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Beneficial effects of Incentive Spirometry on Pulmonary Problems in Patients with sickle cell Anemia

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Abstract
Objective: To detect the effects of incentive spirometry on pulmonary problems in patients with sickle cell anemia.

Subjects: Forty patients with homozygous sickle cell anemia assigned randomly into 2 groups; IS group that received incentive spirometry as well as medical treatment and Control group received medical treatment only. Pulmonary functions were measured before and after 8 weeks.

Results: the results of study showed that there were significant differences after intervention between both groups as p value <0.05.

Conclusion: The study concluded that incentive spirometry increased pulmonary functions and decreased the pulmonary problems in patients with sickle cell anemia.

Keywords: Incentive spirometry, Sickle cell anemia, Pulmonary functions

1. Introduction

Sickle cell disease (SCD) is one of the most common inherited diseases worldwide. The disease is characterized by chronic hemolytic anemia, as well as acute and chronic complications [1]. SCD is a group of inherited red blood cell disorders characterized by the presence of abnormal hemoglobin. The clinical manifestations are diverse and may include vaso-occlusive, hematological and infectious crises [2].

Sickle cell anemia (SCA) is a serious problem placing excessive demands on both the patients and their families. In Africa and the Caribbean, SCA is the commonest inherited disorder with about 200, 000 to 250, 000 children born per year. Among people with SCA (homozygous for sickle haemoglobin), the median age at death is 42 years for males and 48 years for females with haemoglobin SS (Hb-SS) [3].

Onset of SCA occurs in early childhood where symptoms are usually: anemia, restricted range of motion, shortness of breath, fatigue and delayed growth and development. Jaundice, organ damage, pulmonary hypertension and even heart failure can occur in severe cases. As the disease progresses, a common side-effect is extreme pain due to sickles becoming “stuck” in small blood vessels[4,5]. Pulmonary complications are one of the commonest set of complications in SCD. They are also a major cause of acute morbidity and mortality in SCD [6].

Pulmonary function abnormalities in SCD are frequent and are characterized by airway obstruction, restrictive lung disease, abnormal diffusion capacity, and hypoxemia. Sickle cell chronic lung disease is presumably related to recurring episodes of infarction and infection and is characterized by a decrease in radiolucency of the lungs, moderate to severe impairment of pulmonary function, and in its most severe form by evidence of pulmonary hypertension[7,8].

Acute chest syndrome consists of a combination of signs and symptoms including dyspnoea, chest pain, fever, cough, multifocal pulmonary infiltrates on the chest radiograph, and a raised white cell count. It is a form of lung injury that can progress to adult respiratory distress syndrome (ARDS). It is estimated that half of all patients with sickle cell anemia will develop ACS at least once in their lives, and ACS is the second most common cause of admission after painful vaso-occlusive crises[9,10].

Despite clinical advances, SCA remains a difficult, chronic medical condition for many children and youth. Therefore, additional treatment strategies, including complementary and

alternative medicine therapies, would be welcome to enhance the clinical care of SCA patients[9]. Antibiotics including macrolides, liberal use of analgesics and incentive spirometry are mainstay of treatment [11].

physical therapists can play a key role in rehabilitating patients with sickle cell anemia. The involvement of physical therapy in their treatment has resulted in the patients having a shortened hospitalization and in the patients receiving reduced amounts of potentially harmful and addictive analgesics. The mainstay of successful treatment is high quality supportive care. Fluid management, oxygenation, chest physiotherapy, bronchodilators, and intermittent incentive spirometry are essential elements of management in the absence of a specific therapy that consistently ameliorates clinical course[12].

Physical therapy plays an essential role in the multidisciplinary management of SCD symptoms, particularly for ROM, balance, and airway clearance issues. Therapists could play a large role in education, treatment and possible prevention of exacerbations. Patient education is extremely important for individuals with sickle cell. They should be educated on the importance of physical activity and remaining mobile in order to combat serious pulmonary and other systemic complications. Furthermore, the patient should be taught breathing techniques and incentive spirometry to also prevent acute chest syndrome and atelectasis, as well as retain adequate lung capacity [13-15]. The purpose of this study was to evaluate the efficacy of incentive spirometry in the treatment of pulmonary problems developed in patient with SCA.

Patients & Methods:

Subjects:-

Forty eight patients (27 girls, 21 boys) with homozygous sickle cell anemia were admitted to National Cancer Institute, Cairo, Egypt, between May 2011 and August 2012. Reasons for hospital admission were sever pain unrelieved by analgesics; fever; respiratory distress; a sharp decrease in the hemoglobin concentration; and a need for oxygen. Diagnosis, history and clinical examination were done for all patients by hematologist. Subjects who fulfilled the following criteria were eligible for enrollment in the study; age between 7-15 years (2) not in any crisis. Additional criteria also included consent to measurements and availability for re-assessments. They were however, informed of their freedom to withdraw from the study at any point in time.

A complete history was taken and routine physical examination as well as laboratory tests routinely performed by physicians to assess the severity of the disease. The following tests were performed: complete blood and differential counts, reticulocyte count, measurement of hemoglobin F and S concentrations, blood culture if the patient was febrile, measurement of oxygen saturation with a pulse oximeter while the patient breathed room air, and chest radiography. The patients were randomized into two groups of equal number. Group (A); that received incentive spirometer as well as medical treatment (in the form of analgesics, antibiotics) and Group (B); received medical treatment only. Assessment was done before, and after 8 weeks of treatment for all patients by measurement of pulmonary functions. All patients had the same medical care and have no evidence impairment of cardiac, renal, and hepatic function.

Measurement procedures

Zan-680 Ergospiro "Ergospirometry System", (manufactured by ZAN Me Bgerate Gmbh, Germany) is a recording device for synchronous registration of breathing flow, respiration volumes as well as inspired and expired gases. It consists of breath gas (O2 and CO2) analyzer, Gas bottle, Computer unit fed with soft ware required for control the treadmill load, and to manipulate and analyze the measured parameters, thermal printer to print out the results. It also includes rubber mouthpiece, clips, mask and electronic treadmill. It was used to measure pulmonary functions such as Forced vital capacity "FVC", Forced expiratory volume after 1 second "FEV1", Maximum voluntary ventilation "MVV".

Calibration of device

On the testing day; the Ergospirometry System was electronically calibrated by click upon the icon of calibration at the computer monitor. Before starting Gas-calibration, the following steps were checked in advance: (1) Zan testing unit had been ON for a minimum warm up time of 20 min. (2) Gas bottle was opened. (3) Gas-suction tube removed from flow sensor and connected to calibration nipple (Cal) on front panel of Zan Testing Unit (it is recommended that Gas-calibration must be carried out before every measurement). Calibration was started with (Enter); the flow of Calibration-Gas was clearly audible. As Calibration was ok, the results were saved with (Enter) (the recommended composition for calibration gas is: 5% CO2, 15.9% O2). At the end, Gas bottle was closed and Gas-Suction tube was removed to flow sensor.

Fig 1: Zan-680 Ergospiro "Ergospirometry System",

Measurement of pulmonary functions

The height and weight were measured for each patient then the computer unit was fed up by the patient demographic parameters (name, age, weight, height, and sex). Each patient was instructed to lose any tight clothes and to sit in upright position in front of the Ergospirometry system equipment. The patient nose was closed with the nose clips and put the mouthpiece in the patient's mouth.

a) Measurement of Forced vital capacity "FVC "

Forced vital capacity "FVC " is The amount of air which can be forcibly exhaled from the lungs after taking the deepest breath possible. FVC is the most basic maneuver in spirometry tests. Each patient was instructed to inspire slowly and deeply, and then he expired with power as quick and as much as possible. The patient was performed at least three trials and the best performance was used for analysis. The unit of measurement was liters [16].

b) Measurement of forced expiratory volume at 1 second "FEV1":

Forced expiratory volume (FEV) measures how much air a person can exhale during a forced breath. FEV1 is the amount of air exhaled measured during the first second of the forced breath. Predicted normal values for FEV1 depend on age, sex, height, weight and ethnicity as well as the research study that they are based on [17].

c) Measurement of maximum voluntary ventilation "MVV":

Maximal voluntary ventilation (MVV), also known as Maximum breathing capacity is a measure of the maximum amount of air that can be inhaled and exhaled within one minute. MVV is the largest volume that can be breathed into and out of the lungs during a 10-15-s interval with maximal voluntary effort [18, 19].

For the comfort of the patient this is done over a 15 second time period before being extrapolated to a value for one minute expressed as liters/minute. Each patient was instructed to breathe as rapidly and fully as possible for 12-15 seconds. The total volume was obtained through multiplying this volume by the appropriate number (5 if tested for 12 seconds, 4 if tested for 15 seconds) [20,21].

Treatment procedures

Standard incentive Spirometry device used in this study to provide sustained deep breathing exercise for group "A" only. It is a flow type device, consisted of three champers with three balls that provides visual feed back to the patient. Patient was asked to sit on the edge of the bed then hold the incentive spirometer in an upright position. Patient placed the mouthpiece in his mouth and seal his lips tightly around it.

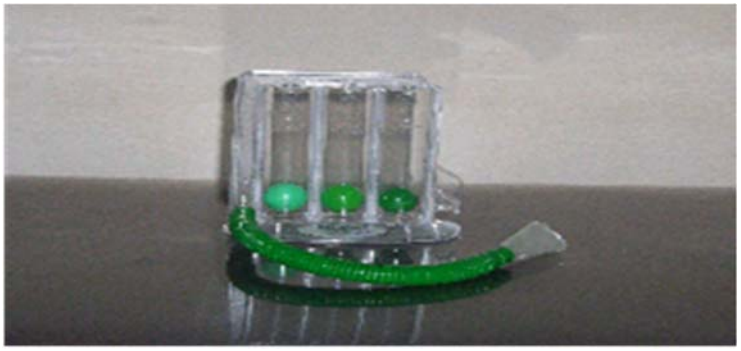

Fig 2: Incentive Spirometry device",

Patient was asked to breathe in slowly and as deeply as possible, raising the balls toward the top of the column then hold his breath as long as possible (for at least five seconds), then allow the balls to fall to the bottom of the column. The exercise was performed twice per day for 15 minutes and 5 times/week for 8 weeks. The exercise was splitted into 5 minutes if the subject was unable to train for 15 minutes without interruption. After each set of 10 deep breaths, practice coughing to be sure his lungs are clear [22].

Statistical procedures:

In this study, the mean, standard deviation was calculated for both groups of patients. Paired t- test was utilized to identify the changes in each group over time for pulmonary function tests (FVC, FEV1, MVV), Independent t test was used to compare the variables between both groups. All hypotheses were tested at 0.05 level of significance.

Results

A total of 48 patients was screened for eligibility, and 41 subjects fulfilled the inclusion criteria. One subject of 41 reported poor adherence to the treatment, (a participant with poor adherence to the program defined as missing more than three consecutive sessions or more than 20% of all sessions) and excluded from the study, and his data were not used in the statistical analysis. A total of 40 subjects completed the study and were initially randomized into two groups of equal number. IS group (n=20), and Control group (n=20). **Table (1**) presents the characteristics of the patients completing the study. Both groups were comparable at the baseline regarding to the demographic and clinical characteristics.

Table 1: Demographic and clinical characteristics

Variables	IS group		Control group		P values
Age (years) (mean ±SD)	11.10±2.77		12.05±2.31		0.246*
Sex (Male - Female)	Male	14(70%)	Male	13(65%)	0.739*
	Female	6(30.5%)	Female	7(35%)	
Height (cm)	1.317±10.56		1.318±7.87		0.973*
Weight (Kg)	26.65±4.64		28.01±4.14		0.304*
Hb($g.ml^{-1}$)	8.50±0.81		8.27±0.80		0.383*
Number of hospitalization	One	14(70%)	One	16(80%)	0.520*
	Two	4(20%)	Two	2(10%)	
	Three	2(10%)	Three	2(10%)	
Initial FVC	1.931±0.534		2.05±0.483		0.437*
Initial FEV1	1.67±0.332		1.69±0.294		0.775*
Initial MVV	55.95±14.45		58.80±13.18		0.519*

* No significant differences between groups pretreatment

SD; standard deviation

Measurement of Forced vital capacity "FVC "

In table (2); The results of study showed significant difference of FVC pre & post treatment in IS group as p value <0.05 while it showed non-significant differences in Control group pre & post treatment as p value >0.05**.** In comparing both groups post treatment, the results of study showed a significant differences p value <0.05.

Table 2: Measurement of Forced vital capacity "FVC "

	IS Group	Control Group	P value between both groups post treatment
FVC Pre	1.931±0.534	2.05±0.483	0.006**
FVC Post	2.464±0.396	2.064±0.477	
P value	0.000**	0.909*	

* No significant difference
* * Significant difference

Measurement of forced expiratory volume at 1 second "FEV1":

In table (3); The results of study showed significant difference of FEV1 pre & post treatment in IS group as p value <0.05 while it showed non-significant differences in Control group pre & post treatment as p value >0.05**.** In comparing both groups post treatment, the results of study showed a significant differences p value <0.05.

Table 3: Measurement of forced expiratory volume at 1 second "FEV1":

	IS Group	Control Group	P value between both groups post treatment
FEV1Pre	1.67±0.332	1.69±0.294	0.002**
FEV1` Post	2.095±0.366	1.71±0.348	
P value	0.000**	0.547*	

* No significant difference
* * Significant difference

Measurement of maximum voluntary ventilation "MVV":

In table (4); The results of study showed significant difference of MVV pre & post treatment in IS group as p value <0.05 while it showed non-significant differences in Control group pre & post treatment as p value >0.05**.** In comparing both groups post treatment, the results of study showed a significant differences p value <0.05.

Table 4: Measurement of maximum voluntary ventilation "MVV":

	IS Group	Control Group	P value between both groups post treatment
MVV Pre	55.95±14.45	58.80±13.18	0.038**
MVV ` Post	73.80±21.21	59.70±17.07	
P value	0.000**	0.705*	

* No significant difference
* * Significant difference

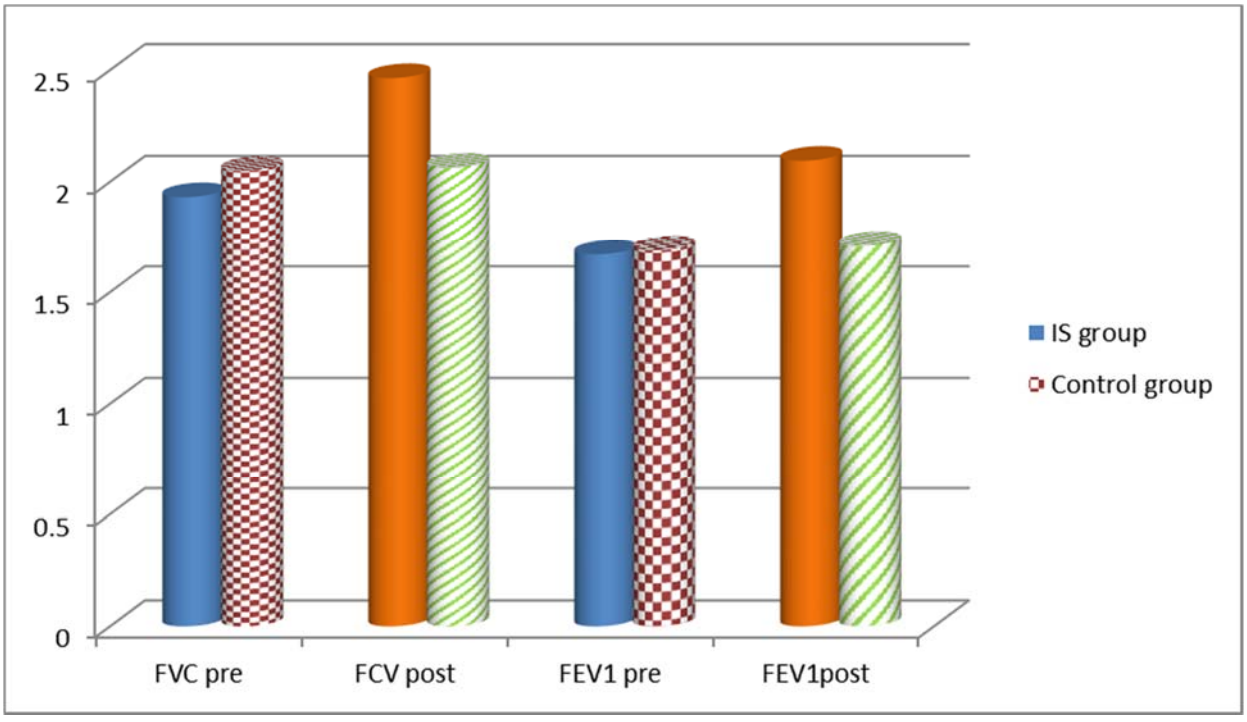

Fig 3: Statistical differences of FCV, FEV1 pre and post treatment in both groups

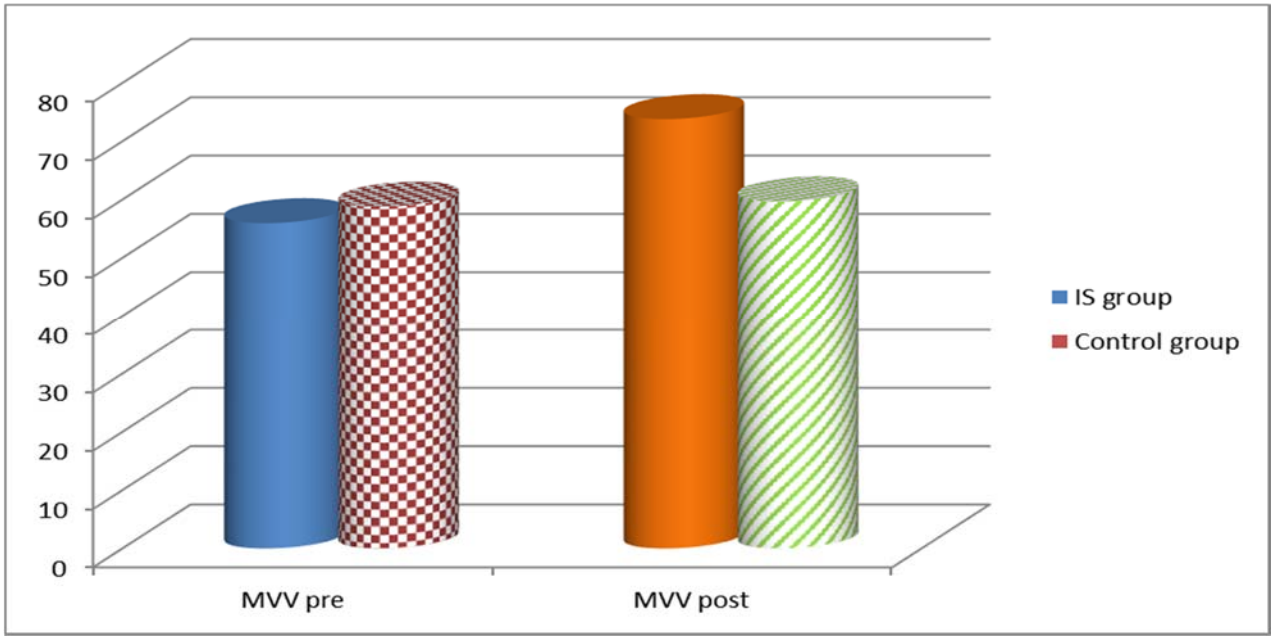

Fig 4: Statistical differences of MVV pre and post treatment in both groups

Discussion

Pulmonary complications are common and account for a large proportion of deaths among adults with sickle cell disease. They result from the complex pathophysiology of sickle cell disease. New therapies are being developed that may prove to be beneficial in the various complications. The purpose of this study was to evaluate the efficacy of incentive spirometry in the treatment of pulmonary problems developed in patient with SCA.

The incentive spirometer has been used successfully for many years to prevent pulmonary atelectasis and its complications in postoperative patients [23,24]. The results of this prospective, randomized trial demonstrated that use of the incentive spirometer significantly increase pulmonary functions in patients with sickle cell diseases, the effect of incentive spirometry was also statistically significant, as the results of the study showed that there were significant differences between incentive spirometry and control group post treatment as regards to Forced Vital Capacity, Forced Expiratory Volume at one second and Maximum Voluntary Ventilation.

Incentive spirometry presumably counteracts the effect of splinting in patients with sickle cell diseases who are unable to take deep breaths because of chest pain and helps prevent the development of atelectasis or infiltrates. The characteristic recurrent pain and organ damage of sickle cell diseases are thought to be due to vaso-occlusion resulting from decreased deformability of sickle cells and their adherence to vascular endothelium[25,26] and to each other[27].

The results of previous studies were support our study results; Bellet and associates [28] reported that the prevention of pulmonary complications by the use of incentive spirometry was clinically beneficial and decreased the average cost of hospitalization from $6,775 to $3,582 by decreasing the length of hospital stay. The mean (±SD) length of hospital stay in the 9 hospitalizations during which pulmonary complications occurred was 6.4±1.9 days, as compared with 3.6±2.1 days in the 29 hospitalizations during which no pulmonary complications occurred (P = 0.001). These pulmonary complications were prevented by the use of incentive spirometry.

Paul *et al* [30] suggested that in many cases thoracic bone infarction with subsequent atelectasis or development of an infiltrate due to chest splinting is the primary pathogenesis of the acute chest syndrome. They found that incentive spirometry can prevent the pulmonary complications (atelectasis or infiltrates) associated with the acute chest syndrome in patients with sickle cell diseases who are hospitalized with chest or back pain above the diaphragm. Also Ahmad *et al*; [29] stated that mandatory incentive spirometry for sickle cell disease patients admitted without respiratory complaints reduces transfusions and ACS, particularly for those presenting with back pain.

Platt *et al*; [31] determined that the acute chest syndrome was a significant risk factor for early death in patients with sickle cell anemia who were 20 years of age or older. It is therefore reasonable to hypothesize that the prevention by incentive spirometry of pulmonary complications (atelectasis or infiltrates) associated with the acute chest syndrome might prevent chronic lung disease and early death.

Saminu and Adeniyiv,[32] reported that the main finding of their study was that teenagers with sickle cell anemia (SCA) had lower peak expiratory flow rate (PEFR) values compared to their age, sex and height matched apparently healthy, non-SCA individuals. However this improved significantly after a six-week period of daily breathing exercises using a local form of incentive spirometry. The improvement, which began to manifest after three weeks of daily use of the apparatus, continued through to the sixth week. However their study has limitation due to small sample size that lead to low sufficient statistical power to detect treatment effect.

From the results of this study and the results of previous studies, It was concluded that incentive spirometry was effective in increasing pulmonary functions so decreasing pulmonary problems in patients with sickle cell anemia.

References

1. Stinson J, Naser B. Pain management in children with sickle cell disease. Paediatr Drugs. 2003;5(4):229-41.
2. El-Hazmi Mohsen. Clinical and hematological diversity of sickle cell disease in Saudi children. Journal of Trop Pediatr. 1992;38:106–112.

3. Panepinto Julie, Pajewski Nicholas, Foerster Lisa, Sabnis Svapna, Hoffmann Raymond. Impact of family income and sickle cell disease on the health-related quality of life. Qual Life Res. 2009;18:5–13.
4. Platt OS, Brambilla DJ, Rosse WF, *et al*. Mortality in sickle cell disease: life expectancy and risk factors for early death. N Engl J Med. 1994;330:1639–1644.
5. Gómez-Chiari M, Tusell Puigbert J, Ortega Aramburu J. [Sickle cell anemia: experience in a center]. An Pediatr (Barc). 2003 Feb;58(2):95-9.
6. Sylvester KP, Patey RA, Milligan P, *et al*. Pulmonary function abnormalities in children with sickle cell disease. Thorax. 2004;59:67–70.
7. S Ahmed, S., Siddiqui, A.K. Pulmonary manifestations of sickle cell disease Postgrad Med J 2003;79:384-390
8. Powars D, Weidman JA, Odom-Maryon T, *et al*. Sickle cell chronic lung disease: prior morbidity and risk of pulmonary failure. Medicine, 1988;67:66–76.
9. Klings ES, Wyszynski DF, Nolan VG, Steinberg MH. Abnormal pulmonary function in adults with sickle cell anemia. Am J Respir Crit Care Med. 2006;173:1264–1269.
10. Mak V, Davies SC. 2003. The pulmonary physician in critical care illustrative case 6: Acute chest syndrome of sickle cell anemia. Thorax 58:726-8.
11. Sibinga EM, Shindell DL, Casella JF, Duggan AK, Wilson MH. Pediatric patients with sickle cell disease: use of complementary and alternative therapies. J Altern Complement Med. 2006;12:291–298.
12. Stuart MJ, Nagel RL. 2004. Sickle-cell disease. Lancet 364:1343-60.
13. Rees DC, Olujohungde AD, Parker NE *et al*. Guidelines for the Management of the Acute Painful Crisis in Sickle Cell Disease. British J of Haematology. 2003; 120: 744-752
14. Stewart M. Sickle Cell Disorder and Physiotherapy. Physiotherapy. 1997; 83: 333-339.
15. Tinti G, Somera R, Valente FM, Domingo CR. Benefits of kinesiotherapy and aquatic rehabilitation on sickle cell anemia: a case report. Genetics and Molecular Research. 2010; 9: 360-364.
16. Perez, LL (March-April 2013). "Office spirometry". Osteopathic Family Physician 5 (2): 65-69.
17. Stanojevic S, Wade A, Stocks J, *et al*. (February 2008). "Reference Ranges for Spirometry Across All Ages: A New Approach". Am. J. Respir. Crit. Care Med. 177 (3): 253–60.
18. Wilkins RL, Krider SJ, and Sheldon RL, (2000): “Clinical Assessment in Respiratory Care”, Mosby, 4th Ed, Ch (7), pp141-160.
19. Ruppel G (1994). Lung volume tests. In: Ruppel G (Editor), Manual of Pulmonary Function Testing. 6th edn. Mosby, St. Louis, 1-25.
20. American Thoracic Society (1991). Lung function testing. Selection of reference values and interpretative strategies. American Review of Respiratory Diseases, 144: 1202-1218.
21. Neder JA, Andreoni S, Castelo-Filho A & Nery LE (1999). Reference values for lung function tests. I. Static volumes. Brazilian Journal of Medical and Biological Research, 32: 703-717.
22. Scherer TA, Spengler CM, Owassapian D, Imhof E and Boulellier U, (2000):“Respiratory Muscle Endurance Training in Chronic Obstructive Pulmonary Disease”, Am J Respir Crit, Care Med, 162(5): 1709-1714.
23. Craven JL, Evans GA, Davenport PJ, Williams RHP. The evaluation of the incentive spirometer in the management of postoperative pulmonary complications. Br J Surg 1974;61:793-797
24. Krastins IRB, Corey ML, McLeod A, Edmonds J, Levison H, Moes F. An evaluation of incentive spirometry in the management of pulmonary complications after cardiac surgery in a pediatric population. Crit Care Med 1982;10:525-528.
25. Hebbel RP, Boogaerts MAB, Eaton JW, Steinberg MH. Erythrocyte adherence to endothelium in sickle-cell anemia: a possible determinant of disease severity. N Engl J Med1980;302:992-995.
26. Fabry ME, Rajanayagam V, Fine E, *et al*. Modeling sickle cell vasoocclusion in the rat leg: quantification of trapped sickle cells and correlation with 31P metabolic and 1H magnetic resonance imaging changes. Proc Natl Acad Sci U S A 1989;86:3808-3812
27. Morris CL, Rucknagel DL, Joiner CH. Deoxygenation-induced changes in sickle cell-sickle cell adhesion. Blood 1993;81:3138-3145
28- Bellet PS, Kalinyak KA, Rucknagel DL. Incentive Spirometry in Sickle Cell Crisis, N Engl J Med 1996; 334:124-127.
28. Ahmad FA, Macias CG, Allen JY; The use of incentive spirometry in pediatric patients with sickle cell disease to reduce the incidence of acute chest syndrome**.** J Pediatr Hematol Oncol. 2011 Aug;33(6):415-20.
29. Paul S. Bellet,., Karen A. Kalinyak,., Rakesh Shukla., Michael J. Gelfand, M.D., and Donald L. Rucknagel, M.D., Incentive Spirometry to Prevent Acute Pulmonary Complications in Sickle Cell Diseases N Engl J Med 1995; 333:699-703
30. Platt OS, Brambilla DJ, Rosse WF, *et al*. Mortality in sickle cell disease -- life expectancy and risk factors for early death. N Engl J Med 1994;330:1639-1644
31. Saminu KS, Adeniyiv AF; Local incentive spirometry improves peak expiratory flow rate in teenage sickle cell anaemia patients: a randomized pilot trial, Afr Health Sci. 2011 September; 11(3): 303–308.