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Pulmonary Dysfunction in Residents of Chronic Care Facilities

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The study was designed to determine the extent of significant pulmonary dysfunction in selected residents of chronic care facilities, to find out any relationhip between pulmonary dysfunction and general disability, and explore pulmonary treatment options.

Thirty-six nursing facility residents over 80 year old, or with a diagnosis of chronic obstructive pulmonary disease or generalized neurological or neuromuscular disease, were surveyed for extent of disability and pulmonary dysfunction. By comparison with age, height, and sex norms, there were significant restrictive pulmonary syndrome, poor inspiratory muscle strength and endurance and inadequate airway secretion clearance mechanisms in all of the residents studied. In addition, at least eight residents manifested chronic alveolar hypoventilation. Four residents with severe restrictive pulmonary syndromes were misdiagnosed as having chronic obstructive pulmonary disease. The extent of pulmonaary dysfunction significantly correlated with the extent of general disability.

We conclude that chronic alveolar hypoventilation and respiratory muscle insufficiency appear to be under diagnosed and under treated in residents of chronic care facilities. The failure to institute appropriate nonivasive interventions may lead to increased pulmonary morbidity in this patient population.

Key words: geriatric medicine, pulmonary function, respiratory insufficiency, pulomonary rehabilitation.

INTRODUCTION

The vital capacity (VC) plateaus at 19 years of age and decreases by about 30 ml per year (1.0% in males, 1.2% in females) thereafter. The maximum voluntary ventilation (MVV) decreases by 0.8% per year after age 30. The PaO2 is equal to 109-0.43 times the age in years.[1] With concurrent pulmonary disease, the rate of loss of pulmonary volumes and function increases. For example, the rate of loss of forced vital capacity may double in patients with chronic obstructive pulmonary disease (COPD),[2] be about 1.8% per year for many patients with postpoliomyelitis,[3] 250-300 ml per year through adolescence for patients with Duchenne muscular dystrophy,[4,5] and can lead to ex-

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treme pulmonary restriction for post-spinal cord injured patients[6] and other aging patients with relatively benign neuromuscular conditions. These and other conditions which lead to dependence in activities of daily living, decreased activity, and long-term residence in chronic care facilities almost invariably have consequences on pulmonary function.[7] Decreased pulmonary function leads to the development of chronic alveolar hypoventilation (CAH) which is often most severe during sleep.

The elderly also have a higher incidence of obstructive sleep apnea syndrome (OSAS) which may be associated with CAH.[8] Residents of nursing facilities often receive medications which can reduce the ventilatory response to hypercapnia and exacerbate CAH. The presence of borderline pulmonary function can lead to pulmonary symptoms, complications and overt cardiopulmonary failure during acute medical illnesses, episodes of stress, or excessive fatigue. Despite this, the pulmonary function of nursing facility residents is not routinely monitored.

PATIENTS AND METHODS

Primary care physicians of a chronic care facility were directed to refer cooperative residents over 80 years of age, residents with significant neuromuscular disability or with a diagnosis of COPD for pulmonary function studies. In all, 36 residents who lived at the facility for a measn of 2 years and 9 months (range= 6 months to 15 years) were referred and studied. Twelve residents, five females, seven males, with a mean age of 70 years (range= 24 to 85 years) had a diagnosis of COPD. The remaining 24 residents, 10 females, 14 males, had a mean age of 59 years (range= 30 to 94 years). Nineteen of these residents had diagnoses leading to generalized neuromuscular disability including nine residents with multiple sclerosis, ten with static central nerve system disorder. Five elderly residents had no generalized neurological or neuromuscular disorder. None of the residents complained of pulmonary symptoms at the time of referral.

The severity of the residents' disability was evaluated by the primary care physician with the use of the PULAEA[10] profile (Table 1). The PULSES scale was designed to measure functional levels of self-care and mobility in the physically impaired. The residents then underwent standard pulmonary function studies (Gould CPI-5000, Sensormedics, Anaheim, CA) with sitting position and nasal clip. The pulmonary function tests included the measurement of maximum imspiratory and expiratory pressures (Hunter manometer 360, Hunter Manufacturing Co, Iowa City, Iowa), end-tidal pCO2 (Beckman LB-3, Sensormedics Inc, Anaheim, CA) and SaO2 (Ohmeda BIox #3760, Ohmeda Inc, Louisville, CO) (Table 2). All results were compared to predicted norms for age, height, and sex.[11] Correlation between the extent of disability (PULSES score) and pulmonary dysfunction (measurements of vital capacity, maximum voluntary ventilation, maximum imspiratory and expiratory pressures) was by Spearman rank correlation analy-

Table 1. Percentage of patients with a pulses[a] profile of 3 or greater in each category and total greater than 18

	P[b]	U[c]	L[d]	S[e]	E[f]	S[g]	>18
COPD	92	34	75	15	42	25	47
Multiple Sclerosis	100	88	100	67	88	22	78
Other	67	66	67	46	66	46	60

a--This profile is based on a scale of 0 to 4 with 3 or greater indicating moderate to severe impairment in any particular category and a total score of 18 or greater indicating severe general disability; b--physical condition including diseases of the viscera; c--upper extremities including the shoulder girdles, cervical and upper thoracic spine; d--lower extremities including the pelvis, lower thoracic and lumbosacral spine; e--sensory impairment including speech, vision and hearing; f--excretory (bowel and bladder) function; g--mental and emotional status.

Table 2. Pulmonary function of 36 nursing facility residents

	COPD	MS	OTHER
Patients	12	9	15
VC*	43 (25-80)	23 (12-54)	42 (17-65)
FVC*	37 (12-69)	16 (7-32)	38 (15-69)
FEV_1/FVC	70 (66-80)	71 (64-75)	70 (67-78)
FEV_1/FVC*	84 (49-122)	86 (73-98)	85 (67-99)
ERV	0.3 (0-1.0)	0.1 (0-0.8)	0.2 (0-0.5)
FRC*	105 (66-139)	65 (28-93)	72 (27-115)
RV*	149 (96-199)	116 (14-163)	109 (18-194)
MVV*	19 (3-53)	12 (2-42)	25 (2-58)
VE%*	181 (90-443)	94 (67-110)	142 (59-274)
Vt	0.6 (0.3-1.3)	0.5 (0.4-0.6)	0.5 (0.2-1.0)
MIP	20 (5-62)	14 (7-22)	19 (5-52)
MEP	22 (5-40)	15 (8-18)	20 (5-65)
PEF	1.3 (0.4-3.7)	1 (0.3-1.6)	1.5 (0.6-2.3)
DLCO%*	44 (10-100)	45 (17-83)	73 (22-134)
HYPERCAP	3	2	3

*as a percentage of predicted norms
VC--vital capacity; FVC--forced vital capacity; FEV1--forced expiratory volume in one second; ERV--expiratory reserve volume (liters); FRC--functional residual capacity; RV--reserve volume, MVV--maximum voluntary ventilation; VE%--minute ventilation; Vt--tidal volume (liters); MIP--maximum inspiratory pressure (mm-Hg); MEP--maximum expiratory pressure (mm-Hg); PEF--peak expiratory flow (liters per second, normal is greater than 6 liters per second); DLCO%--diffusion of carbon monoxide; HYPERCAP--number of patients with continuous elevation of endtidal Pco_2 greater than 50mm Hg.

sis.

RESULTS

The percentage of residents with PULSES index equal to or greater than 3 in each category of disability and with a PULSES total score of equal to or greater than 18 is indicated in Table 1. Since the nine residents with multiple sclerosis made up the single largest diagnostic sample in this population, since they were more disabled and had significantly worse pulmonary function than the other 27 residents, their data is presented separately.

The pulmonary function data are presented in Table 2. Spearman rank correlation analysis indicated significant correlation ($p<0.05$) between the total PULSES score and the four pulmonary function variables tested: vital capacity, r= 0.61; maximum voluntary ventilation, r= 0.44; maximum imspiratory pressure, r=0.58; maximum expiratory pressure, r= 0.05.

DISCUSSION

The results demonstrated significant correlation between general disability and restrictive pulmonary dysfunction. All of the patients had significant restrictive pulmonary syndromes with no patients having vital capacities over 80% of predicted normal. There was little obstructive pulmonary disease in the population as a whole. In fact, four of 12 residents with a diagnosis of COPD were found to

have purely restrictive pulmonary syndromes but three residents were found to have significant undiagnosed obstructive airway disease in addition to a restrictive syndrome. All of the patients had poor respiratory muscle endurance as manifested by maximum voluntary ventilation under 58% of normal. All of the patients had significant weakness of both inspiratory and expiratory muscles with none having maximum imspiratory pressures greater than 62% of predicted normal or maximum expiratory pressures greater than 65% of predicted normal. All of the patients had suboptimal or inadequate cough with less than 60% of normal peak cough expiratory flows. In addition, 25% of the patients had undiagnosed chronic ventilatory insufficiency with chronic hypercapnia. The decrease in diffusion capacity can be explained largely on the basis of the decrease in respiratory exchange memgrane due to the extent of the restrictive pulmonary syndrome.[12]

In the elderly and severely debilitated resident population of chronic care facilities, symptoms of respiratory insufficiency may be absent, or subtle and remain unrecognized unless specifically sought. They had so limited activities in daily lives that the borderline respiratory function could handle them well even in sever reduction of functional reserve capacity. As a result the lack of recognition or misdiagnosis of pulmonary disability may be common. This is true despite the fact that residents with diagnoses not infrequently seen in chronic care facilities such as traumatic quadriplegia, muscular dystrophy, postpoliomyelitis, multiple sclerosis, and Guillain-Barre' syndrome have been recognized to be at risk for the development of late onset ventilatory failure.[3,6,9,13]

In each of the three study groups, the restrictive pulmonary disorder was sufficiently severe to warrant the use of nocturnal noninvasive blood gas monitoring for some patients even without symptom at present.[3,9,15,16] Patients with vital capacities (VC) or forced expiratory volumes in one second (FEV1) less than about 35% of predicted normal, maximum inspiratory (MIP) or expiratory (MEP) pressures less than 30 mm Hg, or symptoms of OSAS or CAH are candidates for daytime and overnight monitoring of oxyhemoglobin saturation.[17] This conveniently screens for the presence of significant hypoxia, CAH, and OSAS. A sawtooth pattern of oxyhemoglobin desaturation during sleep generally indicated the presence of OSAS which can be confirmed by cwpolysomnography.[18] A smooth pattern of oxyhemoglobin desaturation below the normal baseline of 95% is generally an indication of CAH. Chronic hypoxia with pO2 indicated the need for supplemental oxygen therapy.[19] Nocturnal oxyhemoglobin desaturation accompanied by pO2 exceeding 50 mm Hg should not be treated by oxygen therapy unless severe hypoxia persists after normalization of pCO2 by the use of noninvasive methods of assisted ventilation.

A prescription of reconditioning exercise in respiratory muscle is properly to delay detoriating functional reserve capacity in this group of patients. Ventilatory muscle assistance by intermittent positive pressure ventilation (IPPV) delivered via the nose[9,15,16,20] or mouth[3,4,6] is indicated to correct hypoventilation with secondary hypoxia when acute medical illness, episodes of stress, or excessive fatigue. The use of nasal ventilation for inspiratory muscle assistance has recently been described in a large geriatric population.[21] Brief daily periods of mouth IPPV with frequent sighs increase cough efficacy, permit louder speech, and improve dynamic pulmonary compliance.[22] These brief periods also accustom the patient for potentially vital and possibly continuous mouth or nasal IPPV which can in some cases eliminate the need for intubation during respiratory tract infections.[23] Patients with supine VCs less than 30% of predicted normal have been found to commonly require ongoing nocturnal ventilatory assistance and those with 12% or less to require ventilatory assistance up to 24 hours a day.[9]

A minimum of five to seven liters per second

of peak cough expiratory flow is needed to achieve effective airway secretion clearance.[24] All patient groups studied had expiratory reserve volumes, maximum expiratory pressures, and peak expiratory flows that were grossly inadequate to generate an optimal cough. This, too, greatly increases the likelihood of an otherwise mild URI or uncomplicated surgical procedure resulting in life-threatening atelectasis, mucus plugging, pneumonia, and acute respiratory failure. In addition to providing adequate inspiratory muscle assistance and periodic lung expansion to decrease atelectasis,[25] chest percussion, postural drainage, and most importantly, manual assisted coughing[26] can become vital. Since a tidal volume of about 85% to 90% of maximum normal inspiratory capacity[27] is required for an effective cough an adequate insufflation may need to be provided by a blower (Zephyr, Lifecare Inc, Lafayette, CO), portable ventilator, intermittent positive pressure breathing (IPPB) or BiPAP (Respironics Co, Monroeville, PA) machine prior to manually assisting cough.[28] A mechanical insufflation-exsufflation device (Emerson Inc., Cambridge, MA) achieves even greater clearance of secretions with little effort by delivering an adjustable deep insufflation via an anesthesia mask followed by a rapid and sustained drop in pressure of approximately 80 cm H2O.[8,24,29] This creates expiratory flows of 6 to 11 liters per second which is adequate for effective expulsion of airway secretions.[4,24,30] Mechanical exsufflation can be crucial for avoiding intubation for airway secretion clearance.

In conclusion, pulmonary restriction correlates significantly with general disability in residents of chronic care facilities. Misdiagnosis of pulmonary dysfunction may be common. COPD may be over diagnosed as an explanation for pulmonary symptomatology while restrictive pulmonary syndrome appears to be both under diagnosed and under treated. This may lead to excessive morbidity particularly during URIs. Noninvasive inspiratory and expiratory aids have been used to enhance respiratory muscle function and prolong the lives of individuals with severe restrictive pulmonary syndromes. Greater awareness of the applications of these methods may benefit debiliteated individuals with restrictive pulmonary syndromes as well.

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REFERENCES

1. Higgins ITT: Epidemiology of bronchitis and emphysema. In: Fishman AP, ed. Pulmonary Diseases and Disorders, 2nd Ed. New York: McGraw-Hill, 1988;p 1237.
2. Burri PH: Development and regeneration of the lung. In: Fidhman AP, ed. Pulmonary Diseases and Disorders, 2nd Ed. New York: McGraw-Hill, 1988;pp 70-90.
3. Bach JR, Alba AS, Bohatiuk G et al:Mouth intermittent positive pressure ventilation in the management of postpolio respiratory insufficiency. Chest 1987;91:859-864.
4. Bach JR, O'Brien J, Krotenberg R et al: Management of end stage respiratory failure in Duchenne muscular dystrophy. Muscle Nerve 1987;10:177-182.
5. Bach J, Alba A, Pilkington LA et al: Long-term rehabilitation in advanced stage of childhood onset, rapidly progressive muscular dystrophy. Arch Phys Med Rehabil 1981;62:328-331.
6. Bach JR: New approaches in the rehabilitation of the traumatic high lvevl quadriplegic. Am J Phys Med Rehabil 1991;70:13-20.
7. Gorini M, Ginanni R, Spinelli A et al: Inspiratory muscle strength and respiratory drive in patients with rheumatoid arthritis. Am Rev Respir Dis 1990;142:289-294.
8. Bach JR: Mechanical exsufflation, noninvasive ventilation and new strategies for pulmonary

rehabilitation and sleep disordered breathing. Bull N Y Acad Med 1992;68:321-340.
9. Bach JR, Alba AS: Management of chronic alveolar hypoventilation by nasal ventilation. Chest 1990;97:52-57.
10. Moskowitz E, McCann CB. Classification of disability in the chronically ill and aging: J Chronic Dis 1957;5:342-346.
11. Needham CD, Rogan MC, McDonald I: Normal standards for lung volumes, intrapulmonary gas-mixing, and maximum breathing capacity. Thorax 1954;9:313-325.
12. Jack W. Pulmonary Function Testing: A Practical Approach. First edition, William & Wilkins, Baltimore, pp116-118.
13. Bach JR, Alba AS, Mosher R et al: Intermittent positive pressure ventilation via nasal access in the management of respiratory insufficiency. Chest 1987;92:168-170.
14. Slonim NB, Hamilton LH: Respiratory Physiology, 5th Ed. Et. Louis: C.V. Mosby, 1987 pp 235-238.
15. Carroll N, Branthwaite MA: Control of nocturnal hypoventilation by nasal intermittent positive pressure ventilation. Thorax 1988;43:349-353.
16. Leger P, Jennequin J, Gerard M et al: Home positive pressure ventilation via nasal mask for patients with neuromuscular weakness or restrictive lung or chest-wall disease. Respir Care 1989;34:73-79.
17. Welch JP, DeCesare R, Hess D. Pulse oximetry: Instrumentation and clinical applications. Resp Care 1990;35:584-597.
18. Bach JR, Penek J. Obstructive sleep apnea complicating negative pressure ventilatory support in patients with chronic paralytic/restrictive ventilatory dysfunction: Chest 1991; 99:1386-1393.
19. Anthonisen NR: Home oxygen therapy in chronic obstructive pulmonary disease. In: Make B, ed. Clinics in Chest Medicine: Pulmonary Rehabilitation. Philadelphia: W.B. Saunders, 1986,pp673-678.
20. Ellis ER, Bye PTP, Bruderer JW et al: Treatment of respiratory failure during sleep in patients with neuromuscular disease. Am Rev Respir Dis 1987;135:148-152.
21. Daniel Benhamou, Christophe Girault, Colette Faure et al: Nasal Mask Ventilation in Acute Respiratory Failure; Experience in Elderly Patients. Chest: 1992;102:912-917.
22. Bergofsky EH. COr pulmonale in the syndrome of alveolar hypoventilation: Progh Cardiovas Dis 1967;9:414-437.
23. Benhamou D, Girault C, Faure C, Portier F, Muir JF. Nasal mask ventilation in acute respiratory failure: experience in elderly patients: Chest 1992;102:912-917.
24. Barach AL, Beck GJ, Smith H: Mechanical production of expiratory flow rates surpassing the capacity of human coughing. Amer J Med Sci 1953;226:241-248.
25. Bach JR, Alba AS: Rehabilitation of the patient with paralytic/restrictive pulmonary syndromes. In: Haas F, Axen K, Pineda H, eds. Pulmonary Therapy Rehabilitation, 2nd Ed. Baltimore: Williams & Wilkins, 1991,pp339-358.
26. Sortor S, McKenzie M. Toward Independence: Assisted Cough (video), BioScience Communications of Dallas, Inc, 1986.
27. Leith DE. Cough. In: Brain JD, Proctor D, Reid L, eds. Lung Biology in Health and Disease: Respiratory Defense Mechanisms, Part 2. New York: Marcel Dekker, 1977,pp545-592.
28. Bach JR: Pulmonary rehabilitation considerations for Duchenne muscular dystrophy: the prolongation of life by respiratory muscle aids. Crit Rev Phys Rehabil Med 1992;3:239-269.
29. The OEM Cof-flator Portable Cough Machine. Shampaine Industries, St. Louis, MO.
30. Bach JR, Smith WH, Michaels J et al: Airway secretion clearance by mechanical exsufflation for post-poliomyelitis ventilator assisted individuals. Arch Phys Med Rehabil (in press).

慢性療養機構住民之肺功能評估

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肺功能異常是導致慢性療養機構住民活動能力降低的原因之一。在慢性療養機構的住民或因老化現象，或因罹患慢性阻塞性肺疾病，或因有神經肌肉病變，常會有肺功能的異常。但因平時活動有限，不易顯現呼吸系統症狀，易被醫護人員忽略，而沒有及早處理。本文即探討這群住民肺功能異常的嚴重度及提出適當的評估及治療方式。

本研是以36名慢性療養機構住民為對象，其中12名曾被診斷為慢性阻塞性肺疾病，19名有神經肌肉病變或中樞神經損傷，5名為大於80歲之老人。所有36名受試都接受肺功能檢查。利用PULSES scale來評估受試自我照顧及活動的能力，以Spearman rank相關分析來檢定受試自我照顧及活動能力障礙和肺功能異常的關係。

結果顯示所有受試在接受肺功能測試時，主觀上都沒有呼吸系統的症狀，但肺功能檢查都表現出明顯的限制性肺疾病證據。（其呼氣容積都小於預測值之80%）。他們自我照顧和活動能力的障礙和限制性肺疾病之嚴重程度成正相關。所有受試的呼吸肌力，呼吸肌耐力及排除分泌物之能力都有明顯下降的現象。這些呼吸功能的不足使得他們即使只是面對一般的呼吸道感染，也無法有效地克服，而易形成肺部的合併症。早期找出這群高危險住民潛在之肺功能異常，給予適當地胸腔復健，或者利用非侵襲性的呼吸器具幫助他們渡過急性呼吸道感染，是減低這群住民肺罹病率及死亡率的不二法門。

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