Research Article

Optimized CSMRI Algorithm-Based MRI Image Analysis in the Active Rehabilitation Method for Patients with Acute Cerebral Infarction

Chao Zeng, Jing Chen, Wenbing Liu, Kang Liang, Hui Li, Jing Wang, Jingge Li, and Haibo Xu

1Department of Rehabilitation, The Third Affiliated Hospital of Zhejiang Chinese Medical University, Hangzhou 310012, China
2Department of Rehabilitation, Zhejiang Rehabilitation Medical Center, Hangzhou 310051, China
3Department of Traditional Chinese Medicine, Zhejiang Rehabilitation Medical Center, Hangzhou 310051, China

Correspondence should be addressed to Haibo Xu; 2006020130@st.btbu.edu.cn

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This paper combines optimized CSMRI algorithm (CS) and magnetic resonance imaging (MRI) to shorten the scanning time of MRI image data and improve the imaging quality. At the same time, the paper applies functional magnetic resonance imaging (BOLD-fMRI) based on the principle of blood oxygen level dependence to explore the application value of the nerve function reconstruction therapy system for the rehabilitation of active and passive motor functions in patients with acute cerebral infarction. Methods. In this paper, 20 patients with acute cerebral infarction were included. The random drawing method was used to divide them into active group and passive group, each with 10 cases. Both groups were treated with conventional medication and acupuncture. The active group used the active mode of the nerve function reconstruction treatment system to guide the patients’ limb active exercise; all training in the passive group is provided by the nerve function reconstruction treatment system to passively exercise the patients’ limbs; both groups undergo BOLD-fMRI examination before treatment and after 2 weeks of treatment and observe the activated parts of the brain functional area and corresponding parts of the two groups before and after treatment. We observe the activation volume and, at the same time, the ADL score. Results. After treatment, the activation volume and ADL scores of brain functional areas in the two groups were significantly improved compared with those before treatment, and the difference was statistically significant ($P < 0.05$). Conclusion. The combination of optimized CSMRI algorithm (CS) and magnetic resonance imaging (MRI) can be used to evaluate the early rehabilitation efficacy of patients with acute cerebral infarction and has certain guiding value for clinical treatment.

1. Introduction

With the aging of the population, stroke has become a serious public health problem in the world, with high disability, mortality, and recurrence rates, which seriously affect the quality of life and physical health of patients. Resting functional magnetic resonance is a noninvasive brain function detection technology that has been developed rapidly in recent years. Using its blood oxygen level dependence principle, it can accurately and intuitively observe the active parts and ranges of brain functions and study the morphological structure and functional activities of the human brain. In this study, functional magnetic resonance imaging based on the principle of blood oxygen level dependence was used to carry out active and passive exercise rehabilitation before and after the study of patients with acute cerebral infarction. And, clinical value of motor function rehabilitation in patients with acute cerebral infarction was explored [1].

2. Optimized CSMRI Algorithm

The main content of the optimized CSMRI algorithm refers to the following: signals with a sparse representation in a certain transform domain can be collected in a way that is far
below the standard of the Nyquist sampling theorem, sampling can be done through random projections that are incoherent with the transform basis, and the appropriate optimization algorithm can be done to reconstruct the original signal with high probability and accuracy. The theoretical framework mainly includes three aspects: sparse representation, measurement matrix, and reconstruction algorithm (see Figure 1 for details). It can be seen from Figure 1 that the original signal is sparse in some transform domains, which is a priori condition for the optimized CSMRI algorithm to accurately reconstruct the original signal; the selection and design of the measurement matrix directly affects whether the random measurement value retains enough original signal information that can accurately reconstruct the original signal; the reconstruction algorithm is the core of the optimized CSMRI algorithm, which directly affects the signal reconstruction quality and reconstruction speed. At present, the reconstruction algorithms of the optimized CSMRI algorithm are mainly divided into four categories: greedy algorithm, convex algorithms, and the iteration algorithm (see Figure 1 for details). It can be seen from the theoretical framework mainly include three aspects: sparse representation, measurement matrix, and reconstruction algorithm.

The optimization algorithm can be done to reconstruct the original signal with high probability and accuracy. SK_he

On the basis of orthogonal matching tracking algorithm, Donoho and other scholars proposed a piecewise orthogonal matching tracking algorithm based on the phased idea. Then, the existing support set and atoms are updated, and finally, the least square method is used to obtain the approximate accuracy and the iteration margin is updated. In the segmented processing stage, the optimized CSMRI algorithm first constructs a residual vector sequence $r_1, r_2, \ldots$ and then establishes an approximation sequence $X_0, X_1, \ldots, X_s$ marking the coordinate values of nonzero elements in $X_s$, and the initial solution $X_0 = 0, I_s$ is empty:

Step 1: initialize and set the maximum iteration step size and maximum iteration error $e$ and $s = 1$.

Step 2: let the sparse matrix obtained after wavelet transform be $y$ and perform random measurement [3]:

$$\Theta_s = \phi^T r_{s-1}. \quad (1)$$

Step 3: normalize $\Theta$, and $K$ is the signal length of $\Theta_s$:

$$\Theta_s(i) = \frac{\sqrt{m} \Theta_s(i)}{\sqrt{\sum_{1}^{K} \Theta_s(i)^2}}. \quad (2)$$

Step 4: threshold the $\Theta$:

$$\sum s = \{ j : |\Theta_s(i)| > \lambda_s \}. \quad (3)$$

Step 5: the coordinate index of the two latest transformations is merged, and the set $I_s$ is unified:

$$I_s = I_{s-1} \cup \sum s. \quad (4)$$

Step 6: solve the linear equation group

$$\sum (X_s)_{ls} = (\phi_s^T \phi_s)^{-1} \phi_s^T y. \quad (5)$$

Step 7: Calculate the residual $r_s$:

$$r_s = y - X_s. \quad (6)$$

Step 8: determine the accuracy of the sparse solution, such as $r_s < e$, and then $X_0 = X_s$; otherwise $s = s + 1$, return to Step 3. The algorithm flow is shown in Figure 2.

3. Materials and Methods

3.1. General Information. Twenty patients with acute cerebral infarction who were treated in our hospital from October 2017 to May 2019 were divided into 10 cases in the active and passive groups by random drawing and were given different rehabilitation treatments. The study had been approved by the Medical Ethics Committee of the Hospital, and the patients and their families had understood the situation of the study and signed the informed consent forms. In the active group, there were 7 males and 3 females, aged 52–74 years, with an average age of (59.8 ± 8.56) years; the shortest course of the disease was 4 hours, the longest course was 2 days, and the average course was (1.6 ± 0.7) days. In the passive group, there were 6 males and 4 females, aged 57–78 years, with an average of (63.9 ± 7.34) years; the shortest course was 5 hours, the longest course was 2.1 days, and the average course was (1.8 ± 0.5) days. Comparing the general data of the two groups, the difference was not statistically significant ($P > 0.05$) and was comparable [4].

Inclusion criteria included the following: (1) CT excludes cerebral hemorrhage; (2) MRI is diagnosed as unilateral basal ganglia area and/or radiation crown lesion; (3) DWI confirms acute cerebral infarction; (4) Hemiplegia, paralyzed muscle strength I–III, and people with pure motor impairment; (5) people with unconsciousness, speech, and communication impairments; (6) patients and their families voluntarily signing an informed consent form.

Exclusion criteria included the following: (1) disturbance of consciousness; (2) existence of aphasia, apraxia, or neglect; (3) suffering from severe heart, liver, kidney, and other diseases; (4) patients who cannot cooperate to complete MRI examination.

3.2. Sports Rehabilitation. Both groups in the training exercised systematic rehabilitation for active and passive motor functions based on conventional medicine and acupuncture treatment. The active group uses the active mode of the nerve function reconstruction treatment system to guide the active movement of the patients’ limbs; the passive group trains the passive function movement of the patients’ limbs by the nerve function reconstruction treatment system. The schedule was 1 time/d, 30 min/time, and 6 times/week for 2 weeks.
3.3. Brain BOLD-fMRI Scanning Method. SIEMENS Verio 3.0 T magnetic resonance imaging was used to conduct rest functional magnetic resonance imaging on 20 subjects before and 2 weeks after treatment. Scanning sequence and parameters were as follows: The cross-sectional FSE-T2WI image is used as a conventional anatomical positioning image, and the layer thickness is 5 mm; TR/TE: 4650/102 ms; NEX: 2.0; and FOV: 24 cm. The scanning baseline is parallel to the front-to-back joint line, covering a total of 24 layers of the whole brain. Resting-state fMRI sequence: GRE-EPI sequence; TR/TE: 2000/30 ms; flip angle: 90°; matrix: 64 × 64; Nex = 1 time. The subjects lay on their backs on the scanning bed, fixed their heads properly, and used rubber earplugs to reduce noise and were asked to relax, close their eyes and breathe calmly, to keep the whole body still, and to avoid mental activities.

**Figure 1:** Basic theoretical framework of the optimized CS-MRI algorithm.

**Figure 2:** Algorithm flow.
3.4. Resting-State fMRI Data Processing. SPM99-MATLAB image postprocessing software was used to preprocess the original data: remove the image data of the first 10 time points, time and head correction, spatial standardization, registration with standard templates, image smoothing processing, data linear drift, and filter function low frequency after connection. After filtering and functional connection processing, the brain function activation area map is obtained and the region of interest (ROI) is analyzed. The cerebral hemisphere and primary sensorimotor area (SMC) are selected as the areas of interest, and the volume of the brain function activation area is observed [5].

3.5. Scale Assessment. All the subjects underwent daily life activity scale (ADL) measurement before and after treatment and were scored by Barthel index scale. The evaluation standard ranges from 0 to 100 points. The higher the score, the stronger the independence and the smaller the dependence: (1) <20 points: daily life ability needs complete help; (2) 20∼40 points: daily life ability needs great help; (3) 40∼60 points: daily life ability needs help; (4) >60 points: daily life ability can basically be taken care of.

3.6. Statistical Methods. The data were analyzed using SPSS 20.0 statistical software. Normal distribution data were tested by the t test; nonnormal distribution data were tested by the Mann–Whitney U test. \( P < 0.05 \) means the difference is statistically significant.

4. Results

4.1. Brain Functional Area Activation Sites before and after Treatment. Before treatment, the primary ipsilateral sensory motor area (SMC) and secondary motor cortex of the affected limb including premotor area (PMA) and auxiliary motor area (SMA) were activated to varying degrees, and a small area of ipsilateral sensorimotor cortex was activated to a small amount (see Figure 3).

After treatment, the central activation area in the ipsilateral brain of the affected limbs decreased compared with the anterior, while the activation volume of the sensory motor cortex on the opposite side of the affected limb increased. The bilateral secondary motor areas include bilateral SMC, PMA, SMA, and other degrees of activation, as shown in Figure 4.

4.2. Comparison of Activation Volume and ADL Value before and after Treatment in Both Groups. As shown in Table 1, \( D_1 = SMC \) after treatment-SMC before treatment and \( D_2 = ADL \) score after treatment-ADL score before treatment.

5. Discussion

At present, acute ischemic stroke has become a problem that cannot be ignored in the global public health field. The disability rate is high. The recovery of patients after acute cerebral infarction is key, which directly affects the future quality of life of patients. Therefore, it is particularly important to adopt suitable rehabilitation treatment methods and objective evaluation indexes of clinical efficacy. Cerebral infarction is the main cause of vascular dementia. Among the biological factors that lead to vascular dementia, the location and area of cerebral infarction have been regarded as the decisive factor. With the aging population and the increasing use of MRI, this view is worthy of further discussion. The literature reports infarcts in the frontal, temporal, parietal, occipital, thalamus, and basal ganglia areas. No matter which side, there are significant intellectual and memory impairments, mainly expressing language and words, abstract thinking, computing operations, visual memory retention, recent events memory, etc., fronto-temporal lobe damage and abnormal behavior, apathy, and depression. Thalamic and/or basal ganglia area damage may also include thalamus aphasia and lateral neglect, as shown in Figure 5, which is an MRI image of cerebral infarction [6]. There is no statistically significant difference in the location of lesions in this project, which may be related to the following reasons: aphasia, mental symptoms, and other cases that affect neuropsychological examinations. Most of the medical records in this group constituted multiple infarcts. The above two points may affect the specificity and dominant performance of the lesion.

For patients with dementia reported in the literature, our sample consisted of cognitive impairment and borderline
cognitive impairment. To varying degrees, early research focuses on the amount of cerebral infarction. Recent studies have found that the ratio of most cerebral infarction tissues to the brain is not significantly different between patients with cerebrovascular disease dementia and cerebrovascular disease nondementia. Our observations found that in the acute stage of cerebral infarction, cognitive impairment is related to the size of the lesion, but the total infarct volume has not reached 50 ml, which reveals a clinically trending phenomenon, advanced age, and improved medical conditions. Diffuse brain damage was increased [7]. Nerve cell injury and brain edema in the acute phase have become an important cause of cognitive impairment. Another important cause is white matter loosening (LA). During the rehabilitation period of cerebral infarction, brain MRI changes become independent factors that affect cognitive function. This shows that with the resolution of acute cerebral edema and compensation of nerve tissues, limb function recovers at the same time and cognitive function changes accordingly. The main risk factor of cognitive impairment is LA.

Neuropsychological studies in LA have found that spatial structure ability and fine operation skills of the elderly are significantly affected. LA is obviously related to the test results involving the speed of complex information transfer.

### Table 1: Comparison of SMC volume and ADL value of brain function activation area before and after treatment in both groups (\(\bar{x} \pm s\)).

| Group     | Time       | SMC volume (mm\(^3\)) | ADL value (min) | \(D1\) (mm\(^3\)) | \(D2\) (minute) |
|-----------|------------|------------------------|-----------------|---------------------|----------------|
| Active    | Before     | 135.6 ± 32.79          | 27.6 ± 5.62     | 204.3 ± 35.31\(^1\) | 26.5 ± 6.49\(^2\) |
|           | After      | 399.9 ± 28.05\(^1\)\(^2\)| 54.1 ± 5.86\(^1\)\(^2\) |                     |                |
| Passive   | Before     | 130.5 ± 27.25          | 30.0 ± 5.98     | 312.8 ± 61.01\(^3\) | 32.3 ± 4.95\(^1\) |
|           | After      | 443.3 ± 65.13\(^1\)    | 62.3 ± 5.41\(^1\) |                     |                |

Compared with before treatment, \(^1\)\(P < 0.05\); compared with the passive group, \(^2\)\(P < 0.05\).
processing. The group with LA lesions accounted for 60.3%. Among the various tests of the elderly cognitive scale, the cognitive impairment group had the most classification and similar loss points, followed by copying and long memory, with high orientation, digital breadth, and verbal accuracy. It is conceivable that cognitive impairment in the acute phase of cerebral infarction is directly related to the lesion. With the recovery of acute injury, some lesion-related cognitive impairments have also recovered to varying degrees. Figure 6 shows an MRI image of acute-stage cerebral edema. Complex cognitive functions such as spatial structural ability caused by LA failed to recover at 6 months.

The human brain is like a complex network. Each control node in the network has an intricate relationship with the surrounding area. However, due to individual differences, it may cause changes in the control nodes of the entire brain network of different patients after stroke, which eventually leads to differences in the activation area. After cerebral infarction, certain brain function reorganization will naturally occur, but it is relatively limited. The reorganization of motor cortex function after stroke involves a multimechanism and multilevel regulation. Although many scholars have reached a certain consensus on the reorganization and remodeling of brain functional areas after cerebral infarction, the precise mechanism of activation of the brain areas has not yet been fully revealed and further research is needed. Early rehabilitation treatment can accelerate the establishment of medial collateral circulation, promote the reorganization or compensation of brain functional areas, and maximize the potential of the brain’s own “plasticity.”

The results of this study showed that the corresponding brain functional areas of the two groups were activated to different degrees after treatment, the activation volume of the contralateral motor functional area of the affected limb increased after the treatment in the two groups, and the activation volume of the passive group was higher than that of the active group. The two groups were effective after treatment with conventional drugs, acupuncture, and nerve function reconstruction treatment systems, and the passive group had better efficacy of passive exercise on patients with passive function than the active group; after the rehabilitation treatment, all the selected patients’ ADL scores increased significantly, suggesting that the patients’ motor function is significantly improved. Therefore, the nerve function reconstruction system has a significant effect on early rehabilitation of cerebral infarction. Studies have shown that normal people’s resting-state motor network’s bilateral functional connection is symmetrical. A series of studies have found that bilateral cerebral hemispheres inhibit each other and this mutual inhibition is destroyed after cerebral infarction. This study found that the bilateral cerebral hemispheres of patients with cerebral infarction have anatomical and functional asymmetry, and changes in the activation sites of brain functional areas suggest that the rehabilitation process is accompanied by the reconstruction of brain functional areas. After the treatment of acute cerebral infarction, the activation volume of the ipsilateral brain area of the affected limb decreased, while the activation volume of the contralateral brain area increased, indicating that the reorganization of brain function changes with
different periods of cerebral infarction and the activation pattern gradually tends to be original. At present, many studies have proved that resting fMRI can observe the network connection between the functional areas of the entire brain, that is, the functional connection that reflects the subcortex [8].

6. Conclusion

Although some pathological data or neuroimaging evidence still indicate that the volume, number, and location of cerebral infarction tissues are important factors in determining vascular dementia, our research shows that many. There are white matter lesions, mainly found in the frontal lobe, subparietal cortex, and lateral ventricle, which are caused by deep small-vessel diseases and chronic cerebral perfusion insufficiency. There are white matter lesions, mainly found in the frontal, subparietal cortex, and the lateral ventricle, which are caused by deep small vessel disease and chronic cerebral perfusion insufficiency. Therefore, cerebral lesions are independent risk factors for cognitive impairment after infarction. If these patients have cognitive impairment, is it diagnosed asBinswanger disease? Some people think that the current diagnosis of Binswanger disease is too broad, and some people think that it is the opposite. We believe that white matter lesions cause impaired axoplasmic transport and blood-brain barrier function, as well as astrocyte degeneration and microglia activation, which destroys cognitive function, especially integration loops related to complex cognition. Both basic research and clinical epidemiological research data support that LA is a high-risk factor for dementia. For LA patients with mild or marginal cognitive impairment, clinical treatment and observation should be based on Binswanger disease. Due to the shorter observation time of this study and the longer scanning time of BOLD-fMRI, the patients’ compliance and tolerance were greatly affected, so the brain function activation area included in this study is less and the number of cases included is also less. These will have a certain impact on the statistical results, so it is necessary to increase the sample size to improve the accuracy of the study.

Data Availability

No data were used to support this study.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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