Original article

Kana Pick-out Test assesses brain function beyond the frontal cortex

Momoko Kobayashi*, **, Hideyuki Hoshi***, Yoko Hirata****, Keisuke Fukuasawa*****, Sayuri Ichikawa*****,
Yoshihito Shigihara*, ***

Abstract The Kana Pick-out Test is a Japanese neuropsychological test developed to assess the prefrontal cortex. It has been used as a screening test for dementia, especially in the early stage, when frontal dysfunction is often salient. The test is evaluated using four scores: (1) number of correct responses, (2) workloads, (3) errors, and (4) omissions. A previous study demonstrated that the number of correct responses is considered the primary score, which is associated with prefrontal cortex volume. Number of errors is associated with the volume of the prefrontal cortex and caudate nucleus, while number of omissions is associated with the volume of the temporal lobe. However, data regarding the number of workloads remains unclear. In this study, we explored the brain regions whose activities were associated with the number of workloads and the other three scores of the Kana Pick-out Test. These scores were obtained from 106 patients who visited the outpatient department for dementia care at the Kumagaya General Hospital. Resting-state brain activity was measured using magnetoencephalography. The associations between the scores and brain activity were examined using regression analysis. The results showed that the number of correct responses was positively associated with the brain activities in the left superior frontal gyrus, left precentral gyrus, and right postcentral gyrus. It supports the fact that the Kana Pick-out Test was originally designed to evaluate the function of the prefrontal cortex. The number of workloads was positively associated with the brain activity in the right precuneus, located outside the prefrontal cortex. No significant association was identified with the number of errors or omissions. These findings indicate that the Kana Pick-out Test evaluates cognitive functions associated with wider brain regions beyond the frontal cortex when workload scores are included. Careful assessment of workload scores alongside the number of correct responses maximises KPT in comprehensively evaluating brain function and optimises the test as an early-stage dementia screening tool.

Keywords : Kana Pick-out test; Magnetoencephalography; Number of workloads; Precuneus; Resting-state brain activity

1. Introduction

Dementia is characterised by a progressive deterioration in cognitive function due to brain disorders such as Alzheimer's disease (AD), significantly interfering with daily and social life (Mimura, 2018). The treatment goals are to improve the quality of life of people with dementia and their caregivers, facilitating adaptation to their conditions by maximising their residual cognitive function (Midorikawa, 2019; Maki et al., 2018). Cognitive function comprises multiple domains: complex attention, executive function, learning and memory, language, perceptual-motor skills, and social cognition. The residual functions differ per individual and change along with the progress of dementia, even within the same patient. To provide the best treatment, evaluating residual cognitive function using appropriate neuropsychological tests is crucial. The Mini-Mental State Examination (MMSE) is a neuropsychological test widely used in clinical and research fields to assess cognitive impairment associated with dementia (Arevalo-Rodriguez et al., 2015). It mainly evaluates learning and memory functions (Dinomais et al., 2016), which depend on the temporal cortex. However, MMSE is sometimes less sensitive for evaluating cognitive function, especially at the early stage of cognitive decline,

---

Received: September 18, 2024; Accepted: October 9, 2025.

* Precision Medicine Centre, Kumagaya General Hospital, Kumagaya, Japan
** Department of Rehabilitation, Kumagaya General Hospital, Kumagaya, Japan
*** Precision Medicine Centre, Hokuto Hospital, Obihiro, Japan
**** Department of Neurosurgery, Kumagaya General Hospital, Kumagaya, Japan
***** Clinical Laboratory, Kumagaya General Hospital, Kumagaya, Japan

because some patients show decline in cognitive function associated with the prefrontal cortex, such as executive function, rather than learning and memory (Guarimo et al., 2019; Baudic et al., 2006; Hoshi et al., 2022). The MMSE lacks sensitivity to evaluate executive dysfunction (Bak and Mioshi, 2007) and early cognitive impairment (Kawano, 2012; Lacy et al., 2015). Assessing cognitive function at the early stage of decline is especially important because patients have higher residual function, which can better enable clinicians to intervene effectively and potentially improve their cognitive function (Kashimoto, 2012; Shigihara et al., 2020). To compensate for the limitation of the MMSE, additional neuropsychological tests sensitive to executive function should be administered with the MMSE to detect subtle cognitive impairments (Sato et al., 2020). The Kana Pick-out Test (KPT) is a Japanese neuropsychological test originally designed as a screening test for evaluating prefrontal dysfunction (Sahara, 2007), including executive function. It is recommended as the initial screening tool for dementia (Sahara, 2007). During KPT, patients are asked to read a short story and identify vowels as quickly and accurately as possible, and the number of vowels is called the number of correct responses (NCR), which is the primary KPT score (Imamura, 2005). A previous study reported that NCR correlates with the prefrontal cortex volume (Sato et al., 2020). This finding supports the idea that the KPT is a test of frontal lobe function. Additionally, the KPT has four secondary scores: number of workloads (NW), errors (NE), omissions (NO), and comprehension score (CS) (Imamura, 2005). A previous study showed that NE is related to the volume of the left ventral prefrontal cortex and left caudate nucleus, whereas NO is related to the volume of the left and right superior temporal gyri and the right middle temporal gyrus (Sato et al., 2020). These findings suggest that the KPT can assess wider brain regions beyond the prefrontal cortex. For NW, it remains unclear whether NW assesses frontal lobe function or function in other areas. We did not emphasise CS in this study since it is subjective (see Methods section). To properly interpret the primary and secondary KPT scores (including NW) and maximise their use in clinical practice, it is crucial to understand which brain regions commonly influence these scores in actual patient groups, regardless of their causative diseases.

In this study, we explored the neural basis of the KPT scores, especially the NW, and examined their neurological characteristics among patients. Therefore, we compared the four KPT scores with the resting-state brain activity of 106 patients with cognitive impairment who visited our outpatient department for dementia care.

2. Materials and Methods

2-1. Patients

This retrospective study included patients who visited the outpatient department for dementia care at Kumagaya General Hospital. Patients who underwent neuropsychological tests and magnetoencephalography (MEG) assessment as part of standard clinical practice were included. Consequently, the dataset included 106 patients (68 women; mean age ± SD: 78.4 ± 7.0 years, ranging from 59 to 90 years). Sixty four patients were diagnosed with dementia (dementia due to AD, 45; dementia with Lewy bodies, eight; frontotemporal dementia, five; vascular dementia, one; dementia due to argyrophilic grain disease, one; mixed dementia, three; others, one), 25 were diagnosed with mild cognitive impairment (MCI), and 17 were age-related changes or suspected other diseases, although these diagnoses were not considered in the analyses. Dementia diagnosis was made according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) (American Psychiatric Association, 2013), and MCI was defined following the definition by Petersen (2004). The diagnoses were made by a neurosurgeon who was a clinical instructor at the Japanese Society of Dementia Research. According to the medical records, 25 patients had a history or suspected history of head trauma or neurological disorders, including epilepsy. Some had visual impairment, which could potentially influence their KPT scores. Clinical psychologists conducted the KPT as part of clinical practice. Before testing, they carefully explained the KPT to each patient, taking account of the cognitive level of each patient. However, some of them had difficulty comprehending due to their cognitive decline or other factors, which could influence the scores. To minimise selection bias, all eligible patients were included in the analyses, regardless of whether or not they followed the instructions properly. Written informed consent was obtained from all patients or their family members to reuse their clinical data for research purposes. The study was conducted in accordance with the Declaration of Helsinki, following national and international guidelines. This study was approved by the Ethics Committee of the Kumagaya General Hospital (#25).

2-2. Functional localisation hypothesis and functional neuroimaging

This study was conducted based on the ‘functional localisation hypothesis,’ which assumes that each brain area is specialised for particular cognitive functions (Ozkirli et al., 2025). The concept was first introduced by Franz Joseph Gall in the late 1700s and supported by lesion studies in the mid-to-late 19th century. The experimental approach for the localisation emerged using direct electric stimulation to the brain in the mid-20th century. During the 1970s−1980s, development in neuroimaging techniques, such as MEG, positron emission tomography, and functional magnetic resonance imaging, allowed the localisation of brain function in living humans. However, the localisation sensitivity was poor because the data contained large noise. According to Proudfoot et al. (2014), analysing MEG data is like 'hearing a pin drop at a rock concert'. The sensitivity significantly improved by the new analysis concept: statistical parametric mapping (SPM), which was invented in 1991 by Karl Friston and his colleagues. It averages neuroimaging data across time and individuals based on Bayesian statistics (Ashburner et al., 2013) and identifies common brain function across individuals with better sensitivity (Friston et al., 1999). The analysis concept gives importance to the aspect of similar regional function across individuals. Consequently, individual differences are not well considered: it is a trade-off with the sensitivity. Considering the historical background of neuroimaging and SPM, we intentionally included all potential patients regardless of potentially confounding factors such as physical disabilities, comprehension of instructions, and differences in causative diseases of cognitive impairment (i.e., pathologies).

2-3. Cognitive assessment

Clinical psychologists assessed patients' cognitive status using the KPT as an initial assessment of clinical practice. During the test, patients were asked to read a short story of 406 kana characters. Kana is the Japanese alphabet (minimum unit of written Japanese language), which consists of five vowels (あ, い, う, え, and お) and 41 consonants. Patients were asked to read the story and draw circles around the vowels while ignoring any consonants and simultaneously understanding the story for 2 min (i.e., dual-task). Performance was evaluated using five scores (Imamura, 2005): (1) NCR represents the number of correctly circled vowels (ranging from 0 to 61). (2) NW indicates the number of vowels within the part of the story that patients had read, regardless of circling performance (ranging from 0 to 61). The endpoint of the reading was determined by the examiner observing the patient's work or asking the patient. (3) NO represents the number of vowels within the story section read by the patients but missed and not circled (ranging from 0 to 61). (4) NE indicates the number of mistakenly circled consonants (ranging from 0 to 345). (5) CS represents the level of correct understanding of a story. These five scores represent different aspects of performance: NCR for accuracy, NW for speed, NO and NE for carelessness, and CS for memory. In this study, CS was not analysed because it is a subjective score, unlike the other four (see limitations in the Discussion section).

2-4. MEG recording

MEG is a noninvasive functional neuroimaging technique sensitive to changes in cognitive status (Hoshi et al., 2022). It detects changes in magnetic fields caused by the activation of pyramidal cells in the cerebral cortex, and MEG signals contain information associated with brain activity. The scanning and analysis procedures followed the pipeline described in our previous study (Shigihara et al., 2020; Hoshi et al., 2022). Resting-state brain activity was recorded for 5 min for each patient using a 160-channel whole-head MEG system (RICOH160-1; RICOH Co., Ltd., Tokyo, Japan) in a magnetically shielded room. During the scan, patients were asked to remain calm in the supine position with their eyes closed. The sampling frequency was 2,000 Hz with 500-Hz low-pass filtering during recording. Neuropsychological tests and MEG recordings were conducted on the same day for all patients, except for seven patients.

2-5. MEG data analysis

The MEG data were preprocessed offline using the software package SPM-12 (Wellcome Trust Centre for Neuroimaging, London, UK; https://www.fil.ion. ucl. ac. uk/spm/) and the MEAW system (https://www. hokuto7.or.jp/hospital/lang/english-home/meaw/). The cortex was considered as it comprises 900,395 voxels with a size 2.0 mm x 2.0 mm x 2.0 mm. All analyses were conducted in voxel-based comparisons within Montreal Neurological Institute (MNI) space (Chau and McIntosh, 2005). The MEG analysis was performed in two steps: first and second levels (Figure 1). In the first-level analysis, the oscillatory intensity was estimated from the MEG signals of each patient. The first-level analysis was subdivided into three steps: preprocessing, forward modelling, and source inversion. In the preprocessing step, the continuous MEG signals for 5 min were divided into non-overlapping 10-s segments (i.e., 'epoching'). Since the experimental environment generated a utility frequency, a 50-Hz band-stop filter was applied to the epoched data. Forward modelling is a procedure used to calculate the relationship between brain regions and MEG sensors (i.e., so-called lead field matrix), influenced by various assumptions such as the shape of the brain and its relative position against MEG sensors. In this study, this process was performed on the whole brain using a single-shell model with canonical MRI from SPM-12. Source inversion is a process used to estimate the brain regions producing the resting-state-induced components, during which the cortical projections of the preprocessed MEG signals are computed using an algebraic algorithm with the lead field matrix. We employed a maximal smoothness algorithm with a spatially coherent source model (i.e., the COH algorithm implemented in SPM-12) (Friston et al., 2008) as the estimation algorithm. This algorithm is comparable to the standardised low-resolution brain electromagnetic tomography (Pascual-Marqui, 2002) and is often used in clinical settings (Ray and Bowyer, 2010; Terakawa et al., 2008). To determine the source of the MEG signals in each frequency band, the source inversion procedure was applied separately to the delta (0–3 Hz), theta (4–7 Hz), alpha (8–12 Hz), beta (13–25 Hz), and gamma (low-gamma, 26–40 Hz; high-gamma, 41–80 Hz) oscillatory components using corresponding bandpass filters. No source priors were used for the source estimation. The estimated oscillatory intensity for each frequency band in each brain region (i.e., the regional oscillatory intensity) was saved as a source image file in the Neuroimaging Informatics Technology Initiative format. The source images were spatially smoothed (20 mm × 20 mm × 20 mm). These smoothed source images were used for group-level comparisons in second-level analysis.

In the second-level (i.e., group-level) analysis, statistical inference was applied to the source images from all patients to identify the brain regions where the regional oscillatory intensities met the interests of this study. To identify the brain regions where the oscillatory intensity was associated with each of the four KPT scores (NCR, NW, NO, and NE), smoothed source images were subjected to a regression model where each KPT score was used as a predictor (i.e., covariate). The coefficient of the predictor was then computed and stored voxel-wise. Finally, both positive and negative effects of KPT scores on source intensities were evaluated using t-contrasts with +1 and −1 on the predictor. Consequently, we obtained a matrix of t-values mapped to the voxels of the normalised brain, known as a statistical parametric map (SPM).

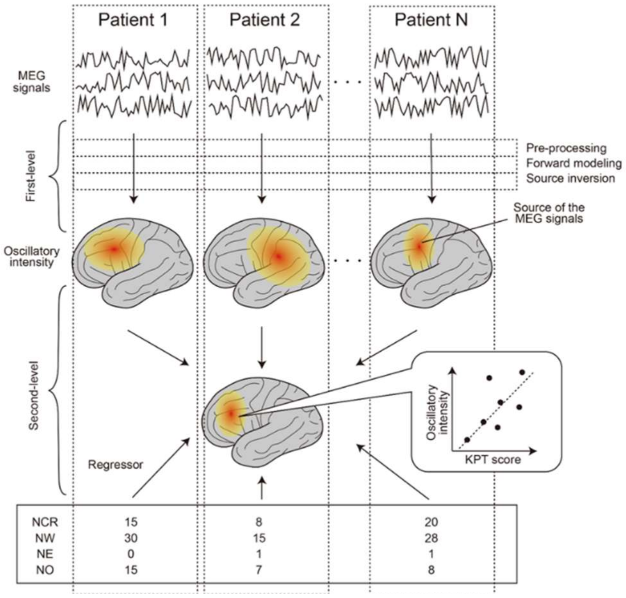

Figure 1. MEG analysis pipeline. The analysis consists of first and second levels. First-level analysis estimates the resting-state regional brain activity for each patient. Second-level analysis identifies the brain regions where the regional oscillatory intensities were associated with the KPT scores. MEG, magnetoencephalography; KPT, Kana Pick-out Test.

2.6. Reporting formats

We reported the voxels where the coefficients of the predictor (i.e., KPT score) statistically deviated (larger or smaller) from zero at a significance threshold of p (corrected for family-wise error) = 0.05. A cluster is a group of adjacent voxels that exceed a predefined statistical threshold. Cluster size refers to the number of voxels within the cluster. If the size is too small, the cluster could be unreliable (Woo et al., 2014). Cortical regions where the peaks of the statistical values were located on SPM-12 were labelled using the default atlas (i.e., the atlas provided by the OASIS project (https://www.oasis-brains.org/) and Neuromorphometrics, Inc. (http://neuromorphometrics.com/). All statistical results were described following the standard SPM reporting formats.

3. Results

The NCR was positively associated with the resting-state oscillatory brain activity in the left superior frontal gyrus at low gamma frequency (Table 1 and Figure 2) and in the left precentral, left superior frontal, and right postcentral gyri at high gamma frequency. NW was positively associated with the resting-state oscillatory brain activity in the right precuneus at high gamma frequency. No other oscillatory intensities were significantly associated with NCR, NW, NO, or NE.

4. Discussion

This study revealed that NW, a secondary KPT score, was associated with brain activity in the right precuneus, outside the prefrontal cortex, at high gamma frequency.

The KPT measures an individual's task performance during a dual-task, which is the ability to perform two tasks simultaneously (MacPherson, 2018). To execute the task, multiple cognitive functions are required, such as selective and divided attention to detect target characters, grasping the content of the story, working memory to store multiple pieces of information temporarily, and controlling their operations performed in parallel (Sato et al., 2020).

These cognitive functions are largely governed by the prefrontal cortex (Ohsugi et al., 2013), as it plays a vital role in selective (Squire et al., 2013) and divided (Loose et al., 2003) attentions, working memory (Lara and Wallis, 2015), and cognitive control (Miller, 2000). Therefore, the KPT is considered a neuropsychological test sensitive to prefrontal function (Sahara, 2007). This assumption is supported by a previous study showing that the NCR is associated with the left dorsolateral prefrontal cortex volume (Sato et al., 2020). Our findings also align with the study: the NCR, which is the primary KPT score, was associated with resting-state oscillatory brain activity in the left superior frontal gyrus, left precentral gyrus, and right postcentral gyrus at gamma frequency (Figure 2, Table 1).

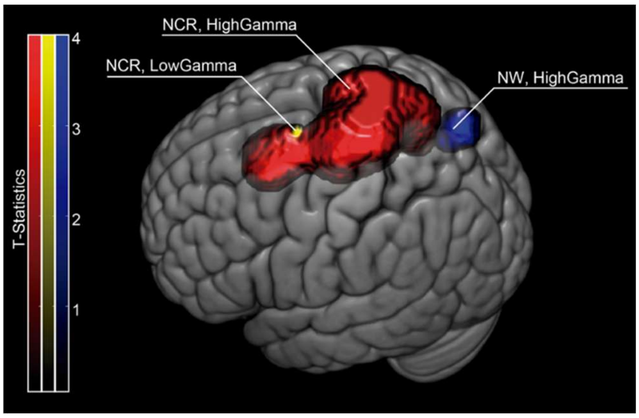

Figure 2. Brain regions where KPT scores are significantly associated with regional neural oscillatory intensity. The yellow and red coloured portions indicate the regions where low-gamma and high-gamma oscillatory intensities, respectively, are associated positively with the number of correct responses. The blue coloured portion indicates the region where high gamma oscillatory intensity is positively associated with the number of workloads. The three-dimensional image was created using MRIcroGL (https://www.mccauslandcenter.sc.edu/mricrogl/). KPT, Kana Pick-out Test; NCR, number of correct responses; NW, number of workloads.

However, these findings do not imply that the KPT performance is limited to the prefrontal cortex (Sato et al., 2020). The task asks patients to mark target letters quickly and accurately for 2 min. This requires visual-motor coordination, which depends on neural activity in the basal ganglia (Kaneko, 2017) and parietal association areas (Murayama et al., 2005), which are outside the prefrontal cortex.

Present result associating NW supported the contribution of wider brain regions beyond the prefrontal cortex. NW was the secondary score of focus in this study. Our results showed that NW was associated with resting-state oscillatory brain activity in the right precuneus, a part of the parietal cortex, not in the prefrontal cortex, at the gamma frequency (Figure 2, Table 1). The right precuneus plays an important role in controlling the spatial aspects of motor behaviour (Cavanna and Trimble, 2006), and motor control performance influences the processing speed during the KPT. Loss of motor function is observed in the preclinical stages of AD (Buchman and Bennett, 2011), and decreased blood flow in the precuneus is a potential biomarker of MCI (Thomas et al., 2019). NW likely reflects motor behavioural performance associated with precuneus function, suggesting its utility in the early detection of dementia. However, involvement of the right precuneus does not imply that the prefrontal cortex is unrelated to NW. The precuneus and medial prefrontal cortex are interconnected as a part of the default mode network (Koshino et al., 2014), whose activities are decreased during attention-demanding tasks and increased during some other cognitive tasks associated with memory or abstract thought (Smallwood et al., 2021). These cognitive functions are required while executing dual tasks, and our findings reflected the activity changes. The prefrontal cortex may influence NW by modulating brain activity in the right precuneus.

Notably, the associations between brain activity and KPT scores (NCR and NW) were found in the gamma frequency, which is

Table 1. Association between source-level oscillatory intensity and KPT scores

		Cluster level		Peak level		Coordinate			
Predictor	Frequency	*p* (FWE)	kE	*p* (FWE)	*T*	X	Y	Z	Brain region
NCR	Low gamma	0.048	43	0.047	3.647	-16	0	68	Lt Superior Frontal Gyrus
	High Gamma	0.016	8358	0.019	3.912	-18	-26	74	Lt Precentral Gyrus
				0.026	3.798	-20	14	56	Lt Superior Frontal Gyrus
				0.027	3.795	6	-30	74	Rt Postcentral Gyrus
NW	High Gamma	0.043	410	0.036	3.682	12	-72	50	Rt Precuneus

KPT, Kana Pick-out Test; NCR, number of correct responses; NW, number of workloads; Coordinate, Montreal Neurological Institute coordinat kE, cluster size; *p* (FWE), p-value corrected for family-wise error; *T*, *T*-statistics; Lt, left; Rt, right.

associated with higher-level cognitive functions such as attention and memory (Omura, 2012; Shigihara et al., 2021). Generally, oscillatory activities in higher frequency often reflect physiological compensation mechanisms such as neuroplasticity (McAllister et al., 2013; Pellegrino et al., 2019) while those in lower frequencies tend to reflect pathological changes such as amyloid deposition and stroke (Kaplan and Rossetti, 2011; Fernandez et al., 2013; Nakamura et al., 2018). If KPT scores reflected cognitive dysfunctions associated with AD pathology, the associations should have been found at lower frequencies. However, this was not the case: the association was found in beta, which is higher. The KPT scores are not likely to directly reflect the pathological changes. This dissociation between cognitive dysfunction and pathology remind us that the fact that the severity of cognitive dysfunction in patients with dementia is not parallel with the severity of causative pathologies (Stern et al., 1992; Iacono et al., 2009): patients with severe AD pathology could be cognitively healthy, while patients with mild AD pathology could suffer from severe cognitive impairment. Cognitive performance is influenced by both pathological damage and physiological compensation effects (Fukasawa et al., 2025). The compensation effects are underpinned by neuroplasticity, which is associated with gamma activity (Pellegrino et al., 2019). Taken together, the KPT evaluates the level of cognitive dysfunction associated with physiological effects rather than the severity of AD pathologies.

For the other two secondary scores (NO and NE), a previous study showed that NE correlated with the volume of the left dorsolateral prefrontal cortex and left caudate nucleus, while NO correlated with the volumes on both sides of the superior temporal gyrus and right middle temporal gyrus (Sato et al., 2020). However, our results did not identify the brain regions whose activities were significantly associated with NO or NE. We used regression analyses to explore the significant associations between the scores and regional brain activities (i.e., oscillatory intensities), where the predictors (KPT scores) were assumed to be continuous measures (i.e., covariates). However, the NE has a rather discrete nature, usually between zero and a few; it ranged from zero to two in this study. Therefore, we inferred that the regression analysis could not detect sufficient variance. For NO, omission refers to failing to complete a step in a task (Beaver et al., 2019). It can be caused by multiple factors such as lapses in attention (Perri et al., 2017; Acosta-López et al., 2021), failures in cognitive control, or sensory processing limitations (Damaso et al. 2022) collectively. This multifactoriality may have resulted in a weak statistical power at the group level and failed to identify the brain regions associated with NO.

Taken together, our results showed that cognitive functions evaluated by KPT depend on both the prefrontal cortex and other regions. Particularly, NW was associated with the function in the right precuneus, which is located outside of the prefrontal cortex. This study assumes the functional localisation hypothesis, a fundamental concept in cognitive neuroscience that plays an important role in both clinical medicine and neuroscience. However, it is just a hypothesis explaining an aspect of brain function. We used the SPM for localising brain function. Its analysis concept was originally introduced by Karl Friston, who ironically introduced the 'functional connectivity theory,' which is a conflicting concept to the functional localisation hypothesis (Friston et al., 1993). We should always keep in mind that the functional localisation hypothesis is simply an interpretation of brain function, and cognitive functions can be distributed across brain regions.

This study has a potential limitation. It was a retrospective analysis based on clinical data, and some patients may have had a limited understanding of the test protocols. In such instances, scores like NCR and NW might not have accurately reflected the intended measurements, which could have affected the results. Future studies should aim to enhance patients' understanding of the test protocols by incorporating training sessions prior to administering the actual KPT.

In conclusion, this study revealed that NW (a secondary score) reflects brain activity in the right precuneus, whereas NCR (a primary score) reflects brain activity in the prefrontal cortex. These findings suggest that secondary scores provide additional information about brain activities in the extra-prefrontal cortices that can contribute to the early detection of dementia and qualify as primary information (i.e., NCR) of prefrontal function.

5. Acknowledgements

We thank the patients included in this study and would like to express our genuine gratitude for their contributions to continued progress in the medical sciences. We thank Ms. Yuki Sakamoto, who performed part of the neuropsychological assessment. We sincerely thank Drs. Hajime Kamada (Chairperson, Hokuto Hospital) and Ikuo Hashimoto (Chairperson, Kumagaya General Hospital) for providing access to their facilities.

6. Conflict of interest

Hideyuki Hoshi was employed by RICOH Co., Ltd (manufacturer of magnetoencephalography).

Funding

This study was partially sponsored by RICOH. The sponsors played no role in the study concept/design, methods, data analysis, or paper preparation. Their financial support was used for English editing fee.

References

1. Acosta-López JE, Suárez I, Pineda DA, Cervantes-Henríquez ML, Martínez-Banfi ML, Lozano-Gutiérrez SG, et al (2021), Impulsive and omission errors: Potential temporal processing endophenotypes in ADHD, Brain Sci, 11 (9), 1218.
2. American Psychiatric Association (2013), Diagnostic and Statistical Manual of Mental Disorders, Fifth edition (DSM-5). American Psychiatric Publication, Washington, D.C.
3. Arevalo-Rodriguez I, Smailagic N, Roqué I Figuls M, Ciapponi A, Sanchez-Perez E, Giannakou A, et al (2015), Mini-Mental State Examination (MMSE) for the detection of Alzheimer's disease and other dementias in people with mild cognitive impairment (MCI), Cochrane Database Syst Rev, 2015 (3), CD010783.
4. Ashburner J, Barnes G, Chen CC, Daunizeau J, Moran R, Henson R, et al (2013), SPM12 Manual. Functional Imaging Laboratory. https://www.fil.ion.ucl.ac.uk/spm/ Last viewed date: November 9, 2023.
5. Bak TH, Mioshi E (2007), A cognitive bedside assessment beyond the MMSE: the Addenbrooke's Cognitive Examination, Pract Neurol, 7 (4), 245-249.
6. Bastos AM, Usrey WM, Adams RA, Mangun GR, Fries P, Friston KJ (2012), Canonical microcircuits for predictive coding, Neuron, 76 (4), 695-711.
7. Baudic S, Barba GD, Thibaudet MC, Smagghe A, Remy P, Traykov L (2006), Executive function deficits in early Alzheimer's disease and their relations with episodic memory, Arch Clin Neuropsychol., 21 (1), 15-21.
8. Beaver J, Wilson KB, Schmitter-Edgecombe M (2019), Characterising omission errors in everyday task completion and cognitive correlates in individuals with mild cognitive impairment and dementia, Neuropsychol Rehabil, 29 (5), 804-820.
9. Buchman AS, Bennett DA (2011), Loss of motor function in preclinical Alzheimer's disease, Expert Rev Neurother, 11 (5), 665-676.
10. Cavanna AE, Trimble MR (2006), The precuneus: a review of its functional anatomy and behavioural correlates, Brain, 129 (Pt 3), 564-583.
11. Chau W, McIntosh AR (2005), The Talairach coordinate of a point in the MNI space: how to interpret it, Neuroimage, 25 (2), 408-416.
12. Damaso KAM, Castro SC, Todd J, Strayer DL, Provost A, Matzke D, et al (2022), A cognitive model of response omissions in distraction paradigms, Mem Cognit, 50 (5), 962-978.
13. Dinomais M, Celle S, Duval GT, Roche F, Henni S, Bartha R, et al (2016), Anatomic correlation of the Mini-Mental State Examination: A voxel-based morphometric study in older adults, PLOS One, 11 (10), e0162889.
14. Fernández A, Turrero A, Zuluaga P, Gil-Gregorio P, del Pozo F, Maestu F, et al (2013), MEG delta mapping along the healthy aging-Alzheimer's disease continuum: diagnostic implications, J Alzheimers Dis, 35 (3), 495-507.
15. Friston K, Harrison L, Daunizeau J, Kiebel S, Phillips C, Trujillo-Barreto N, et al (2008), Multiple sparse priors for the M/EEG inverse problem, Neuroimage, 39 (3), 1104-1120.
16. Friston KJ, Frith CD, Liddle PF, Frackowiak RS (1993), Functional connectivity: the principal-component analysis of large (PET) data sets, J Cereb Blood Flow Metab, 13 (1), 5-14.
17. Friston KJ, Holmes AP, Price CJ, Büchel C, Worsley KJ (1999), Multisubject fMRI studies and conjunction analyses, Neuroimage, 10 (4), 385-396.
18. Fukasawa K, Hoshi H, Hirata Y, Kobayashi M, Shibamiya K, Ichikawa S, et al (2025), Regular physical activity affects brain activities in old individuals: an observational study, PLOS One, 20 (7), e0326163.
19. Guarino A, Favieri F, Boncompagni I, Agostini F, Cantone M, Casagrande M (2019), Executive functions in Alzheimer disease: A systematic review, Front Aging Neurosci, 10, 437.
20. Hornero R, Escudero J, Fernández A, Poza J, Gómez C (2008), Spectral and nonlinear analyses of MEG background activity in patients with Alzheimer's disease, IEEE Trans Biomed Eng, 55 (6), 1658-1665.
21. Hoshi H, Hirata Y, Kobayashi M, Sakamoto Y, Fukasawa K, Ichikawa S, et al (2022), Distinctive effects of executive dysfunction and loss of learning/memory abilities on resting-state brain activity, Sci Rep, 12 (1), 3459.
22. Hoshi H, Kobayashi M, Hirata Y, Fukasawa K, Ichikawa S, Shigihara Y (2023), Decreased beta-band activity in left supramarginal gyrus reflects cognitive decline: Evidence from a large clinical dataset in patients with dementia, Hum Brain Mapp, 44 (17), 6214-6226.
23. Iacono D, Markesbery WR, Gross M, Pletnikova O, Rudow G, Zandi P, et al (2009), The Nun study: clinically silent AD, neuronal hypertrophy, and linguistic skills in early life, Neurology, 73 (9), 665-673.
24. 今村陽子, (2005), 臨床高次脳機能評価マニュアル 2000(改訂第 2 版). 株式会社新興医学出版社, 東京

25. 伊藤健吾・乾好貴・木澤剛・木村泰之・加藤隆司, (2017), 認知症の診療における核医学診断の現状と展望. 臨床神経学, 57, 479-484.

26. 金子満雄, (2008), 認知症の大部分は生活習慣病型. 日本未病システム学会雑誌. 14, 30−33.

27. 金子鋭, (2017), 大脳基底核の機能とドパミン神経の役割, 最新医学. 72, 804−810.

28. Kaplan PW, Rossetti AO (2011), EEG patterns and imaging correlations in encephalopathy: encephalopathy part II, J Clin Neurophysiol, 28 (3), 233-251.

29. 樫本直樹, (2012), 認知症を予防することと受け入れること : MCI(軽度認知障害)という概念を手がかりに. 医療・生命と倫理・社会, 1, 66−72.

30. Koshino H, Minamoto T, Yaoi K, Osaka M, Osaka N (2014), Coactivation of the Default Mode Network regions and Working Memory Network regions during task preparation, Sci Rep, 4, 5954.

31. 河野直子, (2012), 早期発見のために—スクリーニングのためのテストバッテリー— 高齢者こころのケアの実践上巻 認知症ケアのための心理アセスメント. 21-27. 株式会社創元社, 大阪

32. Lacy M, Kaemmerer T, Czipri S (2015), Standardized mini-mental state examination scores and verbal memory performance at a memory center: implications for cognitive screening, Am J Alzheimers Dis Other Dimen, 30 (2), 145-152.

33. Lara AH, Wallis JD (2015), The role of prefrontal cortex in working memory: A mini review, Front Syst Neurosci, 9, 173.

34. Loose R, Kaufmann C, Auer DP, Lange KW (2003), Human prefrontal and sensory cortical activity during divided attention tasks, Hum Brain Mapp, 18 (4), 249-259.

35. MacPherson SE (2018), Definition: Dual-tasking and multitasking, Cortex, 106, 313-314.

36. Maki Y, Sakurai T, Okochi J, Yamaguchi H, Toba K (2018), Rehabilitation to live better with dementia, Geriatr Gerontol Int, 18 (11), 1529-1536.

37. 松田博史, (2018), SPECT 検査. 認知症トータルケア, 147(2), 202−203.

38. McAllister CJ, Rönnqvist KC, Stanford IM, Woodhall GL, Furlong PL, Hall SD (2013), Oscillatory beta activity mediates neuroplastic effects of motor cortex stimulation in humans, J Neurosci, 33 (18), 7919-7927.

39. McKhann GM, Knopman DS, Chertkow H, Hyman BT, Jack CR, Kawas CH, et al (2011), The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease, Aizheimers Dement, 7 (3), 263-269.

40. 緑川晶, (2019), 語られないことの理解—認知症の残存機能. Trends In The Sciences, 24, 44−51.

41. Miller EK (2000), The prefrontal cortex and cognitive control, Nat Rev Neurosci, 1 (1), 59-65.

42. 三村將, (2018), 認知症の概念と DSM-5 の診断基準. 認知症トータルケア, 147, 35−36.

43. 水野敬・上土井貴子・渡辺恭良, (2019), 小児慢性疲労症候群—不登校と脳機能—. Jpn J Rehabil Med, 56, 469−475.

44. Mizuno K, Tanaka M, Tanabe HC, Sadato N, Watanabe Y (2012), The neural substrates associated with attentional resources and difficulty of concurrent processing of the two verbal tasks, Neuropsychologia, 50 (8), 1998-2009.

45. 村山尊司・沼田憲治・高杉潤・宮本晴見, (2005), 頭頂葉の機能的役割とその損傷に起因する全身性の運動障害. 脳科学とリハビリテーション, 5, 29−33.

46. Nakamura A, Cuesta P, Fernández A, Arahata Y, Iwata K, Kuratsubo I, et al (2018), Electromagnetic signatures of the preclinical and prodromal stages of Alzheimer's disease, Brain, 141 (5), 1470-1485.

47. 中塚博貴, (2003), 「物忘れ」患者における、MRI 所見、MMSE、仮名ひろいテストについての脳血流 SPECT を用いた統計学的解析による検討. 認知神経科学, 5, 122−126.

48. 「認知症疾患治療ガイドライン」作成合同委員会編, (2010), 認知症の定義, 概要, 経過, 疫学 認知症疾患治療ガイドライン 2010, 1−23. 株式会社医学書院,東京

49. 「認知症疾患診療ガイドライン」作成委員会編, (2017), 症候, 評価尺度, 検査, 診断 認知症疾患診療ガイドライン 2017. 18−53, 株式会社医学書院, 東京

50. 大村一史, (2012), ガンマ帯域活動を対象とした ADHD 研究の新展開. 山形大学紀要教育科学, 15, 267−278.

51. Ohsugi H, Ohgi S, Shigemori K, Schneider EB (2013), Differences in dual-task performance and prefrontal cortex activation between younger and older adults, BMC Neurosci, 14, 10.

52. Ozkirli A, Herzog MH, Jastrzębowska MA (2025), Computational complexity as a potential limitation on brain-behaviour mapping, Eur J Neurosci, 61 (1), e16636.

53. Pascual-Marqui RD (2002), Standardized low-resolution brain electromagnetic tomography (sLORETA): technical details, Methods Find Exp Clin Pharmacol, 24 Suppl D, 5-12.

54. Pellegrino G, Arcara G, Cortese AM, Weis L, Di Tomasso S, Marioni G, et al (2019), Cortical gamma-synchrony measured with magnetoencephalography is a marker of clinical status and

predicts clinical outcome in stroke survivors, Neuroimage Clin, 24, 102092.

55. Perri RL, Spinelli D, Di Russo F (2017), Missing the target: the neural processing underlying the omission error, Brain Topogr, 30 (3), 352-363.

56. Petersen RC (2004), Mild cognitive impairment as a diagnostic entity, J Intern Med, 256 (3), 183-194.

57. Proudfoot M, Woolrich MW, Nobre AC, Turner MR (2014), Magnetoencephalography, Pract Neurol, 14 (5), 336-343.

58. Ray A, Bowyer SM (2010), Clinical applications of magnetoencephalography in epilepsy, Ann Indian Acad Neurol, 13 (1), 14-22.

59. 佐原陽子, (2007), やさしい早期認知症診断法の実際―浜松二段階方式における神経心理機能テストの活用法 脳リハビリテーションの実際. 早期認知症の予防と改善プログラム, 金子満雄・杉田フミエ(編), 28‑41, 医歯薬出版株式会社, 東京

60. 佐藤透・澤田陽一・北本浩・加藤佳樹・吉田頌子・塩塚芳子・佐場英明・森田和美・村上佳奈・佐々木賜・安部雄大・原野かおり (2020) かなひろいテストによる軽度認知障害のスクリーニング ―MRI 画像の VBM 解析による脳局所領域との相関性―. 脳神経外科速報, 30, 978‑988.

61. Shigihara Y, Hoshi H, Fukasawa K, Ichikawa S, Kobayashi M, Sakamoto Y, et al (2021), Resting-state magnetoencephalography reveals neurobiological bridges between pain and cognitive impairment, Pain Ther, 10 (1), 349-361.

62. Shigihara Y, Hoshi H, Poza J, Rodríguez-González V, Gómez C, Kanzawa T (2020), Predicting the outcome of non-pharmacological treatment for patients with dementia-related mild cognitive impairment, Aging, 12 (23), 24101-24116.

63. Smallwood J, Bernhardt BC, Leech R, Bzdok D, Jefferies E, Margulies DS (2021), The default mode network in cognition: a topographical perspective, Nat Rev Neurosci, 22 (8), 503-513.

64. Squire RF, Noudoost B, Schafer RJ, Moore T (2013), Prefrontal contributions to visual selective attention, Annu Rev Neurosci, 36, 451-466.

65. Stern Y, Alexander GE, Prohovnik I, Mayeux R. (1992), Inverse relationship between education and parietotemporal perfusion deficit in Alzheimer’s disease. Ann Neurol, 32(3):371–375.

66. 鈴木匡子, (2011), 認知症の診断における神経心理学的検査の意義. 老年期認知症研究会誌, 18, 56‑57.

67. 高村好実, (2017), 認知症の画像診断. 医学検査, 66, 22‑38.

68. 武田千絵・岩倉瑞希・能登谷晶子, (2017), 若年健常者におけるかなひろいテスト成績の比較. Journal of Wellness and Health Care, 41, 151‑158.

69. Tachibana A, Noah JA, Bronner S, Ono Y, Hirano Y, Niwa M, et al (2012), Activation of dorsolateral prefrontal cortex in a dual neuropsychological screening test: an fMRI approach, Behav Brain Funct, 8, 26.

70. Terakawa Y, Tsuyuguchi N, Tanaka H, Shigihara Y, Sakamoto S, Takami T, et al (2008), Quantitative analysis of MEG using modified sLORETA for clinical application, Clin Neurophysiol, 119 (8), 1917-1922.

71. Thomas B, Sheelakumari R, Kannath S, Sarma S, Menon RN (2019), Regional cerebral blood flow in the posterior cingulate and precuneus and the entorhinal cortical atrophy score differentiate mild cognitive impairment and dementia due to Alzheimer disease, AJNR Am J Neuroradiol, 40 (10), 1658-1664.

72. 渡邉 慶・船橋 新太郎, (2015), 2 つのことを同時にうまくできないのはなぜか―二重課題干渉を生じるメカニズム. Brain and Nerve, 67, 1215‑1229.

73. Woo CW, Krishnan A, Wager TD (2014), Cluster-extent based thresholding in fMRI analyses: pitfalls and recommendations, Neuroimage, 91, 412-419.