THE EVALUATION OF MEDULLA SPINALIS PERFUSION AT MODERATE HYPOTHERMIA WITH PATIENTS WHO UNDERGO ANTEGRADE SELECTIVE CEREBRAL PERFUSION IN ASCENDING AND ARCUS AORTIC SURGERY

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Abstract

Aim: Antegrade Selective Cerebral Perfusion (ASCP) with lower body circulatory arrest (LBCA) used in aortic arch surgery can lead to postoperative ischemic organ dysfunctions if it lasts long enough. We aimed to evaluate methods that can provide early detection of spinal cord ischemia during aortic arch surgery. **Methods:** Thirty consecutive patients were prospectively enrolled and Near infrared spectrometry (NIRS) data obtained from the 5 th and 10 th thoracic vertebral region, S100β protein, lactate blood levels during various operative phases and postoperative neurological outcomes were evaluated. **Results:** A total of 30 patients underwent elective hemi arch (73.29%) or total arch (23.31%) replacement and with a mean ASCP period of 25.1 ± 19.0 (limits 10-90) minutes. In-hospital mortality was 6.66% (two patients). Paraparesis developed in one patient (3,33%). Thoracic T5 and T10 NIRS values were lowest during the ASCP period ($p<0.001$) with a good correlation between them ($r=0.853$, $p<0.001$). However, a significant difference between the T5 and T10 levels was observed during the same period (55.40 vs 51.07 respectively, $p=0.001$). A moderately negative correlation between the lactate levels in descending aorta and NIRS values at the T10 level was found during ASCP ($r=-0.514$, $p = 0.004$). **Conclusion:** Thoracic 5 th and 10 th level NIRS monitoring for spinal cord oxygenation were significantly lower during ASCP period compared to the other periods of aortic arch surgery with T10 values being lower than T5 values during the same period indicating a more significant flow disturbance at this level. Measuring lactate levels with thoracic NIRS monitoring seems promising for future studies with larger volumes and longer ASCP periods.

Introduction

Antegrade selective cerebral perfusion (ASCP) has a widespread use today for cerebral protection during aortic arch surgery with increasing incorporation of moderate hypothermia (28°C) during lower body circulatory arrest (LBCA). Although a considerable amount of contribution has been made literally addressing the favorable effects of this technique questions remain regarding the protection of distal abdominal viscera and neuronal structures-namely the spinal cord- during lower body circulatory arrest with moderate hypothermia. The nervous tissue is very sensitive to ischemia and ensuing spinal cord complications can be

devastating with a wide spectrum ranging from clinically undetected sensory loss to permanent paraplegia. Animal models of arch surgery at moderate hypothermia revealed insufficient spinal cord blood flow below thoracic 8 and 9 segment to sustain its viability with ASCP only [1]. If distal perfusion is not included during arch surgery, concerns about the margins of the safe period for spinal cord ischemia during distal circulatory arrest exist.

Considering that a real time information system may help raise awareness of the team about ischemic insult of the spinal cord during thoracoabdominal interventions where there is a considerably high risk of paraplegia, pioneering experimental works with near infrared spectrometry (NIRS) yielded acceptable results [2-4]. NIRS could reveal both ischemic and reperfusion related changes below midthoracic level (T7) in procedures involving the proximal descending thoracic aorta in a large animal model [5]. In the clinical setting, after the feasibility of NIRS monitoring in both open, endovascular and hybrid thoracoabdominal interventions has been confirmed and the Leipzig group have reported upgrading their monitoring system to bilateral thoracic and lumbar paravertebral NIRS monitoring today it is used in both open and endovascular thoracoabdominal procedures [4,6,7]. Regarding arch surgery, Kinoshita et al. were the first to use NIRS monitoring in a pilot study and reported that lower part of the spinal cord was not perfused sufficiently during ASCP [8]. We use routine cerebral NIRS monitoring in arch surgery and had experience with spinal cord NIRS monitoring in patients with type B aortic dissection in our hospital previously[9,10]. We therefore planned to monitor spinal cord oxygenation with NIRS in our patients undergoing arch surgery to detect a warning sign regarding spinal cord ischemic insult and looked for a correlation between blood lactate, s100β values and NIRS levels.

Materials and methods

Patients:

Thirty consecutive patients who were to undergo elective ascending and arcus aorta operations by a single surgeon were prospectively evaluated between December 2019 and 2020. Patients younger than 18 years of age, undergoing emergent operation for acute type 1 aortic dissection or receiving antiplatelet-anticoagulant medication were excluded from the study. Demographic parameters, medical history including comorbidities and medications were recorded. Thorough evaluation with echocardiography, carotid-vertebral Doppler imaging and computed tomography were done in all patients. Paraplegia was determined as complete impairment in motor and sensory function of the lower extremities. However, paraparesis refers to partial neurologic dysfunction of the lower extremity.

Transient neurologic dysfunction was defined as postoperative confusion, agitation, delirium, or obtundation with a negative brain computed tomography scan and complete resolution before discharge. In addition, stroke was characterized as a neurological deficit attributed to a persistent acute focal injury of the central nervous system after surgery with positive brain computed tomography.

Ethical approval:

This article had the ethical Approval from the Ethics Committee No:1 of Ankara City Hospital, Ministry of Health of the Turkish Republic in December 2019 with reference number E1/006/2019.

Patient consent statement:

Written consent has been taken from every patient who has contributed to the study.

Surgical technique:

Proximal right brachial artery cannulation with unilateral ASCP and 28°C hypothermia was used in all patients [9]. Back bleeding from left carotid artery was always controlled to test the adequacy of collateral flow from the right carotid system. During ASCP 700-800 ml/min (10 ml/kg/min) blood flow was provided with cerebral NIRS monitoring (right and left frontal regions). If ASCP period was anticipated to last longer than 40-45 minute or a difference between right and left cerebral NIRS values with more than 20% drop from the baseline was observed bilateral perfusion was employed through a separate left carotid artery cannula which was necessary in 3 patients. For spinal cord rSO_2 NIRS monitoring pallets were placed at

the skin over spinous process of 5^{th} and 10^{th} thoracic vertebrae level. Baseline measurement of NIRS value (rSO_2) were done before anesthetic induction and then followed by 5 periods of evaluation; after anesthetic induction, upon commencement of cardiopulmonary bypass (CPB), after cross clamp application, at the end of ASCP period and after conclusion of CPB (SOMANETICS INVOS^{TM} 7100 Cerebral/Somatic Oximeter, Medtronic).

Βλοοδ λαϛτατε ανδ σ100β μεασυρεμεντϛ:

Lactate levels were studied before, during and after ASCP (during the first minute) from the left radial artery cannula. Samples were also taken in the 2^{nd} and 6^{th}postoperative hour. During ASCP an additional blood sample was taken from descending aorta to correlate with the radial artery sample. For s100β determination blood samples were obtained from descending aorta just before terminating ASCP and from radial artery in the 6^{th} postoperative hour. Kits obtained from Cloud Clone Human ELISA were used for s100β determination. All blood samples were centrifuged for 20 minutes with 1000 cycles/minute and the sera obtained were kept at 80°C to be dissolved all at once.

Statistical analysis:

Statistical analyzes were performed using SPSS software version 15. The conformity of the variables to the normal distribution was examined using visual (histogram and probability graphs) and analytical methods (Kolmogorov-Smirnow/Shapiro Wilk tests). Descriptive analyzes were given using median and minimum-maximum values for non-normally distributed variables. In addition, mean, standard deviation values and ordinal variables were given using frequency tables. Wilcoxon test was used for in-group rank pairwise comparisons. Friedman test was used to compare data with more than two repetitive measurements. If required, pairwise comparisons were made using the Wilcoxon test and evaluated using Bonferroni correction. Ordinal variables in 2 related samples were compared with McNemar test. Correlation coefficients and statistical significance for the relationships between parameters were calculated by Spearman's test. The p value was taken as 0.05 for statistical significance.

Results:

Male patients consisted of the majority (80%) with a mean age of 53.47 ± 15.22 years. There were 4 re-operations (13.3%). Hospital mortality is 6.66% with two patients. One patient with additional extensive calcific aortic valve debridement, replacement and coronary artery bypass grafting (CABG) died of multiorgan failure on 19^{th}postoperative day. The other patient with 2 previous operations involving ascending aorta and the arc developed infective endocarditis of the native aortic valve and pacemaker leads. He could not survive his 3^{rd} operation which included aortic valve replacement, arch graft revision, and withdrawal of the pacemaker wires. Demographics and operative procedures are given in Table 1

Table 1. Demographic and Operative Characteristics

Variables	Mean ± SD (Min-Max) or No (%) (N=30)
Age (y)	53.5 ± 15.2 (22-78)
Male sex	24 (80%)
Body mass index (kg/m^2)	26.3 ± 3.9 (19.5-35.9)
Coronary artery disease	11 (36.7%)
History of cerebrovascular disease	1 (3.3%)
Diabetes mellitus	4 (13.3%)
Chronic obstructive pulmonary disease	1 (3.3%)
Diameter of ascending aort (mm)	50 ± 9.6 (32-67)
Diameter of aortic arch (mm)	35.8 ± 6.9 (23-52)
Previous cardiovascular surgery	4 (13.3%)
Surgical procedures	Surgical procedures

Variables	Mean ± SD (Min-Max) or No (%) (N=30)
Aortic arch reconstruction Hemi arch	30 (100%) 22 (73.3%) 7 (23.3%) 1 (3.3%)
Total arch	
Aortic arch revision	
Elephant trunk	2 (6.7%)
Bentall procedure	4 (13.3%)
Valve sparing aortic root replacement	3 (10%)
Coronary artery bypass grafting	7 (23.3%)
Aortic valve replacement	12 (40%)
Operative variables	Operative variables
CPB time (min)	192.6 ± 64.0 (105-371)
Aortic cross clamp time (min)	113.5 ± 33.0 (45-187)
ASCP time (min)	25.1 ± 19.0 (10-90)
MAP – ASCP (mmHg)	49.5 ± 14.0 (15-70)
SaO_2 – ASCP (%)	98.2 ± 0.5 (97-99)
Hct – ASCP (%)	25.6 ± 4.8 (14-33)

ASCP: Antegrade Selective Cerebral Perfusion, CPB: Cardiopulmonary Bypass,

Hct: Hematocrit, MAP: Mean Arterial Pressure, Sd: Standard Deviation

ASCP perfusion was unilateral in 27 patients (90%) with a mean duration of 25.13 ± 19.02 minutes (range 10-53 minutes). Bilateral cerebral perfusion was instituted in 3 patients (10%) whose ASCP periods were 64, 90 and 63 minutes. One of them suffered from transient neurologic dysfunction (agitation and mild obtundation) which regressed completely and his motor function in the lower extremities was graded as 4/5 with no evidence of sensory loss (3.3%). He was discharged with no significant impairment in walking and his daily life activities. Having had a percutaneous coronary intervention 25 days ago, he received ascending and total arch replacement with distal elephant trunk formation due to chronic type I aortic dissection. During ASCP, a drop in T10 NIRS value by 34.73% at the 33rd minute was observed, and bilateral cerebral perfusion was instituted during the rest 57 minutes and the T10 NIRS value returned to 77,8% of baseline (90 minutes of ASCP). There was no significant reduction in cerebral NIRS values (7,5% and 13% reduction in his right and left cerebral NIRS values respectively). There was no other permanent neurologic deficit in any other patient.

Regarding right and left cerebral NIRS values, there was a slight decline with the onset of CPB through cross clamp application and ASCP periods in the whole group, but this difference was not statistically significant in any period throughout the operation ($p<0.05$). T5 and T10 NIRS values on the other hand were significantly lower than the baseline levels after cross clamp application, during ASCP and CPB termination periods ($p<0.001$). These values started to decrease gradually with the onset of CPB and were lowest during ASCP (Figure 1). T5 and T10 levels differed also between each other during ASCP period (55.40 ± 9.21 vs 51.07 ±12.76 respectively, p=0,001). There was a good correlation between T5 and T10 NIRS values during ASCP period ($r= 0.853$, $p<0.001$) but we could not detect any correlation between the cerebral and thoracic NIRS values during the same period (CR-T5 r=0.345, p=0.062 and CL-T5 r=0.344, p=0.062).

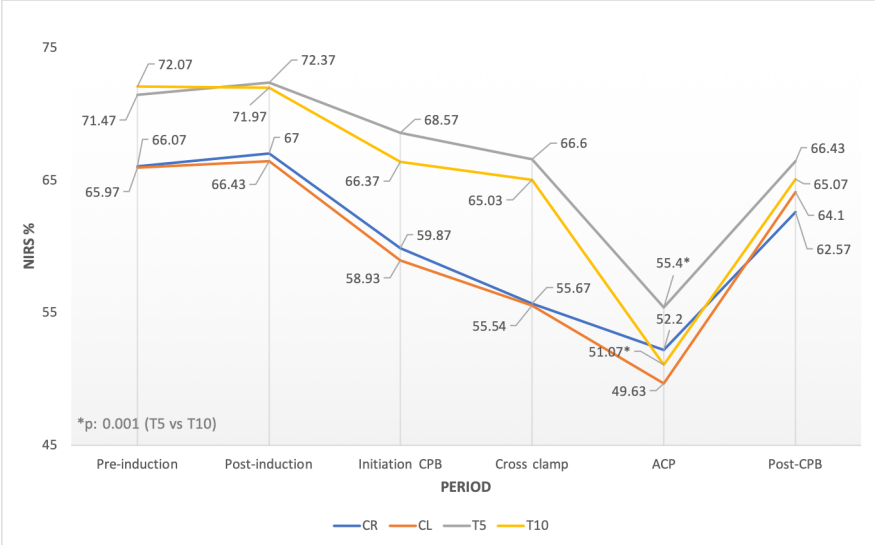

Figure 1 Periodic distribution of NIRS data from CR, CL, T5 and T10 regions

ACP: Antegrade cerebral perfusion; CPB: Cardiopulmonary bypass

When compared to the baseline NIRS levels, more than 20% reduction was observed in 17 patients' T5 NIRS values (56,7%, p<0.001), and 22 patients' T10 values (73.3%, p<0.001) during ASCP period which was statistically more than the number of such patients in the preceding periods. The patient with paraparesis was in this group. A drop more than 36% of baseline occurred less frequently during the same period (in 1 patient's T5 and in 7 patients' T10 NIRS values, p=1,00 and p=0,016 respectively) with none of the patients experiencing any neurologic event. The only patient with more than 36% reduction in both T5 and T10 NIRS values had only 17 minutes of ASCP without any postoperative neurologic complication.

s100β levels at the postoperative 6th hour fell significantly with respect to the period corresponding to the end of ASCP (2.086 ± 0.971 vs 1.082 ± 0.82; p<0.001) (Table 2).

Lactate levels increased with the onset of ASCP reaching its highest level with termination of ASCP and diminishing afterwards in the postoperative period (p^{Σ} <0.001, Table 2, Figure 2). Statistically significant differences were observed in lactate levels between the pairs as before ASCP and during ASCP (p^{Ω} =0.002), during ASCP and at the end of ASCP (p^{Ψ}<0.001) (Table 2). Lactate levels in blood samples obtained from descending aorta during ASCP was significantly higher than the samples drawn concurrently from radial artery with a moderately positive correlation between them (4.52 ± 1.56 vs 3.86 ± 1.61 respectively, p=0.002 and r=0.655, p<0.001).

Ταβλε 2 Περιοπερατιε Δατα οφ Σ100β ανδ Λαχτατε

		Mean ± Sd	p
S100β (ng/ml)	**End of ASCP (Descending Aorta)**	2.09 ± 0.97	p<0.001
	Postoperative 6th hour	1.08 ± 0.83	
Lactate (mmol/L)	**Pre-ASCP**	3.21 ± 1.76$^{\Omega,\Sigma}$	p^{Ω} =0.002
	During ASCP	3.86 ± 1.61$^{\Omega,\Psi,\alpha}$	p^{Ψ}<0.001
	Post-ASCP (1st minute)	6.77 ± 2.38$^{\Psi,\Sigma}$	p^{Σ} <0.001
	Postoperative 2nd hour	5.96 ± 2.53$^{\Sigma}$	
	Postoperative 6th hour	5.55 ± 2.23$^{\Sigma}$	
	End of ASCP (Descending Aorta)	4.52 ± 1.56$^{\alpha}$	p^{α}=0.002

ACP: Antegrade Selective Cerebral Perfusion, Sd: Standard Deviation, p^{Ω}: p value of lactate levels in Pre-ACP and during ACP, p^{Ψ}: p value of lactate levels in during ACP and Post-ACP, p^{Σ}: p value of lactate levels in Pre-ACP, postoperative 2th and 6th hour, p^{α}: Comparison of blood lactate values taken from the descending aorta and radial artery during ACP

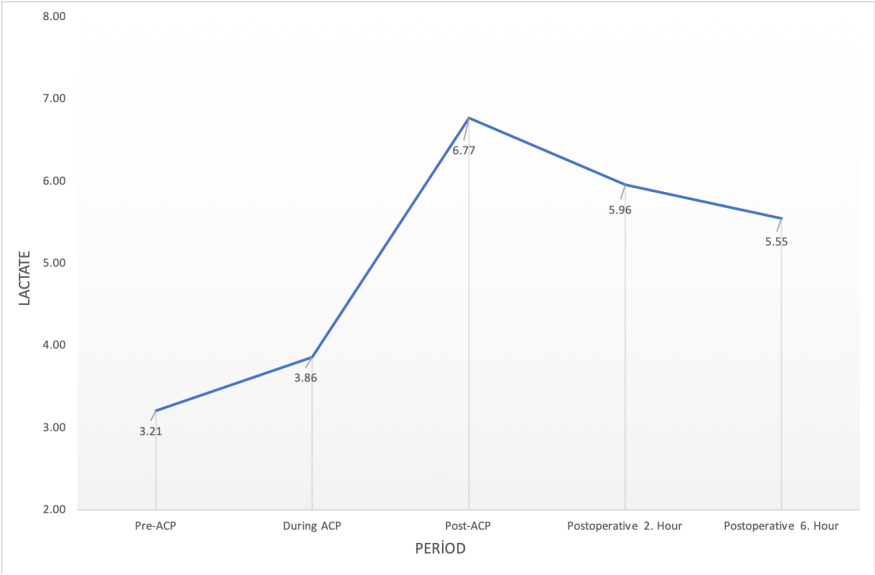

Figure 2 Periodic distribution of lactate levels

ACP: Antegrade cerebral perfusion

s100β and lactate levels at the end of the ASCP period were not correlated with any thoracic NIRS values (r=0.202, p=0.284 and r=-0.040, p=0.833 respectively). On the other hand, lactate levels of descending aorta displayed a moderately negative correlation with T10 NIRS values during the same period (r=-0.514, p=0.004) (Figure 3).

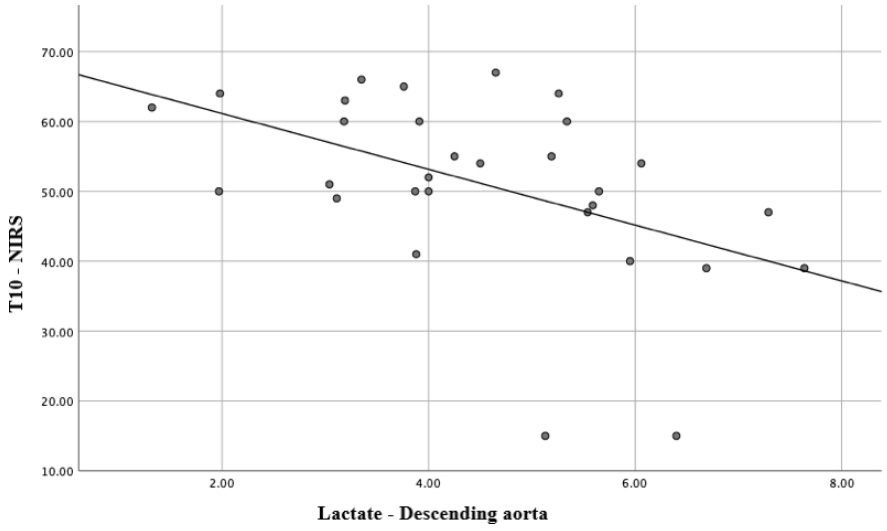

Figure 3 Correlation of NIRS data from T10 level with lactate values in blood sample from descending

aorta during ASCP

Discussion

Despite its benefits in abbreviating side effects of deep hypothermia and longer cardiopulmonary bypass periods, ASCP with moderate hypothermia bears risks of warm ischemia for the abdominal viscera and especially spinal cord during lower body circulatory arrest if prolonged operations times are required or technical challenges occur unexpectedly. Spinal cord injury and ensuing paraplegia after aortic arch surgery is a rare but devastating complication for the patients. Spinal cord perfusion is maintained by segmental arteries from the thoracic and abdominal aorta and increased with the contribution of left subclavian, intercostal, lumbar, hypogastric arteries and the local paraspinous arterial tree embedded in the paraspinous muscles according to the current collateral network concept [6,11,12]. As ASCP eliminates the segmental and hypogastric artery contribution to the spinal cord blood supply it is suggested that nearly two thirds of total flow is lost leaving the vertebral and intercostal arteries only to perfuse the cord [6,11-13]. Routine addition of distal aortic perfusion to overcome this problem may complicate the surgical field and lengthen the procedure with its inherent problems related to retrograde femoral artery perfusion [6]. On the other hand, additional subclavian perfusion may cause significant back bleeding from the intercostal arteries to the descending aorta causing a steal phenomenon and insufficient perfusion of the lumbar segments although blood to the cervical and mid-thoracic segments can be provided[14]. Therefore, when additional distal perfusion is not used, duration of ischemic period and the degree of hypothermia become the critical issues for spinal cord protection during arch surgery.

There is no consensus as to the safe duration of ASCP to rule out spinal cord ischemic injury during arch surgery with lower body circulatory arrest. As most reports are based on experimental studies concerns about safe period remain [1,15-17]. Spinal cord injury was inevitable after 120 minutes and probable after 90 minutes in an experimental study at 28°C. In the clinical setting, when temperatures between 25-28°C are considered, Kamiya et al reported that LBCA duration up to 60 minutes was safe but beyond that limit paraplegia rate increases [18]. The rate was 0% with deep hypothermia (20°C to 24.9°C) with bilateral ASCP but when patients with lower-body circulatory arrest greater than 60 minutes were further evaluated, overall paraplegia rate rose to 2.1% (8/377). They concluded that moderately hypothermic LBCA can be safely applied up to 60 minutes. Beyond that limit they question the safety. Paraplegia rates are usually 5 to 8% in contemporary series but rates as high as 18.2% have been reported with moderate hypothermia (25-28°C) [5,18]. Zierer et al reported 0.3% paraplegia rate in 1002 patients operated at 26-34°C and mostly unilateral ASCP (67.2%). All patients with paraplegia had aortic dissection with a wide range of ASCP period (24, 41 and 127 minutes) [19]. In another large series of patients operated with moderate hypothermia (20-24°C) occurrence of paraplegia was (0.5%) attributed to the creation of elephant trunk[20]. Elephant trunk formation causes a delay for delayed reperfusion injury or inadequate perfusion. Mean ASCP duration was relatively short (25.13 ± 19.02 minutes) in the present study (range 10-90 min) with 3 patients' duration exceeding 60 minutes (63, 64 and 90 minutes). The only patient in the present study with postoperative paraparesis (%3.3) had previous chronic aortic dissection, 90 minutes of ASCP and elephant trunk formation consistent with the above mentioned studies.

NIRS is effectively used for monitoring cerebral oxygen saturation during ASCP in aortic arch surgery and has also been shown to be feasible for spinal cord ischemia detection in procedures involving thoracoabdominal aorta [4]. As it reflects mixed tissue oxygenation -both arterial and venous blood hemoglobin oxygenation- rather than absolute values, usually a decrease of 20–30% from the reference level suggests a possible risk of ischemia. For cerebral NIRS monitoring during aortic surgery, a relative rSO_2decrease to 64–80% of baseline (corresponding to 36-20% decrease of baseline) is suggested to be closely associated with neurologic events by the Japanese Society of Cardiovascular Anesthesiologists[21]. As there is no such predetermined critical NIRS value in the literature for spinal cord injury to occur we evaluated both 20 and 36% reductions in rSO_2 values in our study group. More than half of the patients experienced more than %20 drop in T5 and T10 levels and only one of them had postoperative paraparesis. Less patients had more than %36 drop in their baseline NIRS values during ASCP as can be expected but none of them suffered from paraplegia. The

only patient with paraparesis had NIRS reductions in T5 level by 31.5% and T10 level by 34.7% in T10 which returned to 22.2% drop in baseline after bilateral ASCP was instituted at the 33rd minute. However, his ASCP period was long enough to cause paraparesis (90 minutes). On the other hand, the only patient with more than 36% reduction in both T5 T10 values had only 17 minutes of ASCP with no postoperative complication. It is clear that the time period during which NIRS values remains low is very important. But we could not record instantaneous time related changes in NIRS values during the operation which is one of the limitations of the present study.

As to the optimal NIRS optode placement location for spinal cord, which is surrounded by vertebral bodies, supporters of positioning over the bilateral paraspinal muscles argue that these muscles supplied by the longitudinal collateral network indirectly reflects spinal cord perfusion [22]. On the other hand, it is claimed that placing optodes on both sides of the vertebral column over the paraspinal muscles interfere with each other if the light from the contralateral side is received [23]. Thinking that poorly vascularized adipose tissue overlying the muscles especially in obese patients may obscure light transmission if it is thick enough to exceed the penetration depth of NIRS light (12 to 20 mm). Therefore, we chose to place the optodes over the vertebral bodies distal to the spinal processes.

The level to which the NIRS optodes should be placed is another issue. When proximal aorta is clamped 3 cm below the subclavian artery leaving both subclavian arteries intact, high thoracic region (T4-6) levels do not show major fluctuations in NIRS value owing to extensive collateralization in this area [5]. Mid thoracic (T7-9) and low thoracic (T10-12) values on the other hand drop significantly with high lumbar (T13-L2) and low lumbar (L2-5) values being the most prominent. As there is not such extensive collateralization below T8-9 thoracic level, mid-thoracic to lumbar region monitoring is usually preferred in thoracoabdominal interventions and regarded as a baseline reference to the more caudally placed optodes [1,5]. In aortic arch surgery however, where associated coronary artery bypass grafting and harvesting of the left internal thoracic artery is a common procedure, inclusion of high thoracic levels for spinal cord NIRS monitoring should be undertaken as one major collateral is sacrificed (LIMA use in 23.3% of the cases in the present series). A fairly good positive correlation was observed between T5 and T10 levels in the present study when thoracic NIRS values fell significantly during lower body ischemic period (ASCP). In a pioneering study conducted by Kinoshita et al in patients undergoing arch replacement optodes of NIRS were placed at the level of 3rd and 10th thoracic vertebra over the paraspinous muscles and they have observed that regional oxygen saturations fell significantly with the start of ASCP [8]. While T10 values continued to decrease to about 20% of baseline T3 NIRS levels remained relatively stable unlike the present series. They a had similar rate of CABG (17% and 23.3%) and most probably a similar rate of internal thoracic artery harvesting with our series but we think that additional perfusion of the left carotid and left subclavian artery might have accounted for the higher T3 NIRS values in their practice. They reported that with the advent of lower body circulation via a side branch from the graft the difference between 4 NIRS optodes (right and left forehead, T3 and T10) disappeared. They recovered 30 minutes after resumption of CPB similar to the present study. They did not observe any paraplegia in their 18 patients and concluded that cooling was the most important means of protecting the spinal cord from injury (core temperature about 25°). They had a higher mean lower body circulatory arrest compared to our study (49±14 vs 25.13 ± 19.02 minutes) but when LBCA times are compared in both studies they did not have any patient with more than 90 minutes of LBCA duration (ranges between 24 and 72 minutes). The only patient with postoperative paraparesis in our study had 90 minutes of LBCA. Therefore, we can conclude that when LBCA durations are expected to approach 90 minutes, additional lower body perfusion should be implemented as was also shown in animal studies[1].

One important result of the present study is moderately negative correlation between the lactate levels in blood obtained from descending thoracic aorta and T10 NIRS values during the ASCP period. Lactate levels from the radial artery and descending aorta were moderately correlated (r=0.655) but they were significantly higher in descending aorta probably due to the retrograde blood flow to descending aorta from the visceral, thoracic and lumbar arteries rich in lactate. We think that lactate level measurement during open distal anastomoses accompanied with T10 NIRS monitoring can be a useful adjunct during arch operations with long ASCP periods. However, studies with larger volumes may help to determine a threshold limit to be

used as an alarming sign for spinal cord ischemia.

Regarding s100β levels, in patients with spinal cord injury after thoracoabdominal operations Kunihara et al. observed that serum levels reached peak levels just after the operation and patients with spinal cord injury had a second peak at 24 hours postoperatively (1.3 ± 1.4 μg/L) [24]. We do not have a baseline s100β value preoperatively, but the values in 6^{th} postoperative hour fell significantly compared to values at the end of ASCP. Our values are somewhat higher but serious neurological complication occurred in one patient whose s100β levels were 0.33 ng/ml and 1.929 ng/ml respectively and one patient is not enough to make a reliable statistical evaluation.

One of the most important drawbacks of the present study is failure to monitor lumbar spine NIRS values. We could have observed significant fall in lumbar NIRS values before any reduction occurred in the thoracic level. Another shortcoming is absence of basal s100β level determination before any procedure took place. Patients with acute aortic dissection was excluded but there were 2 patients with chronic aortic dissection with previous intervention to the ascending aorta and aortic valve. Instantaneous display of NIRS during the operation was available however we could not record them. This study was conducted in a small patient group. Large scale studies are needed to arrive at a better conclusion regarding alarming signs for spinal cord injury during aortic arch surgery.

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Figures List:

- **Figure 1** Periodic distribution of NIRS data from CR, CL, T5 and T10 regions
- **Figure 2** Periodic distribution of lactate levels
- **Figure 3** Correlation of NIRS data from T10 level with lactate values in blood sample from descending aorta during ASCP