Adenosine-induced cardiac arrest and EEG changes in patients with thoracic aorta endovascular repair

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Background. We studied haemodynamic and metabolic variables, and cerebral function after cardiac arrest induced by high dose of adenosine in patients undergoing thoracic aorta endovascular repair.

Methods. Arterial blood pressure, blood gas values and EEG were recorded continuously in 15 patients undergoing anaesthesia (isoflurane) for endovascular thoracic aorta repair. Cardiac arrest was induced by different doses of adenosine (Adrekar®, Sanofi-Synthelabo, Berlin, Germany; 0.4–1.8 mg kg⁻¹ body weight). Serum concentrations of neurone-specific enolase (NSE) were determined before and after stent graft implantation. Neurological function was assessed before and after surgery.

Results. After adenosine, the heart beat stopped immediately for 18–58 s in close relation to the adenosine dose. EEG power was significantly reduced to 57%, but reached normal values within 5 min after cardiac arrest. In particular, the fast alpha- and beta-EEG-frequencies sensitively reflected patients’ EEG activity during the procedure. No intraoperative increases in NSE concentrations, and no neurological dysfunctions after surgery, were observed.

Conclusion. After adenosine-induced cardiac arrest, changes in haemodynamic variables and EEG power spectra reversed completely within 1 and 5 min, respectively, without persistent brain dysfunction after stent graft implantation.

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Endovascular reconstruction of the descending thoracic aorta offers significant advantage to conventional open surgery in a selected group of patients. However, despite significant improvement in anaesthesia, surgical techniques and postoperative care the morbidity remains high with both the techniques. Using transluminal techniques, the self-expanding stent-armed vascular endoprothesis is retrogradely passed into the aorta and the systemic pressure is then taken off the aneurysm by precise positioning of the endograft. It is useful to temporarily induce asystole, while the stent graft is being placed via the femoral artery into the thoracic aorta, to prevent distal migration of the device as a result of the propulsive flow during systole. Different methods have been used to achieve this. These include induction of hypotension [mean arterial pressure (MAP) <50 mm Hg] using nitroglycerine or nitroprusside and transient ventricular fibrillation and transient cardiac arrest using high-dose i.v. adenosine. Adenosine-induced cardiac arrest is a desirable method in patients with previous heart surgery, advanced age, or in an emergency setting if traditional open aortic reconstruction is not possible because of significant co-morbidities or the vascular anatomy demonstrates a short length of non-diseased aorta.

Adenosine is a known potent vasodilator of coronary and cerebral resistance vessels. Therapeutically, it is commonly used to treat supraventricular tachycardia. After a bolus injection, the heart rate is dose-dependently reduced until a complete AV-node blockade is reached. However, this effect on the AV node is transient and of short duration because of the rapid inactivation of adenosine in human plasma by uptake into erythrocytes and endothelial cells. In addition, experimental data in animals suggested that adenosine may be a potent endogenous, neuroprotective substance in acute and long-lasting ischaemia.

The development of computerized EEG with conversion of EEG signals by means of Fast Fourier Transformation...
Adenosine-induced cardiac arrest

(FFT), which has been implemented in the CATEEM®, EEG system, provides objective and reproducible information for continuous intraoperative EEG monitoring.

In the present study, the relationship between the duration of adenosine-induced cardiac arrest and brain function was studied intraoperatively using EEG power spectral analysis during intraluminal stent placing in thoracic aorta endovascular repair.

Methods

After having obtained written informed consent, 15 patients (Table 1) undergoing thoracic aorta endovascular repair were included in the present study. This study was approved by the local Ethics Committee (ethic votum 196/2004; Medical Faculty, University of Heidelberg, Germany) and Institutional Review Board and was performed in accordance with the standards of the Declaration of Helsinki.

Three women and twelve men were continuously monitored haemodynamically and with quantitative EEG during surgery. All patients were thoroughly investigated by a neurologist before surgery. Patients with previous neurological disease, based on NIH Stroke Scale (0–22) were excluded from the study. Because the half-life time of adenosine can be significantly prolonged in the presence of dipyridamol (Persantin®), particular care was taken to exclude all patients receiving medication interacting with adenosine.

After oral premedication with midazolam 5 mg, anaesthesia was induced with i.v. midazolam 1–3 mg, fentanyl 2–5 μg kg⁻¹, etomidate 0.15–0.3 mg kg⁻¹ and atracurium 0.5 mg kg⁻¹. After tracheal intubation, anaesthesia was maintained with nitrous oxide (50%) in oxygen and isoflurane 0.2–0.6%. Atracurium and fentanyl were subsequently administered as necessary. The lungs were mechanically ventilated to maintain normocapnia (Paco₂ 5.0–5.5 kPa). In each patient, 5-channel-ECG data and arterial pressure changes, using a 20-gauge radial artery catheter (Abbott, Wiesbaden, Germany), were continuously recorded. For safety reasons, a pacemaker (paceport®, Baxter Germany) was placed in the right ventricle via the internal jugular vein for electrical stimulation of the heart, and its function was verified by electrical stimulation. For EEG recording, an electrode cap with an adjustable head size (Electro Cap, Co., OH, USA) was placed on the patient’s head.

During surgery, arterial blood gas values (electrolytes, haemoglobin, haematocrit, arterial Po₂ and PCO₂) were measured 30 min before and after cardiac arrest using standard blood gas analysis (Radiometer Copenhagen). The neuron-specific enolase (NSE) in serum was determined at specific time points according to Schaarschmidt and colleagues using an immunoluminometric assay (Byk-Sangtec, Germany).

Heparin (3000 IE) was given 30 min before stent placement. Two minutes before inducing cardiac arrest all anaesthetized patients’ lungs were ventilated with 100% oxygen. No other drugs were given to prevent drug-dependent EEG changes. After adenosine, no other medications were used. Arterial pressure changes and EEG power were monitored continuously until the end of surgery.

Adenosine was injected via the central venous catheter to reach a high dose of adenosine at its major site of action, the AV node. From previous studies we know that a bolus of adenosine ranging from 18 to 45 mg can induce cardiac asystole for ~20 s.¹¹ The response of each individual patient to adenosine and to adenosine-induced AV-block

<table>
<thead>
<tr>
<th>Age (yr)/sex</th>
<th>Co-morbidities</th>
<th>ASA</th>
<th>Pathology/indication</th>
<th>Maximum adenosine (mg kg⁻¹)/maximum duration of cardiac arrest (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>32/M</td>
<td>Hypertension, contrast medium allergy</td>
<td>III</td>
<td>Anastomotic aneurysm after open arch repair/emergency</td>
<td>1.15/50</td>
</tr>
<tr>
<td>42/M</td>
<td>Smoking, obesity</td>
<td>III</td>
<td>Contained rupture after patch repair of isthmus stenosis</td>
<td>1.03/52</td>
</tr>
<tr>
<td>52/M</td>
<td>Hypertension, CHD, PTCA, COPD</td>
<td>IV</td>
<td>Thoracoabdominal aortic aneurysm</td>
<td>1.69/53</td>
</tr>
<tr>
<td>69/M</td>
<td>Hypertension, COPD, smoker</td>
<td>III</td>
<td>Thoracoabdominal aortic aneurysm</td>
<td>1.18/58</td>
</tr>
<tr>
<td>60/M</td>
<td>Renal cell carcinoma, hypertension, CHD</td>
<td>III</td>
<td>Chronic expanding aortic dissection type B</td>
<td>1.4/55</td>
</tr>
<tr>
<td>61/M</td>
<td>Hypertension</td>
<td>II</td>
<td>Chronic expanding aortic dissection type B</td>
<td>1.2/57</td>
</tr>
<tr>
<td>60F</td>
<td>Hypertension, CHD</td>
<td>III</td>
<td>Chronic expanding aortic dissection type B</td>
<td>0.51/35</td>
</tr>
<tr>
<td>69/M</td>
<td>CHD, smoking, AVI, TVI</td>
<td>III</td>
<td>Thoracoabdominal aortic aneurysm</td>
<td>1.69/53</td>
</tr>
<tr>
<td>62/M</td>
<td>Hypertension, CHD, COPD, obesity</td>
<td>III</td>
<td>Chronic expanding aortic dissection type B</td>
<td>1.15/56</td>
</tr>
<tr>
<td>61/M</td>
<td>Hypertension, spontaneous pneumothorax</td>
<td>II</td>
<td>Chronic expanding aortic dissection type B</td>
<td>1.4/55</td>
</tr>
<tr>
<td>81/M</td>
<td>Hypertension, hypothyroidism, AAA</td>
<td>II</td>
<td>Thoracic aortic aneurysm</td>
<td>0.95/45</td>
</tr>
<tr>
<td>59/M</td>
<td>Hypertension, CHD, cardiomyopathy, renal insufficiency, COPD</td>
<td>III</td>
<td>Thoracic aortic aneurysm</td>
<td>1.15/50</td>
</tr>
<tr>
<td>49/M</td>
<td>Previous thoracotomy</td>
<td>I</td>
<td>Patch aneurysm after repair of aortic isthmus stenosis</td>
<td>1.6/52</td>
</tr>
<tr>
<td>78/F</td>
<td>Hypertension, AVI, tachyarrhythmia</td>
<td>III</td>
<td>Chronic expanding aortic dissection type B</td>
<td>0.44/30</td>
</tr>
<tr>
<td>70/F</td>
<td>Hypertension, smoking</td>
<td>II</td>
<td>Acute complicated type B dissection</td>
<td>1.5/58</td>
</tr>
</tbody>
</table>
was tested by an initial injection of 0.4–0.6 mg kg\(^{-1}\) body weight. A high dose of adenosine (0.6–1.8 mg kg\(^{-1}\)) was then injected to induce prolonged cardiac arrest. During adenosine injections, patients were adequately ventilated using the Datex-Engström\(^\circ\) (Freiburg, Germany) ventilator. Arterial pressure and heart rate changes were monitored continuously online, and the duration for cardiac arrest was recorded.

During surgery, heart rate and systolic and diastolic blood pressures and MAPs were monitored online using DATEX-Ohmeda system (Freiburg, Germany). All the data were saved onto a computer using the program Hypnos\(^\circ\) (S. Esch, Bielefeld, Germany). The body temperature was monitored using an oesophageal temperature probe and maintained at 36.0–36.5°C using a warm touch system during surgery (Marquette Hellige GmbH, Freiburg, Germany).

The CATEEM\(^\circ\) (computer-aided topographical electroencephalometry system; Medisyst, Linden, Germany) measures cerebral activity by automatic frequency analysis of all 99 (17 real and 82 virtual) electrode positions using FFT. After placing an electrode cap to facilitate accurate electrode placement, the absolute power density (\(\mu V^2 Hz^{-1}\)) was measured continuously with 17 electrodes positioned according to the international 10:20 standard system with the central electrode Cz as the physical reference. The high input impedance of the amplifier (AC 10 MW; DC 20 MW) ensured a sufficient signal-to-noise ratio. Electrode impedance was in the range of ~5 kΩ. The transferred data were simultaneously displayed on a monitor for automatic user-defined identification and the elimination of artifacts (movements of the eyes, surgical electrocoagulation, muscular or sweat effects). The signal frequencies were analysed using the FFT, which is based on 4 s data acquisition sweeps with effective sample rate of 128 Hz. The resulting frequency spectra were divided into six frequency bands (delta: 1.25–4.50 Hz; theta: 4.75–6.75 Hz; alpha 1: 7.00–9.50 Hz; alpha 2: 9.75–12.50 Hz; beta 1: 12.75–18.50 Hz; beta 2: 18.75–35.00 Hz).

The patients were assessed postoperatively by routine CT-scans (Siemens, Germany) within 48 h. To ensure adequate sensory and motor functions and attention, language and orientation, a thorough neurological examination was performed by an independent neurologist when the patient had regained consciousness, 1 and 6 h later, and then daily until the patient was discharged from the intensive care unit or the hospital.

**Statistical analysis**

The results are expressed as mean (SD). In all patients, arterial blood pressure values and EEG data were set at 100% or zero at 10 min before adenosine administration, and all the data from subsequent measurements at 1 min intervals were related to these control values.

Differences within or between the normally distributed data were analysed by analysis of variance (ANOVA) using SPSS 12.0 version followed by post hoc Tukey test. Statistical significance was taken at \(P<0.05\). Pearson’s coefficients of correlation coefficients between the measured parameters were calculated.

**Results**

The mean (range) age of the patients was 60 (32–81) yr and mean (SD) body weight was 84 (13) kg. Arterial pressure (mean, systolic and diastolic) and heart rate were in physiological ranges before the induction of cardiac arrest by adenosine. No significant blood loss was registered during vascular surgery. Adenosine-induced cardiac arrest did not significantly affect arterial blood gas parameters during surgery (Table 2). A representative example of arterial pressure changes is shown in one patient during cardiac arrest (Fig. 1). In this patient, the heart stopped for ~40 s; systolic and diastolic arterial pressures decreased from 126 to 28 mm Hg and from 69 to 27 mm Hg, respectively.

In all patients, depending upon the dose of adenosine, the induction of cardiac arrest was associated with significant

**Table 2** Intraoperative blood gas analysis and biochemical variables. Values are mean (SD). The changes were insignificant

<table>
<thead>
<tr>
<th></th>
<th>Before heart arrest</th>
<th>After heart arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>pH</strong></td>
<td>7.35 (0.4)</td>
<td>7.37 (0.2)</td>
</tr>
<tr>
<td><strong>P(_{O2})</strong> (kPa)</td>
<td>14.9 (1.6)</td>
<td>13.0 (1.7)</td>
</tr>
<tr>
<td><strong>P(_{CO2})</strong> (kPa)</td>
<td>5.0 (0.04)</td>
<td>5.1 (0.03)</td>
</tr>
<tr>
<td><strong>HCO(_3^-)</strong> (mmol litre(^{-1}))</td>
<td>21.8 (1.3)</td>
<td>22.9 (1.3)</td>
</tr>
<tr>
<td><strong>Base excess</strong></td>
<td>−0.5 (1.2)</td>
<td>−2.8 (1.9)</td>
</tr>
<tr>
<td><strong>Haemoglobin (g litre(^{-1}))</strong></td>
<td>12.5 (2.8)</td>
<td>11.9 (3.1)</td>
</tr>
<tr>
<td><strong>Na(^+)</strong> (mmol litre(^{-1}))</td>
<td>134.7 (6.7)</td>
<td>132.3 (4.3)</td>
</tr>
<tr>
<td><strong>Ca(^2+)</strong> (mmol litre(^{-1}))</td>
<td>1.1 (0.1)</td>
<td>1.2 (0.1)</td>
</tr>
<tr>
<td><strong>K(^+)</strong> (mmol litre(^{-1}))</td>
<td>4.3 (1.0)</td>
<td>4.1 (0.9)</td>
</tr>
<tr>
<td><strong>Haematocrit (%)</strong></td>
<td>37.1 (2.7)</td>
<td>35.8 (3.7)</td>
</tr>
<tr>
<td><strong>Glucose (mg dl(^{-1}))</strong></td>
<td>117.7 (11.3)</td>
<td>129.5 (10.1)</td>
</tr>
</tbody>
</table>

**Fig 1** One example of intraoperative DATEX monitoring. Original data were documented every 10 s over a time interval of 4 min. As seen with changes in heart rate (HR), and systolic (syst.), diastolic (diast.) and mean arterial pressure (MAP) after the administration of adenosine bolus (arrow), a prolonged cardiac arrest of 40 s was reached for adequate stent implantation. P, arterial pressure.
reductions in arterial pressure (between −38 and −70%) more than 1 min duration. A close biphasic relationship between adenosine dose and duration of cardiac arrest is shown in Figure 2. In detail, for adenosine dose <0.6 mg a linear relationship was detected, whereas with higher dose a plateau was reached (curve-linear dependency). In all patients, arterial blood pressure values normalized in 59 (22) s after the heart spontaneously started to beat again, and these remained unchanged until the end of the surgical procedure.

No significant differences in intraoperative serum concentrations of NSE were obtained before or after adenosine (Table 3). An example of the original intraoperative EEG data before (Fig. 3A), during (Fig. 3B) and after (Fig. 3C) adenosine are shown in Figure 3. A transient, but significant reduction in all spectral frequencies was observed after adenosine-induced heart arrest reaching an isoelectric line. One minute after administering adenosine (Fig. 4), the fast alpha 1, alpha 2 and beta 1 EEG waves exhibited a drastic reduction (>40%) and reflected more sensitively the functional deficit in the brain than the delta and theta frequencies (<40%). Five minutes after administering adenosine, all EEG frequencies had normalized completely (Fig. 5) and remained unchanged until the end of surgery. A linear relationship between absolute EEG power spectrum and the duration of cardiac arrest was observed (Fig. 6) with correlation coefficients between −0.67 for theta and −0.8 for beta 1 frequencies (P < 0.001). No topographical differences were observed at the fronto-parieto-occipital and central electrode positions when comparing the right and left hemisphere. Thus, adenosine caused a global and transient reduction in total EEG power.

No marked signs of stroke were detected in routinely performed CT-scans within 48 h after surgery. Neurological changes were investigated after operation in the intensive care unit by an independent neurologist. NIH scale presented no marked differences in comparison with preoperative data (P < 0.05). None of the patients showed neurological deficits and changes such as aphasia, hemiparesis, changes in orientation, coordination and paraesthesia. The corneal and light reflexes were normal.

**Discussion**

Adenosine dose-dependently induced a rapid cardiac arrest during stent implantation in thoracic aorta endovascular surgery. This was paralleled by transient (<1 min) and significant (up to 70%) reductions in arterial pressure. Fast systemic regulatory mechanisms and rapid adenosine inactivation could be responsible for this short cardiac arrest. It is well known that adenosine is rapidly inactivated by its uptake into erythrocytes and endothelial cells. The half-life time of adenosine in human plasma was determined to be <10 s at therapeutic doses. Therefore, cardiac arrest can only be reached when high adenosine doses are administered by bolus injection.

A characteristic linear and curve-linear relationship was determined between changes in adenosine dose and the duration of cardiac arrest (Fig. 2). With adenosine doses below 1.0 mg kg\(^{-1}\), there was a linear relationship between the two parameters. In contrast, with higher adenosine doses (>1.0 mg kg\(^{-1}\)) a plateau was reached. Based on the linear relationship, it was calculated that adenosine dose of 0.017 mg kg\(^{-1}\) body weight is necessary to obtain cardiac arrest for 1 s. For example, in a patient weighing 80 kg the mean dose of adenosine required to reach cardiac arrest for at least 20 s in duration would be ~30 mg. In practice, however, sporadic ventricular contractions that can prevent an accurate stent graft implantation have been reported with lower adenosine doses. In three patients in the present study, three to five sporadic ventricular contractions were detected during asystole. However, these contractions were of no clinical relevance for haemodynamic changes in MAP because the vessels were maximally dilated after adenosine administration, as was also shown by Hashimoto and colleagues, and did not disturb adequate stent graft placement. From previous investigations we know that higher adenosine dosages (>30 mg) are necessary during thoracic aorta endografting and accurate stent graft placement to exclude some sporadic ventricular contractions. When a maximum 58 s of cardiac arrest was reached, the hearts started to beat again. As shown in these results, arterial pressure returned to control values in a rather short period of time (~1 min).

![Fig 2 Relationship between dose of i.v. bolus adenosine and the duration of cardiac arrest.](image_url)

**Table 3** Serum concentration of NSE. No significant changes between groups were detected

<table>
<thead>
<tr>
<th>Time After Cardiac Arrest</th>
<th>Before Heart Arrest</th>
<th>10 min After Arrest</th>
<th>20 min After Arrest</th>
<th>30 min After Arrest</th>
<th>50 min After Arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td>(ng ml(^{-1})) Mean (SD)</td>
<td>15.9 (1.7)</td>
<td>11.2 (4.1)</td>
<td>12.6 (2.9)</td>
<td>14.9 (3.4)</td>
<td>13.9 (1.6)</td>
</tr>
</tbody>
</table>
No significant overshoot in the course of arterial pressure was observed after cardiac activity had fully recovered even with high dosages of adenosine. Thus, the results with adenosine clearly demonstrate rapid and complete blockade of AV-node conduction, full reversibility of adenosine’s action within a short period of time and a close adenosine-dose dependency to the time of cardiac arrest. These characteristic features of adenosine are the major advantages of this nucleoside in comparison with induced profound hypotension by different pharmacological substances and ventricular fibrillation. Kahn and colleagues have reported that the induction of ventricular fibrillation can facilitate endovascular stent graft repair of thoracic aortic aneurysms. However, the fibrillating myocardium is characterized by enhanced oxygen consumption, thereby generating a significant mismatch in the oxygen supply-to-demand ratio of the heart. This detrimental condition is even more pronounced when the regular sinus node activity is induced by stressful defibrillation. For similar reasons we do not recommend the application of beta-blockers or potent vasodilators for inducing hypotension during endovascular stent placement. Compared with adenosine the onset and offset of the action of both types of drugs are rather slow and the precise adjustment and control of heart rate and arterial systemic blood pressure is difficult. Therefore, both alternatives are not used in our institution.

Fig 3 Example of original registration of changes in EEG parameters (CATEEM®). Intraoperative EEG changes before (A), during (B) and after (C) adenosine bolus of 120 mg. First the decrease in fast activities can be seen. Then changes in delta activity develop, including the appearance of very slow delta waves, and finally an isoelectrical EEG. The vertical line represents EEG normalization to baseline values after heart arrest.

Fig 4 Changes in EEG frequencies 1 min after adenosine bolus. Percentage (%) reduction in EEG power density compared with values before adenosine bolus administration (reference) is shown in subgroups of patients with low (0.4–0.6 mg kg⁻¹) and high (0.6–1.8 mg kg⁻¹) adenosine (ADO).

Fig 5 Time course of EEG parameters after adenosine bolus. Values before adenosine (ADO) were taken as reference (100%). Means (%) plus sd after adenosine (0.4–1.8 mg kg⁻¹ body weight) induced heart arrest. ANOVA, *P<0.05. α: alpha; β: beta; δ: delta; θ: theta frequencies.
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because of the relatively higher risk of the developing myocardial ischaemia and infarction.

Significant reduction in EEG power with fast restitution in cerebral function has been described after short cardiac arrest during defibrillator implantation by several authors.\cite{13, 14, 15, 16} To our knowledge, the present study is the first study analysing the effect of adenosine-induced cardiac arrest in relation to EEG frequencies. The used EEG methodology can reliably measure global EEG changes. One limitation of the use of a cap may be that the leads are not rigorously and precisely attached to the proper location of the scalp in every patient. However, the results of the present study demonstrate that adenosine-induced cardiac arrest caused a global, significant reduction of up to ~57% in total EEG power without topographical differences. These results are in good agreement with the studies of other investigators.\cite{13, 14, 15, 16} Therefore the limitation of the use of an EEG cap is probably of negligible consequences. In addition, the data of EEG monitoring demonstrate that the initial reduction in EEG power was completely reversible (Figs 3 and 5). Even repeated two short periods of circulatory arrest did not cause cumulative global EEG changes.

It is highly likely that changes in the EEG power in the present study were the result of low cerebral perfusion pressure after cardiac arrest. In addition, it is also possible that changes in EEG power can result from a specific action of adenosine on brain tissue itself. The action of increased endogenous or exogenous adenosine is described as being neuroprotective, especially during cerebral ischaemia acting via different neuronal and glial adenosine receptors.

In this study, adenosine-induced cardiac arrest was associated with differential effects on the spectral EEG frequencies. In particular, fast alpha and beta frequencies were more sensitive to global changes in systemic and cerebral perfusion than other EEG frequencies. Some authors propose that different EEG frequencies can correlate to definite neurotransmitter systems.\cite{18, 19, 20} Because cerebral adenosine can inhibit the release of excitatory amino acids, for example, glutamate, or antagonize dopamine, it may be postulated that changes in fast alpha 2 (dopaminergic) and beta 1 (glutamatergic) EEG frequencies are more sensitive to adenosine than other EEG frequencies.\cite{17} However, more specific pharmacological studies may be helpful to characterize the specificity of this nucleoside in inducing neuroprotective effects.

Pichlmayr and Lips\cite{24} reported that short-term changes in cerebral EEG only lead to reversible functional changes without persistent structural changes. To demonstrate the structural integrity of patients' brain tissue, NSE, one marker of neuronal integrity, was measured at different time points before and after cardiac arrest in accordance with Schaarschmidt and colleagues.\cite{10} No significant changes in serum NSE concentrations were obtained after cardiac arrest. In addition, after operation neurological assessment confirmed that none of the patients included in the present study exhibited neurological signs of ischaemia or stroke, respectively.

To draw more specific conclusions, we are currently investigating the cognitive abilities of patients before and after surgery using different psychometric tests (Nuernberger Altersinventar). Preliminary results of these investigations show no significant differences in consciousness, attention or organized thinking. In line with cognitive function, daily working activities were not disturbed up to 3 months after vascular surgery (Plaschke K and Bardenheuer HJ, unpublished observations).

In summary, we have shown that adenosine-induced cardiac arrest is an alternative technique in patients undergoing thoracic aorta endovascular repair to induce hypotensive phases without persistent cerebral disturbances because EEG changes in our study were transient, totally reversible, and short in nature, and there was adequate haemodynamic and functional stability after termination of adenosine action. Therefore, in our opinion, adenosine-induced asystole is a very useful adjunctive technique that provides unencumbered identification of important aortic branches, precise definition of the local anatomy and an ideal environment for the accurate placement of thoracic endograft systems.
Acknowledgements

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References