Neurophysiological monitoring during thoracoabdominal aortic endovascular stent graft implantation

Ernst Weigang\textsuperscript{a,1,*}, Marc Hartert\textsuperscript{a,1}, Michael P. Siegenthaler\textsuperscript{a}, Katrin Pitzer-Hartert\textsuperscript{a}, Maximilian Luehr\textsuperscript{a}, Ronen Sirca\textsuperscript{b}, Patrick von Samson\textsuperscript{a}, Friedhelm Beyersdorf\textsuperscript{a}

\textsuperscript{a}Department of Cardiovascular Surgery, University Hospital Freiburg, Hugstetter Strasse 55, 79106 Freiburg, Germany
\textsuperscript{b}Department of Neurosurgery, University Hospital Freiburg, Germany

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Abstract

Objective: The aim of this study was to evaluate the benefit of neurophysiological monitoring during thoracic and thoracoabdominal endovascular stent graft implantation.

Methods: The spinal cords of 21 patients undergoing endovascular stent graft implantation on the thoracic and thoracoabdominal aorta were monitored with transcranial motor-evoked potentials (tcMEP) and somatosensory-evoked potentials (SSEP). All patients underwent mild systemic hypothermia (34—35 °C), constant cerebrospinal fluid (CSF) pressure and vital parameter monitoring. If CSF pressure exceeded 15 mmHg, CSF-drainage was carried out.

Results: Three of the 21 patients (14%) exhibited short-term loss of tcMEP and SSEP after the deployment of the self-expanding endoprosthesis. We observed an intraoperative recovery of the evoked potentials in all cases. CSF-drainage was necessary in three of them. One patient, whose potentials were stable intraoperatively, developed paraparesis 3 weeks after the intervention.

Conclusions: Neurophysiological monitoring has proved to be an ideal monitoring method to detect spinal cord ischemia during thoracic and thoracoabdominal endovascular stent graft implantation. Due to the advantages of endovascular therapy (no aortic cross-clamping, continuous distal perfusion, and no reperfusion injury), changes in potentials were seldom observed.

Keywords: Aortic surgery; Endovascular stent graft implantation; Thoracoabdominal aorta; Spinal cord protection; Neurophysiological monitoring

1. Introduction

The main danger of the aortic operation does not come from the heart or from the aorta itself, but from the central nervous system [1].

Spinal cord ischemia is the most dreaded complication of thoracoabdominal aortic aneurysm (TAA) surgery. Despite improvements in surgical technique and the use of complementary methods for spinal cord protection, the risk of postoperative neurological deficits remains significant. Due to its multifactorial etiology, various spinal cord-preserving strategies have been developed in order to improve clinical results. Three main factors contribute to spinal cord injury: ischemia during aortic cross-clamping, unsuccessful reattachment of spinal cord-perfusing arteries, and cytotoxic damage caused by hypotension and reperfusion injury [2]. On account of the experiences of former studies [3—9], neurophysiologically monitored open surgery of TAA has proven to be of positive neurological outcome. However, the open surgical technique is associated with the severe risk of paraplegia and mortality. The implantation of endovascular stent grafts enhances the general prospects of a positive outcome for patients undergoing TAA repair. Due to its limited influence on the patient’s perfusion physiology, stent graft implantation comprises three main advantages: (1) aortic cross-clamping is unnecessary, and thus the negative side effects on cerebrospinal perfusion are avoided. (2) The distal aortic perfusion remains uninterrupted, guaranteeing a continuous blood flow. (3) By neglecting segmental arteries during the phase of reimplantation, we minimize the risk of reperfusion injury. The pathophysiological mechanism underlying the occurrence of spinal cord ischemia after thoracic endovascular aortic repair requires future investigation.

Up to now, only a few studies have reported about the risk of paraparesis or paraplegia after TAA-endovascular stent graft implantation [10—13]. None of them analyze the use of intraoperative, neurophysiological monitoring to detect spinal cord ischemia. In this study, we assess the efficacy
of transcranial motor-evoked potentials (tcMEP) and somatosensory-evoked potentials (SSEP) as measures to reduce the prevalence of spinal cord ischemia during endovascular stent graft implantation. In addition, we illustrate our most recent 5-year experience with TAA-endovascular stent graft implantation and assess the efficacy of prophylactic adjuncts and therapeutic interventions.

2. Materials and methods

Between December 2000 and April 2005, 21 patients (Table 1) underwent neurophysiological monitoring during endoluminal exclusion of their TAA. The 21 patients can be categorized according to the Crawford Classification (CC) system as follows: CC Type I = 7 patients, CC Type II = 9 patients, and CC Type III = 5 patients. Preoperative spiral computed tomography (CT) scanning and angiography were applied to all patients to determine aortic anatomy and obtain measurements for endograft sizing (Fig. 1). All TAA repairs were performed in the operating room. Endografts (Talent, Medtronic AVE, Minneapolis, USA; MN/TAG Excluder, W.L. Gore and Associates, Flagstaff, AZ, USA; Zenith, Cook Group Inc., Bloomington, IN, USA) were placed via femoral arterial access under fluoroscopic guidance.

The night before surgery, all 21 patients underwent lumbar drainage to monitor and regulate cerebrospinal fluid (CSF) pressure followed by routine monitoring until the third postoperative day. The patients were placed on their backs. After exposing the femoral artery and correctly positioning the self-expanding endoprosthesis was deployed in the aneurysmal region of the thoracoabdominal aorta. Intraoperative angiography was used for guidance and confirmation of the stent graft position, excluding endoleaks and dislocation.

Fourteen of the 21 patients were classified as Crawford Type II or III. We performed a bypass on the visceral and renal arteries in 5 of the 14 patients (Table 2) to ensure a sufficient visceral organ perfusion. Access was achieved through an anterior midline, transperitoneal incision. In 9 of the 14 patients, we were able to keep the visceral and renal arteries uninvolved due to a limited regional aortic dilatation. These patients underwent close-meshed controls and — in case of critical dilatation — were treated with a second surgical intervention including a stent graft implantation as well as a visceral and renal arteries bypass. An additional supraaortic bypass was necessary in three patients. Access was achieved via median sternotomy.

2.1. Neurophysiological monitoring

The additionally applied neurophysiological monitoring serves as a control mechanism to identify spinal cord ischemia. It reveals pathologically relevant changes in the functional integrity of neural tissue and creates the possibility for immediate intervention to restore the physiological situation. TcMEP are applied to observe the descending pathways. A motor reaction occurs in the peripheral regions after transcranial stimulation of the motor cortex. SSEP are used to assess the ascending pathways. The electroencephalogram (EEG) output is continuously measured following electrical stimulation of peripheral muscles. Both monitoring methods assess the central gray matter of the spinal cord and have a complementary controlling character. They gauge different anatomical spinal cord structures: tcMEP recordings are sensitive to the corticospinal system, and SSEP recordings document the activity of the posterior and lateral columns of the spinal cord. Using the 10—20 system for EEG recordings,

![Fig. 1. (a) Preoperative angiography. Thoracoabdominal aortic aneurysm with a main dilatation within the distal region of the descending aorta and below the celiac axis. (b) Postoperative CT-image (3D-reconstruction). Endoluminal implantation of two coated stent grafts and an additional iliac-hepatic bypass of the celiac axis.](image-url)
tcMEP stimulation electrodes are attached percutaneously to the C3/C4 region of the motor cortex, and SSEP recording electrodes are positioned at Cz/Fz [14]. TcMEP recording sites on the leg are located at the anterior tibial muscle and gastrocnemius muscle. SSEP stimulating electrodes are laterally and caudally anchored to the medial malleolus in order to excite the tibial nerve (EWACS and ISIS IOM, inomed Medizintechnik GmbH, Teningen, Germany) (Fig. 2).

The tcMEP stimulation impulse consists of a train of five anodal pulses of 200–400 μs duration, spaced by 2–4.5 ms (200–500 Hz). They are executed every 30–60 s by a constant current electrical stimulator (Digitimer D57H, Digitimer Ltd., Welwyn Garden City, Hertfordshire, UK). Earthing occurs through a percutaneous needle electrode fixed between the stimulating and recording site (preferably in the knee region). The resulting electromyographic response does not require signal averaging. To change the stimulation side, we alter the polarity of the stimulus. The basic method to gain SSEP recordings is signal averaging due to the relatively long distance between the recording electrodes and the somatosensory cortex. The system registers the response of 200 consecutive stimuli as well as background noise, and filters the results in order to visualize and interpret the potentials. After the patient has been anesthetized, a baseline recording should be made. The baseline guarantees a correct definition of the patient’s individual neurophysiological output. It is important to obtain the baseline recordings prior to surgery, as interferences from technical equipment preclude the making of viable recordings. The comparison between intraoperatively gained potentials and the patient’s individual baseline values enables the neurophysiological monitoring team to attain an assessment of the current spinal cord function.

2.2. Spinal cord-protecting modalities

A set of spinal cord-protecting modalities improves the neurological results. A perioperative systemic mild hypothermia (34–35 °C) reduces the oxygen demand of the neural tissue (5%/°C) [15]. We perform liquor drainage if the intracranial pressure (ICP) exceeds 15 mmHg. Another important aim was the restoration of a sufficient spinal perfusion pressure. Our objective was to raise mean aortic pressure (MAP) above 60 mmHg via application of noradrenaline. The central venous pressure (CVP) was reduced below 12 mmHg via application of nitroglycerine and restrictive volume management.

2.3. Anesthesia technique

Close collaboration between the neurophysiologist and anesthetist during TAA repair is of vital importance, as complete neuromuscular blockade conflicts with tcMEP monitoring [16,17]. Therefore, vecuronium was applied as a short-term muscle relaxant only once at the beginning of general anesthesia. Benzodiazepine (0.01–0.03 mg/kg body weight) was given for sedation and fentanyl (0.004–0.007 mg/kg body weight) administered as an analgesic. Patients were not given further relaxants or oral anesthetics during TAA repair to prevent major interference with the neurophysiological monitoring.

Postoperative continuation of neurophysiological monitoring during the first hours of ICU stay is an important tool to

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### Table 2

<table>
<thead>
<tr>
<th>CCT</th>
<th>Patients</th>
<th>VRA bypass</th>
<th>SAV bypass</th>
<th>Intraoperative EP loss</th>
<th>Postoperative outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>7</td>
<td></td>
<td></td>
<td></td>
<td>No neurological deficit</td>
</tr>
<tr>
<td>II</td>
<td>9</td>
<td>4</td>
<td>1</td>
<td></td>
<td>2 no neurological deficits</td>
</tr>
<tr>
<td>III</td>
<td>5</td>
<td>1</td>
<td></td>
<td>3</td>
<td>1 paraparesis (25 days postsurgical)</td>
</tr>
</tbody>
</table>

CCT = Crawford Classification Type; VRA = visceral and renal arteries; SAV = supraaortic vessel; EP = evoked potentials.

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![Fig. 2. Topographical site of stimulation and recording electrodes of evoked potentials (tcMEP/SSEP).](image-url)
register delayed changes. It is worth noting that electrical stimulation is painful and cannot be used in postoperatively conscious, sedated patients. The extended control enhances the patient’s security, especially after intraoperative loss of SSEP/tcMEP and the assumed spinal cord ischemia, paraplegia, or paraparesis [4].

3. Results

We performed endoluminal stent graft implantation in 21 patients. TcMEP and SSEP remained intraoperatively stable in 18 of the 21 patients (86%) with no postoperative neurological deficits. We recorded a loss of potentials in 3 of the 21 patients (14%) (all CC Type II) immediately after the stent graft deployment (Table 2). This incident was accompanied by a decrease in blood pressure (34%) and heart rate (27%). Additionally, we recorded an increase in CVP (73%) and ICP (109%). The percentage refers to the patient’s initial, individual baseline values. An intraoperative recovery of tcMEP and SSEP baseline values was recorded in all three patients with loss of evoked potentials due to intraoperative interventions. An increase in blood pressure was obtained through noradrenaline; a decrease of CVP was achieved via nitroglycerine. A liquor drainage is usually applied if ICP exceeds 15 mmHg. In our case, the ICP baseline values were preoperatively registered with 8–10 mmHg. An intraoperative rise above 15 mmHg occurred in three patients and was followed by liquor drainage. No patient revealed postoperative neurological deficits, one of the three patients with intraoperative loss of evoked potentials developed paraparesis 25 days after the operation.

TcMEP react more sensitively to intraoperatively implemented interventions than SSEP. SSEP respond with delayed alterations in amplitude, latency and recovery after changes in evoked potentials. Due to intraoperatively launched interventions, we were able to register a recovery of evoked potentials.

4. Discussion

Our results emphasize the clinical advantages of tcMEP/ SSEP monitoring during endovascular thoracoabdominal aortic repair. Nevertheless, a single, direct procedure for recognizing malperfusion and ischemia of the spinal cord has not yet been established. Therefore, paraplegia can only be prevented by combining neurophysiological procedures and intraoperative vital parameter control.

Changes in tcMEP and SSEP recordings allow an early detection of spinal malperfusion resulting in spinal cord-protecting strategies to reduce the incidence of neurological complications. A distinction between the prognostic values of tcMEP loss versus SSEP loss is obligatory. TcMEP potentials make interpretation of spinal cord function possible within several minutes after intervention, and they regenerate within a short time after loss of potential. SSEP potentials gradually deteriorate, combined with a retarded restoration and an impending long-term loss even after intervention. The prognostic value of tcMEP monitoring must be considered superior to SSEP measurements because of the former’s direct and rapid response to spinal malperfusion.

The advantage of endovascular stentgrafting is its limited influence on the patient’s perfusion physiology [13]. Aortic cross-clamping and therefore proximal hypertension with its negative side effects on cerebrospinal perfusion is unnecessary. Distal aortic perfusion remains uninterrupted, guaranteeing a continuous blood flow. Reperfusion damage is impossible as no reimplantation of segmental arteries occurs. The implantation of a stent graft in the thoracic aorta might be disadvantageous for spinal cord perfusion due to an occlusion of segmental arteries. However, this factor plays a secondary role compared to the overall advantages of stentgrafting. Both the neurophysiological monitoring as well as the neurological outcome of the patients led to improved results.

Regarding the patient with temporary paraparesis 3 weeks after the intervention, we assume that the development of neurological symptoms originates from the thrombosis of a collateral vessel. Studies have underlined the importance of the individual collateral network of the spinal cord in patients with TAA-endovascular stent graft implantation [11,18,19]. An additional tool to minimize spinal cord malfunction is the use of liquor drainage at an early stage. Our experiences during open surgical TAA treatment have shown that this procedure is followed by a quick recovery of evoked potentials, and consequently, has a positive effect on the neurological outcome of our patients [8,9]. Therefore, we conclude that an early adjustment of ICP below 15 mmHg proves to be sensible also during future complex TAA-endoluminal treatment.

The clear benefits of stent graft-supported interventions result in a low incidence of changes in evoked potentials. Consequently, endovascular stent graft implantation is preferable to open TAA repair, as it lowers the rate of paraplegia and mortality [10—12]. However, endoluminal grafting does restrict the opportunity to maneuver in case of impending loss of potential. The only option would be a conversion to open surgery, i.e., stent explantation and implantation of a prosthesis with consecutive reimplantation of all critical segmental arteries. In this instance, the inguinal stent graft access would serve as a tool for swift femoro-femoral cannulation as well as cardiopulmonary bypass with cooled blood.

Monitoring the neurophysiological functions of a patient undergoing TAA repair is an ideal method to detect injuries or changes in spinal cord perfusion during complex endovascular stent graft implantation and serves as a guideline for therapeutic interventions.

References

Appendix A. Conference discussion

Dr R. Bonser (Birmingham, UK): You could say that if you had a protocol in which the blood pressure was kept high, the patient wasn’t overfilled, and you applied continuous CSF drainage, you would have already performed all the possible interventions that you could for those patients in whom you detected an abnormality with your monitoring. If those patients had been having the CSF drainage, if they had had their maintained blood pressure and their central venous pressure, what therapeutic options would still have been open to you?

Dr Weigang: You mean when we did all these interventions.

Dr Bonser: I mean once you have reset your protocol of management in terms of your blood pressure and your CSF drainage and so forth, you then don’t have many therapeutic options available, so one could choose to have either a very strict management protocol for cord protection or to monitor electrophysiologically.

Dr Weigang: We haven’t had such situations so far, but we also think in that sort of situation that you have described to take the same approach from our stent graft in the inguinal region for a cardiopulmonary bypass, and we considered taking the stent out and doing open procedure then, but it has never been done before in our institute. Nobody has described this kind of procedure before. That would be the only reasonable way to go about it.

Dr M. Turina (Zurich, Switzerland): You have stressed that there is very little literature about the subject of this paraplegia after stent graft implantation. There is a basic difference between implantation of the prosthesis and an endovascular procedure. You don’t have an instantaneous drop of pressure which occurs during surgery, when an intercostal artery is obstructed, you have just a gradual reduction, and for me it is probably the mechanism why you have such a low incidence of paraplegia. You give time for the collaterals to develop.

Dr Weigang: Yes, exactly. That’s one of the reasons here. The advantage of stent graft implantation is that there is no aortic cross-clamping necessary, so the proximal hypertension with its injurious side effects on the spinal cord never happens and distal aortic perfusion remains uninterrupted. That means a mechanism why you have such a low incidence of paraplegia. You give time for the collaterals to develop.

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