Journal of Biosciences and Medicines, 2025, 13(9), 1-10
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Progress of Respiratory Dysfunction and Rehabilitation Treatment on Stroke Patients

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How to cite this paper: Zhou, J.L. and Cheng, X.L. (2025) Progress of Respiratory Dysfunction and Rehabilitation Treatment on Stroke Patients. *Journal of Biosciences and Medicines*, **13**, 1-10.
https://doi.org/10.4236/jbm.2025.139001

Received: July 11, 2025
Accepted: August 30, 2025
Published: September 2, 2025

Abstract

Stroke obviously affects the respiratory function of patients, resulting in poor prognosis of patients. Respiratory dysfunction in stroke is manifested as reduced lung function, weakened respiratory muscle strength, respiratory rhythm disturbance, and lung infection. The pathogenesis includes direct damage of primary disease, superimposed damage of complications and iatrogenic aggravated damage. Effective pulmonary rehabilitation measures can significantly improve the respiratory function of patients, and the main rehabilitation measures include respiratory muscle training, airway clearance and diaphragm pacing.

Keywords

Stroke, Respiratory Dysfunction, Respiratory Rehabilitation

1. The Harm of Stroke

Approximately 795,000 people in the United States experience a stroke each year, of which 87% (690,000) are ischemic strokes, 185,000 are relapses, and approximately 240,000 individuals experience a transient ischemic attack (TIA) [1]. Although significant progress has been made in the primary prevention and acute treatment of stroke, stroke is still a major life-threatening disease. Due to population aging, the absolute number of strokes is expected to increase dramatically in the next few years [2]. Three quarters of stroke survivors are left disabled, and one quarter are severely disabled. The functional impairments left behind are usually the following: respiratory dysfunction, limb movement dysfunction, sensory dysfunction, which can lead to limb sensory loss or abnormality, aphasia or dysarthria, emotional disorders, urinary and bowel dysfunction, cognitive dysfunction, etc. Respiratory dysfunction is one of the common complications of stroke, mainly

manifested as reduced ability of lung ventilation and air exchange, decreased muscle strength of respiratory muscle, atrophy of respiratory muscle, abnormal respiratory rhythm and respiratory pattern, etc., which greatly affect the functional recovery of stroke patients [3] [4]. Respiratory training can increase the participation of respiratory muscle in exercise and strengthen the respiratory muscle strength [5], which can effectively improve the respiratory function of stroke patients and improve the exercise ability [6], and is an important method to promote the functional recovery of stroke patients.

2. Manifestations of Respiratory Dysfunction on Stroke

2.1. Reduced Pulmonary Function

Pulmonary dysfunction in stroke patients is usually manifested by significant reductions in maximum spontaneous ventilation, maximum respiratory pressure, maximum vital capacity, second forced breathing volume and peak expiratory flow rate [7]. Teixeira *et al.* [8] found that the muscle strength of respiratory muscle and lower abdominal muscle in stroke patients were weakened, and maximum inspiratory pressure (PImax) and maximum expiratory pressure (PEmax) were lower than those of healthy people of the same age. Neuronopathy often results in decreased PImax and PEmax and functional imbalance between inspiratory and expiratory muscles [9]. In 2000, Khedr *et al.* [10] found that about 40% of stroke patients had reduced diaphragmatic displacement, and forced vital capacity (FVC), forced expiratory volume in one second (FEV1) reduced to 50% of the predicted value. After stroke, respiratory muscle paralysis caused by damage to the critical position of the respiratory center can lead to unilateral ventilation insufficiency, and at the same time, the compliance of the chest lung is changed, and the elastic and inelastic resistance of the lung is increased, thereby reducing the lung ventilation and perfusion capacity, showing restrictive ventilation disorders and the impairment of total lung capacity and vital capacity [11].

2.2. Weakened Respiratory Muscle Strength

Studies have found that the respiratory muscle strength of stroke patients is only half of that of normal individuals [12], and studies have confirmed that the diaphragm of stroke patients is significantly thinner than that of healthy individuals, and the diaphragm thickness on the affected side is significantly lower than that on the healthy side at the end of expiratory breath [13]. In stroke patients, the respiratory muscle muscle strength is decreased, which often leads to shallow and rapid breathing, atelectasis, and dynamic changes in the movement of the rib diaphragm and chest wall, and further leads to restricted thoracic activity and restricted ventilation dysfunction. In addition, weak respiratory muscles can lead to ineffective coughing, retention of secretions, and an inability to keep the airway open. Sutbeyaz [14] pointed out that threshold pressure load training of respiratory muscle can improve the muscle strength of respiratory muscle of stroke patients, and has a significant effect on improving the muscle strength and fatigue

level of respiratory muscle of stroke patients.

2.3. Disordered Respiratory Rhythm

Stroke may affect the central control of respiratory drive and respiratory pattern, airway protection and maintenance, and respiratory mechanisms of inhalation and exhalation [15]. Nerve damage after stroke leads to many changes in respiration, many of which are most prominent during the acute phase of stroke. Immediate changes include abnormal breathing patterns, impaired airways, and reduced respiratory muscle migration. Due to these changes, there is a high incidence of hypoxia, aspiration, and pneumonia in acute stroke patients. At the same time, central nervous system damage can lead to hyperventilation, resulting in relative alkalosis, respiratory muscle weakness and hypoxia symptoms. Sleep apnea (SDB) in stroke patients includes central sleep apnea (CSA) and obstructive sleep apnea (OSA). Severe breathing disorders and respiratory failure after acute stroke usually improve with resolution of cerebral edema, but sleep disnea (SDB) is often common and persistent in stroke survivors. Post-stroke CSA is associated with changes in the breathing rhythm produced by the brain stem during sleep, whereas OSA is primarily the result of reduced muscle activity that maintains airway patency. Many stroke survivors suffer from OSA [16], and OSA is associated with adverse stroke outcomes, including an increased risk of stroke recurrence and worse recovery after stroke.

2.4. Lung Infection

Stroke-associated pneumonia (SAP) is a common post-stroke disease affecting 14% of patients [17] associated with an increased risk of in-hospital death [18], longer hospital stay [19], and considerable economic impact on healthcare resources [20]. Immune deficiency due to stroke increases susceptibility to infection; and the inhalation of oropharyngeal secretions and stomach contents into the lungs, associated with impaired consciousness and difficulty swallowing, may also contribute to the development of infections.

3. Mechanism of Respiratory Dysfunction in Stroke

3.1. Direct Damage from Primary Disease

Stroke can directly damage the respiratory center at all levels and destroy the nerve conduction pathway related to breathing, leading to respiratory system dysfunction such as abnormal pulmonary ventilation and/or pulmonary ventilation [21]. Kim *et al.* [22] believed that abnormal respiratory central rhythm, disruption of nerve conduction pathway and bulbus paralysis caused by stroke are the main causes of hyperventilation and even respiratory failure in patients. Damage to the corticospinal tract can lead to respiratory muscle paralysis, tidal respiration with extensive brain damage, central neurogenic hyperrespiration with damage to the midbrain tegmental area, long inhalation breathing with damage to the first tegmental area of the pontine (apnea after full inspiration), and cluster breathing

with damage to the tail tegmental area of the pontine area (apnea after 4 - 5 breaths). The medulla oblongata is followed by ataxic respiration (breathing rate and amplitude change from time to time, with apnea) [23]. Airway clearance is obstructed due to central cough injury, and decreased CO_2 chemoreceptor reflex of medulla oblongata may induce obstructive or central sleep apnea [24].

3.2. Complications Superimposed Damage

Stroke patients are prone to multiple complications, such as aspiration, secondary pneumonia and sleep apnea [25], which further aggravate respiratory dysfunction. Increased intracranial pressure caused by stroke can lead slow breathing [26]. Pain, depression, and anxiety further limit respiratory muscle exertion [27]. Disturbance of water and electrolyte balance can cause respiratory depression or hyperventilation, resulting in sluggish response to CO_2 ventilation, and then reduced response to hypercapnia or hypoxia [28]. Aspiration and pulmonary infection increase physiological dead space [29].

3.3. Iatrogenic Aggravated Damage

In the treatment of stroke, a variety of controllable or uncontrollable iatrogenic factors such as drug therapy and mechanical ventilation can induce or aggravate respiratory system dysfunction and hinder the rehabilitation process of stroke patients [30]. In the early stage of cerebral infarction, patients need to immobilize and rest in bed, especially rest in supine position, which will inevitably weaken respiratory movement and increase physiological ineffective chamber [31]. In the early stage, the patient is anxious and irritable, and sedation is given to the patient [32], which will inevitably inhibit the patient's breathing and reduce cough, change the respiratory rate and respiratory rhythm, and affect the patient's respiratory function. Patients with pulmonary infection or urinary system infection will inevitably use antibiotics. The application of antibiotics can inhibit and kill beneficial bacteria and reduce body immunity, which is not conducive to rehabilitation and functional recovery of patients [26]. If acute respiratory failure occurs after stroke, artificial airways are usually established by mechanical ventilation to improve oxygenation. Although the use of mechanical ventilation greatly reduces the mortality rate, it causes complications such as ventilator-associated diaphragm dysfunction, ventilator-associated pneumonia, ventilator-induced lung injury and other complications [33], ultimately , the patient's respiratory function decreased, long-term dependence on ventilator, and poor clinical outcome [34].

4. Rehabilitation Measures for Respiratory Dysfunction on Stroke

The American Thoracic Society/European Respiratory Society defines pulmonary rehabilitation as an individualized comprehensive intervention after a comprehensive assessment of the patient's condition, including exercise training, health education, psychological intervention and nutritional support, aiming to improve

the physical and psychological status of patients with chronic respiratory diseases [35]. Mccool *et al.* [36] proposed that when stroke patients are given sufficiently intense training to improve muscle strength in the early stage, the strength and endurance of respiratory muscles will also increase, which can ultimately improve respiratory function. Respiratory rehabilitation includes respiratory assessment and respiratory therapy, which includes exercise training, breathing style training, respiratory muscle training, airway clearance and diaphragm pacing, etc. This article focuses on the progress in the treatment of respiratory muscle training, airway clearance and diaphragm pacing.

4.1. Respiratory Muscle Training

Respiratory muscle training has been shown to be effective in improving inspiratory and expiratory strength, lung function, and dyspnea, and these benefits can also be carried over into exercise [37]. Moreover, respiratory muscle training can effectively reduce the risk of respiratory complications and improve dysphagia by reducing osmosis or aspiration when swallowing fluids after stroke [38]. Karen reported that EMST can improve expiratory muscle strength, reflex cough intensity and cough impulse, so that stroke patients can benefit from the up-regulation of reflex cough by training, thus improving airway protection [39]. Specific respiratory muscle training (IMT) improves the function of the inspiratory muscles [40]. According to literature and clinical experience, there are 3 established methods: resistive load, threshold load and normocapnic hyperpnea.

4.2. Airway Clearance Techniques

Airway clearance techniques are non-drug treatments that acts on the air stream by physical or mechanical means to assist the trachea and bronchus discharge phlegm or induce cough to expel phlegm. ACTs can assist expectoration through mechanical methods, including high-frequency chest wall compression, intrapulmonary tap ventilation, positive expiratory pressure, and mechanical inspiration-exhalation, including postural drainage and spontaneous respiratory circulation techniques [41]. ACT relies on two of the most important physiological principles. First, a mechanism allows air to move after the blockage and ventilate the remote area; secondly, the expiratory air flow is regulated to push the proximal end of the secretion upward into the airway [42]. Common methods of airway clearance include manual techniques or the use of (oscillating) positive expiratory pressure systems. Common methods of airway clearance include manual techniques or the use of (oscillating) positive expiratory pressure systems. In some clinical situations, these techniques may be ineffective and the physiotherapist will need pneumatic instrument support [43]. Zhang [44] reported that non-invasive positive pressure ventilation combined with routine airway clearance is effective in elderly patients with stroke-associated pneumonia, which can shorten hospital stay and reduce cost. Active cycle of breathing techniques (ACBT) is a flexible and autonomically controlled breathing training model for treatment and rehabilita-

tion, which consists of breathing control, chest expansion training and forced breathing techniques [45]. The whole process requires the active participation of patients. Clinical studies have shown that ACBT is effective in clearing bronchial secretions and can improve lung function without exacerbating hypoxemia and airflow resistance.

4.3. Diaphragm Pacing Therapy

Extracorporeal diaphragm pacing (EDP) improves lung function by functional electrical stimulation of the diaphragmatic nerve to increase lung ventilation through up and down movement of the diaphragm following passive electrical stimulation. In addition, functional stimulation can also increase the blood flow of the diaphragm, thicken the diaphragm fibers, increase the muscle strength and endurance of the diaphragm, and improve the quality of life of patients. Yan Bin *et al.* [46] studied the effect of extracorporeal diaphragm pacemaker combined with high-flow nasal intubation oxygen therapy in the treatment of patients with severe cerebral hemorrhage, and the results showed that the combination could increase diaphragm migration, shorten the offline time and the length of stay in the neurological intensive care unit. External diaphragm pacing has the advantages of simple and convenient operation and non-trauma, but the placement of diaphragmatic nerve electrodes during treatment is often difficult to accurately locate due to individual differences and various external factors, and the excessive intensity of current stimulation and fast frequency of stimulation are easy to cause diaphragm fatigue, which is likely to have a certain impact on the reconstruction and recovery process of diaphragm function. Therefore, the feasibility and effectiveness of external diaphragm pacing therapy need to be further studied.

5. Outlook

Respiratory dysfunction in stroke patients is common and the reasons for it are various. Early implementation of respiratory rehabilitation for stroke patients is of great significance for improving respiratory function, motor function, reducing the risk of recurrence and improving the quality of life. However, due to the diversity of manifestations of respiratory dysfunction after stroke, it is suggested that targeted breathing training be provided to patients according to different conditions of the patients after the assessment.

Conflicts of Interest

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

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