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CASE REPORT

Peri-operative Monitoring of an Asystolic Cardiac Arrest Requiring Cardiopulmonary Resuscitation During Carotid Endarterectomy for Symptomatic Carotid Artery Stenosis

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Introduction: A carotid endarterectomy (CEA) has certain risks, of which peri-operative cardiovascular risk is one. Peri-operative neurological monitoring can be done with electroencephalography (EEG) and transcranial Doppler (TCD). No previous reports have been published demonstrating the actual changes in cerebral and cardiac activity during a peri-operative asystole.

Report: The case of a 70 year old man with a symptomatic (bilateral) carotid stenosis is described. The patient complained of amaurosis fugax in both eyes. Duplex ultrasound showed a stenosis of >70% in both carotid arteries. The most severe symptoms were on the right side, so a staged approach was chosen, starting with a right sided eversion CEA (eCEA). Peri-operatively, the patient experienced an asystolic cardiac arrest after external carotid artery revascularisation, requiring brief cardiopulmonary resuscitation, which was recorded on the EEG. Post-operatively, the patient recovered fully, with no post-operative neurological or cardiac sequelae. The (symptomatic) contralateral stenosis was treated conservatively with best medical therapy (BMT; dual antiplatelets and statin). The patient is currently in good clinical condition, 1.5 years later.

Conclusion: This case shows the unique EEG recording of a cardiological event during eCEA. The cause of asystole was most likely a vasovagal syncope as a result of the surgical procedure by iatrogenic damage to the carotid sinus fibres, causing impairment of the baroreflex and chemoreflex mechanisms, which is greater during eCEA. The unilateral eCEA and contralateral BMT in this symptomatic (bilateral) stenosis seemed appropriate when cardiological risk was increased but follow up ruled out any cardiological cause.

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INTRODUCTION

The annual stroke rate in Europe is around 1.4 million, with a mortality rate of 1.1 million per year. Approximately 10%–15% of all strokes are caused by a thromboembolism from an internal carotid artery stenosis that was previously asymptomatic.[1] Patients with bilateral carotid occlusive disease have a higher risk of cerebral infarction and accompanying higher peri-operative risk, which is currently <5%.[2] Peri-operative neurological monitoring can be done with electroencephalography (EEG) and/or transcranial Doppler (TCD). In the current literature no reports of recorded brain activity on the EEG during asystole have been described.

In this report, the case of a man with (symptomatic) bilateral carotid stenosis is described. He had a peri-operative asystolic cardiac arrest for which cardiopulmonary resuscitation (CPR) was performed, all of which was registered on the EEG. This case gives a unique and exact insight into neurological status during and directly after CPR.

Informed consent was obtained from the patient involved.

CASE REPORT

A 70 year old male patient presented to the Emergency Department with complaints of bilateral amaurosis fugax in the previous week, twice on the right side and once on the left. The first event occurred one week prior to the hospital visit. The complaints persisted for about five minutes each and the patient recovered without symptoms. Neurological

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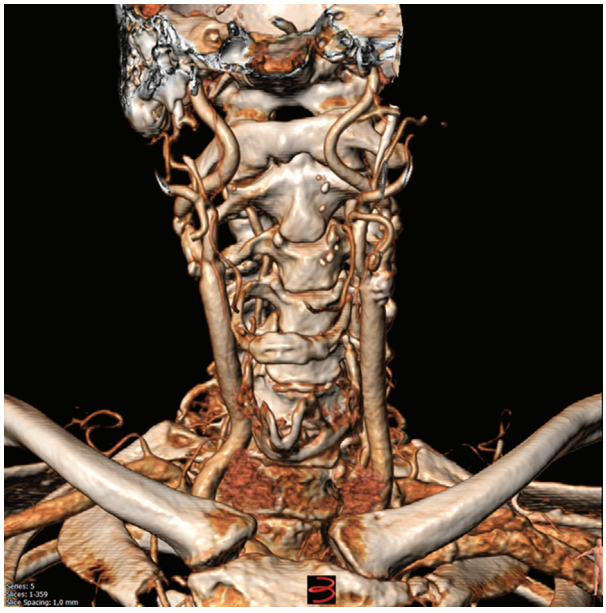

Figure 1. Three dimensional reconstruction of the bilateral carotid artery stenoses.

and physical examination did not reveal any abnormalities. His past medical history consisted of a history of smoking (total of 100 pack years: two packs per day for 50 years), hypertension, and hypercholesterolaemia.

At the time of presentation, the patient was not on any antiplatelet medication. Laboratory results were normal. A contrast enhanced computed tomography scan showed bilateral internal carotid artery stenoses (Fig. 1), with no signs of intracranial haemorrhage or ischaemia. Duplex ultrasound showed a significant bilateral stenosis of >70% (right: peak systolic velocity [PSV] 266 cm/second, end diastolic velocity [EDV] 71 cm/second, and internal carotid artery [ICA]/common carotid artery [CCA] ratio 3.7; left: PSV 332 cm/second, EDV 90 cm/second, and ICA/CCA ratio 5.4), without signs of near occlusion. The patient was started on dual antiplatelet therapy (calcium carbasalate and clopidogrel) and a statin and was discussed with the multidisciplinary vascular team. A staged (two step) bilateral carotid endarterectomy (CEA) was chosen, starting with the right side. Eversion CEA (eCEA) under general anaesthesia was performed one week after clinical presentation. According to the local protocol neurological monitoring was performed with EEG and TCD. No shunt was used. After flushing of the three arteries (ICA, external carotid artery [ECA], and CCA), recirculation of the ECA was performed by removing the clamp of the CCA (at this time the ICA was still clamped). Within seconds the patient went into asystolic cardiac arrest, with a concomitant flat line on the electrocardiogram (ECG; Fig. 2B). After about 16 seconds a diffuse slowing of brainwave activity was registered on the EEG with a decrease in amplitude (Fig. 2C). Eight seconds later the EEG signals reduced in amplitude and frequency even further (Fig. 2D). Following a brief period of chest compressions, the ECG returned, followed by an improvement of the EEG (Fig. 2E). The amplitude recovered first, while the frequency of the EEG was still symmetrically reduced in the theta/delta range (Fig. 2F). About 40 seconds after chest compressions the EEG returned to baseline. The TCD signal was lost completely during asystole. In the meantime, the clamp on the ICA was removed. The operation was finished according to the normal CEA protocol. Details of the time frame and events are shown in Table 1.

Post-operatively, the patient was admitted to the Intensive Care Unit and his recovery was uneventful. The next day he was discharged to the Surgical Ward and transferred to the Cardiology Ward for cardiological monitoring. No cardiac abnormalities were found during the admission. The patient was discharged on post-operative day three without complications. Further cardiological follow up revealed a prolonged QTc for which the patients had multiple tests and did not reveal any other cardiological abnormalities. Further outpatient cardiology follow up is planned in one year.

Due to the unexpected asystolic cardiac arrest, the case was re-discussed within the multidisciplinary team. The main issue was whether contralateral CEA was still feasible and indicated. The consensus was to start a regimen of best medical therapy (BMT) with dual antiplatelets for three months following the last event, a statin, and to postpone any operation until new complaints might occur. This was discussed with the patient, who agreed with this policy. To the authors' knowledge, more than 1.5 years after the procedure, the patient is in good clinical condition without any new neurological events.

DISCUSSION

In this report the case of a man with a (bilateral) symptomatic carotid artery stenosis is described, for which an eCEA was performed (on the right side) and BMT was started to treat the contralateral side. Peri-operative monitoring with ECG, EEG, and TCD enabled registration of an asystolic arrest directly after reperfusion, which recovered after a short period of CPR.

Peri-operative monitoring with EEG and TCD is standard procedure in the authors' hospital when performing a CEA. In this case, a unique recording of the peri-operative asystole and an EEG recording the neurological consequences of asystole (diminished perfusion of the brain) was obtained. Most likely, the cause for this asystole was a vasovagal syncope as a result of post-endarterectomy blood pressure instability, as cardiac function was restored after brief CPR. After consultation with a cardiologist, the highest suspicion was for a vasovagal response, as no cardiac abnormalities were found during post-operative monitoring. Recent cardiological follow up revealed only a minor QTc prolongation on the ECG, most likely caused by a long QT syndrome, for which the patient is being monitored by a cardiologist. To date, the patient has not suffered from any cardiac or neurological consequences resulting from the peri-operative event.

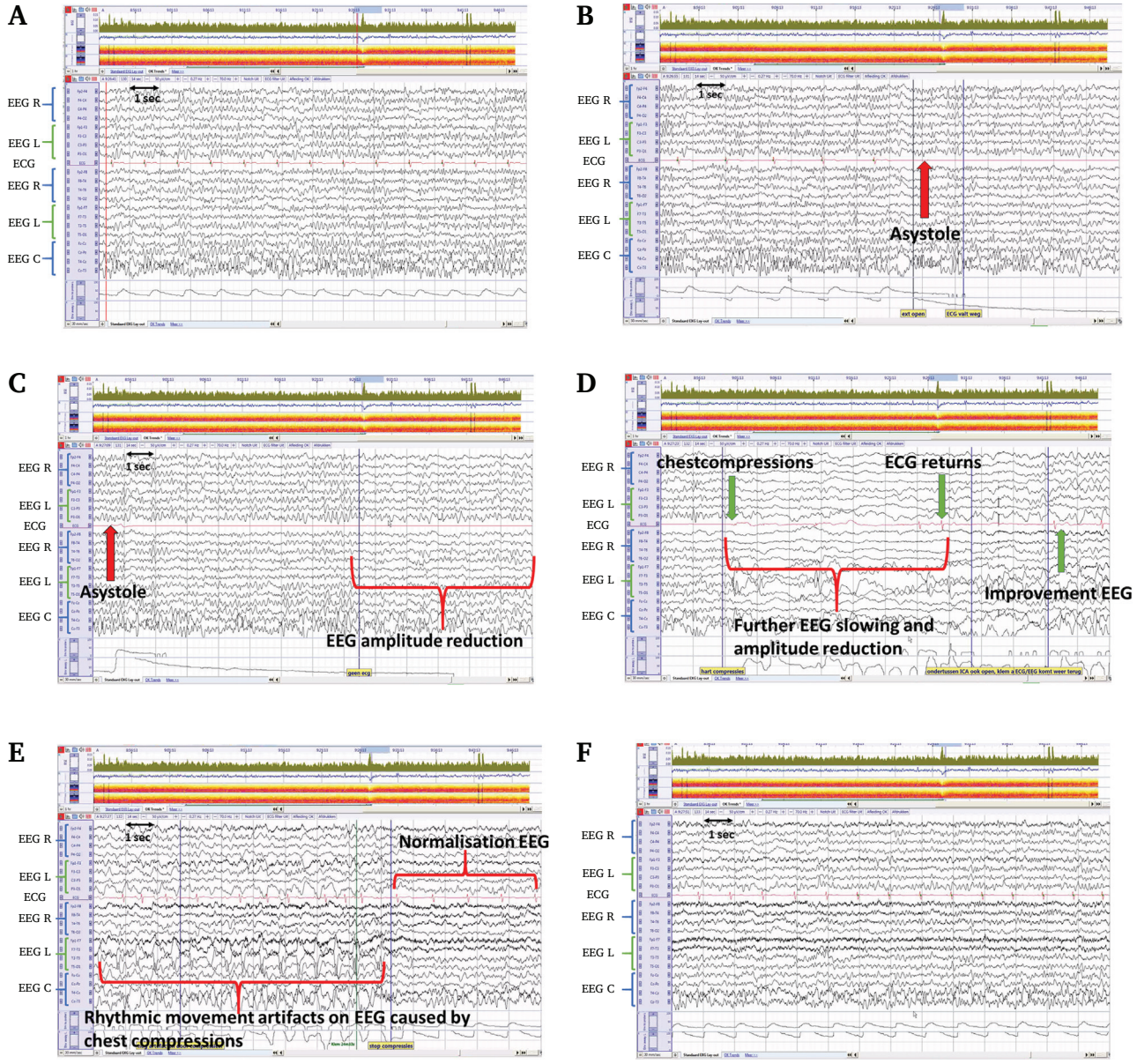

Figure 2. Electroencephalography (EEG) during intra-operative neuromonitoring. In this image a bipolar EEG derivation is used. The first four channels depict brain activity measured over the right (R) cerebral hemisphere. The next four channels show brain activity over the left (L) cerebral hemisphere. The following channel is the ECG. In the next eight channels the first four depict brain activity of the right hemisphere and the next four show brain activity of the left hemisphere. The final four channels show brainwaves measured over the central areas. In this image 14 s of EEG is shown from left to right. (A) EEG registration (09:26 h 41 s—09:26 h 55 s). (B) EEG registration (09:26 h 55 s—9:27 h 9 s) showing the asystolic cardiac arrest, with a concomitant flat line on the ECG. (C) EEG registration (09:27 h 9 s—09:27 h 23 s). About 16 s after asystole a diffuse slowing of brainwave activity was registered on the EEG with a decrease in amplitude. (D) EEG registration (09:27 h 23 s—09:27 h 37 s). Eight seconds later the EEG signals are reduced in amplitude and frequency even further. Following a brief period of chest compression, the ECG returns, followed by an improvement of the EEG. (E) EEG registration (09:27 h 37 s—99:27 h 51 s). In this image slow activity is seen about 2 Hz. This is not caused by slow brain activity but is seen as an EEG artefact caused by the chest compressions. (F) EEG registration (09:27 h 51 s—09:28 h 5 s). About 40 s after chest compression the EEG returns to baseline.

As is known from the literature, blood pressure instability or other haemodynamic changes during CEA can occur as a result of the surgical procedure by iatrogenic damage to the baroreflex response after plaque removal. The iatrogenic damage to the carotid sinus fibres could impair the baro- and chemoreflex mechanisms that the body uses to adjust the circulation and/or ventilation. Peri-operative haemodynamic derangements after eCEA can be attributed to the temporary effects of the procedure.[3,4] The impact of this haemodynamic disturbance could be greater in patients with a contralateral carotid artery stenosis (as was the case in this patient), which might lead to increased

Table 1. Time frame of recirculation and events logged on the electroencephalography registration.

Time	Event	Figure
9:27:03 AM	Recirculation external carotid artery	2B
9:27:04 AM	Loss of ECG signal/asystole	2B
9:27:25 AM	Start manual compressions	2D
9:27:35 AM	Return of ECG signal	2D
9:27:46 AM	Stop manual compressions, cardiac output	2E

ECG = electrocardiogram.

cardiovascular morbidity and mortality.[5] Specifically, eCEA could have a decreasing influence on baroreceptor sensitivity, leading to an increase in sympathetic activity in the early post-operative period, as was described by Demirel *et al.*[6] However, in this study only two patients (6%) with a severe contralateral stenosis were included and peri-operative parameters were not investigated.[6] A comparable case was published in 2019,[7] which describes a transient asystolic cardiac arrest in a patient undergoing a CEA after removal of the clamp from the CCA (as a result of hydraulic stimulation of the carotid baroreceptors). Circulation returned after the administration of atropine. One of their suggestions is a more liberal use of local anaesthetics during the procedure (i.e., prophylactically anaesthetise the carotid sinus and surrounding tissues). However, as they also mention, similar cases are likely to be extremely rare. Another well known cause of intra-operative asystole could be inadvertent clamping of the vagus nerve, making careful dissection and arterial clamping an important issue.

The best treatment strategy for patients with a symptomatic bilateral carotid artery stenosis remains complex. There are several options, among which is the choice of which side should be treated first and if a one or two step approach should be undertaken. A report from 2001 recommended a one step approach for patients with bilateral crescendo transient ischaemic attacks. The advantages of a simultaneous/one step procedure could be a single period of anaesthesia and no delay in the planning of treatment for these patients.[8] The downside of simultaneous bilateral CEA might be the increased risk of neurological complications, such as damage of the hypoglossal or recurrent laryngeal nerves, bilateral vocal cord palsy, or hyperperfusion syndrome.[9] According to the current European Society of Vascular Surgery (ESVS) guidelines and other reports in the literature, a staged CEA remains the method of choice in the management of bilateral carotid occlusive disease.[1,2] This was also confirmed by a 2015 study that showed that the existence of a contralateral occlusion or stenosis was not associated with peri-operative complications.[10]

As for the contralateral symptomatic stenosis, at the time of writing the patient is symptom free with BMT. However, as follow up ruled out any cardiological cause for the asystole, one could debate the decision for BMT. At that time another surgical procedure was cancelled owing to the potentially increased cardiological risk with subsequent risk of impaired outcome. The patient experienced amaurosis fugax and did not have a cerebrovascular accident. However, the patient was still within the symptomatic/treatable period according to the ESVS guidelines,[1] so a contralateral CEA would still have been feasible and a valid treatment option for that symptomatic stenosis. Nevertheless, this decision was the result of a shared decision making process and multidisciplinary meeting. Then again, although BMT is working for this patient at the present time, with a now asymptomatic carotid artery stenosis, the surgical indication can be revisited if the patient develops a new symptom or any of the risk factors for increased incidence of stroke for asymptomatic carotid artery stenosis, as described in the ESVS guidelines for carotid artery stenosis.[1]

Conclusion

In this report a patient with an asystolic arrest after eCEA for a symptomatic carotid artery stenosis is described. The peri-operative EEG recordings showed the asystole (for which CPR was required) and its neurological consequences. Currently, the patient is in good neurological condition. The contralateral symptomatic stenosis was treated with BMT.

CONFLICTS OF INTEREST

None.

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