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COMPARISON OF CHEST EXPANSION IN CHILDREN WITH SPASTIC CEREBRAL PALSY WITH AGE AND GENDER MATCHED NORMALLY DEVELOPED CHILDREN.

Physiotherapy

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ABSTRACT

Background: Cerebral palsy is collection of diverse syndromes characterized by disorders of movement and posture caused by non-progressive injury to immature brain. Respiratory complications may arise in spastic cerebral palsy children due to muscular imbalance that could be a major cause of morbidity and mortality in the long term. Respiratory impairments in children with cerebral palsy is often overlooked and rarely assessed in clinical practice. Chest expansion is important tool to measure compliance of lung. **Aim:** To assess the chest expansion in children with cerebral palsy and compare the same with normally developed children. **Participants and methods**: A total of 20 children diagnosed with spastic diplegic cerebral palsy in age range of 6-12 years and forty age matched normally developed controls were enrolled. Assessment of chest expansion (CE) at three levels and comparison of mean values was done between the two groups. p-value <0.05 was considered as statistically significant. **Results:** Children with diplegic spastic cerebral palsy were found to have reduced chest expansion in comparison to normally developed children at all the three levels (p value <0.05). **Conclusion:** Chest mobility as evaluated by (CE) is decreased in diplegic spastic CP, which may interfere with respiratory function later in their life.

KEYWORDS

Cerebral palsy, Spastic diplegic, Chest expansion, Respiratory impairment, Chest mobility, Tape measurement.

Introduction:
Cerebral palsy (CP) is a disorder of movement and posture caused by nonprogressive injury to the immature brain and is characterized by a collection of diverse syndromes.[1]

Consequences of neuromuscular impairment in a CP child may lead to lung damage and reduced lung function, though there is no direct airway or parenchymal lung dysfunction. [2-5]

There is an increased risk of significant morbidity and mortality in CP children from respiratory infections due to poor nutritional status, drooling, aspiration, gastroesophageal reflux, impairment of airway clearance by muscular weakness or incoordination and poor pulmonary reserve (due to chest wall or spine deformity) and spasticity. [6-8]

Residual chronic lung disease contributing to pulmonary problems may be present in premature infants or who had prolonged neonatal course. [9]

Other than movement issues, there are a variety of other symptoms and breathing problems that may be present in a person with CP such as high incidence of recurrent pneumonia, atelectasis, bronchiectasis, and restrictive lung disease. Compromised pulmonary function and associated respiratory infections may lead to illness and pre mature death. A major role is played by respiratory problems in the quality of life and expectancy of these children. [10,11]

The consequences due to neuromuscular and musculoskeletal dysfunction indirectly compromise the pulmonary functions, though the condition itself does not directly cause respiratory problems. Underlying pathology in cerebral palsy especially the spasticity of muscles (rib cage is not an exception) has a direct effect leading to weakness of respiratory muscles. Intercostals are affected relatively early, causing paradoxical breathing and a bell-shaped chest. Diaphragm weakness occurs later and may lead to the onset of respiratory failure later in the life. [12]

Many changes are observed in the ribcage and thoracic spine structure overtime in spastic cerebral palsy children which may lead to chest wall impairments. The ribcage tends to stay elevated with lack of effective lower trunk activity to anchor it to the pelvis and begins to take its shape because of muscular imbalance. Chest mobility is limited by breathing using an abnormal pattern for a long period and shortening of respiratory muscles and stiffening of costovertebral joints may worsen it. [13]

Moreover, spinal curvatures are observed in cerebral palsy because of imbalance between muscle tone and gravity. Lung function is restricted by decreased chest wall compliance and decreased mechanical advantage of the respiratory muscles due to chest wall deformity secondary to severe kyphoscoliosis. In addition, scoliosis can result in unequal lung expansions (basal atelectasis on the concave side and overexpansion of the convex side), leading to ventilation/perfusion mismatch. These factors together increase the work of breathing and may predispose a cerebral palsy child to respiratory failure in the latter part of their lives . [14]

Having known the above mentioned pathomechanics in CP children, the aim of this study was to compare chest mobility at three different levels by means of chest expansion in spastic CP patients and in normal healthy individuals, which would help to gain a better understanding of the role of respiratory muscle weakness, spasticity, thoracic cage limitations and physical activity level on respiratory dysfunction. [2,4,14]

We believe that the findings of our study may be helpful in rehabilitative efforts focused on respiratory dysfunction, especially in choosing the appropriate exercises for the persons with CP.

MATERIAL AND METHODS:
20 consecutive children with spastic CP with mean age of 8.525±2.2 years [range 6 to 12 years] and 40 healthy controls subjects with a mean age of 8.525±2.18 years [range 6 to 12 years] were evaluated in the study. It was a cross sectional comparative study conducted at Outpatient department of VSPM`s College of Physiotherapy, Nagpur and other Paediatrics Neuro-physiotherapy setups in the city, using the convenient sampling. The controls were evaluated from the residential areas of Nagpur city. None of the CP child had any intrinsic lung disease. The body weight, height and BMI were recorded for each subject. Grades of spasticity according to Ashworth scale [slight, mild, moderate, severe], Level of ambulation [non-ambulatory, ambulatory with orthosis and or assisted devices, ambulatory without orthosis or assisted devices] and presence of any spinal deformity were noted for

the Cerebral Palsy. The patient's chest circumference at maximal voluntary inspiration and at maximal voluntary expiration and chest expansion [difference between the two] were measured in sitting position using a tap measure marked at 0.1 cm increment at the level of Axilla, 4th intercostal ad xiphisternum.

All the children were informed about the procedure and were asked to exhale as much as possible and hold the position for chest expiration measurements and to take as deep breathe as possible and hold it for chest inspiration measurements. Only the children who could follow the commands and who would co-operate throughout the examination were included in the study. The best of 3 attempts was recorded as the final chest expansion.

Formal sample size calculation was carried out by considering the mean difference [Effective size= 1.8 cm] with an α error of 5%. The required sample size was 20 in study group with an allocation ratio of 1:2.

Children who were able to follow the commands were able to sit with or without support and those who can walk with or without support between age group of 6-12 years of diagnosed case of diplegic cerebral palsy were included. Known case of any respiratory disease were eliminated, congenital disease, cognitively impaired or those who had an history of acute respiratory infection in last 6 weeks were excluded. Written informed consent will be taken from parents/ guardians of all participants. Chest expansion was measured in sitting position using a tape at the level of axillary, 4th intercostal, xiphisternum level. Children were asked to take as deep breathe as possible and hold it for initial reading measurement and then asked to exhale as much as possible and hold the position for final reading. The difference was recorded as chest expansion.

RESULTS:

Sixty children, 20 with spastic cerebral palsy (Group 1) and 40 normally developed children (Group 2) were enrolled in study. All children were spastic diplegic, 9 children were ambulatory with the device and support and 11 were ambulatory without device or support Most of the children were having the grade 1 spasticity. The two groups were comparable with respect to demographic characteristics. None of the patients had apparent chest deformity. Both the groups were similar with respect to age, gender, weight, height, BMI.

On comparing the chest circumference at expiration, the mean value of CP group was 60.40, 59.770, 56.85 at axillary, 4th intercostal, xiphisternum level respectively and the mean value of control group was 60.43, 59.695, 57.45 at axillary, 4th intercostal, xiphisternum level respectively and the p value was 0.987, 0.979, 0.817 at 3 levels respectively, which showed no statistical difference between the two sub groups. (Table 1)

Similarly, there was no statistically significant difference observed between the two groups on measurement of chest circumference at maximum inspiration. (Table 2)

However, when the chest expansion (difference between the complete chest expiration to maximum inspiration), the CP group showed reduced chest expansion as compared with controls. Analysis revealed that Chest expansion was significantly decreased in the cerebral palsy group when compared with the controls ($p<0.001$) at all the three levels (Axillary, 4th intercostal and xiphisternum level). (Table 3)

Table 1 showing Comparison of mean values of Chest Expiration in CP and control group.

Chest expiration	Group	N	Mean	Std. Deviation	T value	P value
Axillary	Cerebral Palsy	20	60.40	9.084	-0.017	0.987
	Normal	40	60.43	8.709		
4th intercostals	Cerebral Palsy	20	59.770	10.1818	0.026	0.979
	Normal	40	59.695	10.5751		
Xiphisternum	Cerebral Palsy	20	56.85	8.461	-0.232	0.817
	Normal	40	57.45	9.767		

Table 2 showing Comparison of mean values of Chest Inspiration in CP and control group.

Chest inspiration	Group	N	Mean	Std. Deviation	T value	P value
Axillary	Cerebral Palsy	20	62.345	9.4369	-0.448	0.656
	Normal	40	63.460	8.9237		
4th intercostals	Cerebral Palsy	20	61.73	10.556	-0.504	0.616
	Normal	40	63.18	10.521		
Xiphisternum	Cerebral Palsy	20	58.84	8.806	-0.648	0.520
	Normal	40	60.55	10.075		

Table 3 showing Comparison of mean values of Chest Expansion (CE) in CP and control group.

Chest expansion	Group	N	Mean	Std. Deviation	T value	P value
Axillary	Cerebral Palsy	20	1.925	0.507	-7.433	<0.001
	Normal	40	2.975	0.520		
4th intercostals	Cerebral Palsy	20	1.90	0.809	-8.804	<0.001
	Normal	40	3.49	0.572		
Xiphisternum	Cerebral Palsy	20	2.010	0.607	-7.052	<0.001
	Normal	40	3.113	0.552		

DISCUSSION

The results of present study showed that the chest mobility as evaluated by chest expansion was decreased at all the three levels i.e Axillary, 4th intercostal and xiphisternum level in spastic cerebral palsy children when compared with healthy controls.

Normal function of nervous system, respiratory muscles and costo vertebral joints are needed for chest expansion. As cerebral palsy child does not have articular involvement, the limited chest mobility may be due to impaired neuromuscular control and incoordination, weakness, spasticity and secondary changes in respiratory muscles. [1-4,15,16]

Stiffening of the costo-vertebral joints could be due to the inability of respiratory muscles to adequately increase and decrease the volume of the thoracic cavity which may result in decreased chest expansion.[15]

Shallow and reversed abnormal patterns of breathing have been reported in studies on cerebral palsy which may limit the chest mobility by shortening of respiratory muscles and stiffening of costo-vertebral joints in the long run.[16]

There was a derth of literature on chest expansion measurements especially in Indian populace but studies have reported marked decreased in the vitals capacity in cerebral palsy children as low as 23 to 67 % of the predicted normal values.[1-4] It can be inferred that limited chest expansion observed in our study may be an indirect reflection of reduced vital capacity as seen in cerebral palsy patients.

Studies have reported that spastic cerebral palsy children have a tendency to develop barrel shaped chest which is a chest deformity seen in COPD and could result in chronic air trapping. Also, FEV1% have been found to be low in cerebral palsy children as reported in the literature.[2]

Chest expansion was observed to be decreased in our study subjects when compared with healthy controls even though marked respiratory muscle involvement is not expected in diplegic patient which was our study population. Our results are in line with the findings of Murat Ersoz et al [1].

None of the patients in our study sample were able to run or participate in sports as normal children do. This finding suggest that increased respiratory demand as a result of intensive physical activity plays a major role in development of normal chest mobility. As lung impairment is an important cause of morbidity and mortality for cerebral palsy children[6], evaluation and early management of

respiratory dysfunction should be considered as an integral part of routine rehabilitation.

Conclusion

In conclusion, chest mobility as evaluated by CE is decreased in diplegic spastic CP, which may interfere with respiratory function later in their life.

This finding raise the need of early initiation of pulmonary rehabilitation which may improve and maintain chest mobility and respiratory function.

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