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Prediction of postoperative motor deficits using motor evoked potential deterioration duration in intracranial aneurysm surgery

Zhibao Li [a,b,1], Xing Fan [a,b,1], Mingran Wang [a,b], Xiaorong Tao [a,b], Lei Qi [a,b], Miao Ling [a,b], Dongze Guo [a,b], Hui Qiao [a,b,*]

[a] Department of Neurophysiology, Beijing Neurosurgical Institute, Capital Medical University, Beijing, China
[b] Beijing Tiantan Hospital, Capital Medical University, Beijing, China

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HIGHLIGHTS

- Motor evoked potential (MEP) deterioration duration can be used to predict postoperative motor deficits.
- Patients with MEP deterioration duration ≥13 min have a higher risk of postoperative motor deficits.
- MEP deterioration duration is not associated with postoperative CT abnormalities.

ABSTRACT

Objective: The study aimed to investigate the predictive value of motor evoked potential (MEP) deterioration duration for postoperative motor deficits in patients undergoing intracranial aneurysm surgery.
Methods: Data from 587 patients were reviewed and 92 patients with MEP deterioration were enrolled. MEP deterioration duration was compared between patients with and without postoperative motor deficits. Receiver operating characteristic (ROC) curve analysis was performed to define the threshold value for predicting postoperative motor deficit risk. Additionally, the association between MEP deterioration duration and postoperative CT findings was explored.
Results: Patients with postoperative motor deficits had a significantly longer MEP deterioration duration ($p < 0.01$). An MEP deterioration duration greater than or equal to 13 min was identified as an independent predictor of immediate ($p < 0.01$), short-term ($p < 0.01$), and long-term postoperative motor deficits ($p < 0.05$). There was no significant association between MEP deterioration duration and new CT abnormalities.
Conclusion: MEP deterioration duration could be used for predicting intracranial aneurysm surgical outcome.
Significance: The study first proposed a threshold value of MEP deterioration duration (13 min) for predicting the risk of postoperative motor deficits in patients undergoing intracranial aneurysm surgery.
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1. Introduction

Surgical clipping, as well as endovascular intervention, are both effective therapies for intracranial aneurysms. Nevertheless, multiple factors during the treatment, e.g. surgical procedures or vasospasm, may lead to decreased blood flow in a major cerebral artery, and then result in cerebral infarction. Postoperative cerebral infarction can be observed in 10.40% of patients and is mostly attributable to intracranial aneurysm surgery (Sasaki et al., 2007). Postoperative complications caused by cerebral infarction, including hemiplegia, sensory disturbance, and aphasia, can significantly decrease patients' quality of life. During intracranial aneurysm clipping, cerebral infarction is often induced by excessively prolonged occlusion of the aneurysm-bearing vessel, perforating artery destruction, or inappropriate positioning of the retractor or a permanent clip.

Abbreviations: IONM, Intraoperative Neurophysiologic Monitoring; MEP, Motor Evoked Potential; CT, Computed Tomography; SSEP, Somatosensory Evoked Potential; mRS, modified Rankin Scale; ROC, Receiver Operating Characteristic; AUC, Area Under the Curve.

* Corresponding author at: Beijing Neurosurgical Institute, Capital Medical University, 119 Fanyang Road, Beijing 100070, China.
E-mail address: proqiao@sina.com (H. Qiao).

[1] These authors contributed equally to this work and should be considered as co-first authors.

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To date, various assistive techniques have been applied to intracranial aneurysm surgery to reduce the incidence of cerebral infarction, including angiographic and blood flow studies, as well as intraoperative neurophysiologic monitoring (IONM) (Warren et al., 2001; Florence et al., 2004; Raabe et al., 2005; Hecht et al., 2009; Schichor et al., 2010; Cui et al., 2011; Bacigaluppi et al., 2012). Among these assistive techniques, IONM has already become indispensable in avoiding cerebral infarction in intracranial aneurysm surgery.

Motor evoked potential (MEP) is one of the most sensitive IONM modalities for predicting postoperative motor deficits. Many studies have indicated that MEP is highly sensitive to drops in cerebral blood flow, enabling prediction of postoperative outcome in intracranial aneurysm patients (Neuloh and Schramm, 2004; Wiedemayer et al., 2004; Guo and Gelb, 2011; Shida et al., 2012). However, those studies mainly focused on the decrease in MEP amplitude as a warning criterion (Lopez et al., 1999; Szelenyi et al., 2006; Yue et al., 2014; Thomas and Guo, 2017). To the best of our knowledge, few studies have investigated the predictive value of MEP deterioration duration for postoperative motor deficits. In the current study, we aimed to investigate the correlation of the MEP deterioration duration with postoperative motor deficits and tried to identify an appropriate threshold value for MEP deterioration duration to avoid postoperative motor deficits. The association between MEP deterioration and CT findings was also explored.

2. Methods

2.1. Patients

Data from 587 patients undergoing intracranial aneurysm clipping with IONM from January, 2016 through December, 2017 at our hospital were retrospectively reviewed. The inclusion criteria were: (1) absence of preoperative motor dysfunction, (2) absence of preoperative MEP deterioration, and (3) existence of intraoperative MEP deterioration. 92 patients (40 male, 52 female) were enrolled. The mean age of the patients was 54 years (range 18–77). Among all 92 intracranial aneurysms, 82 (89%) were in the anterior circulation, 37 (45%) were ruptured and 45 (55%) unruptured Ten (11%) of the intracranial aneurysms were in the posterior circulation, 4 (40%) were ruptured and 6 (60%) unruptured. The study was approved by the ethics committee of our hospital. Fig. 1 shows the flow diagram for participants.

2.2. Anesthesia

Total intravenous anesthesia was induced by sufentanil (25 μg) combined with etomidate (20 mg) and was maintained by propofol (4–6 mg/kg/h) combined with remifentanil (0.05–0.2 μg/kg/min). Rocuronium bromide, a short-duration muscle relaxant, was used during tracheal intubation and scalp incision. The patients' vital signs, blood-oxygen concentration, and carbon dioxide concentration were monitored continuously during surgery.

2.3. Neurophysiologic monitoring

Monitoring protocols included bilateral upper and lower extremity MEPs and somatosensory evoked potentials (SSEPs). For MEP monitoring, transcranial electric stimulation was conducted by a constant-voltage stimulator using 8-pulse trains (interstimulus interval 2–5 ms, pulse duration 0.5–1 ms, stimulus intensity 100–400 V), and the intensity level chosen for monitoring was specified as near-threshold. Corkscrew electrodes were placed at C1 and C2, and the anode-cathode switch was used (C1-C2 for right and C2-C1 for left). MEPs were recorded by subcutaneous needle electrodes from the abductor pollicis brevis and abductor hallucis muscles. The filter bandwidth was set as 30–3000 Hz.

The baseline MEP was acquired before opening the dura mater. MEP was recorded every 3–5 min during the entire surgery. The warning criteria were defined as a reduction over 50% in MEP amplitude or MEP disappearance. Once a warning was given, the neurosurgeon would change the surgical strategy, e.g. loosening the clip or unblocking the occlusion, to restore the MEP amplitude. MEP deterioration was divided into reversible and irreversible deterioration by whether the reduced amplitude or missing MEP could return to more than 50% of baseline amplitude. For reversible deterioration, MEP deterioration duration was defined as the time from which the MEP amplitude decreased by more than 50% of baseline or the MEP disappeared to when the MEP amplitude returned to more than 50% of baseline. For irreversible deterioration, the MEP deterioration duration could not be acquired because of permanent deterioration. IONM data were collected and evaluated by one of two experienced IONM technicians.

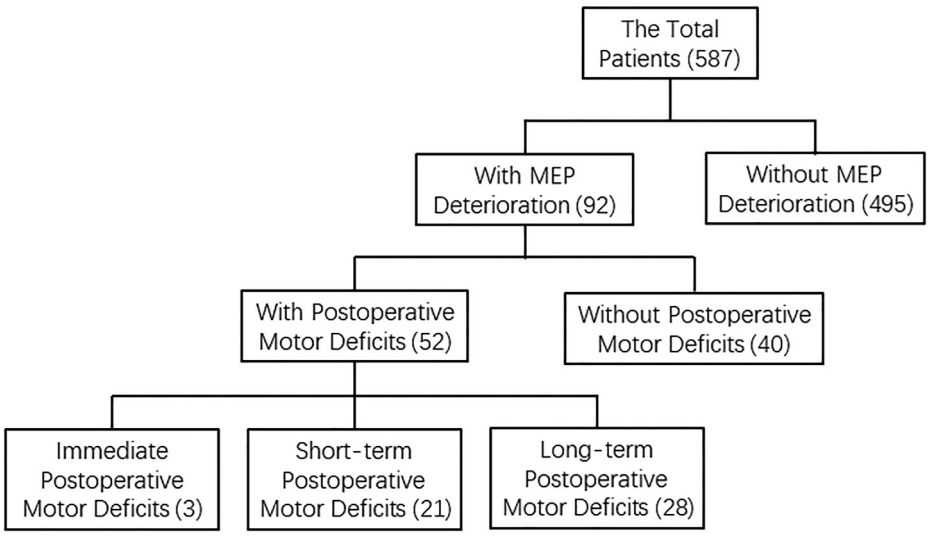

Fig. 1. Flow diagram of the study.

2.4. Clinical assessment and follow-up

Postoperative motor deficits were evaluated by the modified Rankin Scale (mRS, Supplementary Table S1). The assessment of neurological function was performed at three time points: 24 h, 7 days, and 3 months after surgery. Immediate postoperative motor deficits were defined as motor dysfunction that occurred within 24 h but returned to normal within 7 days after surgery. Short-term postoperative motor deficits were defined as motor dysfunction that still existed 7 days after surgery but returned to normal within 3 months after surgery. Long-term postoperative motor deficits were defined as motor dysfunction that had not returned to normal by 3 months after surgery. Preoperative and postoperative neurological conditions were examined by one of two experienced neurosurgeons.

2.5. Radiological assessment

Postoperative CT scanning was performed and assessed by one of two professional radiologists within 24–72 h after surgery. CT findings were recognized as positive when new low-attenuation areas were observed compared with preoperative images. The radiologists were blinded as to whether the patient had experienced any IONM deterioration.

2.6. Statistical analysis

Continuous data were expressed as the mean ± standard deviation, while categorical data were described in terms of frequency or proportions. A p value < 0.05 was considered statistically significant. MEP deterioration duration was compared by the Mann-Whitney U-test between patients with postoperative motor deficits and those without. MEP deterioration duration in patients with short and long-term postoperative motor deficits was further analyzed by the Mann-Whitney U-test.

Receiver operating characteristic (ROC) curve analysis was performed to calculate the optimal threshold value for MEP deterioration duration that could avoid postoperative motor deficits. The rates of new CT abnormalities between patients with postoperative motor deficits and those without was compared. Comparison were also made between patients with MEP deterioration duration less than and greater than or equal to the threshold value, using the chi-squared test. Multivariate binary logistic regression analysis was performed to describe the correlation of relative parameters with immediate, short-term, and long-term postoperative motor deficits.

3. Results

3.1. MEP deterioration and postoperative motor deficits

In the 92 patients enrolled in the current study, 76 patients had reversible MEP deterioration and 16 patients experienced irreversible MEP deterioration. In the 76 patients with reversible MEP deterioration, 3 (8%) had immediate postoperative motor deficits only, 19 (51%) had short-term and 15 (41%) had long-term postoperative motor deficits. The MEP deterioration duration was 9 ± 5 min (mean ± standard deviation, here and elsewhere, range 4–14), 27 ± 20 min (range 3–96), and 49 ± 62 min (range 5–255) in patients with immediate, short-term and long-term postoperative motor deficits, respectively. For patients without postoperative motor deficits, the MEP deterioration duration was 11 ± 9 min (range 3–55). Patients with postoperative motor deficits had a significantly longer MEP deterioration duration than patients without postoperative motor deficits (p < 0.01, Mann-Whitney U-test, Fig. 2).

In 16 patients with irreversible MEP deterioration, all five patients with irreversible MEP disappearance had postoperative motor deficits (one with short-term and four with long-term postoperative motor deficits), and most patients with irreversible reduction in MEP amplitude also experienced postoperative motor deficits (one with short-term and nine with long-term postoperative motor deficits), except one patient. The incidence rate of postoperative motor deficits in patients with irreversible MEP deterioration was significantly higher than in patients with reversible MEP deterioration (37/76 vs. 15/16, chi-squared test, p < 0.01).

The details of the 52 patients with postoperative motor deficits are presented in Table 1. Of these, there were 37 patients with reversible MEP deterioration, and the mean mRS scores were 3.5 and 4.7 at 7 days after surgery. The scores were 0.1 and 3.9 at 3 months after surgery in patients with short-term and long-term postoperative motor deficits, respectively. No significant difference was identified in MEP deterioration duration between patients with short and long-term postoperative motor deficits (27 ± 20 vs. 49 ± 62, Mann-Whitney U-test, p = 0.30).

The optimal threshold value for avoidance of postoperative motor deficits was 13 min (AUC = 0.80, 95% CI 0.70–0.91, Fig. 3). In addition, the patients with MEP deterioration duration greater than or equal to 13 min had a significantly higher risk for postoperative motor deficits (30/41 vs. 7/30, chi-squared test, p < 0.01). In 41 patients with an MEP deterioration duration greater than or equal to 13 min, 30 (73%) had postoperative motor deficits (one with immediate, 17 with short-term, and 12 with long-term postoperative motor deficits). In 35 patients with an MEP deterioration duration less than 13 min, only 7 (20%) had postoperative motor deficits (two with immediate, two with short-term, and three with long-term postoperative motor deficits).

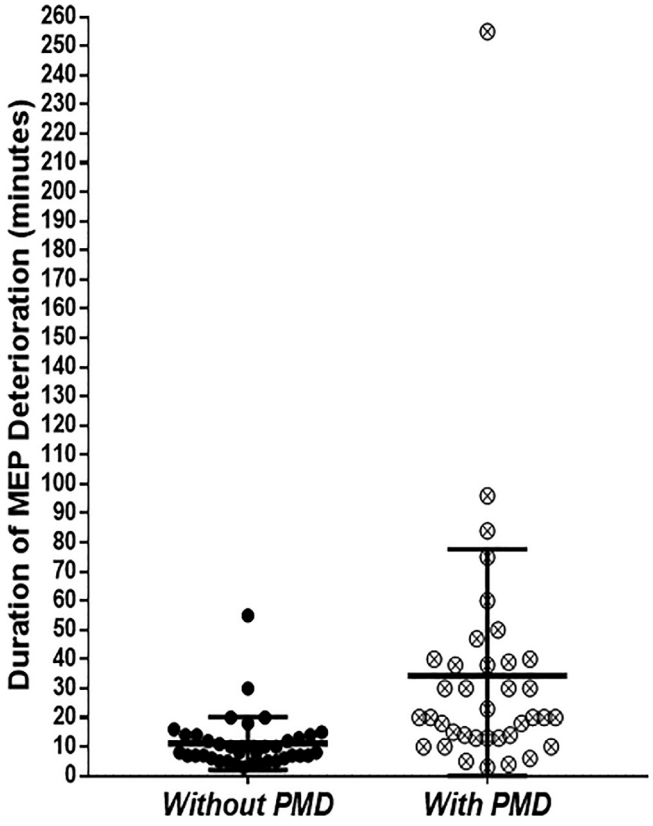

Fig. 2. Comparison of the MEP deterioration duration between patients with and without postoperative motor deficits. MEP deterioration duration in patients with postoperative motor deficits is significantly higher than those without (p < 0.01, Mann-Whitney U-test). MEP, motor evoked potential; PMD, postoperative motor deficit.

Table 1
More details of patients with postoperative motor deficit.

Num	Age	Sex	Aneurysm location	Aneurysm feature	CT presentation	Location of NLA on CT	MEP change deterioration (min)	PMD	MRS 7 days/3 months
		Male = 1/ female = 2	AC = 1/PC = 2	Rup = 1/ Unrup = 2	With NLA = 1/ without NLA = 2	No NLA = 0 Superficial = 1 Subcortical = 2		Immediate PMD = 1/ Short-term PMD = 2/Long-term PMD = 3	
1	59	2	1	2	2	0	10	1	2/0
2	57	2	1	2	2	0	14	1	0/0
3	68	1	2	2	1	2	4	1	2/0
4	60	2	1	1	1	1	5	3	5/4
5	54	1	1	2	1	2	10	2	4/0
6	67	2	1	1	1	2	10	3	5/4
7	53	2	1	1	1	2	13	2	2/0
8	58	1	1	2	1	1	18	3	4/2
9	67	2	1	2	1	2	18	2	3/0
10	49	2	1	1	1	2	20	2	3/0
11	61	1	1	2	1	2	20	2	3/0
12	45	2	1	2	1	2	20	3	3/2
13	39	2	1	2	1	2	23	3	5/3
14	67	2	1	2	1	2	38	3	5/4
15	50	2	1	2	1	2	40	2	5/0
16	46	2	1	2	1	1	50	3	4/3
17	66	2	1	2	1	2	60	3	5/5
18	40	2	1	1	2	0	3	2	3/0
19	42	2	1	2	2	0	6	3	5/5
20	64	1	1	2	2	0	13	2	3/0
21	48	2	1	2	2	0	13	2	3/0
22	72	1	1	2	2	0	14	3	5/4
23	65	2	1	1	2	0	15	2	5/0
24	67	2	1	1	2	0	20	2	4/2
25	54	2	1	1	2	0	30	2	4/0
26	53	1	1	1	2	0	30	2	5/0
27	40	1	1	1	2	0	38	3	5/4
28	52	2	1	2	2	0	39	2	4/0
29	60	2	1	2	2	0	47	2	3/0
30	56	2	1	1	2	0	84	3	5/5
31	47	1	1	2	2	0	96	2	3/0
32	57	1	2	2	1	1	20	2	3/0
33	60	2	2	1	1	2	40	3	5/4
34	60	1	2	2	1	2	75	3	5/5
35	66	2	2	1	1	2	255	3	5/4
36	58	2	2	2	2	0	30	2	3/0
37	71	2	2	1	2	0	30	2	4/0
38	54	1	1	1	1	1	Irreversible reduction	3	5/4
39	42	2	1	2	1	2	Irreversible reduction	3	4/3
40	55	1	1	1	1	1	Irreversible reduction	3	5/3
41	48	2	1	2	1	2	Irreversible reduction	3	4/3
42	40	1	1	2	2	0	Irreversible reduction	2	3/0
43	54	1	1	2	2	0	Irreversible reduction	3	5/4
44	56	2	1	1	1	1	Irreversible reduction	3	5/4
45	78	1	1	1	1	2	Irreversible reduction	3	4/4
46	67	1	1	1	1	2	Irreversible reduction	3	5/4
47	45	2	2	2	1	1	Irreversible reduction	3	5/5
48	49	1	1	1	1	2	Irreversible disappearance	3	5/4
49	56	1	1	2	1	2	Irreversible disappearance	2	4/0
50	67	2	1	2	1	2	Irreversible disappearance	3	5/4
51	46	2	1	1	1	1	Irreversible disappearance	3	5/5
52	41	2	1	2	1	2	Irreversible disappearance	3	5/3

AC: Anterior Circulation; PC: Posterior Circulation; Rup: Ruptured; Unrup: Unruptured; NLA: new low-attenuation areas; PMD: postoperative motor deficits; MRS: modified Rankin scale.

3.2. MEP deterioration and postoperative CT imaging

New low-attenuation areas were observed on postoperative CT images in 40 (43%) patients; 32 (80%) of them had postoperative motor deficits. Among these 32 patients, one (3%) had an immediate postoperative motor deficit, eight (25%) had short-term and 23 (72%) had long-term postoperative motor deficits. By contrast, in 52 (57%) patients without new CT abnormalities, 20 (38%) patients had postoperative motor deficits, and immediate, short-term and long-term postoperative motor deficits were observed in two (10%), 13 (65%) and five (25%) patients, respectively. The new CT abnormalities were significantly associated with postoperative motor deficits (32/40 vs. 20/52, chi-squared test, $p < 0.01$). Additionally, among the 40 patients with new CT abnormalities, rever-

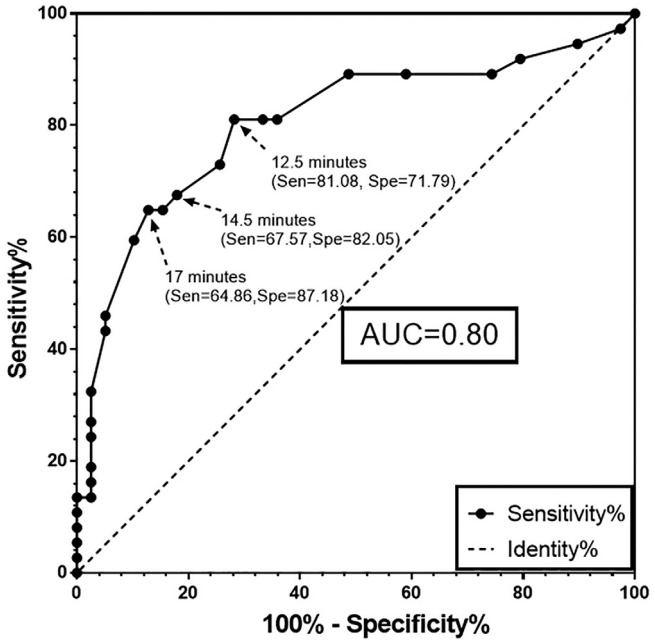

Fig. 3. Receiver operating characteristic curve showed the area under the curve of the duration of MEP deterioration in predicting postoperative motor deficits was 0.80. When MEP deterioration duration was equal to or greater than 13 min, the sensitivity was 65%, the specificity was 87%, the positive predictive value was 83%, the negative predictive value was 72%, and the diagnosis rate was 76%. MEP, motor evoked potential.

sible MEP deterioration was observed in 27 patients (20 with low-attenuation areas located in the subcortex or basal ganglia region, seven with low-attenuation areas located in the superficial cortex), while irreversible MEP deterioration was observed in 13 patients (eight with low-attenuation areas located in the subcortex or basal ganglia region, five with low-attenuation areas located in the superficial cortex); no significant difference was identified (20/27 vs. 8/13, chi-squared test, $p = 0.66$). Interestingly, the occurrences of new CT abnormalities in patients with irreversible reduction in MEP amplitude or MEP disappearance were significantly higher than those with reversible reduction in MEP amplitude or MEP disappearance (13/16 vs. 27/76, chi-squared test, $p < 0.01$).

In patients with new CT abnormalities, the MEP deterioration duration was 32 ± 48 minutes (range 4–255); in patients without new CT abnormalities, the duration of MEP deterioration was 17 ± 18 min (range 3–96). There was no significant difference between the groups. (Mann-Whitney U-test, $p = 0.06$). Additionally, we also found that patients with an MEP deterioration duration greater than or equal to 13 min did not have a higher incidence rate of new low-attenuation areas on postoperative CT images (10/35 vs. 17/41, chi-squared test, $p = 0.24$).

3.3. Multivariate analysis

Multivariate binary logistic regression analysis was performed to identify whether the MEP deterioration duration is an independent predictive factor for surgical outcome. The period of MEP deterioration was divided into a binary variable using the previously obtained threshold value of 13 min. The results are presented in Table 2. Compared with patients with an MEP deterioration duration less than 13 min, patients with an MEP deterioration duration greater than or equal to 13 min have a higher risk of developing immediate (OR 15.12, 95% CI 4.11–55.58, $p < 0.01$), short-term (OR 29.08, 95% CI 5.58–144.61, $p < 0.01$), and long-term postoperative motor deficits (OR 4.40, 95% CI 1.06–18.22, $p < 0.05$). Additionally, intraoperative aneurysm rupture is also an independent risk factor for short-term postoperative motor deficits (OR 6.24, 95% CI 1.20–32.46, $p < 0.05$). No other variable (including sex, age, and location) was identified as a predictive factor for postoperative motor deficits.

4. Discussion

4.1. MEP deterioration duration and postoperative motor deficits

The results of this study showed that the duration of MEP deterioration in intracranial aneurysm surgery was significantly associated with postoperative motor deficits, similar to previous studies (Suzuki et al., 2003; Komatsu et al., 2017). These results could reflect the relationship between the duration of ischemia and ischemic cerebral infarction indirectly.

Early in the 1970s, Sundt et al. took the lead in identifying the relationship between brain infarction and reduced cerebral blood flow in carotid endarterectomy (Sundt et al., 1974, 1981). However, reduced cerebral blood flow is not the only factor that leads to cerebral infarction. Morawetz et al. demonstrated that the infarction of gray and white matter did not occur in short-term regional blood flow reduction, but did occur in long-term regional blood flow reduction (Morawetz et al., 1978). This evidence could support the idea that the duration of ischemia also plays a key role in the process of initiating infarction. In our study, we indicated that the duration of ischemia was significantly associated with ischemic cerebral infarction in intracranial aneurysm surgery, which provided further evidence for this view.

Additionally, consistent with previous studies (Ferch et al., 2002; Ha et al., 2009; Griessenauer et al., 2014), no difference was found in MEP deterioration duration between patients with short-term and long-term postoperative motor deficits. This finding suggested that the MEP deterioration duration can predict the occurrence but may not predict the duration of postoperative motor deficits.

4.2. Threshold value of MEP deterioration duration

To date, a variety of clinical studies have investigated the relationship between MEP deterioration duration and cerebral infarction in intracranial aneurysm surgery. Suzuki et al. reported five patients with postoperative motor paresis among 20 patients with MEP deterioration, and the MEP deterioration duration was only 8–16 min in the affected patients (Suzuki et al., 2003). Moreover, another study reported hemiparesis in four patients whose MEP deterioration durations were 8, 10, 12, and 35 min (Szelenyi et al., 2006). Irie et al. reported that postoperative hemiparesis

Table 2
Results of multivariate analysis.

Variables	Immediate postoperative motor deficit			Short-term postoperative motor Deficit			Long-term postoperative motor deficit		
	OR	95% CI	P-Value	OR	95% CI	P-Value	OR	95% CI	P-Value
Sex	2.54	(0.79–8.17)	0.12	0.44	(0.13–1.47)	0.18	0.55	(0.15–2.04)	0.37
Age	1.02	(0.97–1.08)	0.44	1.01	(0.95–1.07)	0.76	1.00	(0.94–1.07)	0.94
Location	0.23	(0.03–1.79)	0.16	0.40	(0.05–2.96)	0.37	0.57	(0.11–3.11)	0.52
Aneurysm Feature	0.11	(0.77–12.18)	0.11	6.24	(1.20–32.46)	0.03	1.60	(0.44–5.85)	0.48
MEP Deterioration Duration	15.12	(4.11–55.58)	<0.01	29.08	(5.85–144.61)	<0.01	4.40	(1.06–18.22)	0.04

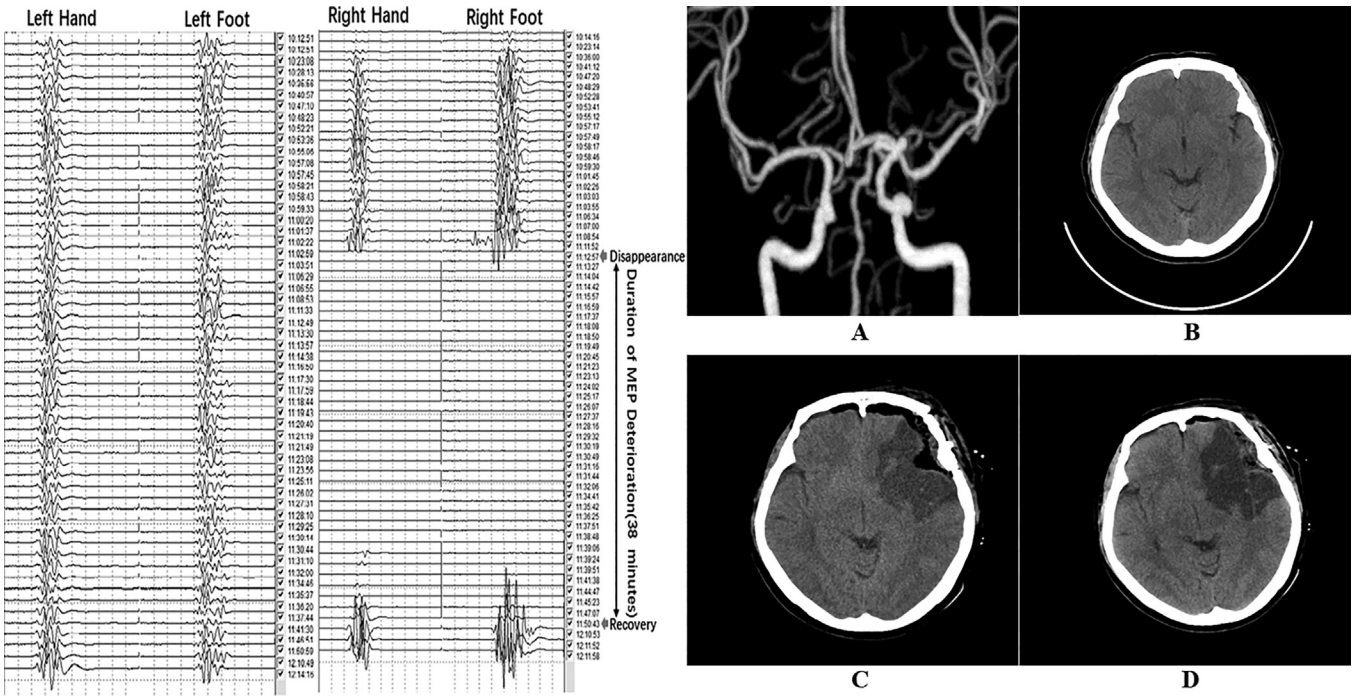

Fig. 4. A 67-year-old female complained of a headache for 1 month. Left, intraoperative MEP monitoring; right, image before (A, B) and after (C, D) surgery. Preoperatively, the patient had normal muscle strength and normal somatosensory function. CT angiography showed a left medial cerebral artery aneurysm (A). Intraoperatively, the patient underwent intracranial aneurysm clipping through the left frontotemporal approach with MEP monitoring. MEP disappearance for 38 min was reversed. At 1 day after surgery, the patient was unconscious, and CT scans showed a large low-attenuation area in the left frontotemporal region (C). At 3 days after surgery, her condition was unchanged (D). Follow-up at 7 days and 3 months after surgery showed mRS scores of 5 and 4, respectively. MEP, motor evoked potential; mRS, modified Rankin Scale.

occurred only in one patient with an MEP deterioration duration greater than 50 min among five patients with MEP deterioration (Irie et al., 2010). In general, however, because of the lack of cases, all those previous studies could not summarize a critical time point for the tolerance of cerebral ischemia induced by surgical procedures.

In the current study, analyzing the data from 52 patients with postoperative motor deficits, 13 min was identified as a threshold value of MEP deterioration duration for predicting postoperative motor deficits. Patients with an MEP deterioration duration greater than or equal to 13 min has a significantly higher incidence rate of postoperative motor deficits (73% vs. 20%). Multivariate analysis also identified that the MEP deterioration duration could serve as an independent predictor of postoperative motor deficits. To our knowledge, the current study has the largest sample size in this field, which significantly increases the clinical significance of the threshold value. According to our results, in clinical practice, we suggest that every effort by the surgical team should be made to control the MEP deterioration duration as much as possible so that it is less than 13 min. Of course, this does not mean that postoperative motor deficits will never occur, however, the incidence rate would be significantly reduced.

It is also worth mentioning that in 2013, Kang et al. assessed the relationship between changes in SSEP and MEP and ischemia tolerance in intracranial aneurysm surgery (Kang et al., 2013). They had an ingenious design and found that their self-defined "ischemia tolerance ratio" of SSEP was valuable in predicting postoperative neurological deficits. However, in their study, MEP recordings were considered as a supplement to SSEP monitoring, and MEP deterioration was only observed in four patients. In our study, we mainly focused on the duration of MEP deterioration, and showed that it was more direct in predicting the postoperative motor deficits.

4.3. MEP deterioration duration and postoperative CT

Previous studies had indicated that long periods of MEP deterioration were associated with new CT abnormalities in intracranial aneurysm patients (Irie et al., 2010; Yeon et al., 2010). However, in the current study, we did not find that patients with longer MEP deterioration duration had a significantly higher incidence rate of new low-attenuation areas on postoperative CT images. Meanwhile, 20 patients with postoperative motor deficits did not have abnormal CT findings. This observation might be because it was too early to detect changes in CT imaging or because the infarction was too small. Additionally, there were eight patients with abnormal CT findings without postoperative motor deficits; this might be because the infarct areas did not involve motor structures.

Additionally, Szelényi, et al. investigated the relationship between the type of MEP deterioration (reversible or irreversible) and the location of new postoperative signal alterations on magnetic resonance imaging in patients with intracranial tumors. They found that MEP deterioration was significantly correlated with new signal alterations located in the precentral gyrus, while MEP loss was more often followed by subcortically located new signal alteration (Szelenyi et al., 2010). However, in the current study, no such association was found, most of the new low-attenuation areas were located in subcortex or basal ganglia region. This is probably because the areas involved in intracranial aneurysm surgery were usually in the deep region perfused by the perforating branches of cerebral arteries.

4.4. Reversible MEP deterioration and postoperative motor deficits

In the current study, there was a phenomenon that piqued our interest: cerebral infarction with motor deficits could occur despite full MEP amplitude restoration in some patients (Fig. 4). In other

words, the corticospinal neurons involved in MEP generation initially suffered ischemic failure, later showed apparently full neurophysiologic functional recovery with reperfusion, and yet a motor deficit still occurred. One possible explanation is that the infarction of corticospinal neurons or peri-Rolandic supplementary motor neurons dose not contribute to MEP directly. The other possible explanation is that the transcranial electrical stimulation might not detect cortical and/or superficial subcortical cerebral infarction because of supramaximal penetrating current that could active the subcortical or brainstem area (Macdonald et al., 2013), so even though the MEP amplitude could be restored, cortical and/or superficial subcortical cerebral infarction had already occurred.

4.5. Limitations

There are limitations to our study. First, the sample size was relatively small. Second, our study was limited by its retrospective nature. Future prospective studies are needed to validate our results.

5. Conclusion

In the current study, we revealed the predictive value of MEP deterioration duration for postoperative motor deficits in patients undergoing surgery for intracranial aneurysm. Moreover, we first proposed a threshold value of MEP deterioration duration (13 min) for predicting the risk of postoperative motor deficits in these patients, which has possible clinical significance in guiding the neurosurgical team.

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Conflict of interest

None of the authors have potential conflicts of interest to be disclosed.

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Appendix A. Supplementary material

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