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Is surgery in acute aortic dissection type A still contraindicated in the presence of preoperative neurological symptoms?[†]

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Abstract

OBJECTIVES: Severe neurological deficit (ND) due to acute aortic dissection type A (AADA) was considered a contraindication for surgery because of poor prognosis. Recently, more aggressive indication for surgery despite neurological symptoms has shown acceptable postoperative clinical results. The aim of this study was to evaluate early and mid-term outcomes of patients with AADA presenting with acute ND.

METHODS: Data from 53 patients with new-onset ND who received surgical repair for AADA between 2005 and 2012 at our institution were retrospectively reviewed. ND was defined as focal motor or sensory deficit, hemiplegia, paraplegia, convulsions or coma. Neurological symptoms were evaluated preoperatively using the Glasgow Coma Scale (GCS) and modified Rankin Scale (mRS), and at discharge as well as 3–6 months postoperatively using the mRS and National Institutes of Health Stroke Scale. Involvement of carotid arteries was assessed in the pre- and postoperative computed tomography. Logistic regression analysis was performed to detect predictive factors for recovery of ND.

RESULTS: Of the 53 patients, 29 (54.7%) showed complete recovery from focal ND at follow-up. Neurological symptoms persisted in 24 (45.3%) patients, of which 8 (33%) died without neurological assessment at follow-up. Between the two groups (patients with recovery and those with persisting ND), there was no significant difference regarding the duration of hypothermic circulatory arrest (28 ± 14 vs 36 ± 20 min) or severely reduced consciousness (GCS <8). Multivariate analysis showed significant differences for the preoperative mRS between the two groups ($P < 0.007$). A high preoperative mRS was associated with persistence of neurological symptoms ($P < 0.02$). Cardiovascular risk factors, age or involvement of supra-aortic branches were not predictive for persistence of ND.

CONCLUSION: More than half of our patients recovered completely from ND due to AADA after surgery. Severity of clinical symptoms had a predictive value. Patients suffering from AADA and presenting with ND before surgery should not be excluded from emergency surgery.

Keywords: Aortic dissection type A • Neurological deficit • Coma • Recovery • Malperfusion

INTRODUCTION

Outcome of surgical repair for acute aortic dissection type A (AADA) complicated by neurological deficit (ND) has improved significantly over the past decade, but surgeons remain hesitant to operate in view of generally poorer prognosis and difficult predictability [1–4], and some still advocate medical management or delayed surgery.

AADA is complicated in about 16–33% by malperfusion [2, 5, 6], which implies poorer prognosis [7, 8]. Neurological symptoms are observed in 17–44% [9, 10], and arise from either occlusion of the supra-aortic vessels by the dissection flap, from hypoxic

encephalopathy secondary to shock or tamponade, or thromboembolism originating from the false lumen [4]. Surgery itself, requiring anticoagulation and hypothermic circulatory arrest (HCA), additionally increases the risk of a pre-existing neurological dysfunction.

Cerebral malperfusion in patients with AADA was demonstrated to be a predictor for detrimental outcome, and thus long considered as a contraindication for emergent surgery [11, 12]. Recently, more liberal indication for surgery has been adopted, and acceptable clinical results have been reported. Several newer studies and case reports put the evidence for adverse outcome in patients with AADA and preoperative ND in question [1, 13]. Even in patients presenting in a comatose state, fortunate outcomes have been observed [13, 14]. Early diagnosis and therapy of AADA are crucial, since lack of improvement has been associated with the time to

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surgery >9 h [15], and treatment within 5 h after the onset of symptoms has been reported to result in favourable outcome [13].

The aim of our study was to evaluate early and mid-term outcomes of patients with AADA and preoperative new-onset ND and to find predictive factors, which would allow a better preoperative assessment of risk and benefit of surgical repair in these patients.

METHODS

Patients and data collection

We reviewed our institutional database identifying all patients who underwent surgical repair for AADA, diagnosed according to the Stanford Classification, between January 2005 and December 2012. All clinical data were obtained by retrospective review of hospital records. This study was approved by the Institutional Review Board of the Inselspital, University Hospital Berne. Of 300 patients treated because of AADA, 53 (17.6%) showed new-onset ND due to AADA. Patient demographics, clinical history and presentation, imaging findings, surgical management and postoperative course were available. We were not able to precisely identify the time between the onset of symptoms and surgery in the majority of the patients, but surgery was never intentionally delayed because of ND at our institution.

Neurological assessment

Neurological symptoms were assessed by the emergency medicine team, anaesthesiologist and surgeon performing the operation (Table 1), before strong sedative or narcotic medication was initiated. Neurological symptoms were evaluated using the Glasgow Coma Scale (GCS) [16] and modified Rankin Scale (mRS) [17] preoperatively (Table 1). Additionally, the initial neurological symptoms were qualitatively classified.

Postoperatively, neurological evaluation contained GCS, mRS and the National Institutes of Health Stroke Scale (NIHSS) [18] at discharge, and again at follow-up (mRS and NIHSS). The symptoms were compared with the ND described preoperatively, and a correlation between pre- and postoperative symptoms was possible in all but the 6 patients who died. Follow-up was achieved in 92.5% of discharged patients (2 died not disease-related within

the next 8 months and 2 were transferred back to their home country after discharge). Follow-up consisted of consultations at our institution including CT scan at 3 and 12 months and regularly thereafter.

Surgical management

All patients underwent median sternotomy and cardiopulmonary bypass (CPB) was initiated via right axillary artery and right atrium. Femoral artery cannulation was used in 8 patients.

The extent of aortic arch repair was adapted to the site of the intimal tear and the size of the aortic arch. An open anastomosis with replacement of the small curvature of the arch was performed in the majority of patients (Table 3). Surgical repair was performed in moderate or deep HCA in all but 2 patients. Thiopental was administered before initiation of circulatory arrest at a bladder temperature of 26°C. Acid-base balance was managed following 'alpha-stat' strategy.

During circulatory arrest, bilateral selective antegrade cerebral perfusion (ACP) was performed in all patients at a flow rate of 6–9 ml/kg/min, and cerebral oxygenation was monitored using near-infrared spectroscopy throughout the surgery since 2009.

Statistical analysis

Continuous variables are presented as mean \pm standard; categories as *n* and %. Pre- and intraoperative variables were compared with the χ^2 test for categorical and unpaired Student's *t*-test for continuous variables. *P*-values were validated with non-parametric Mann-Whitney *U*-test. Logistic regression analysis was performed with persisting neurological symptoms as a dependent variable. We included at most two independent variables into the logistic regressions with respect to the small number of patients: on the basis of the descriptive statistics, we included preoperative mRS together with age, cardiovascular risk factors, duration of HCA, involvement of the carotid arteries (dissection or occlusion) and the clinical neurological presentation preoperatively evaluated by GCS into a logistic regression analysis.

A *P*-value of <0.05 was considered significant; all *P*-values and 95% confidence intervals were two-sided. All analyses were performed using the Stata software (version 12, StataCorp., College Station, TX, USA).

RESULTS

Based on the postoperative neurological assessment, we divided our cohort into two groups. All patients without focal ND at the time of follow-up (NIHSS = 0) were considered fully recovered N0 (*n* = 29) and compared with patients with persistent ND at follow-up (NIHSS \geq 1), termed group N1 (*n* = 24). Group N1 included patients who died with signs of severe cerebral damage/oedema as assessed by CT scan or electroencephalogram but without possibility for clinical neurological assessment postoperatively.

Neurological outcome

In our cohort of patients with new preoperative ND, more than half of the patients (N0, 54%) recovered with no motor or sensory

Table 1: Neurological assessment

	N0 (<i>n</i> = 29)	N1 (<i>n</i> = 24)	<i>P</i> -value
GCS preoperative	12.5 (\pm 3.9)	10.9 (\pm 4.9)	0.17
GCS \leq 8	6 (20%)	9 (37%)	0.17
GCS postoperative	15.0 (\pm 0.0)	10.9 (\pm 5.7)	<0.001
mRS preoperative	3.3 (\pm 1.6)	4.4 (\pm 1.0)	0.007
mRS postoperative	1.1 (\pm 1.3)	4.4 (\pm 1.7)	<0.001
mRS follow-up	0.8 (\pm 0.8)	2.8 (\pm 1.5)	<0.001
NIHSS postoperative	0.5 (\pm 0.8)	17.8 (\pm 17.8)	<0.001
NIHSS follow-up	0.0 (\pm 0.0)	3.6 (\pm 4.4)	<0.001

GCS: Glasgow coma scale; mRS: modified Rankin Scale; NIHSS: National Institutes of Health Stroke Scale.

deficit according to the NIHSS at 3 months postoperatively. With respect to the predictive value of neurological status, there was no difference between the groups regarding preoperative GCS. The severity of impairment with respect to the ability to perform daily routine actions independently, assessed by mRS, was predictive of neurological recovery ($P < 0.007$) as depicted in Table 4. Patients who recovered had a significantly lower preoperative mRS score of 3.3 ± 1.6 compared with those in Group N1, who were severely impaired, exemplified by a score of 4.4 ± 1.0 . We identified an mRS of >3 as a cut-off value for the lack of neurological recovery (odds ratio [OR]: 5.3, $P = 0.01$, 95% CI 1.46–19.61).

In all patients receiving surgery for AADA within the 8-year period ($n = 300$), we observed a new postoperative stroke rate of 12%. These permanent strokes were diagnosed by postoperative CT scan and neurological assessment.

Neurological recovery

In both groups, clinical impairment improved significantly until discharge and further at follow-up (Fig. 1). Although the NIHSS in Group N0 was 0 by definition at the time of follow-up, the mRS showed minimal residual impairment in some patients due to cognitive deficit such as poor concentration leading to partial disability. In the group lacking full recovery (N1), 41% of patients showed improved symptoms at follow-up, and 25% remained similarly impaired after surgery, whereas 33% had devastating outcome and died. Of the 15 patients completing follow-up in this group, 10 had an mRS of ≤ 3 , which classifies moderate disability in the sense of 'requires some help, but able to walk unassisted'. Thus, we could assume that of all surviving patients presenting with ND, 71% were living with an acceptable neurological outcome and quality of life.

Regarding the outcome of 14 patients, who presented with a severely reduced level of consciousness (Table 1), we did not find GCS level predictive for neurological outcome (Table 4), but cannot conclude that it is not important. There was no correlation between the time from the onset of symptoms to the beginning of surgery and the severity of ND (mRS) in N1 patients either. Time to surgery was not statistically different or shorter in the comatose patients who fully recovered (N0: 3.1 ± 1.3 h vs N1: 5.8 ± 3.0 h, $P = 0.07$),

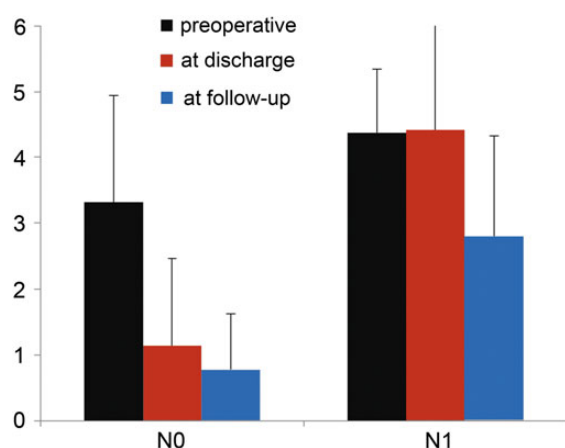


Figure 1: Development of neurological symptoms resulting in clinical impairment of patients with neurological deficit due to acute aortic dissection type A. N0: group with postoperative NIHSS = 0; N1: patients with NIHSS ≥ 1 at follow-up; mRS: modified Rankin Scale; NIHSS: National Institutes of Health Stroke Scale.

although there was a trend and the 2 patients with time to surgery of more than 8 h died with devastating cerebral damage.

Patient- and surgery-related parameters

Age and gender were not different between the two groups (Table 2). Concerning the cardiovascular risk, there was a significantly higher incidence of hypertension, smoking and family history of cardiovascular disease in Group N0. Obesity and diabetes appeared more frequently in Group N1. Cardiovascular risk factors did not predict outcome in our cohort. There was a trend towards longer operation and CPB time (Table 3), as well as the duration of HCA in patients of Group N1 ($P = 0.095$). These factors may be confounding factors, and might be explained by the fact that surgical repair could have been more complicated in these cases as a consequence of larger extent of the dissection. When mRS was adjusted for the duration of HCA, it was still a significant predictor of outcome (Table 4).

Involvement of the supra-aortic vessels

One significant difference between both groups was a higher incidence of occlusion of the right carotid artery by the dissection in the

Table 2: Patient-related parameters

	N0 (n = 29)	N1 (n = 24)	P-value
Age	58.6 (± 13.0)	61.0 (± 10.5)	0.46
Gender			0.50
Male	18 (62%)	17 (71%)	0.50
Cardiovascular risk factors			
Hypertension	22 (76%)	16 (67%)	0.07
Obesity	4 (14%)	5 (21%)	0.04
Dyslipidaemia	8 (28%)	5 (21%)	0.07
Smoking	13 (45%)	5 (21%)	0.03
Diabetes mellitus	1 (3%)	1 (4%)	0.07
Family history	8 (28%)	3 (13%)	0.05

Table 3: Surgery-related parameters

	N0 (n = 29)	N1 (n = 24)	P-value
Ascending aorta repair	1	1	
Hemi-arch repair	24 (83%)	19 (79%)	
Total arch repair	4	4	
Operation time (min)	263.8 (± 74.2)	295.8 (± 120.3)	0.25
CPB (min)	152.9 (± 52.0)	173.1 (± 85.6)	0.29
Cross-clamping time (min)	94.5 (± 35.4)	102.1 (± 51.6)	0.52
Reperfusion (min)	36.1 (± 17.5)	41.4 (± 25.8)	0.38
HCA (min)	28.4 (± 14.6)	36.6 (± 20.1)	0.09
With ACP (min)	23.3 (± 13.6)	30.6 (± 17.3)	0.09
Without ACP (min)	6.2 (± 5.8)	7.6 (± 6.8)	0.42
Hypothermia ($^{\circ}\text{C}$)	21.3 (± 3.0)	20.8 (± 2.8)	0.51
Cannulation site			
axillary	25 (86%)	19 (79%)	0.47
femoral	4 (14%)	3 (13%)	
axillary + femoral	0 (0%)	1 (4%)	

CPB: cardiopulmonary bypass; HCA: hypothermic circulatory arrest; ACP: antegrade cerebral perfusion.

Table 4: Logistic regression analysis for predictive factors of persistent neurological deficit

	Odds ratio	P-value	95% Confidence interval
mRS preoperative	2.0	0.005	1.09–3.41
GCS preoperative	1.0	0.80	0.86–1.20
GCS ≤ 8	2.3	0.18	0.67–7.79
Time of HCA	1.0	0.37	0.98–1.05
Age	1.0	0.14	0.98–1.08
Cardiovascular risk factors	0.8	0.49	0.50–1.39
Dissection/occlusion carotid arteries	1.0	0.94	0.51–2.03
Dissection and/or occlusion of right carotid artery	2.7	0.078	0.89–8.32

mRS: modified Rankin Scale; GCS: Glasgow coma scale; HCA: hypothermic circulatory arrest.

Table 5: Involvement of the carotid arteries

	N0 (n = 29)	N1 (n = 24)	P-value
Occlusion RICA	1 (4%)	5 (23%)	0.049
Dissection RICA	11 (42%)	15 (68%)	0.073
Occlusion LICA	1 (4%)	1 (5%)	0.90
Dissection LICA	9 (35%)	8 (36%)	0.90

RICA: right internal carotid artery; LICA: left internal carotid artery.

group showing persistent ND ($P < 0.05$), but it had no predictive value (Table 4). There was also a trend when taking into account dissection of the right carotid artery, but neither dissection nor occlusion of the left carotid artery was different (Table 5). We did not find a correlation between reconstitution of blood flow in the right carotid artery postoperatively and at follow-up to a favourable neurological outcome. Although symptoms of a right-sided stroke were more frequently found in the whole cohort, there was no difference between groups regarding this parameter (34% in N0 vs 46% in N1). About 51% (15 in Group N0 vs 12 in Group N1) out of all patients had preoperative cranial CT or MRI with or without angiogram, ruling out haemorrhage or demarcation of territorial stroke.

Mortality

In the complete cohort of 300 patients with AADA who received surgical repair between 2005 and 2012 at our institution, the 30-day mortality rate was 10% (32 patients). In patients with preoperative new onset of neurological symptoms, this rate increased to 15% (Table 6). At the time of follow-up, two additional deaths were recorded, and both patients died from non-disease-related causes, 1 of them residually impaired (N1).

Of the 8 patients who died in-hospital (Table 6), 6 had disease-related, neurological detrimental outcome and life-support was withdrawn. Two patients with preoperative visceral malperfusion died from disease-related multiorgan failure, and the cerebral imaging showed severe cerebral lesions as well. Thus, all deceased

Table 6: Postoperative course and mortality

	N0 (n = 29)	N1 (n = 24)	P-value
Intubation (days)	1.1 (± 1.2)	2.7 (± 2.2)	0.003
Hospitalization (days)	12.6 (± 5.1)	16.3 (± 12.1)	0.15
Mortality			
In-hospital	0 (0%)	8 (33.3%)	0.002
At follow-up	1 (3.4%)	1 (4.2%)	

patients showed devastating neurological status postoperatively, resulting from cerebral oedema and/or haemorrhage in conjunction with stroke.

DISCUSSION

Neurological symptoms are frequently found in patients with AADA, and the potential for recovery is difficult to predict: similar to the findings of larger registries and other single-centre studies [6, 19], every fifth patient presented with new onset of neurological dysfunction in our cohort. Repeated observations of neurological recovery after surgery have been made in about 50% of these patients [3, 15]. Therefore, surgeons are more likely to perform the extensive surgery necessary to repair the ascending aorta and re-establish antegrade blood flow. But, how can we distinguish patients who will recover their ND from those with detrimental outcome?

In view of the very poor prognosis of medical therapy with in-hospital mortality of more than 60% [20, 21], surgery was recently deemed appropriate even in high-risk patients such as octogenarians [22] and in those with more complex AADA (with coronary ischaemia or visceral malperfusion for instance). On the other hand, preoperative ND seemed to predict in-hospital death with an OR of 7.7 [23]. Other studies did not find preoperative ND alone to be a predictor of mortality or even of postoperative ND [6].

At our institution, the decision whether to operate on a patient presenting with ND is made by the surgeon's clinical judgement on a case-to-case basis, fairly liberal but not overly aggressive as indicated by the average GCS of 11.8 (± 4.4). This strategy resulted in an overall favourable outcome with the rate of persistent ND and mortality comparable with or even exceeding previously published results [3, 6, 8].

We identified an mRS of >3 as an independent predictor of poor neurological improvement. Similarly, Morimoto *et al.* [15] also defined an NIHSS score of >11 to be associated with an unfavourable outcome. In their study, patients in the group without recovery even deteriorated from an average NIHSS score of 17 ± 7.2 – 22 ± 14.7 . In contrast, the majority of patients in our study improved significantly over the course of the follow-up (Fig. 1). It has been suggested, in a recent publication of the German Registry for Acute Aortic Dissection Type A (GERAADA), that preoperative ND alone does not predict postoperative ND, and that malperfusion of three organ systems was predictive of persistent postoperative ND [6].

In accordance with the study by Morimoto *et al.* [15], the present analysis confirmed that cerebral malperfusion supposedly resulting from either dissection or occlusion of the carotid arteries is not predictive of persistent ND and neither is recovery associated with a higher degree of postoperative regression of the

involvement of the supra-aortic vessels. This speaks in favour of clinical assessment rather than putting too much emphasis on the radiological evidence. But, in many previous reports, cerebral malperfusion was defined as a significant risk factor for detrimental neurological outcome and mortality [1, 6]. More detailed information on intracranial cerebral vessel perfusion may provide an additional value as intracranial Doppler may become more available in the future.

As to how the immediacy of surgery influenced our results cannot be derived from our data, because we did not have sufficient data to correlate outcome with the time to surgery in all patients. It is generally accepted now that an intentional delay of surgery, which results in 15–25% mortality, cannot be advocated anymore [24].

We observed a trend towards longer operating time, including cross-clamping time, time of circulatory arrest and ACP in the N1 group, but none of these parameters were predictive for recovery. Operative procedures of longer duration suggest more extensive repair and/or a more demanding intraoperative situs. However, there was no difference between both groups when the need for total arch repair was analysed. The majority of patients received an open distal anastomosis, consisting of an excision of the concave portion of the aortic arch and a slanting suture line towards proximal of the brachiocephalic trunk.

A further important aspect is the underlying pathophysiological mechanism leading to the ND. It seems important to distinguish between focal and generalized symptoms, such as coma. In patients with a generally reduced level of consciousness, this can be due to stroke, a prolonged post-ictal phase after epileptic seizure induced by general hypoxia, but also an effect of cardiac tamponade with consecutive deterioration of the cardiac function. Comatose patients represent an interesting cohort, for which an acceptable outcome has recently been published as well [13]. In our study, surgery was performed on a minority of patients presenting in a comatose state. Of these 14 patients, 6 recovered to various extent and were in Group N1, 6 of them recovered fully (N0). Although our findings in this cohort are limited by small numbers, we did not identify a correlation between the severely reduced level of consciousness with neurological outcome, but saw a trend towards shorter time to surgery and thus may conclude, that surgery might prove beneficial even in these patients, when carried out immediately.

Cerebral protection strategies, including the arterial cannulation strategy, ACP, hypothermia and transcranial oxygen saturation monitoring, have been considered to play a major role in the neurological outcome following surgery for AADA. Cannulation of the right subclavian artery first is a strategy that was implemented at our institution throughout the whole period of observation, and was only quit after frustrane trial of perfusion via this access. A report from the GERAADA registry stated that the type of cerebral protection did not impact neurological complication rate [25], which is in contrast to earlier reports, which found that ACP led to superior outcome [4].

Limitations

This study has certain limitations, mainly the limited sample size and the method of retrospective data analysis. Neurological assessment was limited to GCS and mRS, both rather crude assessment scores that are not validated in this specific situation.

For obvious reasons, the performance of randomized trials is highly unlikely in these patients, so the community will rely on

data from registries and case series such as this one. We believe that the highly defined and standardized approach at our institution constitutes an advantage over more heterogeneous data acquired in registries and therefore single-centre studies should complement them.

We unfortunately do not have reliable data for all patients on the time between onset of symptoms and surgery. The diagnosis of AADA was frequently made by CT scan in an outside hospital, and the patient was transferred, resulting in 2–3 h delay between imaging and skin incision. Furthermore, some patients delayed admission to a hospital themselves and whether this contributed to neurological outcome is difficult to evaluate, since it is known that the dissection can progress over time.

CONCLUSION

We report an 8-year experience of treating AADA patients, of which roughly 20% showed new ND of a large variation from comatose to focal motor deficit only. A favourable outcome with either full recovery or limited incapacitation (mRS <3) was achieved in more than 70% of this cohort. Crude assessment of neurological impairment via mRS was predictive of detrimental outcome, but the majority even of comatose patients showed recovery to some extent, were discharged from our hospital and concluded the first follow-up. We conclude that even severely incapacitated patients should not *per se* be excluded from life-saving surgery.

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APPENDIX. CONFERENCE DISCUSSION

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Dr E. Saadi (Porto Alegre, Brazil): We know neurological symptoms secondary to arch vessel malperfusion are documented in up to 20% of the cases with acute aortic dissection. Although stroke and coma are traditional contraindications for immediate surgery, and the criteria to define reversible brain damage remain unclear, successful emergency repair has been reported in your paper. We would like to comment on some aspects of this challenging situation.

We have some concerns about evaluating the patient's neurological condition using the Glasgow Coma Scale score after aortic dissection. In fact, this score was developed to evaluate the level of consciousness in patients after head injury who might have organic damage to the brain tissue and neurological system and may not apply to this specific disease. The wide range of neurological dysfunction presented, from small sensory deficit or convulsion to hemiplegia and coma, may compromise the evaluation of the whole group, which is not homogenous.

I would like to ask you, what was the type of cerebral protection used? You told us it was antegrade cerebral perfusion. Was this unilateral, just on the right, or bilateral? Do you use any special cerebral protection in patients with previous neurological deficit?

In your experience, is there any situation in which you would contraindicate surgery in acute type A aortic dissection because of the neurological deficit?

Dr Most: As for the first question, we don't do any special treatment to the patients who present with neurological deficit. All of them receive antegrade cerebral perfusion via catheters bilaterally. As soon as we go into hypothermic arrest, the perfusion via the arterial cannula is stopped, and we don't clamp the brachiocephalic trunk, but we insert perfusion catheters into both carotid arteries. We always do near-infrared spectroscopy to assess the success of our cerebral perfusion.

There is no special treatment for those patients with signs of cerebral malperfusion, such as preoperative carotid revascularization or selective cannulation, but we are very conscious of inserting the perfusion catheters carefully so as not to make the damage progress or cause thrombo- or air embolism.

Dr Saadi: And the contraindications?

Dr Most: As to the contraindications, we did not have reliable data on the patients who were refused surgery because sometimes this is also done while the patient is still at the referring centre.

With the previous speaker, I would agree that the time from the onset of coma is relevant and that the group from Japan also showed that coma of more than six hours was detrimental and had worse outcomes. Of course, the general situation in which the patient presents is also playing a role. Obviously not only cerebral malperfusion but malperfusion of other organ systems like ischaemia of the intestines is an important factor in this setting and can be a contraindication.

But I think in isolated brain malperfusion, the presence of the coma or of a generally reduced level of consciousness, at least in this cohort, did not as such predict a detrimental outcome.