Near-infrared Spectroscopy Monitoring of the Collateral Network Prior to, During, and After Thoracoabdominal Aortic Repair: A Pilot Study

C.D. Etz [a,b,*,d], K. von Aspern [a,d], S. Gudehus [c], M. Luehr [a], F.F. Girrbach [a], J. Ender [c], M. Borger [a], F.W. Mohr [a]

[a] Department of Cardiac Surgery, University of Leipzig, Heart Center Leipzig, Saxony, Germany
[b] Department of Cardiothoracic Surgery, Mount Sinai School of Medicine, New York, NY, USA
[c] Department of Anesthesiology, University of Leipzig, Heart Center Leipzig, Saxony, Germany

WHAT THIS PAPER ADDS
This study evaluates, for the first, time non-invasive monitoring of the collateral network oxygenation by means of near-infrared spectroscopy prior to, during, and after thoracoabdominal aortic repair in a clinical series. Although shown to be a feasible monitoring method for routine utilization in a clinical setting, specific studies are needed before it can be used to facilitate guidance in peri- and intraoperative perfusion management.

Objective: The aim of this study was to evaluate the feasibility of non-invasive monitoring of the paraspinous collateral network (CN) oxygenation prior to, during, and after thoracoabdominal aortic repair in a clinical series.
Methods: Near-infrared spectroscopy optodes were positioned bilaterally—over the thoracic and lumbar paraspinous vasculature—to transcutaneously monitor muscle oxygenation of the CN in 20 patients (age: 66 ± 10 years; men $= 11$) between September 2010 and April 2012; 15 had open thoracoabdominal aortic repair (Crawford II and III), three had thoracic endovascular aortic repair (TEVAR; Crawford I), and two had a hybrid repair (Crawford II). CN oxygenation was continuously recorded until 48 hours postoperatively.
Results: Hospital mortality was 5% ($n = 1$), 15% suffered ischemic spinal cord injury (SCI). Mean thoracic CN oxygenation saturation was $75.5 \pm 8\%$ prior to anesthesia ($=$baseline) without significant variations throughout the procedure (during non-pulsatile cooling on cardiopulmonary bypass and with aortic cross-clamping; range $= 70.6-79.5\%$). Lumbar CN oxygenation (LbS) dropped significantly after proximal aortic cross-clamping to a minimum after 11.7 ± 4 minutes ($74 \pm 13\%$ of baseline), but fully recovered after restoration of pulsatile flow to 98.5% of baseline. During TEVAR, stent-graft deployment did not significantly affect LbS. Three patients developed relevant SCI (paraplegia $n = 1$/paraparesis $n = 2$). In these patients LbS reduction after aortic cross-clamping was significantly lower compared with patients who did not experience SCI ($p = .041$).
Conclusions: Non-invasive monitoring of CN oxygenation prior to, during, and after thoracoabdominal aortic repair is feasible. Lumbar CN oxygenation levels directly respond to compromise of aortic blood circulation.
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INTRODUCTION

Paraplegia remains the most devastating complication after repair of extensive descending thoracic aneurysms (DTA) and thoracoabdominal aortic aneurysms (TAAA).[1] The maintenance of adequate spinal cord oxygenation is critical to the success of open and endovascular repair of TAAAs to prevent spinal cord ischemia when blood flow to the spinal cord is impaired (e.g., by segmental artery occlusion or aortic cross-clamping). Monitoring of spinal cord function using motor evoked potentials (MEP) or somatosensory evoked potentials (SSEP) is widely accepted in the assessment of intraoperative spinal cord viability during aortic procedures, but it requires significant technical effort and invasiveness.[2,3] It has been shown that paraplegia may be reduced by sustaining a *supranormal* mean arterial pressure peri-operatively and by routine use of cerebral fluid (CSF) drainage.[4–7] Despite these and other strategies and technical improvements spinal cord ischemic injury remains significant with an incidence of 5–11% in contemporary series.[8]

The recent introduction of "the collateral network (CN) concept" by Etz et al.[9]—based on experimental and clinical data—demonstrates that blood supply to the spinal cord is provided by a rich network of paraspinous arterial collaterals enabling sufficient blood flow in chronic ischemia or acutely after extensive sacrifice of segmental arteries.

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[d] These authors contributed equally to this study.

* Corresponding author. C.D. Etz, Heart Center Leipzig, Department of Cardiac Surgery, Struempellstrasse 39, 04289 Leipzig, Germany.
E-mail address: christian.etz@med.uni-leipzig.de (C.D. Etz).

Monitoring of spinal cord integrity, however, remains challenging and difficult to interpret. According to the CN concept, blood supply to the paraspinous vasculature correlates with spinal cord blood supply. Critical spinal cord ischemia can occur not only during extensive thoracoabdominal aortic repair, but can also be delayed owing to inadequate postoperative spinal cord perfusion.[10,11] It is therefore of great importance to measure the adequacy of spinal cord blood supply intra- and postoperatively.

We theorize that oxygenation of the paraspinous CN—which is fed by the segmental arteries, as well as the spinal cord—may directly correlate with spinal cord blood supply.

Near-infrared spectroscopy has been shown to effectively monitor cerebral oxygen saturation during cardiopulmonary bypass and selective cerebral perfusion.[12–15] Analogous to this, we used conventional near-infrared spectroscopy (NIRS) optodes to monitor tissue oxygenation of the thoracic and lumbar paraspinous muscles—hence the paraspinous CN—to provide real-time, non-invasive spinal cord monitoring, potentially indicating pending spinal cord ischemia.

With the results of this study we prove feasibility of this new non-invasive monitoring tool in clinical practice.

MATERIALS AND METHODS

Between September 2010 and April 2012 a total of 73 patients was treated for thoracoabdominal aortic pathologies (32 open aortic repair and 41 stent interventions). Twenty patients were included in this study (age: 66 ± 10 years; men = 11): fifteen who received open thoracic or thoracoabdominal aortic (TAAA) repair (Crawford II + III), three thoracic endovascular aortic repair (TEVAR) (Crawford I), and two who received hybrid repair (second stage, Crawford II). All patients were enrolled non-consecutively depending on the presence of trained research personnel for documentation on site. Each enrolled patient is reported on; no patient was excluded in the course of this study. During open TAAA repair cooling to a mean rectal temperature of 31 ± 3 °C (median: 32 °C) was performed. In Crawford II aneurysms, visceral perfusion via a balloon catheter was utilized after distal aortic clamping. CSF drainage was only used during and after open TAAA repair to maintain an intrathecal pressure <12 cmH₂O or below opening pressure. The CSF catheter was removed 72 hours postoperatively.

NIRS optodes were positioned bilaterally—above the upper thoracic (T5—T7) and lumbar (L1—L3) paraspinous vasculature of the CN (Fig. 1, left)—for non-invasive transcutaneous monitoring of regional tissue (muscle) oxygen saturation of haemoglobin, as indicated on the NIRS device interface (Fig. 1, upper right). CN oxygenation was continuously recorded—prior to, during, and after—TAAA repair until 48 hours postoperatively (Fig. 2). Arterial blood pressure was measured at all times invasively via a catheter placed in the radial and femoral artery. Arterial blood pressure is expressed in mmHg, oxygenation saturation in percent calculated as the mean of left and right optode measurements, respectively. Data of four patients—all of whom were from the open TAAA repair group—were of limited use for postoperative analysis, as acquisition could not be continued beyond 5 hours (all acquired data up to this point was equally included in the analyses). One of these patients needed to be manually resuscitated intraoperatively, after compromise of the right coronary artery during heart luxation due to a distinct pectus excavatum. In this patient, both optodes measuring thoracic and one optode measuring the lumbar CN oxygenation dislocated, and thoracic CN oxygenation saturation (ThS) was not available throughout the procedure. Mean calculations at the start of distal perfusion include measurements of the 12 patients where distal perfusion was performed (see Table 1). Statistical analysis of NIRS measurements and blood pressure for patients undergoing TEVAR was performed and reported on separately. As NIRS for CN monitoring has only recently been adopted into clinical practice for TAAA repair at our institution and is still in preliminary testing, measurements were only analyzed retrospectively, without decisive influence on intra- or postoperative management. Demographic data and procedural details of all patients and subgroups are listed in Table 1.
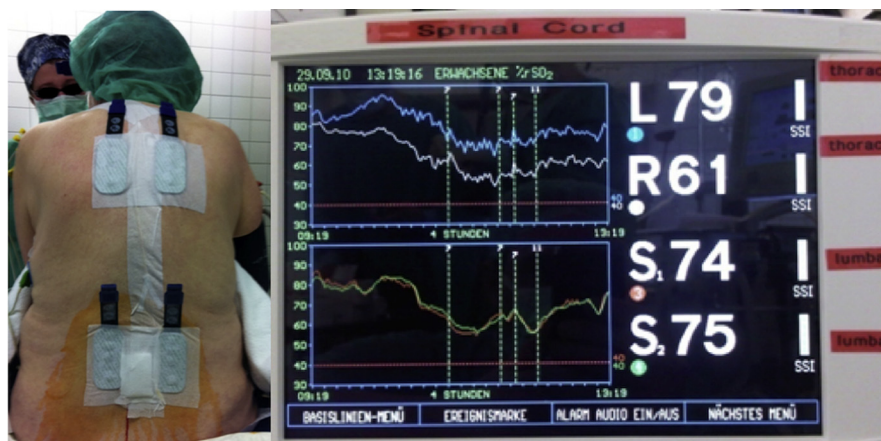

Figure 1. Positioning of the thoracic and lumbar near-infrared spectroscopy (NIRS) optodes (left); real-time NIRS monitor (right).
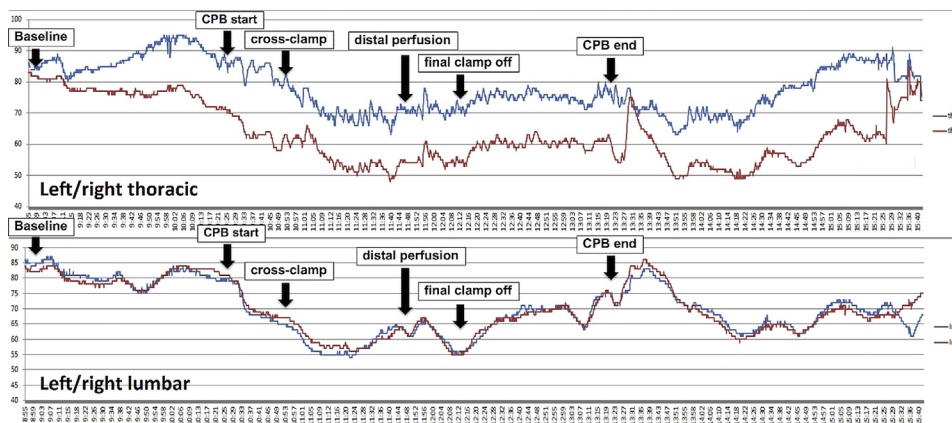
Figure 2. Monitor excerpt of real-time near-infrared spectroscopy (NIRS) measurements over 7 hours showing the thoracic (ThS, top) and lumbar (LbS, bottom) collateral network oxygenation. *Note.* CPB = cardiopulmonary bypass.

Data were imported to SPSS (version 17.0; SPSS, Chicago, IL, USA) for description and analysis. All tests were performed as two-sided at a significance level of 5%. Mean differences were demonstrated using the Wilcoxon signed-rank test and chi-square test as appropriate.

RESULTS

All patients survived the initial operation, but one patient suffered from refractory low cardiac output failure and died as a result of multi-organ failure after having already been manually resuscitated intraoperatively. Hospital mortality was 5% (n = 1). The incidence of permanent paraplegia (n = 2) or paraparesis (n = 1)—indicating severe intraoperative or early postoperative spinal cord ischemia—was 15% including all patients.

Patients with open and hybrid repair

Paraspinous NIRS optodes measured a mean ThS of 75.5 ± 8% prior to anesthesia (range: 60.0–88.5%; median: 77.5%), which was individually set as a reference baseline in each patient (=100%). Throughout the entire operation, ThS remained steady without significant variations (p = .1–9). From the introduction of anesthesia until non-pulsatile cooling on cardiopulmonary bypass (CPB) ThS gradually increased to a mean of 109.5 ± 10% of baseline (range: 92.9–125.4%; median: 105.9%; p = 112), dropping at aortic

Table 1. Patient demographics and procedural details.

Variable	All	TAAA	TEVAR	Hybrid
	n	n	n	n
Patients	20	15	3	2
Male gender	11	7	2	2
Age (y ± SD)	66 ± 10	68 ± 11	57 ± 8	56 ± 5
Connective tissue disease	1	1	0	0
Diabetes (IDDM)	1	1	0	0
Chronic obstructive pulmonary disease	3	2	0	1
Hypertension	14	9	3	2
Coronary artery disease	6	5	1	0
Previous aortic surgery	7	6	1	0
Aneurysm diameter (mm ± SD)	66.1 ± 9	67.1 ± 8	59.7 ± 3	70.0 ± 14
Crawford I	8	5	3	0
Crawford II	10	8	0	2
Crawford III	2	2	0	0
Type B dissection	8	5	3	0
CPB time (min ± SD)	127.9 ± 61	148.8 ± 40	0	207.5 ± 77
Cross-clamp time (min ± SD)	31.9 ± 18	35.1 ± 15	0	64.5 ± 40
Lowest body temperature (°C ± SD)	32.1 ± 2	31.7 ± 2	36.4 ± 1	25.5 ± 1
Distal perfusion	12	11	0	1
Tracheotomy	4	4	0	0
Paraplegia	1	0	0	1
Paraparesis	2	2	0	0
Died	1	1	0	0

Note. IDDM = insulin-dependent diabetes mellitus; CPB = cardiopulmonary bypass; TAAA = thoracoabdominal aneurysm repair, TEVAR = thoracic endovascular aortic repair.

cross-clamping to 99.4 ± 13% of baseline (range: 79.1–126.2%; median: 97.5%; $p = .443$), remaining at 97.7 ± 10% (range: 81.0–116.9%; median: 97.7%), and returning to 101.2 ± 10% of baseline (range: 82.2–116.6%; median: 103.2%; $p = .638$) after releasing the aortic cross clamp (partial pulsatility). After re-establishment of full pulsatility (with the end of cardiopulmonary bypass) a reduction in ThS to 97.4 ± 11% of baseline occurred (range: 82.1–116.2%; median: 98.0%; $p = .308$).

The individual mean lumbar CN oxygenation (LbS) was 79.2 ± 9% prior to anesthesia (range: 63.5–94.5%; median: 75.5%) and was set as the reference baseline (=100%). Analogous to ThS, an initial increase in LbS to 107.8 ± 10% of baseline (range: 95.1–124.8%; median: 104.9%; $p = .117$) occurred from anesthesia introduction until cooling on CPB. LbS showed steady saturation levels only during non-pulsatile cooling on CPB, significantly dropping after proximal aortic cross-clamping to a minimum after 11.7 ± 4 minutes (74.1 ± 15% of baseline; range: 52.1–100.5%; median: 68.1%; $p = .002$).

The start of distal perfusion raised lumbar CN oxygenation to 80.9 ± 16% of baseline (range: 58.3–103.6%; median: 81.7%; $p = .046$). Releasing the aortic cross clamp further raised LbS to 93.9 ± 12% (range: 73.5–113.5%; median: 95.4%; $p = .158$). Complete pulsatility fully restored lumbar CN oxygenation after an overall delay of 12.7 ± 4 minutes (98.5% ± 8%; range: 92.4–116.7%; median: 97.2%; $p = .388$).

The mean radial artery blood pressure—representing the perfusion pressure to the upper body—demonstrated no significant fluctuations during the operation (range of mean: 64.8 ± 5–87.6 ± 6 mmHg, $p = .285–.834$). In contrast, the mean femoral arterial blood pressure (fMAP; range of mean: 48.2 ± 14–84.9 ± 8 mmHg) dropped significantly after aortic cross-clamping compared with baseline levels (before clamping = 93.1 ± 14%; after clamping = 58.7 ± 27%; $p = .003$) and returning to 94.9 ± 17% after restoration of pulsatile flow ($p = .289$).

Postoperatively, ThS and LbS were not statistically different from baseline levels with 97.7 ± 11% (range: 89.7–106.1%; median: 99.5%) and 99.1 ± 10% (range: 94.2–108.6%; median: 98.9%) at 48 hours, respectively ($p = .575$ and $p = .889$). Mean ThS and LbS for all patients ($n = 17$, TEVAR excluded) are illustrated in Fig. 3.

One patient presented with a short drop in ThS and LbS (5 minutes) after experiencing a seizure 24 hours postoperatively (ThS = 80.0%; LbS = 80.7%). Oxygenation levels returned to baseline levels after successful treatment and seizure termination.

The patient who had required CPR intraoperatively owing to heart failure died 72 hours postoperatively from multiorgan failure despite all measures, including extracorporeal membrane oxygenation. The patient never regained consciousness postoperatively; therefore, no conclusive statement on motor function can be made.

Comparison of ThS and LbS with regard to their baseline levels throughout the procedure showed a significant reduction after aortic cross-clamping for LbS only (Fig. 3),
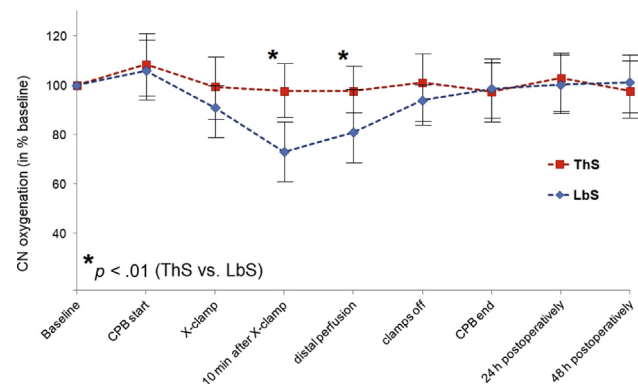

Figure 3. Mean thoracic (ThS) and lumbar (LbS) collateral network (CN) oxygenation levels of all patients prior to, during, and after thoracoabdominal aortic aneurysm (TAAA) repair (from left to right). *Note.* CPB = cardiopulmonary bypass; X-clamp = proximal aortic cross-clamping.

rising with the initiation of distal perfusion until complete restoration after the re-establishment of pulsatile flow (mean difference baseline to cross-clamp: ThS = 1.7 ± 7%; $p = .433$ vs. LbS = 20.6 ± 13%; $p = .002$).

As measurements of ThS and LbS have been performed laterally on both sides of the vertebrae, we analyzed saturation levels for potential differences. No statistically significant difference was evident between ThS and LbS ($p = .249–1.000$).

Comparing patients who developed paraplegia/paraparesis with those who did not showed no significant differences in ThS throughout the procedure ($p = .195–1.000$). The difference of LbS between the two groups, however, approached statistical significance 10 minutes after aortic cross-clamping until the end of cardiopulmonary bypass. At the time of aortic cross-clamping LbS of patients who developed paraplegia/paraparesis as opposed to those who did not was 80.1 ± 12.0% (range: 58.0–102.2%; median: 97.1%) versus 92.8 ± 9.0% (range: 76.9–119.8%; median: 90.1%) of baseline, respectively ($p = .539$), decreasing to 65.6 ± 8% (range: 63.1–80.5%; median: 67.4%) versus 76.2 ± 7.0% (range: 60.9–99.3; median: 69.3%) of baseline after 10 minutes ($p = .073$), and reaching the most distinct difference just before distal perfusion initiation with 58.3 ± 15% (range: 56.2–81.2, median: 58.3%) versus 85.2 ± 10% (range: 69.4–103.5%, median: 89.7%) of baseline ($p = .041$). In these patients LbS was below 75% of-baseline for 15 ± 9 minutes (range 6.8–26.1; median: 13.2 minutes). Beyond that point no statistically significant differences between LbS in patients with and without paraplegia/paraparesis were evident ($p = .371–.859$).

Patients undergoing TEVAR

In patients undergoing TEVAR, significant changes in ThS or LbS were not detected despite extended stent-graft coverage of the descending and thoracoabdominal aorta: one patient was treated with four size-progressive stents (diameter: 24–38 mm) covering the descending/abdominal

aorta from the subclavian artery downwards to the mesenteric artery; one patient received two stents (diameters: 26 and 28 mm) ranging from the descending thoracic aorta to the mesenteric artery; and one patient's descending thoracic aorta was stented directly distal to the subclavian artery (diameter: 38 mm). Short periods of reduced fMAP after stent deployment occurred in the two patients with abdominal aortic involvement. Following stent deployment mean LbS only dropped by 2.6 ± 10% without reaching statistical significance ($p = .681$; mean difference of fMAP before/after stent deployment: 7.0 ± 3, $p = .048$).

DISCUSSION

Paraplegia remains the most devastating complication after open or endovascular repair of the thoracic and thoracoabdominal aorta. It has been shown that intraoperative or early postoperative (intermediate-delayed <48 h) spinal cord ischemia is likely responsible for at least 60% of cases of paraplegia/paraparesis after TAAA surgery.[4] Therefore, *supranormal* mean arterial pressure is now considered mandatory to effectively reduce the incidence of postoperative paraplegia. However, the pathology of early and delayed postoperative paraplegia after aortic surgery is still not fully understood, as several factors seem to contribute to the development of spinal cord ischemia.

A reliable non-invasive methodology to clinically detect peri-operative spinal cord ischemia is not available. The use of MEP and SSEP monitoring is timely, cost intensive, and demands adequate interpretation skills.[2,3]

Postoperative management on the intensive care unit focuses mainly on (1) stable—*supranormal*—mean arterial pressures and (2) a decrease of intrathecal pressure via a routinely placed CSF drainage to ensure adequate spinal perfusion via the collateral network despite lower postoperative perfusion pressures.[16]

Monitoring of the spinal cord blood supply remains a difficult, but extremely important, in eliminating paraplegia. Non-invasive direct monitoring of spinal cord tissue oxygenation may not currently be possible owing to technical limitations. Although a few clinical reports on cases utilizing NIRS for direct monitoring of spinal tissue oxygenation have been published,[17,18] the disadvantageous neuronal tissue to bone ratio in humans may restrict its clinical or experimental interpretation. Direct pressure measurement in critical segmental arteries supplying the cord is technically feasible, but has not yet been clinically evaluated.[19]

Thoracic paraspinous CN oxygenation

The finding that the thoracic paraspinous CN muscle oxygenation does not significantly drop during aortic cross-clamping and stent-graft deployment correlates with previous experimental results and may reflect on the extensive arterial collateralization supporting thoracic spinal cord perfusion via the vertebral and intercostal arteries, and, to some extent, via the upper thoracic segmental arteries originating from the subclavian artery.[5] The clinical experience that open TAA repair, despite extensive segmental

artery sacrifice from Th4 to Th12 is safe, and the fact that endovascular stent-graft deployment for the treatment of descending pathologies are both associated with a paraplegia rate of 1—3% in experienced aortic centers, supports this result.[6,7]

Lumbar paraspinous CN oxygenation

The NIRS response on distal perfusion diminution—at a time when the thoracic optodes do not detect a significant saturation drop—may reflect the limited collateral backup of the lumbar region of the paraspinous arterial network, while thoracic CN blood supply is still maintained. Monitoring the lumbar paraspinous vasculature therefore appears most important.

Interestingly, both LbS and ThS increase from introduction of anesthesia to cooling on CPB despite of a drop in mean arterial blood pressure. This finding can best be explained by the surplus in the oxygenation—pressure/oxygenation—saturation ratio during the introduction period coupled with the vasodilatative effects of the intravenous medication.[20]

The delay at which the NIRS detection reacts to cross-clamping, while mean arterial blood pressure already dropped significantly, might reflect the presence of a vast CN serving as a blood reservoir averting pending ischemic danger to the spinal cord. However, without adequate input the CN cannot provide sufficient blood flow to the lumbar spinal cord, most likely resulting in severe spinal cord ischemic damage if longer operation times at higher core temperatures are required. With regard to these observations, distal perfusion as a neuroprotective adjunct by providing lumbar spinal cord blood supply may play a role. Safi et al.[21] showed that distal perfusion does, in fact, reduce the incidence of spinal cord ischemic damage during TAAA repair.

Another notable observation is that reduction of mean arterial blood pressure after stent-graft deployment was not detected by NIRS at the thoracic or lumbar level. An explanation might be the presumed reservoir function of the paraspinous CN bridging short irregularities in blood supply. However, no circulatory arrest or cross-clamping was required during TEVAR, which allowed for continuous retrograde blood supply via the hypogastric arteries and directly via the non-covered segmental aortic arteries. This might be of great importance, especially in cases of extensive coverage of segmental arteries by multiple stent-grafts.

These initial results suggest that NIRS may allow intraoperative detection of significant decreases in spinal cord perfusion by means of the paraspinous CN. However, further experiments need to be conducted in order to shed light on the questions resulting from these observations.

Direct correlation between regional NIRS monitoring and spinal cord perfusion via the CN needs to be proven in an experimental setting to validate the concept and to allow for reproducibility. The reaction time—what is thought to be real-time monitoring—between NIRS signaling and CN perfusion and, subsequently, spinal cord tissue oxygenation, needs to be investigated also with regard to distribution

within the spinal cord (anterior vs. posterior horns). As SSEP and MEP are already clinically validated for functional spinal cord monitoring, NIRS needs to be evaluated against these methods. Last but not least, potential confounders such as the reaction of spinal cord/muscle arteriols on vasoactive and tonus-altering drugs/conditions are crucial items to be addressed before conclusions regarding its clinical relevance can be drawn.

Through demonstration that NIRS is a feasible monitoring method for clinical routine, additional experiments and trials on larger/homogenous cohorts can be performed and are underway.

Limitations of this study

The small number of patients restricts statistical analyses in terms of significance, especially in the TEVAR and hybrid group. Furthermore, the overall diverse nature of these procedures (open DTA and TAAA repair vs. TEVAR vs. hybrid) makes concerted interpretation of the data difficult.

CONCLUSION

Non-invasive monitoring of the paraspinous CN oxygenation prior to, during, and after open, endovascular, and hybrid thoracoabdominal aortic repair is feasible. Lumbar CN oxygenation levels directly respond to aortic cross-clamping, but demonstrate a delay. Further studies are needed to answer various key questions before conclusions with regard to the reduction of ischemic spinal cord injury can be drawn.

CONFLICT OF INTEREST

FUNDING

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