 4	29 ± 2	10 ± 1	7.28 ± 0.09	6.0 ± 0.6	19.1 ± 3.7
Group I	3.2 ± 0.8	99 ± 12	92 ± 7	11 ± 1	23 ± 2	7 ± 2	7.40 ± 0.06	5.0 ± 0.5	27.3 ± 4.5
Group IS	3.2 ± 0.5	77 ± 14	73 ± 9	8 ± 2	16 ± 1	7 ± 1	7.44 ± 0.04	5.0 ± 0.2	30.8 ± 1.3

Values are given as means ± SD. MAP, Mean arterial pressure; PCWP, pulmonary capillary wedge pressure; MPAP, mean pulmonary arterial pressure; CVP, central venous pressure; a-, arterial.

*These values are given in millimeters of mercury; 1 mm Hg = 1.36 cm H₂O.

†These values are given in kilopascals; 1 kPa = 7.5 mm Hg.

Physiological Society.⁴ The experiments conformed to the national guidelines for animal care and were approved by the Institutional Animal Care and Ethics Committee.

Data are presented as means ± SD (WinStat version 3.1, 1995) (Tables I and II). The data in Table II show the significance of the within-group changes in intrathecal PO₂ compared with baseline PO₂ in the consistently responding groups P, D, DS, and I. A cross-tabulation for intrathecal PO₂ reduction of more than 50% would allow us to compare the effect of different clamping techniques between groups. In group IS individual data are presented because 2 different patterns of response to vascular interruption were seen.

Results

The recordings of hemodynamic data are presented in Table I. In groups P, D, and DS typical reactions to clamping were recorded, although the increase in afterload was attenuated by the use of a vasodilator. Later, a massive systemic vasodilatation and pulmonary vasoconstriction followed as a response to declamping. To block some of the effects of reperfusion, the vasopressive action of dopamine was used, as was buffer treat-

ment of metabolic acidosis. The pharmacologic interventions proved to be essential in the DS group, where most of the arterial system, except for the carotid arteries, was clamped. Even so, 2 of the animals in this group did not survive the reperfusion phase as a result of cardiovascular distress. In the 2 groups in which the aorta was not clamped (groups I and IS), the animals remained stable throughout the experiments, with no other treatment than substitution for volume losses.

The Paratrend 7-derived intrathecal data are presented in Table II. No changes in intrathecal variables could be explained by corresponding changes in arterial blood gases (Table I). The continuous recordings in Fig 2 visualized how proximal clamping (group P) and combined double and subclavian clamping (group DS) created complete spinal ischemia. In the double-clamped group (group D) collateral circulation was apparently sufficient because of the high proximal perfusion pressure and prohibited steal phenomenon in the excluded part of the aorta. Additional experiments with distal clamp placement (Fig 2, c) showed how important the distal thoracic intercostal arteries were for steal

Abbreviations:

TC = Celiac trunk
SMA = Superior mesentery artery
SA = Subclavian arteries
VA = Vertebral arteries
CA = Carotid arteries (from bicarotid trunk)

Location of clamping:

1 = Proximal aorta
2 = Distal aorta
3 = Subclavian arteries
4 = Segmental intercostal and lumbar arteries
5 = Supreme intercostal arteries

Groups:

P = 1	Proximal aortic clamping
D = 1 + 2	Proximal + distal aortic clamping
DS = 1 + 2 + 3	Proximal + distal aortic clamping + subclavian clamping
I = 4	Ligation / clamping of all lumbar segmental arteries and intercostals below T5
IS = 4 + 5	Ligation / clamping of all lumbar segmental arteries and intercostals below T5 + selective clamping of supreme intercostals

Fig 1. Clamping techniques. Schematic shows what combinations of numbered clamping locations were used in the various experimental groups. For orientation, larger arteries and important vertebral bodies are delineated.

prevention and thereby for the sufficiency of the collateral circulation. The reversal of flow caused by the steal phenomenon during proximal clamping was also documented by means of ultrasonographic examinations (Fig 3).

In group I, where only the segmental arteries were interrupted, no significant changes in the cerebrospinal PO_2 , PCO_2 , or pH were observed (Fig 4, *a*). Adding selective clamps on the supreme intercostal arteries and leaving vertebral circulation intact resulted either in ischemia (Fig 4, *b*) or only a PO_2 reduction by half (Fig 4, *c*). To achieve complete ischemia, interruption of all collaterals was crucial, as confirmed by moving the clamps to a proximal position on the subclavian arteries (Fig 4, *d*).

At conclusion of the experiments, the vascular anatomy was examined. By inspecting the interior aspects of the aorta, we looked for all segmental arteries, but no preparations were made for the intraspinal radicular vessels. The intercostal arteries originated from a left-sided common stem, with only a few exceptions. In 2

animals bilateral arteries were found in the region of T4-T7. There were 2 arteries at each lumbar segmental level, but the origins of both were very close and easy to reach with one clamp from the left-sided dissection. The number of identified intercostal arteries arising from the aorta was 8 to 9, and there were 5 lumbar pairs. The largest calibers were consistently found close to the celiac trunk (ie, the 2-3 arteries above and 1-2 arteries at or below the trunk, corresponding to T12-T14 and L1-L2). Preparation of the proximal anatomy was part of the experimental dissection. We could identify and clamp the supreme intercostal artery (T3-T5, arising from the subclavian arteries) with the first intercostal (T1) close to it on the right side. On the left side both arteries branched off from the costocervical trunk. We were not able to dissect the second intercostal arteries (T2) intraoperatively because of its deep location at the base of the vertebral or dorsal scapular arteries. The only variations in the major vessels were separate aortic origins of the carotid arteries in 2 cases (instead of a bicarotid trunk) and of the left-sided sub-

Fig 2. Paratrend 7 recordings and trend printouts (examples from groups P, D, and DS). *Single arrows* indicate the start of clamping (1 kPa = 7.5 mm Hg). Trend registrations of 30 minutes show changes in intrathecal pH, PCO_2 , and PO_2 in response to aortic crossclamping and other manipulations of the spinal cord circulation. **a** shows an example from group P in which the proximal aorta was crossclamped for 30 minutes. Within minutes, PO_2 fell to undetectable levels, and the changes in the metabolic variables pH and PCO_2 were pronounced. The reverse blood flow in segmental arteries with unimpeded runoff to the distal aorta explains why ischemia develops in the spinal cord. The result of double-aortic crossclamping (group D) is seen in **b**. PO_2 was reduced to an intermediate level, and the changes in pH and PCO_2 developed slowly and never reached convincingly dysoxic levels. The explanation for this is that steal circulation is prevented in the excluded thoracoabdominal aortic segment, which makes the proximal collateral circulation more effective. The importance of the distal clamp location is demonstrated from a separate experiment in **c**. *Double arrows* indicate that the distal second clamp (with a time interval of 5 minutes) was placed 2 segments above the celiac trunk. This increases PO_2 to some extent, but when the clamp is moved close to the celiac trunk 5 minutes later (*triple arrow*), PO_2 improves further as a result of prevention of steal in important segmental arteries (Adamkiewicz artery). **d** shows the effect of combined clamping (double aortic and both subclavian arteries, group DS). Although aortic steal was partly prevented, this combination of aortic clamping and interruption of both the subclavian-intercostal and vertebral collateral circulation creates ischemia comparable with that of the group with only proximal aortic crossclamping.

clavian and vertebral artery in one case. The pertinent topographic anatomy regarding the proximal collateral circulation is shown in Fig 5.

Discussion

For better understanding of the pathophysiology of spinal cord ischemia, reliable and reproducible experi-

mental and clinical monitoring techniques are needed. One problem with many of the existing aortic clamping models is that the ischemia is not selective for the spinal cord nor is it possible to study reperfusion without confounding systemic changes. Aortic crossclamping also induces hemodynamic changes and necessitates vasoactive interventions, adding further confusion

Fig 3. Two-dimensional image and Doppler ultrasonography. *Arrows* indicate aortic lumen and the proximal part of the largest segmental artery close to the celiac trunk. The direction of intercostal blood flow is shown in relation to proximal aortic crossclamping.

to the study of spinal cord ischemia. Another problem is how to evaluate the relevance of experimental models for human conditions. This requires well-founded data on comparative anatomy and physiology. For the interpretation of the present study, an outline of the spinal cord circulation and some comparative aspects is warranted.

Vascular anatomy of the human spinal cord. In the late nineteenth century, Adamkiewicz⁵ drew attention to the vascular anatomy of the human spinal cord. He stated that neither was there a symmetrical bilateral distribution of intercostal arteries nor did the existing segmental arteries give off radicular anastomoses to the anterior spinal artery at all levels. The arterialization of the spinal cord and the advancement of knowledge over time have been reviewed by Luyendijk.⁶

The spinal cord receives its blood supply from an anterior and 2 posterior longitudinal arteries with bidi-

rectional flow capacities. Abundant anastomoses exist in the circumference, although not intraspinally. This longitudinal system is in turn supplied by a limited number of radicular arteries formed by subclavian vertebral and segmental aortic branches. The anterior spinal artery supplies the anterolateral columns and the central gray matter of the spinal cord. The artery runs continuously but with varying caliber in its longitudinal orientation. There are also great variations in its feeding arteries at different spinal levels, both between individuals and between species. At the cervical level, the spinal cord has a relatively rich blood supply from the vertebral and subclavian arteries. Of the many segmental arteries originating from the aorta, only a few give rise to medullary feeding radicular arteries. In two thirds of cases, the most prominent one (the Adamkiewicz artery) is branched off from a left-sided segmental artery of the T8-L2 level. When its origin is

Fig 4. Paratrend 7 recordings and trend printouts (examples from groups I and IS). Arrows indicate the start of clamping (1 kPa = 7.5 mm Hg). Trend registrations of 30 minutes (2 hours in *d*) show changes in intrathecal pH, PCO₂, and PO₂ in response to interruption of segmental arteries and other manipulations of the spinal cord circulation. On clamping or ligation of all segmental arteries below T5 (group I), we could not demonstrate any significant changes in the intrathecal variables, as shown in *a*. Maintained proximal pressure assures adequate collateral circulation through the subclavian-intercostal and vertebral pathways. *b* shows an example from group IS (IS:1), in which adding selective proximal intercostal clamping creates ischemia as a result of insufficient vertebral feeding of the longitudinal spinal arteries. In *c* another example of the same group (IS:4) shows only a 50% reduction in PO₂, with no change in the metabolic parameters. The interpretation of this is that the remaining collateral circulation is sufficient to prevent ischemia in this case. This is confirmed in the same experiment, as shown by the 2-hour trend in *d*. After the 90-minute experiment, the selective clamps on the supreme intercostal arteries were moved to the subclavian arteries (double arrow), whereby the interruption of the remaining collateral circulation resulted in ischemia.

lumbar, an additional artery is often found in the midthoracic portion. At the upper thoracic level, a narrow radicular artery is generally present. In angiographic studies Djindjian⁷ was able to localize the main radicular feeding artery in the T9-T12 region, with about 75% of them originating from left-sided intercostal arteries.

Comprehensive studies of the human spinal cord blood supply were presented by Pisco.⁸ He noted the number of radicular arteries to be 2 to 3 at the cervical, 2 to 3 at the thoracic, and 0 to 1 at the lumbar level. The

largest feeding artery was found at T9, T10, or L1, with 80% of left-sided origin. Two consistent areas of vascular distribution were identified, the lower cervical and the thoracolumbar transitional zone. Other areas showed great variability but with left-sided dominance (except for the cervical region). Most studies of the distribution and calibers of the spinal arteries referred to anatomic preparations. How they correlate to the functional aspects of the dynamic and richly anastomized vascularization of the spinal cord has not yet been fully elucidated.

Fig 5. Topographic anatomy of supreme intercostal arteries.

Some of the established views about the spinal cord blood supply have been questioned. Svensson and colleagues⁹ studied functional aspects of the spinal cord circulation in baboons by measuring spinal cord blood flow (microspheres) at different levels in relation to proximal aortic clamping and the use of a shunt. Without a shunt, the lumbar spinal cord is the area most sensitive to ischemia because the perfusion is dependent on proximal feeding. A distal shunt was shown to increase lumbar spinal cord blood flow more than at the thoracic level. The explanation is the difference between the diameter of the anterior spinal artery above and below the entry of the *arteria radicularis magna*. The higher resistance to flow in the narrow ascending anterior spinal artery favors distal flow.

McCormick and Stein¹⁰ reviewed the functional anatomy of the spinal cord. They stated that the anterior spinal artery extends the entire length of the spinal cord, but it may appear discontinuous in the midthoracic region because of variation in the diameter. He considered the longitudinal system a functional analogue to the intracranial circle of Willis because it allows continuity and reversal of flow.

On the basis of spinal cord blood flow measurements (microspheres) during aortic occlusion, Kaplan and colleagues¹¹ questioned the existence of an "anterior spinal artery syndrome." They could not verify the "anterior two thirds" distribution of ischemic morphologic changes in the spinal cord. In a radiologic study of 24 patients with spinal cord ischemia after vascular surgery, Mawad and colleagues¹² used magnetic resonance tomography to characterize patterns of signal abnormalities. All abnormalities were found to be in the low thoracic cord and distal of it. In postoperative complete spinal cord stroke, the whole cross-section of the cord was infarcted.

Comparative vascular anatomy. Wissdorf¹³ also discussed the left-sided dominance among other species and the predominant lumbar localization of the main radicular artery in many animals in comparison with the low thoracic origin in human subjects and primates. Dapunt and colleagues¹⁴ examined the importance of retrograde steal circulation versus antegrade selective perfusion of segmental arteries in pigs. Changes in latency and total amplitude of somatosensory evoked potentials were recorded. In surviving animals the neurologic outcome was documented on the third day. Simple aortic crossclamping for 1 hour produced paraplegia in all animals of that group. The best results were recorded in a group in which the thoracic segment was perfused and the crucial arteries preserved. Venting of the same segment was detrimental because of the steal phenomenon. The authors argued that the spinal cord blood supply is plurisegmental in pigs, whereas the *arteria radicularis magna* plays a relatively less-important role. The most important arteries were found in the caudal thoracic aorta. He even questioned the existence of an Adamkiewicz artery in pigs.

Regarding the collateral circulation that feeds the proximal longitudinal system, some comparative aspects deserve notice. In human subjects only the first 1 or 2 intercostal arteries originate from the main brachiocephalic branches. The subclavian arteries give off supreme intercostal arteries (T1-T2 through the costo-cervical trunks) distal to the vertebral arteries. The vascular distribution in this area is generally considered less important than that in the midthoracic portion in human subjects, as mentioned by Tveten.^{15,16} Lazorthes and his group¹⁷ depicted the midthoracic zone (T4-T8) as the critical one because of the poor vascularization and scarcity of anastomoses compared with the cervicothoracic and thoracolumbar areas.

Table II. Intrathecal data

Measuring point	Paratrend		
	pH	PCO ₂ *	PO ₂ *
Groups P, D, and DS			
Baseline			
Group P, n = 6	7.26 ± 0.06	7.6 ± 0.5	5.6 ± 0.9
Group D, n = 4	7.25 ± 0.08	7.9 ± 1.0	5.1 ± 0.5
Group DS, n = 6	7.20 ± 0.06	7.7 ± 1.1	5.8 ± 1.2
Clamping, 30 min			
Group P	6.81 ± 0.08	16.5 ± 2.0	0.1 ± 0.2
Group D	7.23 ± 0.07	7.9 ± 1.4	2.5 ± 1.4
Group DS	6.72 ± 0.15	16.4 ± 2.6	0.2 ± 0.3
Declamping, 30 min			
Group P	7.14 ± 0.06	8.1 ± 1.3	6.6 ± 1.3
Group D	7.22 ± 0.06	8.1 ± 1.9	7.0 ± 1.9
Group DS, n = 4	6.97 ± 0.10	8.7 ± 1.6	8.1 ± 3.0
Declamping, 60 min			
Group P	7.20 ± 0.04	7.8 ± 1.1	5.8 ± 1.2
Group D	7.22 ± 0.03	7.9 ± 1.6	5.9 ± 1.8
Group DS, n = 4	7.06 ± 0.07	7.6 ± 1.1	7.2 ± 2.5
Groups I and IS			
Baseline			
Group I, n = 4	7.25 ± 0.05	7.2 ± 1.0	6.9 ± 0.9
IS:1	7.12	9.5	5.2
IS:2	7.26	7.8	4.8
IS:3	7.32	6.8	7.9
IS:4	7.32	6.4	7.2
30 min			
Group I	7.25 ± 0.06	7.3 ± 1.1	7.2 ± 1.6
IS:1	6.95	15.4	0
IS:2	7.23	8	2.5
IS:3	6.91	14.9	1.2
IS:4	7.29	6.5	3.7
60 min			
Group I	7.26 ± 0.06	7.1 ± 0.9	8.0 ± 1.8
IS:1	6.87	18.7	0
IS:2	7.17	8.7	2.4
IS:3	6.74	18.1	0.9
IS:4	7.22	7.1	3.6
90 min			
Group I	7.28 ± 0.05	6.8 ± 0.8	8.3 ± 2.7
IS:1	6.85	19.9	0
IS:2	7.18	8.2	3.2
IS:3	6.71	17.4	1.5
IS:4	7.17	7.3	3.6

Values are given as means ± SD or individual data as indicated.

*These values are given as kilopascals; 1 kPa = 7.5 mm Hg.

They argued that there is no continuity in this middle area of the longitudinal system in human subjects. Svensson and colleagues,⁹ on the other hand, stated that the anterior spinal artery is continuous in both human subjects and primates. Dommissie¹⁸ pointed out that the anterior longitudinal arterial system is crucial rather than any single medullary feeder and that preservation of such an artery will not ensure satisfactory cir-

culation. Jellinger¹⁹ claimed the upper thoracic segments to be the sensible border zone between the main subclavian-vertebral artery and aortic sources of spinal cord blood supply. In many animals used in experimental studies, the proximal source can be even more important. In dogs, cats, and rabbits the dorsal intercostal arteries T1-T3 have a proximal origin. In pigs the subclavian-vertebral contribution to the segmental intercostal circulation is extended to T4 or even T5. This is well documented in the extensive works by Wissdorf¹³ and Ghoshal.²⁰ The supreme intercostal artery divides into T3-T5 and is a branch from the right subclavian artery and left costocervical trunk, respectively. Furthermore, there are side differences in the anatomy of T1 and T2, but they are branched off separately from the base of the subclavian or the vertebral arteries.

Discussion of results. On the basis of the outline above, we will discuss the interpretation of our results for each experimental group separately. The present study demonstrated that single thoracic aortic cross-clamping in pigs (group P) caused a rapid decrease of intrathecal PO₂ to undetectable levels. Wadough and colleagues²¹ reported similar findings when measuring spinal cord surface PO₂. Free runoff to the distal aorta caused a steal phenomenon, which made the collateral circulation insufficient. The explanation offered was that blood tended to drain away from the spinal cord rather than to supply it longitudinally. We used spectral Doppler analysis to document the reversal of flow through the intercostal arteries after application of the proximal clamp. Angiographic studies by DiChiro and colleagues^{22,23} have demonstrated both ascending and descending blood flow currents in different sections of the longitudinal arterial system of the spinal cord. They could also show reversal of blood flow in pathologic conditions (obstructions or change in pressure gradients) as an explanation for the steal phenomenon.

In double clamping (group D) there was a decrease of approximately 50% in intrathecal PO₂. This difference, in comparison with that of proximal clamping, can be explained by the prevention of steal circulation through the intercostal arteries after application of the distal aortic clamp. The moderate decrease of CSF PO₂ in this group might be explained by remaining distal aortic steal through the lumbar arteries. It is important to note that CSF PCO₂ and pH changes did not indicate ischemia. In further studies of the mechanism of spinal cord injury, Wadough and his group²⁴ recorded pressures at various levels in the aorta, as well as in intercostal and lumbar arteries. They found that exclusion of the thoracic aorta by double clamping restored intercostal

vascular bed pressure almost to control level. In a recent study of a double-clamp model, Ishizaki and colleagues²⁵ could successfully identify spinal cord feeding arteries by using intrathecal oxygen monitoring. Perfusion of these arteries with arterial blood improved intrathecal PO_2 and spinal evoked potentials, which had been significantly depressed by ischemia.

Group DS was designed to create complete spinal cord ischemia without the need for dissection of the segmental arteries. It was possible to exclude all proximal collateral circulation by means of double subclavian and aortic clamping. The hemodynamic consequences of this were deleterious. In combination with aortic shunting, this model will make studies of selective spinal cord ischemia and reperfusion possible without deleterious systemic effects.²⁶

Interruption of the segmental arteries (group I) did not reveal any significant changes of the CSF oxygenation, possibly because of well-developed collateral circulation from intracranial and vertebral arteries and the subclavian-intercostal pathways. In a study of 16 patients, Bassett and colleagues²⁷ tried to identify crucial radicular arteries before spinal surgery. The effect of temporary occlusion of 9 segmental arteries in half of the patients was evaluated by means of somatosensory evoked potentials. No sign of ischemia was recorded. The anterior spinal artery was in continuity from the upper thoracic spine to the conus medullaris in all patients. Griep and colleagues²⁸ recently questioned the importance of the localized segmental blood supply of the spinal cord. In their series no patients with fewer than 10 severed segmental arteries had postoperative paraplegia. In some patients spinal cord ischemia could be reversed by adjunctive measures, such as distal perfusion, and maintenance of high-to-normal blood pressures.

Maliszewski and colleagues²⁹ performed postmortem studies of 50 deceased subjects with no symptoms of spinal cord disease. Occluded anterior radicular arteries were found in 4 cases. They concluded that at least gradually occurring occlusions could be compensated for by the development of an efficient collateral circulation. In another postmortem study, Koshino and colleagues³⁰ could not demonstrate any significant correlation between the diameter of the main radicular artery and that of the intercostal or lumbar arteries. In the commentary on the same article, Svensson states that this lack of correlation also exists for the size of segmental arterial ostia or the amount of backflow from the ostia during aortic surgery. In concordance with Griep and colleagues,²⁸ Svensson³¹ argues that reimplantation of segmental arteries in the proximal and

midthoracic aorta is not critical. However, with more extensive aortic repairs, the risk of spinal cord injury increases with the number of sacrificed segmental arteries. The same group³² demonstrated a strong correlation between the incidence of neurologic deficits and the number and level of oversewn patent arteries in a series of 99 patients. They concluded that every effort should be made to reattach distal intercostal arteries and that intraoperative atriofemoral bypass appeared protective. The problem remains of how to best decide which artery is crucial and how to perform reimplantation without early thrombotic occlusion.

In group IS the proximal (subclavian) intercostal blood supply was selectively clamped in addition to interruption of all segmental arteries. The internal thoracic arteries were also ligated to exclude the possibility of collateral circulation between the anterior and posterior intercostal arteries (T3-T7). This left the second intercostal arteries (branches of vertebral or dorsal scapular arteries) as the only remaining intercostal vessels. The sufficiency of the vertebral and intracranial feeding of the anterior spinal artery was decisive for the degree of ischemia as shown in Fig 4, *b* through *d*. In similar experiments in dogs, Spencer and Zimmerman³³ produced paraplegia by interrupting all segmental arteries and clamping the left subclavian artery. Luosto³⁴ noted increasing incidence of paraparesis by consecutively adding subclavian artery clamping to interruption of intercostal arteries.

We have previously shown²⁶ that selective ischemia and reperfusion of the spinal cord without systemic changes can be studied in a pig model that combines double aortic crossclamping with right-sided subclavian occlusion and a shunt from the left subclavian artery to the distal aorta. In this study we demonstrate that interruption of all segmental arteries and proximal collateral pathways without aortic clamping also offers a hemodynamically stable model for the study of selective spinal cord ischemia. The critical level of intrathecal PO_2 is dependent on the duration of an ischemic injury to the spinal cord at a given spinal cord temperature. The presented model (group IS) would enable such studies of ischemia with various durations. Additional variables, such as CSF PCO_2 and pH, might indicate the anaerobic threshold, where dysoxia with irreversible cellular damage to the spinal cord threatens to develop.

The presented technique for continuous CSF monitoring might provide better understanding of the mechanism of spinal cord ischemia during aortic crossclamping. When interpreting experimental studies, we must recognize the comparative aspects of

vascular anatomy regarding the differences in collateral circulation.

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