Research Article

Cuestiones de Fisioterapia

Medicine

EFFECT OF HIGH FREQUENCY (HF) AND LOW FREQUENCY (LF) NEUROMUSCULAR ELECTRICAL STIMULATION (NMES) OF LOWER LIMB MUSCLES ON SYMPTOM PERCEPTION AND FUNCTIONAL WALKING CAPACITY IN SEVERE COPD

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Abstract

Background and Purpose: Neuromuscular Electrical Stimulation (NMES) is a new and emerging modality in the rehabilitation of severely disabled patients with COPD. The objective of the study was to compare the effects of HF (50Hz) and LF NMES (15 Hz) on symptom perception and functional walking capacity in severe COPD patients. Methods: 30 patients, who fulfilled selection criteria were included in this study & were randomly divided into group a & b. group a (n=15) was given conventional physiotherapy & high frequency neuromuscular electrical stimulation & group b (n=15) was given conventional physiotherapy & low frequency neuromuscular electrical stimulation, for a period of 6 weeks. mrc dyspnea scale and vo2 max were taken before and after the treatment in both the groups. results: There was a significant reduction in MRC Dyspnea score and improvement in VO2 MAX in both the groups and a significant difference was found between group A and B ($p<0.05$). CONCLUSION: The result of the present study suggests that Low Frequency NMES is more effective than High Frequency NMES in reducing symptom perception and improving functional walking capacity, and thereby Low Frequency NMES can be recommended in treating severe COPD patients.

Keywords: Chronic Obstructive Pulmonary Disease, Neuromuscular Electrical Stimulation, MRC Dyspnea Score, VO2 MAX

Introduction

According to GOLD Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking[1].

The chronic airflow limitation, characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Chronic inflammation causes structural changes and narrowing of the small airways. Destruction of the lung parenchyma, also by inflammatory processes, leads to the loss of alveolar attachments to the small airways and decreases lung elastic recoil; in turn, these changes diminish the ability

of the airways to remain open during expiration[2]. Risk factors for COPD include exogenous factors such as smoking and air pollution, as well as endogenous factors of the patients themselves. The greatest exogenous factor for COPD is smoking, in addition, there are occupational dusts and chemical materials (vapors, irritant substances, smoke), passive smoking and respiratory infectious diseases The most definite endogenous risk factor is the genetically inherited a-1 antitrypsin deficiency.[3] This causes an inflammatory response in the lungs. In addition to inflammation, an imbalance of proteinases and antiproteinases in the lungs, and oxidative stress are also important in the pathogenesis of COPD. The different pathogenic mechanisms produce the pathological changes which, in turn, give rise to the following physiological abnormalities in COPD: mucous hyper secretion and cilliary dysfunction; airflow limitation and hyperinflation; gas exchange abnormalities; pulmonary hypertension; and systemic effects.[4]

Patients with COPD often complain of chronic cough and sputum production, dyspnea on exertion, reduced exercise capacity, and develop a progressive decline in lung function with increasing age. These symptoms have been attributed to increases in the work of breathing and impairments in gas exchange that result from airflow limitation and dynamic hyperinflation. With progression of the disease, loss of weight and anorexia can become problematic and these are poor prognostic factors.[5]

The physiotherapists play a very important role in management of COPD. The interventions used by physiotherapist include enhancing lung capacity by thoracic mobility exercises, improving expiration with pursed lip breathing exercise and airway clearance intervention such as postural drainage, coordinated with breathing control and vibrations followed by forced expiratory maneuvers. [8] Exercise training as a component of pulmonary rehabilitation, and positive effects of exercise training are now well-documented. However, standard rehabilitation such as aerobic training with cycle or treadmill walk exercise, is harder to perform in severe COPD patients with major muscle deconditioning due to chronic resting breathlessness, repeated exacerbations, and associated loss of muscle mass, high values of oxygen uptake and ventilation, which exact a relatively high claim on the peak aerobic capacity of patients and exercise training is thus not always possible to perform. The improvement observed after exercise training is smaller in patients with severe dyspnea compared to patients with low-to-moderate dyspnea[9]

Neuromuscular Electrical Stimulation (NMES) involves the application of an electrical current through electrodes placed on the skin over the targeted muscles, depolarizing motor endplates inducing skeletal muscle contractions.[10,11]

High frequency Neuromuscular Electrical Stimulation consist of electrically stimulating muscles with frequency of≥ 50 HZ.[13]

Low frequency Neuromuscular Electrical Stimulation consist of electrically stimulating muscles with frequency of ≤ 50 HZ.[13]

NMES is a method of augmenting muscle performance. This technique has been shown to increase the capillary/fiber ratio, the fiber cross sectional area, as well as muscle mass and the number of type I and type II fibers in humans. It has also been shown to improve oxidative potential of the muscle.[14]

The major advantage of NMES over conventional exercise training in severe COPD patients is the virtual absence of ventilatory stress during passive exercise, reflecting the smaller muscle mass involved. Patients would therefore be comfortably able to cope with a training regimen which, if provided by voluntary dynamic contractions, would be demanding and probably not tolerable by patients. Thus, the beneficial changes in muscle function could be translated into more general benefit.[7]

METHODOLOGY:

This is a study of Quasi experimental design, with randomized sampling method.30 patients are divided into group A and group B.

Inclusion criteria:

Male patients with age group between 40 and 60 years with clinical diagnosis of severe COPD, according to GOLD (30% FEV, <50% of predicted) as referred by concerned physician.

Patients with incapacitating breathlessness according to the Medical Research Council scale that is, scores of 4 or 5 are included. Patients with no acute respiratory failure at the time of study, who have discontinued smoking for at least past one year, Patients with skeletal muscle weakness, and those who agree for participation. Patients whose disease stability as indicated by no change in medication dosage or acute exacerbation of symptoms in the preceding 4 weeks. Self reported exercise limitation despite pharmacological treatment and Absence of associated locomotor or neurological conditions are included.[10,11,12]

Exclusion Criteria:

COPD patients with history of Cardiovascular disease, Other pulmonary disease, Neuromuscular disease, Active or debilitating joint disease, History of thoracic, abdominal or limb surgery, Skin condition that may interfere with the application of the NMES are excluded.

Patients who had been treated with systemic corticosteroids and neuromuscular blocking agents and those who were not willing to participate are excluded.[10,11,12]

PROCEDURE:

A total number of 34 subjects diagnosed to have COPD were screened out of which 30 subjects were selected for the study. Each patient was screened initially by using a simple selection Performa relevant to the inclusion and exclusion criteria. Those who fulfilled this symptomatic criterion underwent a detailed assessment. Then the selected patients who were willing to participate were randomly divided into two groups of 15 each in Group-A and Group-B. The details and the purpose of the study were explained to all the patients and informed consent was obtained from each patient was assigned to group-A, second patient was assigned to group-B.

Standard care and medications, as advised by the concerned physician, was strictly implemented throughout the intervention. After receiving the medical request and signed consent form by physician for intervention, patients were randomly assigned into 2 groups. Group A patients received conventional physiotherapy and (HF) NMES for 6 week Group B patients received conventional physiotherapy and (LF) NMES for 6 weeks After 6 weeks of interventions, post treatment scores of MRC dyspnea scale and VO, MAX which was calculated on the basis of 6 minute walk distance were measured. Results were compared and analyzed statistically.

1. MRC Dyspnea scale: Each participant was asked indicate the the level dyspnea.The score was taken and after treatment. The patient would be marking on the scale himself indicating as a subjective experience.[22]

2. VO2 MAX: The six-minute walking distance (6MWD) was measured according to standardized ATS protocol.[23]In 6 MWT, shape the walking course (continuous straight) appears be determinant of distance walked not length of course, (Frank Sciurba et 2003).[24] Patients were asked walk as along 10 meter corridor

in 6 minute. Subject's walking pace self-determined and down or for rest if needed. Neither encouragement, nor time information were given during the test. The total walking distance was measured at the end of 6 minute. [27]Based on the amount of distance walked in 6MWT, VO2 MAX was calculated from following equation VO2max = 9.99-0.096 * age(yr) + 0.022* 6 min walk(m) by Daniel starobin et al (2006).[20]

STATISICAL ANALYSIS:

The statistical analyses were done using software called Statistical Package for Social Science (SPSS 16) for windows. The statistical analyses used were Descriptive analysis to find Means and standard deviation. Intergroup comparison of pre treatment and post treatment scores of MRC dyspnea score between groups was analyzed using non parametric Mann Whitney U test. Intra group comparison of pre & post treatment scores of MRC dyspnea score for group A and B was analyzed using non parametric Wilcoxon Signed Rank Test. Inter group comparisons pre treatment and post treatment scores for VO2 Max between groups was analyzed by using parametric independent t-test. Intra group comparison of pre & post treatment scores for VO2 Max was analyzed by using parametric Paired Samples t test.

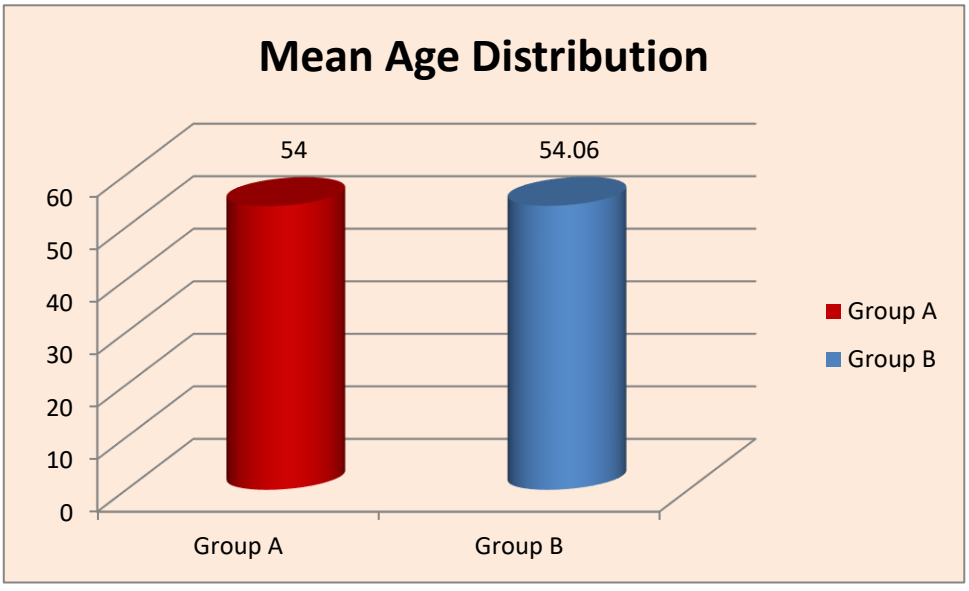
RESULTS:

Total 30 patients took part in study. Non parametric Wilcoxan signed rank test and Mann Whitney U test for intra and inter group comparison is used for MRC Dyspnea score. Parametric paired t test and independent samples t test is used for intra and inter group comparison for VO2 MAX.

Table 1: Age distribution of group A (HF NMES) and Group B (LF NMES)

Group	N	Mean age	Standard deviation	p value
Group A	15	54	±3.047	0.949
Group B	15	54.06	±3.034	

Table 1 shows the Age distribution among Group A and Group B. The mean age and Standard deviation of Group A patients was 54.00 ± 3.04. The mean age and standard deviation of Group B patients was 54.06 +3.03. P value (p>0.05) shows no significant difference between the two groups

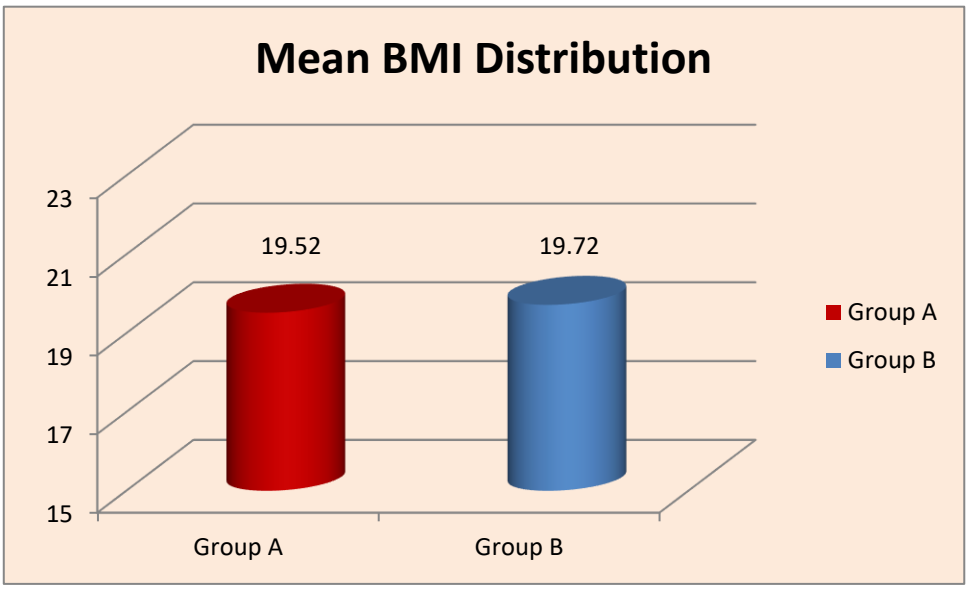


Graph 1: Age Distribution

Table 2: BMI distribution of group A (HF NMES) and group B (LF NMES)

Group	N	Mean BMI	Standard deviation	p value
Group A	15	19.52	±1.59	0.743
Group B	15	19.72	±1.46	

Table 2 shows the BMI distribution among Group A and Group B. The mean and Standard deviation of Group A patients was 19.52 1.59. The mean and standard deviation of Group B patients was 19.72 + 1.46. P value (p>0.05) shows no significant difference between the two groups.



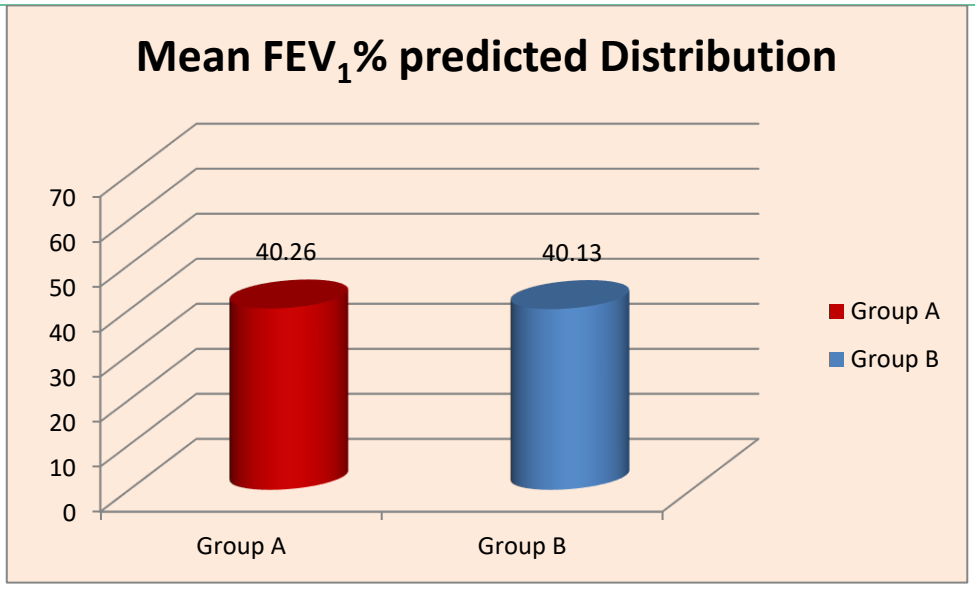
Graph 2: BMI Distribution

Table 3: FEV1% predicted distribution of group A (HF NMES) and Group B (LF NMES)

Groups	N	Mean FEV1 predicted	Standard deviation	p value
Group A	15	40.26	±4.13	0.937
Group B	15	40.13	±4.99	

Table 3 shows the FEV1% predicted distribution among Group A and Group B.

The mean and Standard deviation of Group A patients was 40.26 ± 4.13. The mean and standard deviation of Group B patients was 40.13 +4.99. P value (p>0.05) shows no significant difference between the two groups.



Graph 3: FEV_1% predicted Distribution

Table 4: Inter Group Pre Mrc Dyspnea Score Comparison (Between Group A and B)

Group	Duration	Calculated U value	Table U value	P – value (p<0.05)
A	Pre	105	64	0.77
B	Pre			

Interpretation of the results:

After analyzing the data with Man Whitney U Test, the calculated U-value is 105 and table U-value is 64 for MRC Dyspnea score. The results show that calculated U-value is greater than table U-value and P value is 0.77, showing that there is no significant difference between pre values of MRC Dyspnea score of Group A and B.

Table 5: Intra Group Pre and Post Values of Mrc Dyspnea Score of Group A

Group	Duration	Mean	SD	Calculated Z-value	Table Z-value	p-value (P<0.05)
A	Pre	4.46	±0.51	2.106	25	0.03
	Post	3.6	±1.32			

Interpretation of the results:

The mean and standard deviation of MRC Dyspnea Score of Group A is measured before and after the treatment. The Mean of MRC Dyspnea Score before and after the treatment is 4.46 and 3.6. The Standard deviation of MRC Dyspnea Score before and after the treatment is ±0.51 and +1.32. According to Wilcoxon Signed Ranks Test, in Group A, calculated Z-value is 2.106 and table Z-value is 25. The result shows that calculated Z-value is lesser than tabulated Z-value and p-value is 0.03, showing that there is significant difference between pre and post values of MRC Dyspnea score of Group A.

Table 6: Intra Group Pre and Post Values of Mrc Dyspnea Score of Group B

Group	Duration	Mean	SD	Calculated Z-value	Table Z-value	p-value (P<0.05)
B	Pre	4.4	±0.50	2.948	25	0.003

	Post	**2046**	**±1.40**			

Interpretation of the results:

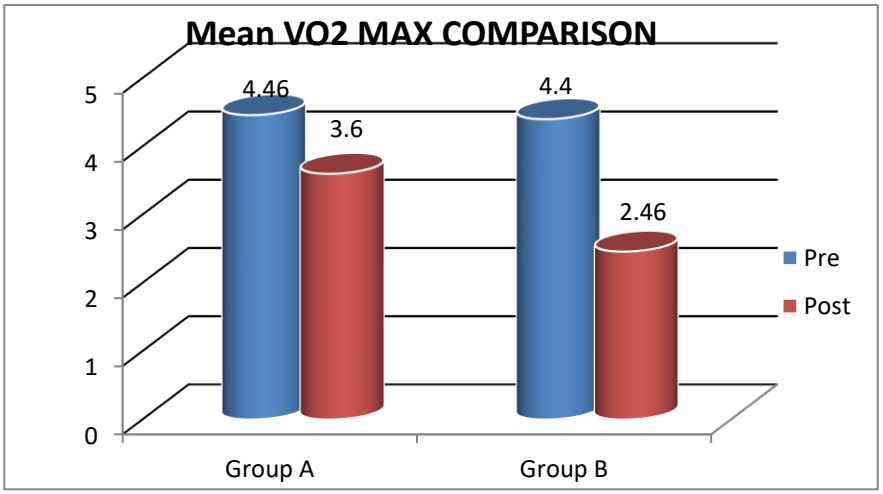
The mean and standard deviation of MRC Dyspnea Score of Group B is measured before and after the treatment. The Mean of MRC Dyspnea Score before and after the treatment is 4.4 and 2.46. The Standard deviation of MRC Dyspnea Score before and after the treatment is 10.50 and +1.40. According to Wilcoxon Signed Ranks Test, in Group B, calculated Z-value is 2.948 and table Z-value is 25. The result shows that calculated Z-value is lesser than tabulated Z-value and p-value is 0.003, showing that there is significant difference between pre and post values of MRC Dyspnea score of Group B.

Table 7: Inter Group Post MRC Dyspnea Score Comparison (Between Group A and B)

Group	Duration	Calculate	Table U value	P-value (P<0.05)
A	**Post**	**62**	**64**	**0.04**
B	**Post**			

Interpretation of the results:

After analyzing the data with Man Whitney U Test, the calculated U-value is 62 and table U-value is 64 for MRC Dyspnea score. The results show that calculated U-value is lesser than table U-value and P value is 0.04, showing that there is significant difference between post values MRC Dyspnea score of Group A and B.



Graph 4: Shows Mean Pre and Post Comparison of MRC Dyspnea Score of Group A and Group B

Table 8: Inter Group Pre Vo2 Max Comparison (Between Group A and B)

Group	Duration	Calculate	Table U value	P-value (P<0.05)
A	**Pre**	**0.004**	**2.048**	**0.997**
B	**Pre**			

Interpretation of the results:

After analyzing the data with independent sample t test, the calculated t-value is 0.004 and table t-value is 2.048 for VO; MAX. The results show that calculated t-value is lesser than table t-value and P value is 0.997 showing that there is no significant difference between pre values of VO, MAX of Group A and B.

Table 9: Intra Group Pre and Post Values of Vo2 Max of Group A

Group	Duration	Mean	SD	Calculated Z-value	Table Z-value	p-value (P<0.05)
A	Pre	8.0	±0.53	3.852	2.145	0.002
	Post	9.00	±0.81			

Interpretation of the results

The mean and standard deviation of VO, MAX of Group A is measured before and after the treatment. The Mean of VO_2 MAX before and after the treatment is 8.04 and 9.00. The Standard deviation of VO, MAX before and after the treatment is ±0.53 and +0.81. According to Independent sample t test, in Group A calculated t-value is 3.852 and table t-value is 2.145. The result shows that calculated t-value is greater than tabulated t-value and p-value is 0.002, showing that there is significant difference between pre and post values of VO, MAX of Group A

Table 10: Intra Group Pre and Post Values of Vo2 Max of Group B

Group	Duration	Mean	SD	Calculated Z-value	Table Z-value	p-value (P<0.05)
B	Pre	8.04	±0.40	4.499	2.145	0.001
	Post	10	±1.20			

Interpretation of the results:

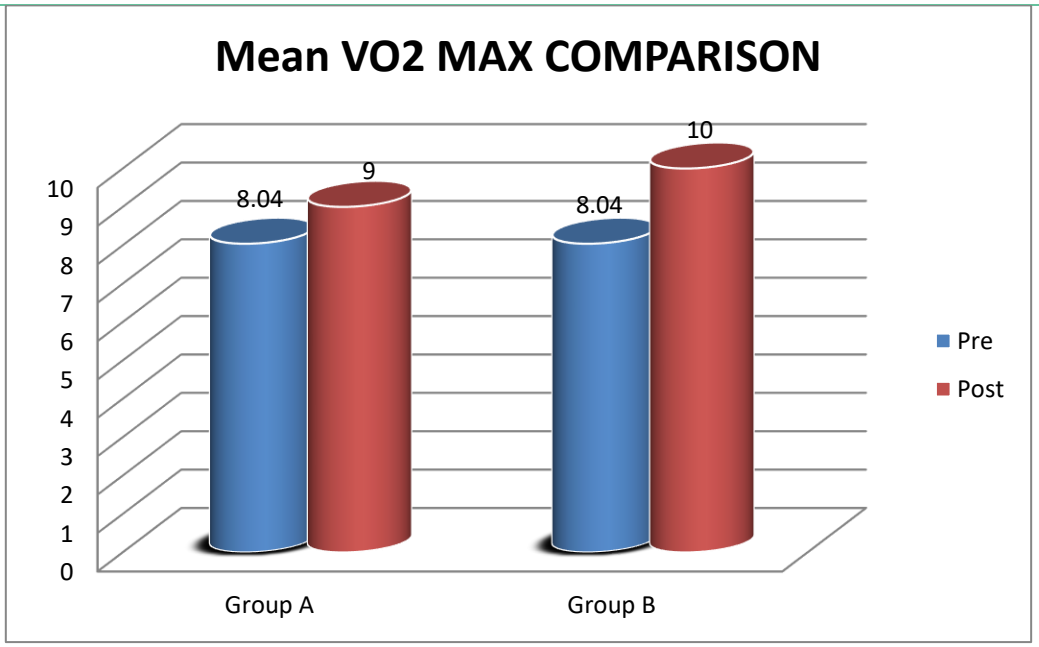
The mean and standard deviation of VO, MAX of Group B is measured before and after the treatment. The Mean of VO, MAX before and after the treatment is 8.04 and 10 The Standard deviation of VO, MAX before and after the treatment is ±0.40 and 1.20. According to Independent sample t test, in Group B calculated t-value is 4.499 and table t-value is 2.145. The result shows that calculated t-value is greater than tabulated t-value and p-value is 0.001, showing that there is significant difference between pre and post values of VO; MAX of Group B.

Table 11: Intra Group Post Vo2 Max Comparison (Between Group A and B)

Group	Duration	Calculate	Table U value	P-value (P<0.05)
A	Post	2.646	2.048	0.013
B	Post			

Interpretation of the results:

After analyzing the data with Independent sample t test, the calculated t-value is 2.646 and table t-value is 2.048 for VO, MAX. The results show that calculated t-value is greater than table 1-value and P value is 0.013, showing that there is significant difference between post values of VO, MAX of Group A and B



Graph 5: shows mean pre and post comparison of VO2 MAX of Group A and Group B

DISCUSSION:

Neuromuscular Electrical Stimulation is a new and emerging modality in the habilitation of severely disabled patients with COPD.[17]Exercise training and pulmonary rehabilitation are integral parts of the medical management of patients with COPD. However, some patients with severe ventilatory limitation may be unable to tolerate it due to chronic resting breathlessness, repeated exacerbations, and associated loss of muscle mass, Hence there is a growing interest in new and alternative rehabilitative strategy such as NMES.[14] The study was detailed and tailored on 30 severe COPD patients to find the efficacy of which type of Frequency was comparatively better among the two groups comprising of 15 patients each, Group A receiving conventional physiotherapy and HF NMES and Group B receiving conventional physiotherapy and LF NMES, using two evaluating tools comprising of MRC Dyspnea Score and Maximal oxygen consumption (VO2 MAX) which is calculated based on 6 minute walk distance.

The results found in this study disclosed that after a six-week treatment, both the groups attained a significant reduction in MRC Dyspnea Score with P-0.03 for Group A groups a and P-0.003 for Group B, compared with the normal confidence interval value of $P<0.05$. Moreover, both the groups attained a significant improvement in VO, MAX with P-0.002 for Group A and P-0.001 for Group B with ($P<0.05$). These findings are in accordance with the ones found by JA Neder et al, 2002, who investigated the effect of Home based NMES as a new rehabilitative strategy for severely disabled patients in COPD with frequency of 50 HZ and concluded that, for severely disabled COPD patients electrical stimulation can improve muscle strength and endurance, whole body exercise tolerance, and breathlessness during activities of daily living.[7]The findings are also in accordance with the study by Martin J. Nuhr et al, 2004 on Beneficial effects of chronic low-frequency stimulation of thigh muscles in patients with advanced chronic heart failure using frequency of 15 HZ and found that there was an improvement in VO2 max and in the 6-min walking test performance and LF NMES is a stable treatment to counteract detrimental changes in skeletal muscle and to increase exercise capacity in patients with severe CHF.[16]

Hence Passive training of specific locomotor muscle groups by means of neuromuscular electrical stimulation might be better tolerated than whole body exercise in patients with severe COPD. At structural

level NMES has been shown to increase the capillary/fibre ratio, fibre cross sectional area, muscle mass, and the number of type I and type II fibers in humans.[14] Skeletal muscle structural and functional changes patients. This also has been noted by,

Whittom and colleagues (1998) demonstrated that type fiber proportion by 20%, whereas the proportion of type Ilb fibers by 10% with severe COPD.[8] Maltais and colleagues (1999) confirmed shift toward type fibers and reduction in the proportion of type 1 fibers in lateralis muscles patients with severe COPD.[25] It should be emphasized that fiber type shifting more glycolytic fibers in limb muscles of COPD patients is associated with reductions in muscle oxidative capacity, as shown by significantly lower levels of COX and SDH in the quadriceps of patients with severe COPD (Gosker et al 2002)[21]. Many alterations occur at structural, metabolic and functional level in skeletal muscles following HF and LF Neuro Muscular Electrical Stimulation training. This can be explained by following studies, Nuhr M et al (2004) found that NMES with Low frequency increases the level of oxidative enzymes whereas HF NMES increases level of glycolytic enzymes. [15,16] Moreover there is strong evidence that NMES with low stimulation frequencies increase type fibers and decrease type II fibers. Also there is strong evidence that NMES with high stimulation frequencies decrease type fibers (Dal es et 2007).[19]

According to Eller et al (1998) VO2 max is the best index of aerobic capacity and the gold standard for cardio respiratory fitness. It represents the maximal achievable level of oxidative metabolism involving large muscle groups. Determinants of VO2 MAX include O carrying capacity of blood and oxidative capacity of muscle fibers.[26] Gaëlle Deley, et al 2008 found that low frequency NMES for a period of 6 weeks is similar to 21 aerobic training and improves walking capacity.[18] As previously noted, LF NMES improves oxidative capacity of muscles, moreover increasing number and density of capillaries and mitochondrial enzymes, all of this an important indicator of improvement in VO2 MAX.[13]

LIMITATIONS

No blinding was done, no long term follow up was done, the study consisted of smaller sample size and all the patients were not on same medicine.

CONCLUSION

The study concludes that Neuro Muscular Electrical Stimulation is effective in reducing symptom perception and improving functional walking capacity in severe COPD patients and also it has been proved that Low Frequency NMES is more effective than High Frequency NMES, and thereby Low frequency NMES can be recommended in treating severe COPD patients.

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