Cerebrovascular Diseases

11

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Abstract

Measuring local cerebral blood flow and metabolism by various mapping methods, such as PET (positron emission tomography), SPECT (single-photon emission computed tomography), perfusion computed tomography, MRI, and so on, helps us to evaluate detailed functions of brain areas containing a focal ischemic lesion, but does not necessarily represent neural activities of the areas. Scalp electroencephalography (EEG), reflecting volume-conducted neural activities, demonstrates that slow wave activity is dominant in an acute ischemic cerebral region, but this technique presents major problems with the lack of objective indices for brain functions and low spatial resolution. Magnetoencephalography (MEG), an important new method in neuroscience to directly detect neural activities with high spatial resolution, has been applied in stroke patients. This chapter mainly describes the relation between magnetic responses and cerebral ischemic changes from several stroke-related manuscripts.

Some papers stressed the clinical usefulness of MEG, for example, that slow wave activities occur on the affected cerebral hemisphere and somatosensory evoked potential becomes indicator of brain plasticity. However, other neurophysiological signal changes after stroke are various and not consistent. The usage of MEG for assessing neural activities in an ischemic brain area has not been fully established as yet. Therefore, more objective analysis of MEG findings in ischemic conditions is needed for future development.

Keywords

Magnetoencephalography • Stroke • Cerebrovascular disease • Ischemia • Brain plasticity

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11.1 Introduction

There are various correlations between electrophysiological and hemodynamic responses in stroke, epilepsy, brain tumor, dementia, rehabilitation process, etc.

There are two main clinical approaches for cerebral vascular disease, especially stroke. The first is measurement of cerebrovascular reserve capacity, evaluation of which is very important to treat cerebral infarction. The other approach involves determination of brain plasticity, which is an indicator of rehabilitation after stroke [1].

Cerebral blood flow and metabolism can be measured by nuclear medicine studies, such as single-photon emission computed tomography (SPECT) and positron emission tomography (PET), as well as by perfusion CT, MRI, and near-infrared spectroscopy (NIRS). These methods have been established for diagnosing and assessing cerebrovascular disease. Some reports have indicated that brain plasticity can be evaluated by diffusion-weighted MRI or fMRI [2].

However, hemodynamics in cerebrovascular disease as assessed by these imaging studies only indirectly reflects brain function.

On the other hand, electrophysiological studies like electroencephalography (EEG) more directly reflect neural activity. It is well known that the slow wave activity in EEG occurs in ischemic areas of the brain. On the other hand, scalp-recorded EEGs are greatly affected by the skull itself. Electrophysiological signal in small infarct lesions may be buried within normal signal findings in surrounding areas and go undetected. Thus, routine EEGs are problematic because of low spatial resolution and a lack of objective indices for quantitative measurement of *brain function*. Magnetoencephalography (MEG) quantitatively measures magnetic activity in the brain and may overcome some of these limitations of EEG. This chapter mainly describes the relations between magnetic responses and cerebral ischemic changes.

MEG is now performed with devices that can cover the entire head, thus providing improved spatial resolution. MEG was first used in clinical research to assess brain function and search for epileptic foci preoperatively in patients undergoing neurosurgery. The usefulness of MEG for brain function evaluation in other diseases has also been frequently reported, but its clinical use in diseases other than epilepsy has been sparse, so it is still unclear what level of scientific evidence exists for its utility outside of epilepsy.

Therefore, trends in clinical research of MEG in ischemic cerebral disease were examined in a literature search to evaluate the level of scientific evidence for using MEG in clinical evaluation.

11.2 Methods

A PubMed database literature search (http://www.ncbi.nlm.nih.gov/pubmed/) was conducted with the keywords (stroke or cerebral ischemia) and (MEG or magnetoencephalography) for publications dated between January 1990 and July 2014. A

total of 62 papers were retrieved from this search regarding MEG based on the titles, 28 of these papers were reviewed based on their abstract contents, and then, focusing on the level of evidence, 14 papers that measured cerebral blood flow (CBF) or used controlled comparisons were selected and reviewed.

Although it was difficult to directly compare these reports due to the nonuniformity of presentation and varied ischemic conditions, some common results are described.

11.3 Results 1

A total of 62 papers about MEG and cerebral ischemia were retrieved from the literature search. These were then limited to original manuscripts, and 28 papers were selected based on their abstract contents. These included reports about diagnostic criteria for cerebral ischemia and functional recovery/neuroplasticity after stroke. They were further divided into reports about spontaneous cerebral magnetic fields and evoked cerebral magnetic fields.

Evaluation of the use of MEG in functional recovery after stroke is reported separately, and some reports about the evaluation of ischemia are described.

11.3.1 Diagnostic Criteria for Cerebral Ischemia in MEG

1. Spontaneous magnetic fields: slow waves occur after stroke

Affected side

- In areas surrounding lesions after stroke, MEG shows a decrease of high-frequency components (gamma band) and an increase of low-frequency delta (theta band) [3, 4].
- Increased theta waves are seen in the temporoparietal area of the affected side [5] and correlate with misery perfusion [6].
- Slow waves are seen on the affected side in transient ischemic attack (TIA) patients [7].
- The power of delta waves is correlated with the National Institute of Health Stroke Scale (NIHSS) [8].
- Slow wave components are correlated with decreased *N*-acetylaspartate (NAA) on MR spectroscopy [9].
- Slow wave activity during re-buildup in areas of impaired circulation in moyamoya disease is seen in deep cortical sulci [10, 11].

Unaffected side

- Increased slow waves are also seen on the unaffected side [4, 12].
- Increased slow waves (delta) on the unaffected side may be clinically correlated with symptom improvement [2].

2. Evoked magnetic fields

SEF (somatosensory evoked fields)

- In stroke cases, asymmetry of localization of equivalent current dipoles (ECDs) in N20m of the affected side [13], prolonged latency [14, 15], abnormal waveforms [16], and decreased [12, 17] or increased ECD strength [11, 15] are seen. This asymmetry is correlated with improvement of clinical symptoms [12, 18].
- The SEF ECD component at N20m is correlated with the NIHSS [8].
- In parallel with decreased CBF, N20m dipole moment decreases and P30m increases [19].

MEF (motor evoked fields)

- Shifts in source position and latencies are seen [20].
- Beta band event-related desynchronization (ERD) is seen in motor areas and the ipsilateral hemisphere [12].
- The amplitude of motor-related direct current (DC) signal (infra-slow MEG signal) decreases in the affected hemisphere [21].

AEF (auditory evoked fields)

- The temporal lobe response on the affected side is decreased [22].

11.3.2 Functional Recovery, Plasticity, and Indices of Reorganization

1. Spontaneous magnetic fields (slow waves)

- Slow waves on the affected side decrease with improved circulation after surgical treatment [6, 23].
- Normalization of slow waves leads to a clinical improvement in symptoms [12].
- Revascularization improves CBF and decreases theta bands, but there are no changes in delta bands [23]. In another report, delta waves near the lesion did not change during follow-up, and there was no correlation with clinical symptoms [3].
- In TIAs, normalization of slow waves from the somatosensory cortex may be an index of short-term functional recovery after stroke [24].
- In cases of aphasia due to stroke, delta waves (1–4 Hz) were seen near the lesion, and a decrease of delta waves after speech therapy was associated with good outcomes [25].
- Delta waves on the unaffected side and gamma waves on the affected side may be indices of functional recovery.

2. Evoked magnetic fields

- Improved SEF latency correlates with sensory improvement [14].
- Source power localized to S1 (SEF) and to M1 with finger tapping showed a correlation with sensory and motor function improvement [26].

11.4 Results 2

Of the abovementioned 28 papers, 14 with high evidence levels, including measurement of CBF or comparisons with normal controls, were further reviewed. Table 11.1 describes their content in detail.

11.4.1 Target Diseases and Comparisons

Most papers discussed internal carotid artery or middle cerebral artery occlusion or stenosis. One paper dealt with TIAs, and one paper was about moyamoya disease. There were normal controls in 11 papers.

11.4.2 Analysis Methods

Spontaneous magnetic fields were measured in six studies, evoked cerebral magnetic fields were measured in six studies, and both spontaneous and evoked cerebral magnetic fields were measured in one study. Among papers dealing with evoked cerebral magnetic fields, four used median nerve stimulation, one used tactile finger stimulation [15], one used auditory stimulation [4], and two used motor-related magnetic fields [27, 28].

Most papers on evoked magnetic fields used a single equivalent current dipole (ECD) method, but one used a spatial filtering technique (synthetic aperture magnetometry, SAM) [28]. Measurement of spontaneous magnetic fields included analysis of the waveforms themselves, analysis of magnetic field distribution using ECDs, analysis using a spatial filtering technique (standardized low-resolution brain electromagnetic tomography, sLORETA) [23], and analysis using power spectral density (PSD) [4].

One paper showed the relation between direct current (DC)-MEG signal and near-infrared spectroscopy (NIRS) for a finger movement task [27].

Table 11.1 Summary of some meritorious manuscripts

No.	Reference no.	Authors	Journal	Title	PubMed PMID	Clinical diagnosis	Case number	Age
1	4	Tecchio F, Zappasodi F, Pasqualetti P, Tombini M, Salustri C, Oliviero A, Pizzella V, Vernieri F, Rossini PM	Neuroimage. 2005 28:72–83	Rhythmic brain activity at rest from rolandic areas in acute mono-hemispheric stroke: a magnetoencephalographic study	16023869	Unilateral MCA ischemia	32	30–86
2	5	Seki S, Nakasato N, Ohtomo S, Kanno A, Shimizu H, Tominaga T	Neuroimage. 2005 25:502–10	Neuromagnetic measurement of unilateral temporo-parietal theta rhythm in patients with internal carotid artery occlusive disease	15784429	ICA occlusive disease	48	40–76
3	6	Ohtomo S, Nakasato N, Shimizu H, Seki S, Kanno A, Kumabe T, Tominaga T	Clin Neurophysiol. 2009 120:1227–34	Temporo-parietal theta activity correlates with misery perfusion in arterial occlusive disease	19539523	ICA or MCA occlusive disease	56	40–75
4	8	Assenza G, Zappasodi F, Squitti R, Altamura C, Ventriglia M, Ercolani M, Quattrocchi CC, Lupoi D, Passarelli F, Vernieri F, Rossini PM, Tecchio F	Neuroimage. 2009 44:1267–73	Neuronal functionality assessed by magnetoencephalography is related to oxidative stress system in acute ischemic stroke	19010427	MCA ischemia	18	Mean: 73
5	11	Qiao F, Kuroda S, Kamada K, Houkin K, Iwasaki Y	Childs Nerv Syst. 2003 19: 760–764	Source localization of the re-build up phenomenon in pediatric moyamoya disease-a dipole distribution analysis using MEG and SPECT		Moyamoya disease	4	8–16

6	16	Forss N, Hietanen M, Salonen O, Hari R	Brain. 1999 122:1889–99	Modified activation of somatosensory cortical network in patients with right-hemisphere stroke	10506091	Right MCA infarction	6	45–65
7	17	Tsutada T, Ikeda H, Tsuyuguchi N, Hattori H, Shimogawara M, Shimada H, Miki T	J Neurol Sci. 2002 198:51–61	Detecting functional asymmetries through the dipole moment of magnetoencephalography		Various lesion	34	37–39
8	19	Bundo M, Inao S, Nakamura A, Kato T, Ito K, Tadokoro M, Kabeya R, Sugimoto T, Kajita Y, Yoshida J	Stroke. 2002 33:61–6	Changes of neural activity correlate with the severity of cortical ischemia in patients with unilateral major cerebral artery occlusion	11779890	"ICA or MCA occlusive disease no apparent clinical symptom"	7	18–75
9	22	Toyoda K, Ibayashi S, Yamamoto T, Kuwabara Y, Fujishima M	J Neurol Neurosurg Psychiatry. 1998 64:777–84	Auditory evoked neuromagnetic response in cerebrovascular diseases: a preliminary study	2170114	"Major branch stenosis and occlusion no apparent infarct area in temporal lobe"	24	43–75
10	28	Oshino S, Kato A, Hirata M, Kishima H, Saitoh Y, Fujinaka T, Yoshimine T	Stroke. 2008 39:2769–75	Ipsilateral motor-related hyperactivity in patients with cerebral occlusive vascular disease	18635836	"Occlusive cerebrovascular disease (arteliosclerosis:28 cases、others:10 cases)with right handedness and no apparent motor weakness	38	55–79

(continued)

Table 11.1 (continued)

No.	Reference no.	Authors	Journal	Title	PubMed PMID	Clinical diagnosis	Case number	Age
11	23	Sakamoto S, Tanaka H, Tsuyuguchi N, Terakawa Y, Ohata K, Inoue Y, Miki Y, HaraM, Takahashi Y, Nitta K, Sawa H, Satone A, Ide W, Hashimoto I, Kamada H	Neuroimage. 2010 49:488–97	Quantitative imaging of spontaneous neuromagnetic activity for assessing cerebral ischemia using sLORETA-qm	19632340	"ICA stenosis: 4 ICA occlusion:1"	5	60–77
12	24	Stippich C, Kassubek J, Kober H, Sörös P, Vieth JB	Neuroreport. 2000 11:3309–13	Time course of focal slow wave activity in transient ischemic attacks and transient global amnesia as measured by magnetoencephalography	11059893	"TIA: 6 cases TGA: 2 cases"	8	44–82
13	27	Leistner S, Sander-Thoemmes T, Wabnitz H, Moeller M, Wachs M, Curio G, Macdonald R, Trahms L, Mackert BM	Biomed Tech (Berl) 56:85–90	Non-invasive simultaneous recording of neuronal and vascular signals in subacute ischemic stroke	21299378	Subcortical infarction: 4cases	4	44–77
14	21	Forss N, Mustanoja S, Roiha K, Kirveskari E, Mäkelä JP, Salonen O, Tatlisumak T, Kaste M	Hum Brain Mapp. 2012 33:534–41	Activation in parietal operculum parallels motor recovery in stroke	21425393	MCA infarction	18	46–85

No.	Control number	MEG and device	Evoked field/ spontaneous field	Other modality	Task Mesurements	Analysis	Result	Clinical approach
1	14	28 channel	SF	None	"MEG was performed between 2 and 10 days after stroke onset 0.48–250Hz sampling:1000Hz 3 min examination"	"Power spectral density (PSD) spectral entropy"	The perilesional increase of the low frequency, 2–3.5Hz 4–7.5Hz, is detect whithin the AH rolandic areas, the same effect is present in the UH. This phenomenon may show interhemispheric diaschisis. An increased of the toital power and a reduction of the spectral entropy were detected in the AH. This effect suggests a high synchrony of local neuronal activity, areduction of the intra-cortical inhibitory networks efficiency and an increase of neuronal excitability	C
2	27	"Neuromag 204 planar-type gradiometer"	SF	None	"0.03–100Hz sampling 300Hz awake 6 min examination"	Wave form	6–8Hz temporoparietal theta wave (in 14 of 48 patients)	C
3		"Neuromag 204 planar-type gradiometer"	SF	XeCT CBF, CVR	"0.03–100Hz sampling 500Hz awake 10 min examination"	"Fourier analysis ECD analysis"	"The presence of TPTA (6–8Hz) was detected in 14 patients, and significantly correlated with both reduced rCBF and reduced rCVR. After anastomosis, TPTA disappeared in 7 of the 10 studied patients. The area of leptpmeningeal collateral correlated with the region of TPTA"	C

(continued)

Table 11.1 (continued)

No.	Control number	MEG and device	Evoked field/ spontaneous field	Other modality	Task Mesurements	Analysis	Result	Clinical approach
4	20	28 channel	"SF(bilateral rolandic area 3min,eye open state) EF (median nerve stimulation)"	None	"SEF:0.48–250 Hz sampling rate 1000 Hz"	"NIHSS for ishemia evaluation ECD analysis Power Spectral Density"	"NIHSS has positive correlation with delta power and negative correlation with N20mECD stremgth. Serum transferrin also correlated with affected hemisphere N20m ECD strength and inversely with its delta power"	C
5		"204 channel Vectorview"	SF	"Xe-SPECT, IMP-SPECT acetazolamide loading"	"High pass filter 0.1Hz sampling 600Hz awake 10 min examination"	"1–6Hz filter extract 50ms data at slow wave activity caliculate ECDs at each 2ms interval"	"During hyper ventilation, slow wave activity on rebuild-up was detected in the area of poor response for acetazolamide loading, especially, deep sulcus region. After surgical anastomosis, rebuild-up was disappeared"	C
6	7	122 channel Neuromag	EF(median nerve stimulation)	None	"0.03–320Hz accumulation: 200 times"	"Neurological evaluation for ischemia ECD analysis"	AH SI showed abnormal response, and AH S2 responce decreased. UH S2 responce showed no change	C
7	22	"160 channel MEG Vision"	EF (median nerve stimulation)	SPECT	"3–200Hz accumulation: 400 times"	ECD analysis	ECD moment showed latelarity between AH and UH	C

8		"37 channel BTI"	EF (median nerve stimulation)	"$H2O^{15}$-PET CBF study"	"Sampling rate: 1041.7 Hz accumulation: 200 times"	"ECD analysis Comparison CBF with the amplitude and latensy of dipole moment at 20ms, 30ms, 40ms"	"The rCBF in the primary sensory area and N20 m were significantly reduced and the second component (P30 m) was significantly augmented in AH as compared with UH. P30m activation may indicate the compensatory change or the inhibitory system"	C
9	12	"37 channel BTI"	EF(AEF)	PET	"Auditory stimulation 1Hz tone and burst Orgogozo score, functional independence score, two points discrimination test MEG was performed within 1 week, at 3months, at one year after stroke onset"	Evaluation for responces at P50m, N100m	"In nine cases, the accurate magnetic sources of P50m or N100m were not identified. Cases with abnormal P50m responses had decreased supratemporal and hemispheric blood flow"	C
10	8	"64 channel CTF"	EF(motor related evoked field)	None	"Clench and un clench during 10 s 60 times index finger tapping"	"SAM analysis for event related synchronization at βband (13–30Hz) comparison between right and left t volume"	"Abnormal ipsilateral dominant distribution of beta ERD was observed significantly more often during contralesional hand grasping in patients with atherosclerotic vascular lesion. This may indicate the inhibitory system of bilateral cerebral hemishere connectivity"	C

(continued)

Table 11.1 (continued)

No.	Control number	MEG and device	Evoked field/ spontaneous field	Other modality	Task Mesurements	Analysis	Result	Clinical approach
11	5	"160 channel MEG Vision"	SF	"$O_2{}^{15gas}$-PET IMP-SPECT"	"0.16–200Hz sampling 500Hz awake 10 min examination"	"sLORETA extract 45 s data from each band, 0.3–2, 2–4, 4–6 6–8Hz comaprison between pre and post operative activated position, signal intensity of slow wave"	"In all 5 patients, slow waves in every frequency band were distributed in the area of cerebrovascular insufficiency. Slow-wave intensities in theta bands decreased postoperatively along with improvements in CBF and metabolism, whereas delta bands showed no significant differences between pre- and postoperatively"	C
12	8	"27 × 2 channel BTI"	SF	None	MEG was performed at 4 days, at 11 days, between 31 and 68 days after stroke onset	ECD analysis	"Slow wave (2–6Hz) appeared in ipsilateral sensorymoter for for TIA (6 cases) and bilateral temporal for TGA (2 cases). These wave disappeared within 11 days after onset"	P
13	4	49 channel	EF (motor related evoked field)	NIRS	"0.16–200Hz sampling 500Hz finger movement task (30-s period movemnt and 30-s rest period, total 30-min)"	Motor relatede DC-field, simultaneous to the NIRS	The motor-related activation shows a trend towards stronger infraslow neuronal signals over the unaffected hemispheres compared with the affected hemisphere	P

14	18	“Neuromag 306 planar-type gradiometer”	EF (tactile finger stimulation)	None	“0.03–308Hz sampling 941Hz MEG was performed at 1–7 days, at 3–4 weeks, ane at 3 months after stroke onset”	“ECD analysis signal space separetion method”	The S1-SEF (contralateral primary somatosensory evoked field) amplitude or latency did not correlate with any of the functional outcome scores. The cPO-SEF (contralateral parietal opercula somatosensory evoked field) amplitude correlated with hand function in the acute phase and during recovery	P

TPTA: Temporo-parietal theta activity
CBF
CBV
NIHSS: National Institute of Health Stroke Scale
AH: affected hemisohere
UH: unaffected hemisphere
N20m: the strength of the initial component of somatosensory evoked magnetic field
P30m: the second component
C: serebral metabolism and circulation relatede matter
P: neuronal plasticity related matter

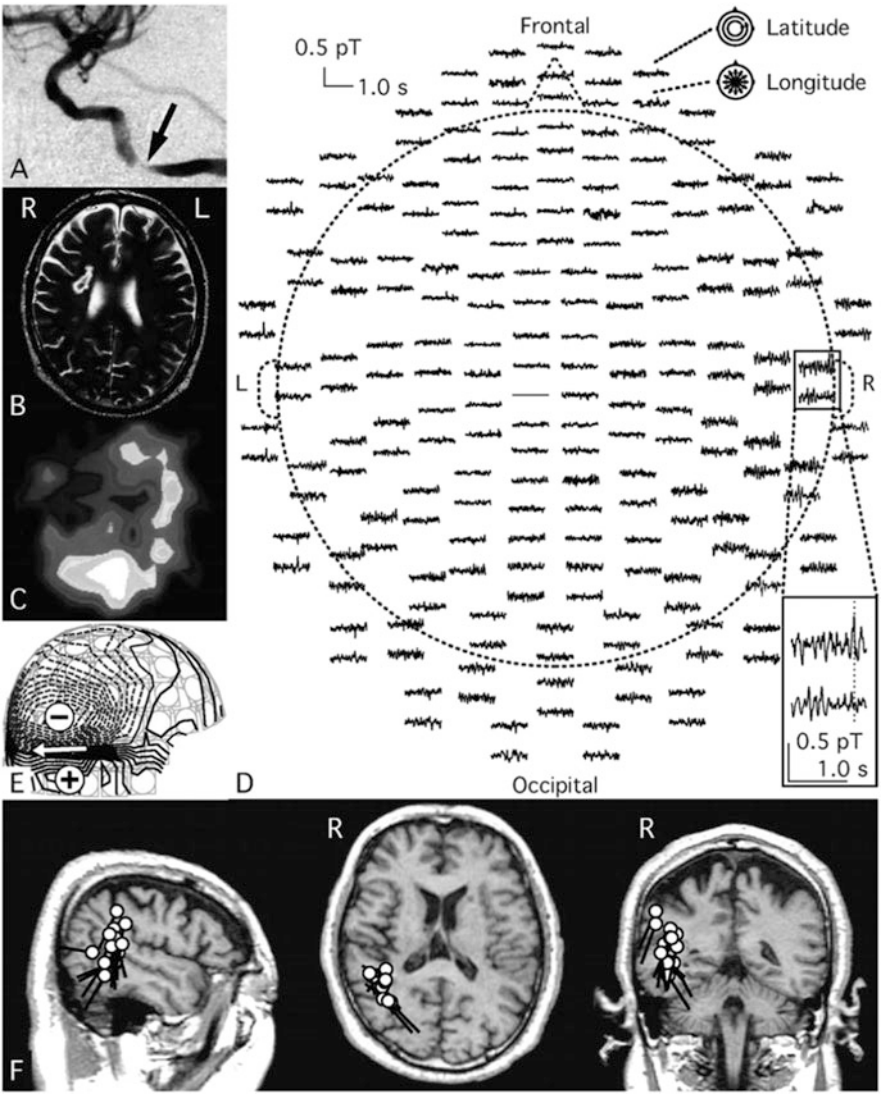

Fig. 11.1 A 54-year-old male with temporoparietal theta activity (TPTA) detected by MEG. (**a**) Digital subtraction angiogram showing stenosis (*arrow*) of the right internal carotid artery. (**b**) T2-weighted MR image showing a right striatocapsular infarct lesion. (**c**) SPECT scan showing flow reduction in the territory of the right middle cerebral artery. (**d**) MEG waveforms, detected in the awake condition with the eyes closed using the latitudinal and longitudinal tangential derivatives of 204 planar-type gradiometers, showing TPTA in the right hemisphere. (**e**) Isofield map at a typical peak of TPTA (*broken line* in the inlet square of D), showing a single dipole pattern. The *arrow* shows the approximate location and orientation of the equivalent current dipole (ECD) of TPTA over the right temporal area. (**f**) ECDs at ten similar peaks of TPTA to (**d**) and (**e**) projected onto three orthogonal MR images. *Circles* and *bars* indicate the ECD location and orientation, respectively (Ohtomo et al. [15])

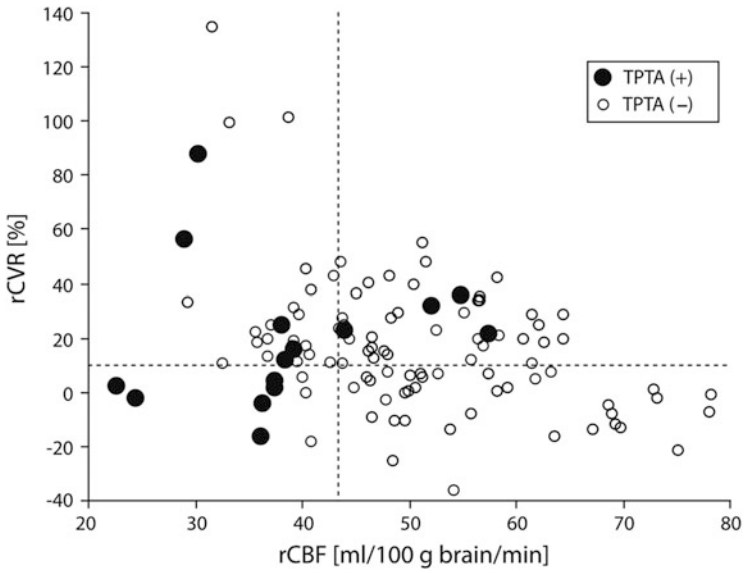

Fig. 11.2 Resting regional cerebral blood flow (rCBF) and regional cerebrovascular reactivity (rCVR) in the middle cerebral artery territory of 112 hemispheres in 56 patients with or without temporoparietal theta activity (TPTA). *Dashed vertical* and *horizontal lines* indicate the criteria for reduced rCBF (43.4 ml/100 g brain/min) and reduced rCVR (10 %) as defined in normal subjects in our institute. Note that TPTA was detected in six of nine hemispheres in the patients with both reduced rCBF and reduced rCVR (Ohtomo et al. [15])

11.4.3 Cerebrovascular Measurements

Six papers described measurement of CBF, including five that measured cerebrovascular reserve capacity. Three papers discussed areas of decreased cerebrovascular reserve (penumbra). They mainly described the correlation with areas of slow wave appearance and discussed postoperative improvement. Ohtomo and coworkers [6] demonstrated typical temporoparietal theta activity (TPTA) in ischemic lesion in Fig. 11.1 and the relation between cerebral circulation and TPHA in Fig. 11.2. Sakamoto and coworkers [23] showed the changes in slow wave activity between pre- and postsurgical anastomosis after stroke in Fig. 11.3.

11.5 General Remarks

Low-frequency activity often appears near ischemic foci and in an ischemic hemisphere, and these waves also tend to occur in the temporoparietal area [5]. In addition, with improved cerebral blood flow (CBF), slow waves, particularly theta band activity, usually disappear [6, 23]. However, this phenomenon is not

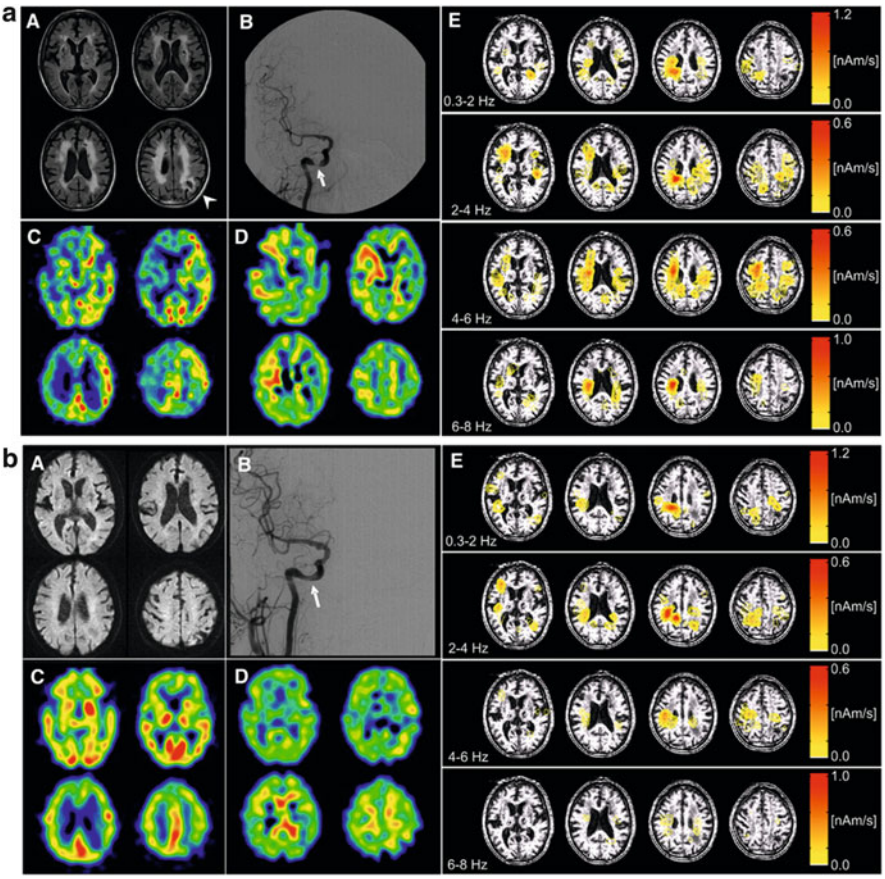

Fig. 11.3 (**a**) A 70-year-old man with the severe stenosis of right internal carotid artery showed slow wave activity in preoperative state by sLORETA of MEG. (*A*) MRI (FLAIR) showed no severe cerebral infarction, excluding old infarct lesion in the left parietooccipital area (*arrowhead*) and multiple lacunar infarctions in bilateral basal ganglia and periventricular white matter. (*B*) Cerebral angiography showed N90 % stenosis of the C5 segment of the right internal carotid artery (*arrow*). (*C*, *D*) 15O-gas PET showed reduced CBF (C, 32.5 ml/100 g/min) and increased OEF (D, 73.3 %) in the right cerebral cortex. (*E*) Quantitative imaging of cerebral neuromagnetic fields using sLORETA-qm in four frequency bands showed distribution and increased intensities of slow waves dominantly in the parietooccipital area of the right cerebral hemisphere. Distribution of slow waves was also recognized in the left parietooccipital area and periventricular white matter corresponding with old infarct lesion (Sakamoto et al. [19]). (**b**) Postoperative investigations in same case. (*A*) MRI (DWI) showed no additional infarction in any area of cerebrum. (*B*) Cerebral angiography showed recovery of stenosis after CAS of the right internal carotid artery (*arrow*). (*C*, *D*) 15O-gas PET showed increased CBF (C, 38.0 ml/100 g/min) and decreased OEF (D, 47.1 %) in the right cerebral cortex. (*E*) Quantitative imaging of cerebral neuromagnetic fields with sLORETA-qm in each frequency band showed decreased intensities at 2–4 Hz, 4–6 Hz, and 6–8 Hz in the parietooccipital area of the right cerebral hemisphere, although no apparent change of intensity at 0.3–2 Hz was observed compared with preoperative data (Sakamoto et al. [19])

necessarily seen in all cases, so using changes in slow waves as an objective index of a penumbra can be unreliable.

Many investigators have reported a decreased SEF N20m response to median nerve stimulation in an ischemic hemisphere, but it is difficult to determine whether this signifies complete ischemia or a penumbra.

MEG reflects activity in the sulcal cortex, whereas EEG also reflects activity in the sulcal and gyral cortex activity. On the other hand, MEG uses large number of channels ($>$ 100 sensors on the whole head), so MEG can detect brain function over a wider area. Furthermore, its spatial resolution is much better than that of EEG. MEG directly detects magnetic fields because it is unaffected by the skin and bones and is, therefore, quantitatively superior to EEG. Moreover, MEG and MRI images can be fused, making it convenient for clinical application, such as neurosurgical navigation techniques.

MEG is a noninvasive technique to quantify neural activity, but it is not yet an established method for evaluating cerebral ischemia. The use of new analytical methods, including spatial filtering, not only measuring spontaneous magnetic fields but also objectively assessing neural activity when patients perform simple tasks [28], is a novel approach using cerebral magnetic fields to evaluate ischemic cerebrovascular disease.

11.6 Summary

1. Slow waves appear on the affected side in patients with stroke.
2. Slow waves are seen in areas of decreased blood perfusion (often the temporoparietal area) in patients with cerebrovascular occlusion.
3. The first peak of the somatosensory evoked field (SEF) in the ischemic hemisphere may decrease, may disappear, or be abnormal.

11.7 Conclusion

This section examined trends in clinical research and reviewed the scientific evidence, based on a literature search, for the use of MEG in evaluating ischemic cerebrovascular disease. MEG could not absolutely evaluate brain plasticity and circulation. Although it is complete as an alternative device to other modalities, a few papers reported a relatively high level of evidence for using MEG in diagnosis and treatment planning. MEG may be superior to scalp EEG in assessing ischemic changes in neural function.

MEG may also be useful to objectively evaluate neurological function, other than from the perspective of cerebral blood flow and metabolism, in patients with ischemic cerebrovascular disease.

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