Optic Nerve Sheath Diameter Measurement to Identify High-Risk Patients for Spinal Ischemia after Endovascular Thoracoabdominal Aortic Aneurysm Repair

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ABSTRACT

BACKGROUND & PURPOSE: **Thoracic endovascular aortic repair (TEVAR) is associated with a reasonable risk of spinal ischemia. As cerebrospinal fluid pressure (CSFP) is correlated with the rate of paraplegia, a non-invasive method to estimate CSFP could help to estimate the patient's individual risk and guide the therapeutic approach. The quantification of the optic nerve sheath diameter (ONSD) using ocular sonography (OS) could be a suitable technique and was examined in the present study.**

METHODS: **28 patients with TEVAR were included. Five consecutive measurements of the ONSD were performed in each patient. The first before the intervention ("baseline"), the next immediately postinterventional at the intensive care unit (post1), measurements 3, 4 (post2, post3) on day 1 and 2 after the intervention and number 5 (post4) before discharge. Statistical analysis was done using the Wilcoxon-test. A p-value < 0.05 was considered statistically significant.**

RESULTS: **A significant increase between baseline and post1-measurements (right eye: $p = 0.006$; left eye: $p = 0.02$) could be detected. A significant decrease was detected between post1 and post3 (right eye: $p = 0.02$; left eye: $p < 0.01$). A group of 5 patients had an additional increase of ONSD from post1 to post2, one of these patients developed a permanent paraplegia. Patients with spinal catheters had significantly lower ONSDs at nearly all time points.**

CONCLUSION: **The present study is the first to prospectively examine and prove the possibility to monitor CSFP changes in patients with TEVAR associated transient spinal edema using OS. Systematic factors as artificial ventilation and body positioning did not have a significant effect.**

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Background and Purpose

Thoracoabdominal aortic aneurysms (TAAAs) are potentially devastating conditions with a high mortality. The risk depends essentially on the diameter of the vessel, with the risk of rupture of 7% per year at a diameter of 6 cm.[1] TAAAs are categorized using the Crawford classification dependent on the length of dilatation and the number of affected visceral arteries.[2]

Two different therapeutic approaches exist: open surgery and thoracic endovascular aortic repair (TEVAR). One of worst complications with both techniques is spinal cord ischemia (SCI). As the brain, the spinal cord is extremely sensitive to reduction of oxygen and inadequate blood flow can lead to irreversible tissue infarction.

The blood supply of the spinal cord is warranted by a network of arteries, which run longitudinally along the cord and mainly consist of a single anterior and two posterior spinal arteries.[3] These arteries are supplied by spinal branches of vertebral, deep cervical, sacral, and segmental aortic branches. Frequently, there is a dominant artery arising between the ninth thoracic and the second lumbar vertebrae, called great radicular artery or artery of Adamkiewiez.[4]

The rate of paraplegia after open surgery of the aorta is largely dependent on the extent of the covered aorta. Incidences range from 38% with Types I and II, and 12% with Types III and IV aortic aneurysms, according to the Crawford classification.[5] Paraplegia risks after TEVAR are respectively lower (0-13%[6]), mainly because of the lacking necessity to cross-clamp the aorta, which leads to an immediate change of the spinal perfusion pressure in open surgery.

In TEVAR, the immediate coverage of intercostal arteries might lead to reduction of blood supply to the spinal cord. Additionally, plaques on the inside of the aortic vessel wall can be dislocated by the guidewire and lead to thromboembolic occlusion of segmental arteries. The length of the covered aorta seems to be associated with a higher risk for paraplegia.[7,8]

Spinal cord perfusion pressure (SCPP) is defined as the difference between blood pressure and cerebrospinal fluid (CSF) pressure. Thus, a reduction of CSF-pressure by CSF-drainage might be therapeutic. In a recent Cochrane review,[9] the only three available prospective studies in humans with CSF-drainage were compared, only one of them reasonably applied the CSF-pressure reduction theory by maintaining a CSF-pressure of 10 mm Hg or less.[10] In this single study, a significant reduction of paraplegia rates with CSF-drainage was demonstrated. As the occurrence of spinal cord ischemia remains unpredictable, the authors saw "a need to develop better techniques for measuring spinal cord perfusion directly" and therefore "a need for more clinical trials."[9]

An ideal technique to estimate SCPP should be noninvasive, safe, repeatable, reliable, and easy to perform at the patient's

bedside or intraoperatively. Ocular sonography (OS) of the eye and retrobulbar structures is a method more and more established in a variety of neurological disorders associated with raised intracranial pressure as well as vascular disorders of the eye.[11] OS is able to easily identify the optic nerve sheath diameter (ONSD) in B-mode, a hyperechogenic structure surrounding the optic nerve and representing the subarachnoid space, that responds quickly to changes of the intracerebral pressure (ICP).[12] For the ONSD, a reliable correlation with invasive ICP measurements could be demonstrated[13] and, in addition, it has a high intra- and interobserver reliability.[14]

In this study, we investigate whether raised spinal CSF-pressure, caused by ischemia-associated edema of the spinal cord after TEVAR, leads to an elevation of CSF-pressure identified by enlargement of the ONSD. Thus, ONSD might be used as a surrogate marker for increased intraspinal pressure (ISP), and its therapy.

Methods

During November 2012 and July 2013, 28 patients designated to TEVAR were included into our prospective study after provision of written informed consent. The study was approved by the local ethics committee (volume number 12–101–0121) according to the Declaration of Helsinki. In 22 patients, the indication for TEVAR was aortic aneurysm, 3 patients were operated because of dissection, and 3 patients had a combination of both. The main vascular risk factor was arterial hypertension in 26 of 28 patients (93%). The mean age was 65 (range 77-50 years), 24 (86%) of the patients were male.

Five consecutive measurements of the ONSD were performed in each patient during inpatient stay. The first before the intervention ("baseline"), the next immediately after arrival at the intensive care unit (post 1), measurements 3, 4 (post 2, post 3) on day 1 and 2 after the intervention, and number 5 (post 4) before discharge.

The ONSD was examined six times at each time point, mean values and standard deviations were calculated. Measurements were performed by a single experienced examiner (vascular surgeon [CB], accredited by the German medical Ultrasound Society [DEGUM]), the correctness of all scans was re-evaluated by a second DEGUM accredited neurologist (FS) blinded to the patients specific disease and clinical outcome. Concomitantly, facultative parameters such as positive end-expiratory pressure (PEEP) in ventilated patients, mean arterial pressure, ISP, and spinal perfusion pressure were monitored and documented.

In 20 of 28 patients, a spinal catheter was placed before surgery. The remaining 8 patients either declined their consent for placement or a placement was not possible because of the need to continue anticoagulation. After surgery, the spinal drains remained open and were only closed for pressure measurements every 4 hours. At "post 1," spinal drains were open in every single patient, at "post 2," six catheters could be clamped and one could be removed, at "post 3," only 6 of 20 catheters were still open. Finally, at "post 4," all catheters could be removed.

For OS, the patients were lying supine with eyes closed. A layer of acoustic gel was applied to the closed eyelids, after which the transducer was placed on the upper lid with the examiner's hand resting on the orbital margin. The optic nerve appears as a hypoechogenic structure beyond the retina and optic disc, and provides an anatomical landmark for the ultrasound examination. The region of interest for an investigation of raised ICP is the optic nerve sheath (ONS), which is demonstrated as a thin bilateral hyperechogenic line surrounding the hypoechogenic optic nerve. The ONSD is measured 3 mm behind the optic disc by measuring the distance between the hyperechogenic borders of the ONS (illustration: Fig 1A, ultrasound: Fig 1B). For safety considerations and machine parameters, see Ref 11.

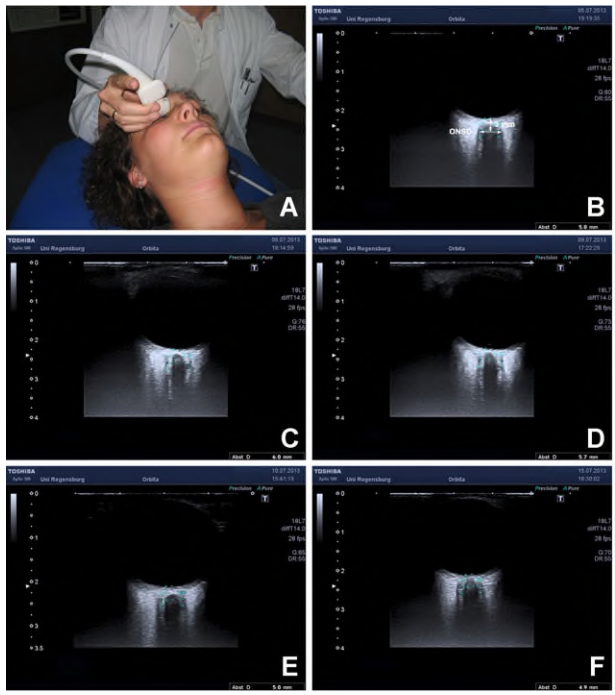

Fig 1. (A) Illustration of the anatomic retrobulbar structures in relation to an ultrasound transducer (© M. Ertl). (B) The ONSD is measured 3 mm behind the optic disc by measuring the distance between the hyperechogenic borders of the ONS. (B–F) Left eye: development of ONSD of a representative patient during inpatient stay. A relevant increase of ONSD could be detected at post 1 with a gradual decrease over the time, reaching baseline values once again before discharge.

Statistical analysis was performed by using Microsoft Office Excel 2007 (Microsoft Corporation, Redmond, WA, USA) and SPSS 13.0 (SPSS Inc, Chicago, IL, USA). The statistical significance of different ONSD values after TEVAR in comparison to baseline values was calculated using the Wilcoxon test. A P value $<.05$ was considered statistically significant, a P value of $<.01$ represents a highly significant difference. Correlations were calculated with Pearson's correlation coefficient, with a positive correlation defined as $P < .05$.

Results

In ONSD measurements, each eye was evaluated separately. At each time point, measurements were taken six times: minimum and maximum values, mean values, and standard deviations are listed in Table 1. High interindividual differences could be detected at all time points (range 3.5-6.5 mm [left eye], 3.7-6.7 mm [right eye]).

Analysis of the Complete Study Population

Statistical comparison of mean values (both eyes) did not show any significant differences, regardless of the time point, at which they were recorded.

Table 1. Tables show Minimum, Maximum, and Mean Values of ONSD

	Minimum	Maximum	Mean	Standard Deviation	Confidence Interval (95%)
***n* = 28 (Left Eye)**					
Baseline	4.0	5.8	4.851	.3979	4.784-4.917
Post 1	3.9	6.5	5.119	.6453	5.014-5.225
Post 2	3.5	6.5	4.967	.6851	4.855-5.078
Post 3	3.9	6.2	4.856	.5708	4.763-4.950
Post 4	4.1	6.5	4.882	.5224	4.796-4.969
***n* = 28 (Right Eye)**					
Baseline	3.8	5.7	4.861	.4104	4.793-4.930
Post 1	4.0	6.4	5.030	.5703	4.937-5.123
Post 2	3.8	6.4	4.915	.6097	4.817-5.014
Post 3	3.9	6.2	4.819	.5468	4.729-4.908
Post 4	3.7	6.7	4.829	.5501	4.738-4.920

Values are given in mm.

Table 2. Comparison of Baseline and Post 1–ONSD on both Eyes

ONSD	Baseline	Post 1	Δ	Percentage Increase	Significance
Right	4.861	5.030	.169	3.47	$P < .01$
Left	4.851	5.119	.268	5.52	$P = .021$

Paired comparison of ONSDs revealed highly significant differences between baseline and post 1-measurements (right eye: baseline: mean = 4.861 mm, post 1: mean = 5.03 mm; $P = .006$; left eye: baseline: mean = 4.851 mm, post 1: mean = 5.119 mm; $P = .02$, see Table 2). At all other time points, there were no significant differences between baseline measurements. A box-plot diagram depicts the gradual decrease of ONSD to baseline values during the time course of consecutive measurements (Fig 2A). A highly significant decrease was detected between post 1 and post 3 (right eye: post 1: mean = 5.03 mm; post 3: mean = 4.82 mm, $P = .02$; left eye: post 1: mean = 5.12 mm; post 3: mean = 4.86 mm, $P < .01$, see Table 3), that is, 48 hours after the first postoperative measurement (Fig 2B). At this time, 27 of 28 patients were extubated breathing spontaneously. Consecutive ultrasound examinations of a representative patient are depicted in Figure 1B–F.

Analysis of Subgroups

Apart from the above-mentioned ONSD differences, there was a group of 5 patients with an additional increase of ONSD from post 1 to post 2 (post 2 > post 1), which was statically not significant. Seven patients, however, showed a highly significant decrease in ONSD comparing post 1- and baseline values (post 1 < baseline, $P < .01$). Respective data are depicted in Figure 3A and B. One of the patients with a further diameter increase at 48-hour post-TEVAR developed an irreversible paraplegia due to SCI despite the application of a spinal drain.

Credibility of ONSD Measurements

As mentioned in the in the "Methods" section, a high interrater reliability of ONSD measurements could already be demonstrated.[14] Blinded assessment of the measurements taken by the main examiner revealed correct values. Intrarater variations were minimized by taking the mean value of six measurements.

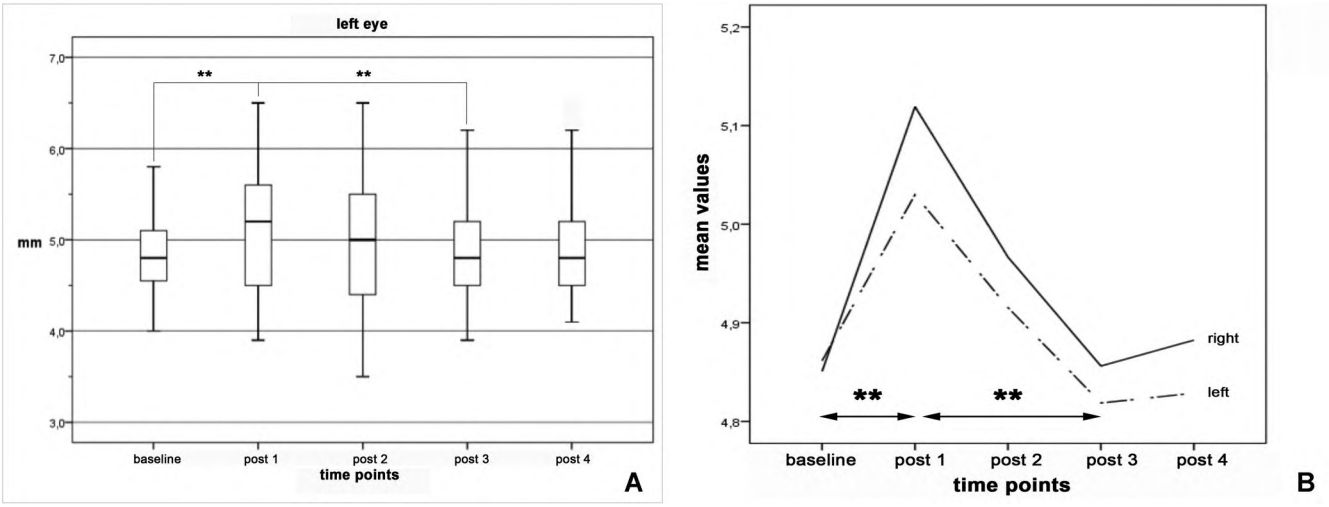

Fig 2. (A) Left eye: Box-plot diagram with mean values of ONSD. Values are calculated in mm. *X*-axis: measurement time points, *Y*-axis: ONSD in mm. **Highly significant differences. (B) Line diagram of the left and right eye with mean values of ONSD. Values are calculated in mm. *X*-axis: measurement time points, *Y*-axis: ONSD in mm. **Highly significant differences.

Table 3. Comparison of Post 1 and Post 3–ONSD on both Eyes

ONSD	Post 1	Post 3	Δ	Percentage Increase	Significance
Right	5.030	4.819	.211	3.98	$P = .02$
Left	5.119	4.856	.263	5.13	$P < .01$

Ventilation/Sedation

Comparing the ventilated with the extubated patients did not reveal any significant differences of ONSD values at any time (Fig 4A). After extubation and removal of the spinal drain, consecutive measurements showed a further decrease in ONSD independent from artificial ventilation parameters.

Spinal Drainage

As before ONSD values on each eye were compared, now in patients with or without spinal catheter. Despite the small sample size, significantly lower values were detected in patients with a spinal drain at nearly all time points (for details, see Table 4, Fig 4B), especially on the left eye with similar results on the right side (data not shown). In patients with spinal catheter, the ONSD increase in post 1 compared to baseline was also highly significant (right eye: baseline: mean = 4.8 mm; post 1: mean = 5.01 mm, $P = .016$; left eye: baseline: mean = 4.8 mm; post 3: mean = 5.03 mm, $P = .001$).

A positive correlation with the amount of drained CSF and ONSD differences over the time could only be detected in the subgroup of patients with further enlargement of ONSD at post 2 (post 2 > post 1) and only on the left eye ($P = .09$). Removal of the spinal drain did not lead to another increase in ONSD at any time.

Postoperative Complications

After TEVAR, the following neurological disorders could be examined: 1 patient with persistent paraplegia (respective ONSD values: baseline: 4.9 mm; post 1: 5.4 mm; post 2: 5.5 mm; post 3: 5.1; post 4: 4.4 mm) and 2 patients with hypesthesia at the bottom of both feet, one of them remittent before discharge. All other patients did not suffer from any postoperative deficits.

Discussion

The present prospective "proof of principle" study was destined to examine the possibility to evaluate the ISP in patients designated to TEVAR noninvasively by using OS. OS has so far been validated as a tool to reliably monitor changes of ICP caused by a variety of diseases leading to elevated ICP.[15–18]

In our study, a highly significant increase of the mean ONSD could be registered directly after postoperative arrival at the ICU compared to baseline values gathered before the intervention. The identical observation could be made in the subgroup of patients with spinal catheters, which were open at any time. The most likely explanation is an increase of the ISP caused by an ischemia related edema of the spinal cord in combination with a reperfusion edema after partial reconstitution of the spinal microcirculation.[19] The vascular supply of the spinal cord is complex and can be compromised by implantation of an endovascular aortic stent as the spinal cord receives segmental contributions from intercostal arteries. A long-segment thoracic aortic coverage had been widely identified as a significant risk factor in predicting clinically evident SCI.[7,8,20,21] Moreover, the involvement of multiple vascular territories was identified as an additional risk factor for SCI.[22]

Within 24 hours after the first postoperative measurement, a significant decrease of the mean ONSD was detected. This effect might partly be explained by CSF-pressure reduction via the spinal catheter, which obviously is only one component. One argument for additional ones is the fact, that patients with spinal catheters certainly had lower ONSD values at any time, but presented the same diameter curve as patients without a spinal drain. Second, ONSD values further decreased spontaneously after the spinal catheters were removed. The reason might be a combination of ISP regression due to partial resolution of the intramedullar edema and a higher net efflux fraction of the CSF reabsorbed into the venous system by a change of the pressure gradient. In contrast to intracranial pathologies, where venous drainage pathways might be also compressed,

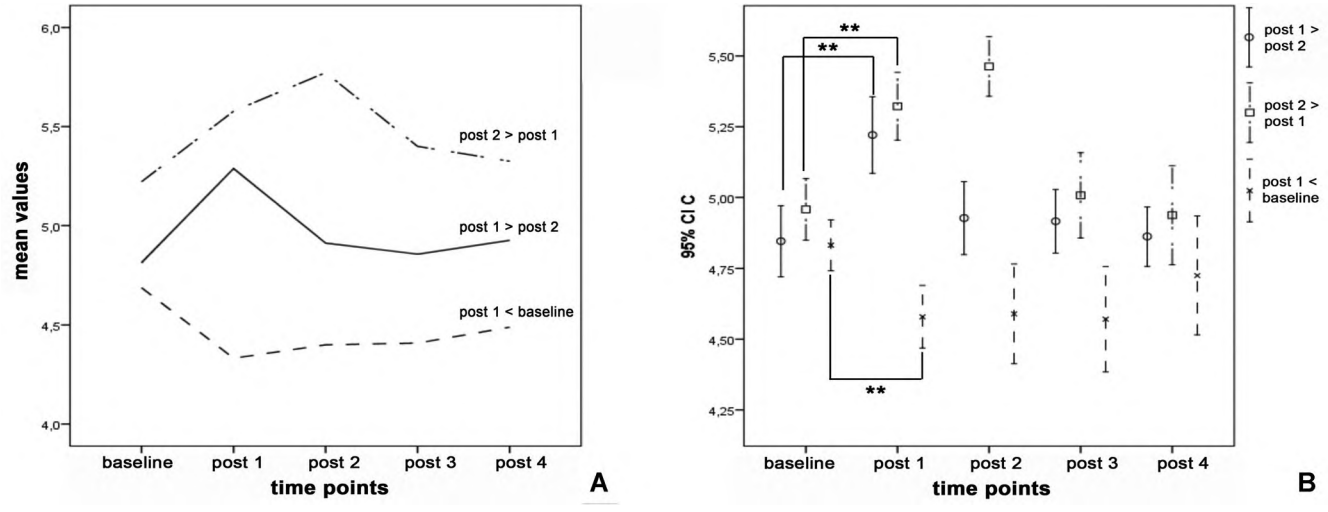

Fig 3. (A) Diagram of subgroup analysis. Depicted are different characteristics of the groups post 1 > post 2, post 2 > post 1, and post 1 < baseline. *X*-axis: measurement time points, *Y*-axis: ONSD in mm. (B) Mean values and 95% confidence intervals of subgroups post 1 > post 2, post 2 > post 1, and post 1 < baseline. **Highly significant differences.

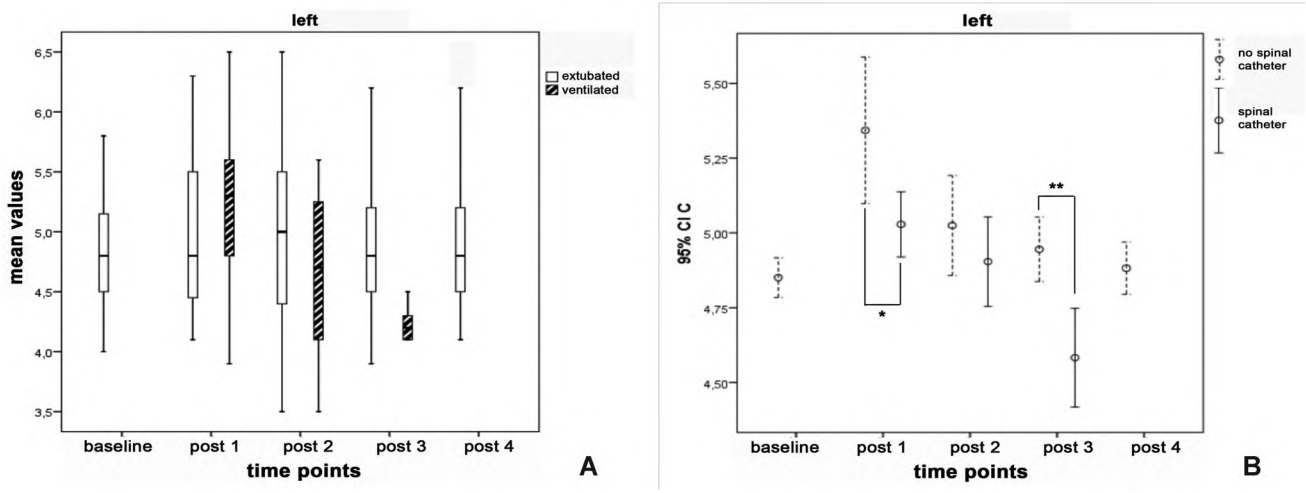

Fig 4. (A) Box-plot diagram of ONSD values dependent on artificial ventilation at consecutive time points. No significant differences were detected. (B) Mean values and 95% confidence intervals dependent on the presence of a spinal catheter. *Significant differences; **highly significant differences.

Table 4. Left eye: Mean values, Standard deviation, 95% Confidence Intervals, and Significance of ONSD in Patients with (+) and without (–) Spinal Catheter (SPC)

ONSD	Mean	Standard Deviation	Confidence Interval (95%)	Significance
Post 1 – SPC	5.343	.7853	5.098-5.588	
Post 1 + SPC	5.029	.5587	4.920-5.138	$P = .04$
Post 2 – SPC	5.025	.7321	4.858-5.192	
Post 2 + SPC	4.904	.6301	4.755-5.053	n. s.
Post 3 – SPC	4.945	.5695	4.838-5.053	
Post 3 + SPC	4.583	.4879	4.418-4.748	$P = .004$

n.s. = not significant.

an isolated spinal pressure increase does not lead to an affection of physiological intracranial CSF-flow. A subset of patients with spinal catheters, however, did not show a postinterventional ONSD increase, but post 1 values were even smaller than baseline. This could, therefore, be a population of patients without significant spinal edema, who potentially do not profit from a spinal drain and could be spared from possibly harmful complications of the spinal catheter itself. In another subgroup of patients with even higher ONSDs in post 2 measurements compared to baseline and post 1, there was 1 patient with an irreversible paraplegia. Of course, this is not sufficient to draw any conclusions, but in theory, a prolonged increase of ONSD despite a functional spinal drain could identify "high risk" patients for permanent SCI. This hypothesis needs further to be investigated in following trials.

To prove a direct relation between ONSD values and ISP alterations, it is mandatory to rule out the effect of other influential factors. One might be the application of PEEP, while the patients were artificially ventilated. This issue is discussed controversially in the literature. Most of the studies, however, did not find an effect of PEEP on the intracranial pressure, as long as the values did not exceed 12-15 mm Hg.[23–25] In the present patient population, a mean PEEP of 6.8 mm Hg with a maximum value of 7.5 mm Hg was applied. At the point of the first postoperative measurement (post 1), 8 of 28 patients (29%) were already extubated. In this study, there was no significant ONSD difference comparing ventilated and nonventilated patients at any time. A significant effect of PEEP, thus, seems very unlikely. Another systematic factor might have been the body position of the patients. So far, there are only very limited data about the influence of the position on the ONSD. Kim et al examined the ONSD in patients with laparascopic prostatectomy dependent on the position and the iatrogenic pneumoperitoneum. The comparison of ONSDs examined 10 minutes after intubation in a sitting position to the values with pneumoperitoneum in Trendelenburg's position thereafter revealed an increase of ONSD of 12.5%.[26] However, it was not clear, if the position alone or the combination with the pneumoperitoneum caused the effect, an influence of the body position seems likely. As all patients in our study were examined in a supine position at all time points, a systematic failure due to a change in body position can be ruled out. In our opinion, there were no further influential factors, which might have distorted the effect of ISP alterations on the ONSD.

To the best of our knowledge, this study is the first to prospectively examine and prove the possibility to monitor ICP changes in patients with TEVAR-associated transient spinal edema. The next step would necessarily be a study to evaluate possible effects on the clinical outcome and the possibility to potentially identify "high-risk" patients, who could benefit from a more aggressive CSF drainage, for example. In the present patient population, there was one patient with a complication of postoperative paraplegia. A statistical comparison of the respective ONSD values with those of the not affected patients would obviously not have been legitimate. The identification

of a certain cutoff value, which could support the decision to open the spinal drain, was not one of the purposes of this study and might be a general problem. According to other ONSD studies,[14,27,28] we also found a high interindividual spread of baseline and consecutive values explaining the variance also depicted in our box-plot diagrams. The bottom line, therefore, is the importance of consecutive measurements and the comparison to baseline values.

We recognize several shortcomings of this study: the lack of a control group without endovascular stent implantation and the first examiner, not having been blinded for the pathology and specific stent configuration for example. Only the second examiner, who confirmed the accuracy of the ONSD measurements, was blinded regarding specific patient information. We relinquished to examine a correlation of the ISP values with the ONSD, as there already exists sufficient evidence of correlation with raised intracranial pressure.[14,29,30] Another aspect of ONSD dynamics might not only be the absolute values at different time points but also the velocity of the changes. As we could demonstrate, the most striking relative differences occurred in the period between baseline measurements and 48 hours thereafter. The contraction of measurement intervals in that period and the application of intraoperative examinations might be helpful to learn more about the immediate pressure dynamics in different patients. One hypothesis might be that patients with a quick increase of ONSD might be more prone to clinically relevant spinal edema.

In conclusion, this study was able to show that changes of the ISP caused be an endovascular aortic stent application can be noninvasively estimated by using OS. The relevance for the clinical decision-making process should be evaluated in further studies.

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