Near-infrared spectroscopy to monitor spinal cord oxygenation in open thoraco-abdominal aortic surgery. A case series.

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Abstract:

Objectives. We adopted the near-infra-red spectroscopy (NIRS) technology to monitor the spinal oxygen supply through the paraspinous muscles oxygenation in agreement with the concept of "collateral network" circulation. We retrospectively investigated our database of subjects who underwent Thoraco-abdominal aorta open repair assessing for the reliability of this monitoring to predict spinal cord injury.

Methods. Consecutive Patients who underwent elective thoraco-abdominal aorta open repair between March 2019 and September 2021. In addition to standard monitoring, patients received the monitoring of the paraspinous muscles oxygenation by NIRS.

Results. In one patient a significant drop of the mean arterial pressure (49 mmHg) and the spinal-cord perfusion pressure (31 mmHg) occurred after the aortic clamping, with a contemporary lowering of the left-side oxygenation of paraspinous muscles (<40%). Both the blood pressure and the spinal cord perfusion pressure were restored within 10 minutes, but the oxygenation remained at an unsafe level (<55%) until the end of the surgery. This same patient experienced a lower-limbs paralysis post-operatively. It did not happen in the other 11 cases of the sample.

Conclusions. The main finding of our retrospective analysis indicates reliability of this technology to monitor the spinal cord oxygenation during open thoraco-abdominal aortic surgery and possibly predict spinal cord injury. Still, several questions need to be addressed about the suitability of this technology to the anatomic and pathophysiology of the spinal cord circulation.

Key Words: Thoraco-abdominal aortic surgery; spinal cord injury; paraplegia.

INTRODUCTION

Thoraco-abdominal aortic (TAA) open repair is a complex surgical procedure with a relevant impact due to the great "surgical invasiveness" and anesthesiologic issues linked to it. Consequently, the patient is at high risk for a complicated outcome (respiratory failure, acute kidney injury, severe intraand/or post-operative bleeding, ileus, liver disfunction, cardiac adverse events, spinal cord injury) or even death.

Spinal cord injury (SCI) due to ischemia during aortic sur-

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gery still has a significant incidence (5-11%) and is one of the most distressing complications in this surgical setting^{1,2}. It impacts on patients' quality of life because of the paraplegia *per se* and the psychological aftermath.

Spinal cord (SC) viability during thoracic aortic surgery can be monitored by motor evoked potentials (MEP) or somato-sensory evoked potentials (SSEP), two systems requiring invasive applications, dedicated personnel, and technical skills³. Therefore, MEP and SSEP are not routinely employed. The most widely adopted estimation of SC perfusion is the Spinal-cord perfusion pressure (SC-PP) resulting from the difference between the mean arterial pressure (MAP) and the sub-arachnoid pressure (sAP). In addition, the catheter that permits the monitoring of sAP allows the anesthetist to drain the cerebro-spinal fluid (CSF). This action, causing the sAP reduction, contributes to increase the SC-PP, if MAP does not change or increase.

A decade ago, Etz *et al.* described the "Collateral Network", a concept whereby the blood circulation of paraspinous muscles should provide blood supply to the spinal cord too⁴.

Following this assumption and considering the anatomic characteristics of the spinal cord circulation (as also described

by Melissano *et al.* - Figure 1)⁵, we started to monitor the near-infra-red spectroscopy (NIRS) of the paraspinous muscles oxygenation during open thoracic and thoraco-abdominal aortic surgery. The present study reports the results of the retrospective analysis of a small case series.

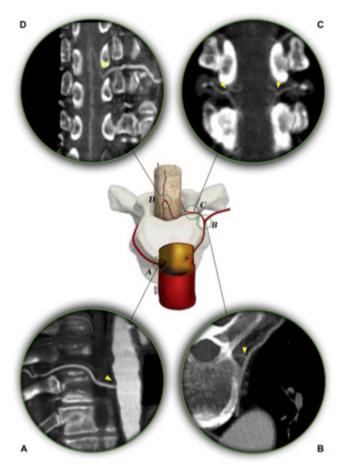


Figure 1. The spinal cord circulation

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METHODS

Between March 2019 and September 2021 we collected the perioperative data of consecutive patients who underwent elective open surgery repair of thoraco-abdominal aorta, at Humanitas Research Hospital in Milan, (Italy). The open approach was indicated and consequently performed upon multidisciplinary consultation.

Emergent operations and endovascular procedures were not included.

This retrospective observational study received the permission of the local Ethical Committee (CE n. 39/21).

STROBE guidelines, and the STROBE checklist were used (http://strobe-statement.org/index.php?id=strobe-home).

The type of TAA in our Hospital is classified according to

the Crawford classification⁶.

Lower limbs palsy was defined by ASIA classification (America Spinal Injury Association; see https://asia-spinalinjury.org).

Before general anesthesia, we inserted a sub-arachnoid catheter (Hermetic™ Lumbar Catheter Open Tip, 80 cm, Integra NeuroSciences, Plainsboro, NJ, USA) at lumbar level (L3-L4) aimed at monitoring and managing the sub-arachnoid pressure (sAP) and measuring the cerebro-spinal fluid lactate concentration (Lac_{CSF}) by a point-of-care machine (GEM Premier 3500-Instrumentation Laboratory Company, Bedford, MA, USA). Then, we placed two NIRS sensors (INVOS™ 5100 Somanetics - Medtronic, Minneapolis, MN, USA) at the thoraco-lumbar region (T9-L2) over paraspinous muscles, (Figure 2).

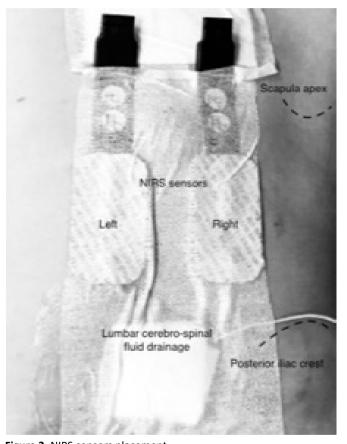


Figure 2. NIRS sensors placement NIRS=Near Infra-Red Spectroscopy

An arterial line had been placed into the radial artery to monitor the invasive blood pressure (IBP_{rad}). Moreover, a four-lines central venous catheter was inserted into the left internal jugular vein for drugs and fluids administration, while another 8Fr-central venous catheter was inserted into the right internal jugular vein for fluids and blood products at high-flow rate.

The surgeon provided a second arterial line into the right femoral artery to monitor the blood pressure (IBP $_{\rm fem}$) supplied by the by-pass pump (Bioconsole560-Medtronic, Minneapolis, MN, USA) draining blood from the left atrium and returning it into the left femoral artery.

The patient is placed in the left lateral decubitus position. Incision, aortic dissection, proximal and distal cross-clamping and anastomosis, and need for arterial reattachment, depend on aneurysm type.

For type 1 and 2 aneurysms, we perform the incision at the 5th intercostal space level, with posterior displacement of the ribs; extension of the incision anteriorly and along the median line of the abdomen is required for type 2 aneurysms. It allows the exposure of the abdominal aorta in the retroperitoneum by mobilizing spleen, left kidney, and left colon to the left side.

For type 3 and 4 aneurysms, we chose the skin incision at the 8th or 9th intercostal space.

For type 2 and 3, the diaphragm is sectioned.

Once the exposure is complete and the aorta is adequately isolated, we perform proximal cross-clamping after the left-bypass pump started.

Regarding the distal clamping, a fixed or a sequential clamp technique can be chosen depending on the level of the distal aneurysmal neck.

Sometimes, we use Fogarty balloon catheters to aid in occluding intercostal, visceral, or renal arteries. Considering ischemic time might be lengthy, we generally provide these arteries with a cooled perfusion solution.

When needed, we adopt a Carrel patch technique for visceral and renal arteries reattachment: most commonly, the celiac trunk, the superior mesenteric artery and the right renal artery are kept on the same patch, while the left renal artery is reimplanted individually. Sometimes, endoarterectomy of the arterial ostia might be required. Typically, the inferior mesenteric artery can be safely ligated after ensuring colonic perfusion is maintained by the arc of Riolan.

Thereafter, we perform the distal anastomosis at a level depending on the extension of the aneurysm. If the abdominal aorta is involved, we prefer to execute it at the level of the iliac bifurcation. Eventually, some previously selected intercostal arteries can be reattached to the aortic graft using a patch - if they are anatomically close enough - or a button technique - if they are too far to be positioned on a unique patch⁷.

Intra-operative monitoring included standard measurements (non-invasive blood pressure, NIBP; Electrocardiogram of D2 and V5 lines; heart rate, HR; end-tidal $\mathrm{CO_2}$, et $\mathrm{CO_2}$; peripheral oxygen saturation, $\mathrm{SpO_2}$), mean invasive blood pressure from both lines (mIBP $_{\mathrm{rad}}$ and mIBP $_{\mathrm{fem}}$) and blood-gas analysis. Further monitoring included cerebro-spinal lactate concentration (Lac $_{\mathrm{CSF}}$), spinal-cord perfusion pressure (i.e., SC-PP=mIB- $_{\mathrm{rad}}$ - sAP) and spinal-cord oxygenation derived from the NIRS (rSO $_{_{2}}$) sensors.

All patients received general anesthesia (Propofol+Fentanyl for the induction; O_2 +Air+Sevoflurane for maintenance; Rocuronium for neuromuscular blockade).

Our monitoring targets consisted of mIBP $_{\rm rad}$ >65 mmHg, sAP \leq 15 mmHg, SC-PP \geq 50 mmHg and mIBP $_{\rm fem}$ 60-80 mmHg. Despite Literature suggests a mIBP $_{\rm fem}$ >80 mmHg 8 , in agree-

ment with surgeons, we adopted a little lower level as a safe compromise between providing the spinal cord perfusion and the limitation of intraoperative blood loss.

In case of mIBP $_{\rm rad}$ <65 mmHg, Norepinephrine was the first-choice drug. When inotropic support needed, we administered Dobutamine. To reach the target SC-PP we increased mIBP $_{\rm rad}$ and/or reduced sAP by CSF drainage as appropriate: 5-10 ml of CSF within 1-2 minutes.

Alongside spinal-cord perfusion pressure, Lac_{CSF} and rSO_2 were used as warnings of SC hypoperfusion/ischemia. Particularly, CSF lactate concentration \geq 4 mmol/L was the cutoff value, since Lac_{CSF} >3 mmol/L had been described to be associated with SCI 9 .

All the patients were actively warmed. Nevertheless, mild intra-operative hypothermia (34.5-35.5 °C) was tolerated.

Baseline data were recorded before the aortic clamping and subsequently collected at standardized times: T1, upon starting of the extracorporeal by-pass; T2, just after clamping at the thoracic level; T3, 15 minutes after thoracic aorta clamping; T4, passing from thoracic aorta clamping to aortic prosthesis clamping; T5, final aortic unclamping; T6, end of the operation and starting wound closure. Generally, 1-3 minutes were spent between T1 and T2.

After the operation, patients were admitted to intensive care unit (ICU) sedated and mechanically ventilated. When normal body temperature had been reached (36°C), the intensivist performed a basic neurological assessment (awareness and legs' motility and sensitivity) during a free-of-sedation time--window. Then, sedation restarted along with respiratory support ventilation. The weaning from mechanical ventilation was generally achieved within the following 12 hours if no complications occurred.

In case of lower limbs paralysis, after the provisional diagnosis of SCI, patients would have received a magnetic resonance imaging (MRI).

Upon discharge from our hospital, all the patients were sent to a specialized rehabilitation center.

Statistics

Data are reported as either mean ± standard deviation, median and range, or number and percentage, as appropriate.

The Pearson correlation test was used to explore linear association between parameters. Pearson's coefficient (r) between variables has been reported as value within the Confidence Interval 95%.

We assumed p<0.05 for statistical significance.

Analysis was performed with Prism 8.2.1 Software - Graph-Pad 2356 Northside Dr. Suite 560 - San Diego, CA 92108 United States.

RESULTS

We retrospectively analyzed 12 cases admitted to our hospital for open repair of thoraco-abdominal aorta (TAA).

The sample age was 67 ± 9.5 yrs (median 68 yrs; range 49

÷ 82 yrs) and according to the Crawford classification⁶, their aneurysm resulted as follows: 4 type-1, 6 type-2, 1 type-3 and 1 type-4. Preoperative ASA (American Society of Anesthesiology physical status classification) score resulted as follows: six subjects in class 2 and six in class 3. Nine of them were males, (Table 1).

The mean total duration of the aortic clamping was 101 \pm 46 min. The average intraoperative CSF drainage was 73 \pm 29 ml.

Before the aortic clamping, the baseline data (T0) are summarized in Table 2.

The type-4 case that had not been considered at a risk for SCI and then the surgeon did not perform any intercostal artery reimplantation to the prosthesis. Conversely, 6 patients received at least two paired intercostal arterial branches reimplantation, and 2 patients received a single pair of intercostal arteries reattached to the aortic graft (one of these two patients experienced SCI).

Out of the 12 patients, one case (Crawford type 2), indicated as Pt2, showed a reduction of rSO₂ to <40% on the left

side at T1 and only at T6 it reached a value >50%. In this case a significant hypotensive event (mIBP $_{rad}$ = 49 mmHg; duration 5-10 minutes) occurred at T1 and was promptly and successfully treated by fluids and NE administration.

Except for Pt2 case, the whole-sample average of mIBP $_{\rm rad}$ was always >70mmHg and the SC-PP had been maintained >50mmHg. Pt2 showed a SC-PP = 31 mmHg at T1, with both mIBP $_{\rm rad}$ and SC-PP restored during the interval T2-T3 (mIBP $_{\rm rad}$ >80 mmHg and SC-PP >60 mmHg).

In Pt2 we observed a high serum concentration of lactate (sLac). Specifically, at T6, sLac = 7.2 mmol/L and Lac_{CSF} = 2.4 mmol/L. At the same time-point the average sLac and Lac_{CSF} in the whole sample were 2.7 \pm 1.8 mmol/L and 2.1 \pm 0.3 mmol/L, respectively.

Figure 3 shows the trends of NIRS, blood pressure and lactatemia both in the serum and CSF, of each single patient. In Pt2, the trends showed some differences. Specifically, at T1 mIBP_{rad} decreased below 65 mmHg, the left-side NIRS dropped with a nadir at around 30% of oxygenation. Then we ruled out the sensor displacement since the right-side oxygenation was stable.

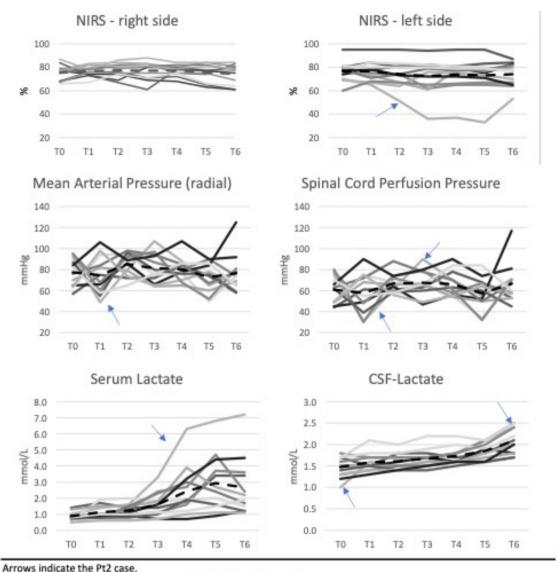
Table 1. The sample characteristics and general perioperative results

Sex		9 Male, 3 Female
Age (years)		67 ± 9.5
ASA		2-3
Crawford classification (n, %)		
	Type 1	4 (33.3)
	Type 2	6 (50.0)
	Type 3	1 (8.3)
	Type 4	1 (8.3)
Aortic clamping duration (minutes)		101 ± 46
Total intra-operative CSF drainage (ml)		73 ± 29
Hospital length-of-stay (days)		15 ± 11
ICU length-of-stay (days)		7 ± 8
Duration of mechanical ventilation (hours)		26 ± 27
Patients who experienced post-operative complications (n,%)		9 (75.0)
Deaths during hospitalization (n,%)		0 (0.0)
Deaths during rehabilitation program (n,%)		1 (8.3)

Table 2. Baseline values

Mean radial arterial pressure (mmHg)	78 ± 14
Sub-Arachnoid pressure (mmHg)	17 ± 5
Spinal-Cord Perfusion Pressure (mmHg)	61 ± 13
Serum Lactate (mmol/L)	0.9 ± 0.3
CSF - Lactate (mmol/L)	1.5 ± 0.2
NIRS right side (%)	75 ± 7
NIRS left side (%)	77 ± 8
Cardiac Index (L/min/m² body surface area)	2.1 ± 0.5
Serum Hemoglobin (g/dL)	10 ± 2

CSF: Cerebro-Spinal Fluid; NIRS: Near Infra-Red Spectroscopy.



Arrows indicate the Ptz case.

The dotted line represents the average trend of the whole sample.

Figure 3. Intra-operative trends The arrow indicates the Pt2 case

In addition, when the Pt2's blood pressure dropped (T1: 49 mmHg), the spinal-cord perfusion pressure reduced accordingly (31 mmHg) and returned to normal value as blood pressure was restored (T2: mIBP $_{\rm rad}$ = 80 mmHg; SC-PP = 61 mmHg).

Three more patients experienced a transient hypotensive event during aortic clamping along with SC-PP dropping, but none of them showed a corresponding reduction in ${\rm rSO}_2$.

We did not find any correlation between SC-PP and Lac_{CSF} SC-PP and rSO_2 , Lac_{CSF} and rSO_2 , and rSO_2 and sLac, from T0 to T6, except for rSO_2 and sLac from T3 to T6. Table 3 lists the Pearson coefficients and statistical significance of these correlations. Conversely, we observed a significant correlation between NIRS and sLac, (Figure 4).

Out of the 12 cases, three cases (25%) had an uneventful outcome. The most frequent complication was respiratory impairment: three cases of post-operative pneumonia (25%) and two cases of atelectasis were also diagnosed (16.6%). All of these had tested positive to a Covid-19 swab more than one month before and underwent surgery after two consecutive negative swabs along with a pre-operative negative chest X-ray

One case of pneumonia needed ICU admission for 30 days and received a tracheostomy too. He died of a septic shock due to urinary tract infection during the rehabilitation phase.

Other complications consisted of one case (8.3%) of acute kidney injury (AKI) recovered after temporary continuous renal replacement therapy (CRRT), one case of acute pancreatitis (8.3%), one case of transient sub-arachnoid fistula (8.3%), and one case of hematemesis due to stress-related gastric ulcer (8.3%) and finally one case (Pt2) of post-operative paraplegia (8.3%). In addition, two patients experienced two simultaneous adverse events post-operatively (one pneumonia+AKI and the other pneumonia+stress-related gastric ulcer).

The other three patients with transient intraoperative hy-

Table 3. Correlation between rSO₂ and serum Lactate

Time	Right side		Left side	
	r	р	r	р
T0	0.2098	0.5127	0.1882	0.5580
T1	0.4170	0.1774	0.1658	0.6065
T2	0.1055	0.7717	-0.4357	0.2081
T3	-0.2271	0.5281	-0.7219	0.0184
T4	-0.1666	0.6684	-0.7373	0.0234
T5	-0.6020	0.0384	-0.7785	0.0029
T6	-0.7444	0.0055	-0.7640	0.0038

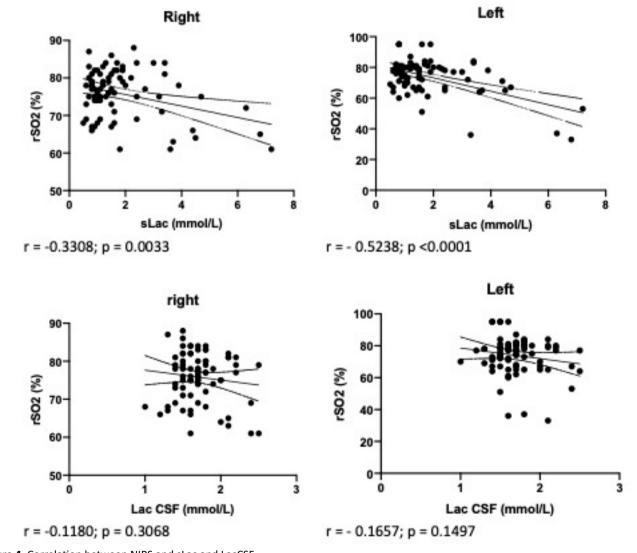


Figure 4. Correlation between NIRS and sLac and LacCSF NIRS=Near Infra-Red Spectroscopy; LacCSF = Cerebro-Spinal Fluid lactate concentration.

potension did not show spinal cord injury post-operatively.

None of the twelve patients had any complication associated with the insertion of the CSF drainage.

DISCUSSION

The main finding of our retrospective analysis indicates a possible reliability of NIRS to monitor the spinal cord oxygenation during open thoraco-abdominal aortic surgery. To corroborate

this assumption, we can consider that only the patient who experienced postoperative lower limbs paralysis showed an even unilateral reduction of ${\rm rSO}_2$ for most of the intraoperative time. Conversely, when ${\rm rSO}_2$ drop was transient it resulted harmless.

Spinal Cord Injury in TAA repair may occur in a percentage that reach 11% or even more depending on the type of procedure (endovascular or open surgery), the extension of the

aorta substitution and the type of the aneurysm^{1,2}. Aiming at limiting the occurrence of SCI, different and combined strategies are applied: spinal fluid drainage, optimization of blood pressure, neuroprotection by drugs and/or hypothermia and arterial branches reimplantation¹⁰. Nevertheless, such strategies need to be supported by spinal cord oxygen supply monitoring and nowadays it may be possible only indirectly.

The oxygen saturation over paraspinous muscles has been considered as corresponding to the oxygenation of the spinal cord based on the assumption that they are supplied by the same circulatory blood flow^{4,11}. Consequently, if paraspinous oxygenation reduces, a simultaneous deficit of spinal cord oxygen supply deficit would be expected.

Although some different levels of sensor positions have been described, the optimal site is under debate¹². Nevertheless, Etz *et al.* reported that the most appropriate site to monitor the SC oxygenation should be at thoraco-lumbar region, because at this level the spinal cord circulation is the most jeopardized in terms of blood perfusion¹³. Such a report made us choose the thoraco-lumbar region for NIRS sensor placement.

Contrarily to what Vanpeteghem *et al.* reported in their review¹¹, after by-pass pump starting and aortic clamping, we did not observe a significant reduction of the rSO₂, except for the Pt2 case, in which, rSO₂ fell almost immediately after the aortic clamping, even if only unilaterally.

Moreover, three co-existing warning signs can be considered as consistent with hypoperfusion of the spinal cord: 1) the systemic hypotension; 2) the SC-PP drop; 3) the rSO_2 reduction.

Even though both blood pressure and SC-PP were promptly restored, the CSF-Lactate concentration started to rise progressively but reached a similar level of other cases.

Anyway, based on these findings, some questions arise. First, why would only unilateral rather than bilateral oxygen desaturation explains the hypoxia of the spinal cord? Second, why did ${\rm rSO}_2$ not recover after blood pressure and spinal-cord perfusion pressure were restored? Third, if CSF-Lactate is a marker of spinal cord ischemia, why did it not rise to a level higher than the other cases?

To answer the first question, we could speculate that the patient developing SCI there might have been a left-side predominance in the arterial circulation supplying both spinal cord and paraspinous muscles. In such a context, we could speculate that since the neural tissue is more sensitive to hypoxia compared to muscle, neurons would suffer whilst myocells hypoxia could be clinically silent, except for the increased lactatemia. This until collateral circulation restored the muscle perfusion. It might justify the late recovery of rSO₃. Accondingly, Luehr et al. reported a delay of rSO2 variation of paraspinous muscles after aortic cross-clamping¹⁴. Furthermore, we are aware that the serum hyperlactatemia we observed in the Pt2 case could be multifactorial in such a major surgery. Nevertheless, the paraspinous muscles ischemia-reperfusion could be the main factor. We expected a progressive clearance of serum lactate provide the hemodynamics was stable.

Moreover, Rojas *et al.*, in a study including 32 cadavers, found asymmetry of the spinal cord arterial circulation both in terms of anatomic disposition and caliber of the vessels, in up to 81.25% of the cases¹⁵.

In few words, the single pair of arterial branches reattached to the aortic graft might have not been sufficient to sustain the spinal cord circulation.

Depending on several vascular and metabolic factors, a safe pressure level does not always ensure an adequate flow. Indeed, in animal models, spinal cord autoregulation is less robust than cerebral autoregulation and more pressure-dependent, hence SC is more sensitive to hypotension¹⁶.

Noteworthy, Vanpeteghem *et al.*, found different effects of phenylephrine and ephedrine on cerebral oxygen saturation and paraspinal oxygen saturation. Following vasoactive drug administration resulted in a steal phenomenon: an increased blood flow to the spinal muscles, masking the spinal cord hypo-oxygenation. The authors concluded that the application of the "Collateral network" concept in case of vasoconstrictive drug use remains to be confirmed ¹⁷.

In our case, NE was able to restore blood pressure promptly, but left side rSO₂ remained at low levels. Then, we might assume that the phenomena observed by Luher and Vanpeteghem's could explain our findings.

With regards to the Lactate concentration, we know that if the Blood-Brain Barrier (BBB) is intact, there is no mixing of blood and CSF. The Lac_{CSF} is a marker of the central nervous system anaerobic metabolism, like during an ischemic injury. When the BBB is disrupted (as in the case of prolonged ischemia) a mix of blood and CSF should occur^{18,19}. In Pt2 case sLac increased more than in CSF, possibly because lactatemia was also depending on the paraspinous muscles ischemia.

When spinal cord ischemia occurred due to a transient hypotension, ${\rm rSO_2}$ reduced, marking the hypoxic state on the left side. Even when blood pressure was restored on the left-side, spinal ischemia persisted. The late normalization of ${\rm rSO_2}$ was only due to the muscles restored oxygenation through any collateral circulation.

In our experience, we already found a significant increase of Lac_{CSF} in a patient submitted to open TAA repair who experienced SCI post-operatively^{20,21}. Unfortunately, at that time we did not monitor rSO₂, and consequently we cannot compare the two cases.

Experimental evidence about NIRS as an index of spinal cord circulation is conflicting. Suehiro et al., reported that NIRS may be used to detect changes in spinal cord circulation following aortic clamping and de-clamping in animal model, whereas it may not reflect changes in spinal cord circulation due to cerebrospinal fluid drainage²².

If both rSO_2 and Lac_{CSF} are markers of spinal cord ischemia, the inconsistency between the drop of the former and the hampered rise of CSF-lactate concentration in the Pt2 patient is unexpected and difficult to explain. The lacking correlation between rSO_2 and Lac_{CSF} may be due to the fact that dead cells (neurons in this case) do not have any metabolism and then

do not produce lactic acid.

Our case series has several limitations. First, its retrospective nature along with the small sample size do not permit definite conclusions and consequently these findings should be considered mostly speculative. Moreover, a single case of deoxygenation observed may have been accidental. Finally, as discussed above, our results leave some important open questions.

In conclusion, Near Infra-Red Spectroscopy application at the thoraco-lumbar region over paraspinous muscles may be helpful to indirectly monitor the spinal cord oxygenation during open repair of thoraco-abdominal aorta. Anyway, we are aware that further specific investigations are desirable to better understand how to interpret and integrate paraspinous muscles rSO₂ in the setting of thoraco-abdominal aorta open surgery.

Assessing the reliability NIRS in this field, could provide a helpful and non-invasive tool to monitor the spinal cord oxygen supply. The surgeon might use it as a tool to decide for further collateral branches re-implantation to the prosthesis. The anesthetist might use it to guide the strategies to prevent spinal cord injury.

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