

POST-POLIO LUNG FUNCTION

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ABSTRACT. Post-polio patients sometimes complain about the occurrence of breathing difficulties decades after the polio infection. We have examined 40 post-polio patients who have had respiratory or non-respiratory poliomyelitis for at least 30 years in an attempt to elucidate whether hypoventilation is common and to what extent certain symptoms and simple lung function tests are related to hypoventilation or incipient hypoventilation. We measured arterial blood gases, vital capacity (VC), maximal expiratory and inspiratory pressures (MEP, MIP) and CO₂ rebreathing response. Symptoms were assessed by a yes/no questionnaire. Six patients required respiratory assistance at the onset of the disease. At present, two require nocturnal assisted ventilation. Two patients showed manifest hypoventilation; one of which required night-time ventilator, whereas the other patient had not required ventilator assistance even at the onset of the disease. Significant correlation ($p < 0.05$) was found between arterial carbon dioxide tension (a-PCO₂) and VC, MEP and ventilation increase during CO₂ rebreathing. A significantly higher a-PCO₂ was found among those who required respiratory assistance at the onset of the disease, who admitted headache and who felt the cough ineffective. Low VC and low ventilatory increase during CO₂ rebreathing and the presence of headache explained 45% of the variation in a-PCO₂ in a multiple regression analysis. We conclude that manifest hypoventilation is rare in this unselected material of post-polio patients and that a vital capacity below 45-50% of predicted normal and the presence of frequent headaches indicate an increased risk to develop hypoventilation.

Key words: arterial blood gases, hypoventilation, post-polio syndrome, respiratory function.

Late effects from the muscle-skeleton system of post-polio patients have been referred to as the "post-polio syndrome" (6-10). These patients had their infection more than 30 years ago and they experience increased tiredness, muscle weakness and/or pain. Hypoventilation may occur as a consequence of increased weakness of the breathing muscles and could explain why

some patients complain about breathing difficulties. These symptoms may, however, represent physiological changes which are only partly understood (13).

To be able to recognize those who hypoventilate or run an increased risk to develop hypoventilation is important for the care of these patients.

Symptoms like headache, tiredness and dyspnoea have been considered to be associated with the occurrence of hypoventilation (9, 10). These symptoms are unspecific, however, and may be explained by many other causes than hypoventilation. It is therefore unclear how useful these symptoms are in the assessment of hypoventilation in post-polio patients. Simple lung function tests indicating developing hypoventilation would also be clinically useful for the recognition of risk patients.

This study was aimed to show whether hypoventilation is common among post-polio patients and to what extent certain clinical symptoms and/or simple lung function tests are related to hypoventilation or incipient hypoventilation. Abnormally high arterial carbon dioxide tension (a-PCO₂) indicates hypoventilation and we have assumed that a-PCO₂ within the normal range (12) is related to the risk to develop hypoventilation. For example, a patient with a-PCO₂ close to the upper normal limit, is assumed to run an increased risk compared to someone with a lower a-PCO₂. Thus, symptoms and lung function measurements have been related to a-PCO₂.

MATERIAL

We aimed at including all post-polio patients in the age of 40-66 years, living in the region of Göteborg (approximately 450 000 inhabitants). From the Association for Traffic and Polio Injured Patients we got the names of 46 patients. Fourteen dropped out (seven patients did not contact after repeated requests and seven did not want to participate). Thus, 32 of these patients took part in the study. Through the register of the Epidemic Hospital in Göteborg we identified 54 eligible patients, who had been taken care of at the hospital in the years 1950-1954. Twenty of these patients were asked to

Table I. *Working capacity and need for devices*

Sex		Working capacity		Not working	Wheel chair		Walking devices	No walking devices	Breathing devices
		1/1	1/2		Electric	Manual			
F	M	1/1	1/2						
24	16	17	8	15	3	8	19	11	2

participate (the others could not be found in data register, had died or were living outside the region of Göteborg). Nine patients did not answer the offer, while four patients did not want to participate. Thus, seven patients from the register of the Epidemic Hospital took part in the study. Two additional patients were found and included.

One patient was found to suffer from rheumatic arthralgia and was excluded. Accordingly the present study is based on 40 patients.

Mean age was 56 years (range 42–66 years). The mean duration of illness was 40 years (range 30–62 years).

METHODS

Routine physical examination was performed. Scoliosis, if present, was graded in small, medium and severe. A questionnaire with most answers in yes/no form was constructed and sent out together with the invitation to participate in the study. The questions concerned the occurrence of infections, dyspnoea, tiredness, chest pain, headache, symptoms of gastritis, change in appetite, and smoking habits. An attempt to rank dyspnoea was made. Slight to moderate dyspnoea was defined as dyspnoea walking or driving wheel chair on plain level or on a slight slope. Medium to severe dyspnoea was defined as dyspnoea at primary ADL (dress- and undressing, washing) and at rest.

Arterial oxygen tension (a-PO₂), arterial carbon dioxide tension (a-PCO₂) and standard bicarbonate (SBC) were measured in samples of blood obtained from the radial artery. Hypoventilation was defined as a-PCO₂ > 6 kPa. Lung function examination was done with the patients in sitting position. Three acceptable recordings of vital capacity (VC) and forced expiratory volume in one second (FEV₁) were obtained and the maximum value of each variable was used in the further analysis. Normal values were predicted according to Berglund et al. (2). Maximal inspiratory pressure (MIP) reflecting the strength of the diaphragm and maximal expiratory pressure (MEP) reflecting the strength of the abdominal muscles were obtained at functional residual capacity, according to the technique previously described (3). Normal values were predicted, according to Decramer et al. (3) and Lurie et al. (11).

The CO₂ rebreathing technique, according to Read (14), was used as a possible way to test the ability to increase ventilation. End-tidal PCO₂ and ventilation in l/min were registered in two runs with a few minutes' interval. The slope of the curve relating PCO₂ to ventilation ($\Delta V_e/\Delta PCO_2$) was

calculated after exclusion of the initial curved part and expressed in l/min \times kPa.

In addition, eleven of the patients with the lowest VC's were examined by nocturnal transcutaneous oxymetry and an extended questionnaire. These patients spent one night at a sleeping laboratory. Transcutaneous oxygen saturation (Biox 3700) was monitored from about 11 p.m. to about 7 a.m.

RESULTS

Six patients required respiratory assistance at the onset of the disease. Eleven patients were smokers, 17 ex-smokers and 12 non-smokers. Four patients showed slight scoliosis, eight moderate, and ten severe scoliosis. Table I presents working capacity and means of assistance. One patient had a Cuirass ventilator and another had a Dahlbom respirator.

Symptoms

Cooperation in answering the questionnaire was 100%. Fig. 1 illustrates the prevalence of various symptoms. Headache was reported by 14 patients, most of whom (11 patients) now and then used analgetics. Abnormal tiredness was admitted by 12 patients. Ankle swelling had been noted by 20 patients. None complained of bad appetite. Gastritic symptoms were admitted by 17 patients and 13 of them used antacids. Chest pain at effort was admitted by eight persons. One of these used β -adrenergic blockers but none of them used nitroglycerine. Mild dyspnoea was found in 21 patients and moderate to severe dyspnoea in eight. Eleven patients did not complain of dyspnoea. Feeling of adequate cough function was reported by most patients but ten reported the sensation of inadequate cough.

In the extended questionnaire answered by the eleven patients who had the lowest VC's and were examined during sleep, the following was noted: night mares 1/11, experienced memory deterioration 1/11, day time drowsiness 1/11 and the feeling of thoroughly rested in the morning 9/11.

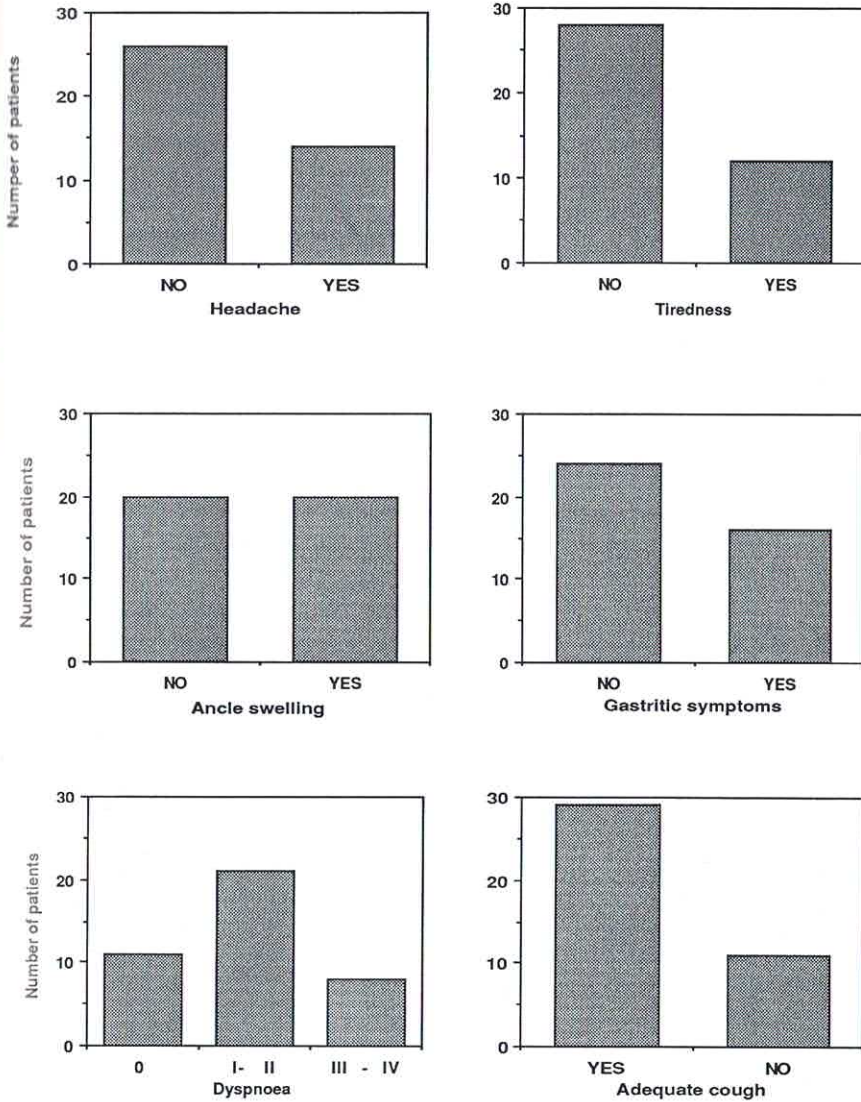


Fig. 1. Prevalence of various symptoms among 40 post-polio patients according to questionnaire results.

Lung function

Two patients showed increased a-PCO₂, one patient required night-time ventilator, whereas the other patient had not required ventilatory assistance even at the onset of the disease. Mean a-PCO₂ in the group as a whole was 5.1 kPa. Spirometric and respiratory muscle strength results are illustrated in Fig. 2. VC was less than 50% of predicted normal value in five of the patients and was within the predicted normal range in 30 patients. All patients had a normal FEV₁.

As a rule, MIP was within the normal range as only

four patients showed reduced pressures. Regarding MEP, twelve patients (30%) showed values below the predicted normal range.

In an attempt to find factors predicting the risk to develop hypoventilation correlations against a-PCO₂ were calculated. Significant correlation ($p < 0.05$) was found to VC, MEP and ventilation increase during CO₂ rebreathing (Fig. 3). MIP, however, was not significantly correlated to a-PCO₂.

Among those who had admitted headache and the feeling of ineffective cough, a significantly higher a-

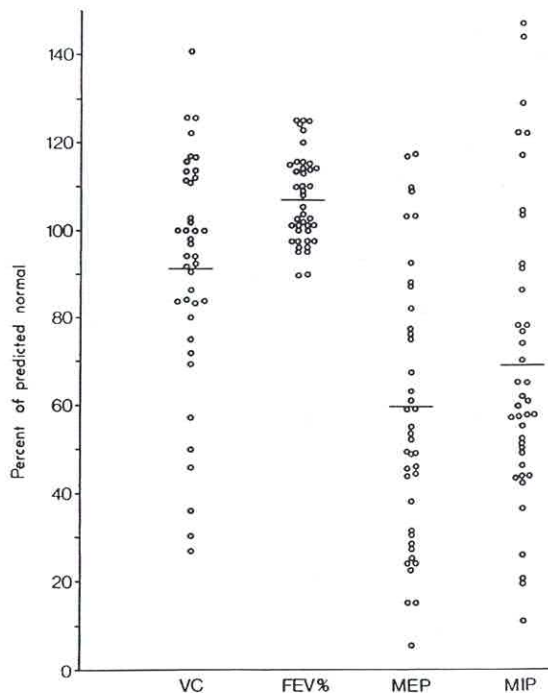


Fig. 2. Individual lung function results among 40 post-polio patients showing VC <50% predicted in five patients and normal FEV% in all. Twelve patients showed maximal expiratory pressure below predicted normal range and four patients reduced maximum inspiratory pressure. The horizontal lines give mean values.

PCO₂ was found (Fig. 4), whereas there were no such differences regarding the other symptoms. Furthermore, those patients who required respiratory assistance at the onset of disease, showed significantly higher a-PCO₂ than the others.

Low VC and respiratory increase during CO₂ re-breathing and the presence of headache explained 45% of the variation in a-PCO₂ in a multiple regression analysis.

Nocturnal oxymetry showed no signs of obvious hypoventilation as none of the patients showed arterial oxygen saturation (a-SO₂) <90% for more than 3% of the sleeping time.

DISCUSSION

Patient selection

The population of post-polio patients in the Göteborg area is unknown. In a previous survey in Sweden (Malmö/Lund) the prevalence is estimated to 70/100 000 (16). In the present study, we have found

15/100 000 individuals but we do not know the number of eligible patients that we were unable to trace. Furthermore, there was a substantial drop-out from those eligible patients who were traced (25/68). Thus, the representativity of the present material is unknown. It seems likely, however, that patients with severe hypoventilation would have been known to us and that those with minor disease tend not to participate. In our opinion therefore, the present material is probably weighted towards over-representation of those patients with the more severe involvement. Anyhow, those who were examined demonstrated large variations in distribution of paresis/paralysis. Also working capacity, as well as need for aid, varied considerably in the group.

Symptoms

Answers about symptoms like headache and tiredness are difficult to interpret, since these symptoms are non-specific. For example, the feeling of tiredness might be influenced by several factors, one of which is mental well-being. Headache can be influenced by anxiety as anxiety can give increased muscle tone and eventually develop tension type headache. Thus, when interpreting these symptoms the patients total situation and signs of mental insufficiency should be considered. This was not done in the present study.

Dyspnoea is a symptom also hard to interpret. The degree of effort producing dyspnoea was hard to estimate due to variations in the degree of disability. Dyspnoea, however, turned out to correlate to an objective variable, i.e. VC. In the group complaining of headache more patients with high level of a-PCO₂ were found ($p=0.02$). Among members of Association for Traffic and Polio Disabled, from where a majority of patients were recruited, discussion about the post-polio syndrome and the risk of hypoventilation had been active and strong consciousness was noted. It is possible therefore that worry-agony might have strengthened some of the unspecific components in the syndrome. Furthermore, among those eleven patients who had the lowest VC's a low frequency of symptoms was noted.

Lung function

In a previous study from Sweden (1) in an area of 750 000 inhabitants a questionnaire was answered by 267 post-polio patients. Eighty-four of these polio patients, who were supposed to be most handicapped, were offered a control of their lung function. Twenty-nine patients were examined, supposedly represent-

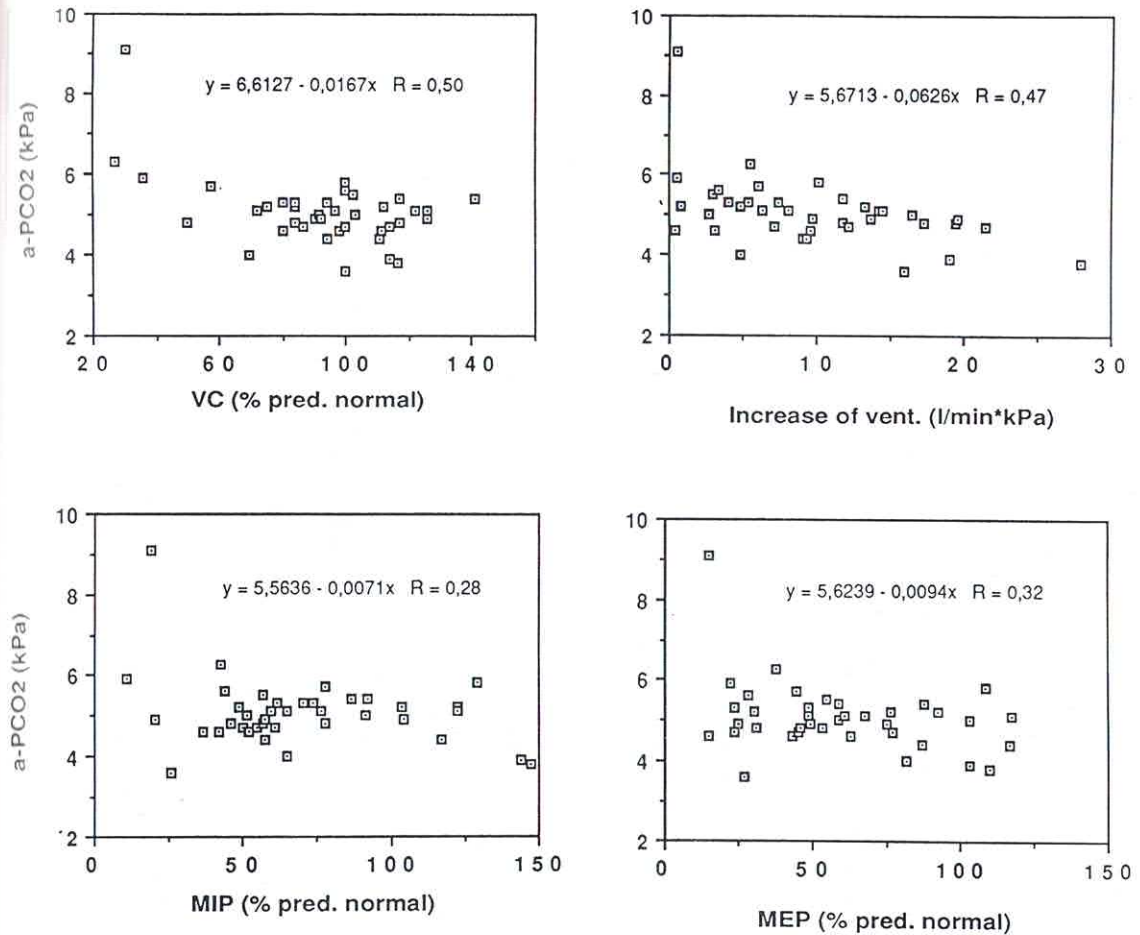


Fig. 3. Relationships between a-PCO₂ and lung function variables among 40 post-polio patients. Significant correlation

($p < 0.05$) was found to VC, MEP and ventilation increase during CO₂ rebreathing.

ing the most seriously handicapped, and four patients were identified with hypoventilation. In the present study, only two patients with a-PCO₂ >6 kPa were identified. These findings and the circumstances related to the patient selection speak in favour of an uncommon occurrence of hypoventilation among post-polio patients.

We have assumed that a-PCO₂ within the normal range is related to the risk to develop hypoventilation. This assumption seems plausible at least to the extent that a-PCO₂ in the highest percentiles are more likely to develop hypoventilation, i.e. abnormally high a-PCO₂, than those patients who have a-PCO₂ in the lower percentiles. Besides the general relation to hypoventilation a measurement of a-PCO₂ suffers from variations caused by measuring errors, pain-induced hyperventilation and differences in the sensitivity of

the respiratory center. A single measurement is therefore uncertain as a measure of the risk to develop hypoventilation. In a group of patients with a range of a-PCO₂, we have assumed that the effects of irrelevant factors cancel out and that the remaining variation in a-PCO₂ is related to hypoventilation. The possibility that pain caused by the arterial puncture, systematically reduces a-PCO₂, is unlikely because pH was within normal limits in all but one of the patients and the SBC was not above normal limits in patients with normal a-PCO₂.

Two lung function variables obtained by simple methods, i.e. VC and MEP, were significantly correlated to a-PCO₂. VC is a standardized and reliable method, likely to reveal reduced respiratory muscle function quite in agreement with the present results (4, 15). Besides reduced respiratory muscle function,

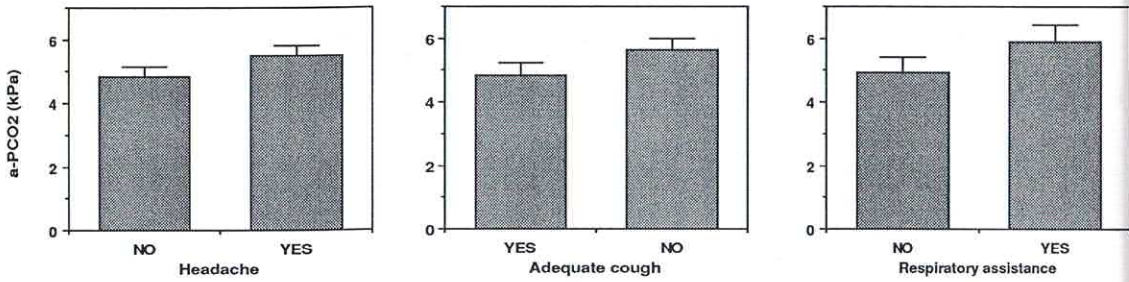


Fig. 4. a-PCO₂ in 40 post-polio patients according to the occurrence of headache, the perception of adequate cough

function and of respiratory assistance at the onset of the disease. The differences are statistically significant ($p < 0.05$).

scoliosis present in some of the patients is likely to have contributed to the reduction of VC (5).

It is important in neuro-muscular diseases to get objective measurement of the strength of the muscles engaged. The test method employed in the present study to assess the respiratory muscles is simple but the normal values show wide variation (9, 10). MIP was not correlated to a-PCO₂ and in the multiple regression analysis MEP did not contribute to explain the variation in a-PCO₂, presumably because of the rather strong correlation to VC ($r = 0.60$). The assessment of the strength of the respiratory muscles by MEP and MIP measurements appears therefore not to be helpful in assessment of the risk to develop hypoventilation. Respiratory muscle endurance might have been more useful but is hard to measure.

We speculated that an inability to increase ventilation might be related to the risk to develop hypoventilation in post-polio patients. We chose to provoke increased ventilation by CO₂ rebreathing rather than by ordinary physical exercise due to the various handicaps. The results showed a significant negative correlation between a-PCO₂ and $\Delta V_e/\Delta PCO_2$ and that $\Delta V_e/\Delta PCO_2$ contributed to explain the variation in a-PCO₂ in the multiple regression analysis. The mechanisms can, however, not be fully explained on basis of the present results. Reduced dynamic force in respiratory muscles combined with reduced endurance capacity is a possible mechanism. Alternatively, a high a-PCO₂ *per se* causes a lowered central drive to increase in ventilation when intracerebral PCO₂ is rising.

In order to reveal possible concealed hypoventilation we added nocturnal oxymetry among those with the lowest VC's. No obvious signs of hypoventilation were found.

In conclusion, hypoventilation seems to be rare

among post-polio patients. In clinical practice, low VC (below 40–50% predicted normal) and the presence of frequent headache should motivate repeated a-PCO₂ determinations and result in an awareness of increased risk to develop hypoventilation. Adequate information to the patient concerning respiratory capacity is of importance and consecutive measurements may be helpful. Continuous assessments of a-PCO₂ during day/night would probably be a useful diagnostic method, especially for patients who are thought to be in the risk zone.

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APPENDIX

Some questions selected from the questionnaire which were answered by the participants

Do you have more than five respiratory tract infections per year?

Coughing needs muscle strength. Have you got enough strength to get an effective coughing?

Are you dyspnoic?

At rest?

At primary ADL?

Walking or driving wheel chair on plain ground?

Walking fast or in a slope?

Do you need to stop to breathe when you walk/drive wheel chair in your own pace on plain ground?

Have you felt augmented tiredness last months/years?

Do you feel tired more than normal in relation to age and life situation?

At what time of the day do you feel tired?

Have you felt chest pain?

During day-time?

During night?

At rest?

At stress?

Do you regularly suffer from headache?

In the morning?

In the afternoon?

Is the headache of such intensity that you must use analgetics?

REFERENCES

1. Andersson, G., Bake, B., Blomstrand, A. et al.: Andningsproblem vid thorakal restriktivitet, ryggmärgsskada och neuromuskulär sjukdom. *Läkartidningen* 85: No 32-33, 2543-2547, 1988.
2. Berglund, E., Birath, G., Bjure, J., Grimby, G., Kjellmer, I., Sandqvist, L. & Söderholm, B.: Spirometric studies in normal subjects. I. Forced expirograms in subjects between 7 and 70 years of age. *Acta Med Scand* 173: 185-206, 1963.
3. Decramer, M., Demedts, M., Rochette, F. & Billiet, L.: Maximal transrespiratory pressures in obstructive lung disease. *Bull Europ Physiopath Resp* 16: 479-490, 1980.
4. De Troyer, A., Borenstein, S. & Cordier, R.: Analysis of lung volume restriction in patients with respiratory muscle weakness. *Thorax* 35: 603-610, 1980.
5. De Troyer, A. & Heilporn, A.: Respiratory mechanics in quadriplegia. *Respiratory function of intercostal muscles*. *Am Rev Respir Dis* 122: 591-600, 1980.
6. Einarsson, G. & Grimby, G.: Strengthening exercise program in post-polio subjects. *In Journal of Research and Clinical Aspects of the late Effects of Poliomyelitis*. Birth Defects: Original series (ed. Halstead & Wichers), 23 (4): 165-274, 1987.
7. Grimby, G., Einarsson, G., Aniansson, A. & Hedberg, M.: Muscle morphology with special reference to muscle

strength in post-polio subjects. *In Journal of Research and Clinical Aspects of the late Effects of Poliomyelitis*. Birth Defects: Original series (ed. Halstead & Wichers), 23 (4): 273-283, 1987.

8. Grimby, G., Einarsson, G., Hedberg, M. & Aniansson, A.: Muscle adaptive changes in post-polio subjects. *Scand J Rehab Med* 12: 19-26, 1989.
9. Hamilton, E. A., Nichols, P. J. R. & Tait, G. B. W.: Late onset of respiratory insufficiency after poliomyelitis. *Ann Physic Med* 5: 223-291, 1970.
10. Lane, D. J., Hazelman, B. & Nichols, P. J. R.: Late onset respiratory failure in patients with previous poliomyelitis. *Quart J Med, New series XLIII*, 172: 551-568, 1974.
11. Lurie, M., Caidahl, K., Johansson, G. & Bake, B.: Respiratory function in chronic primary fibromyalgia. *In press*.
12. Mellemegaard, K.: The alveolar-arterial oxygen difference: its size and components in normal man. *Acta Physiol Scand* 67: 10-20, 1966.
13. Perry, J., Barnes, G. B. & Gronley, J. K.: The post-polio syndrome. An overuse phenomenon. *Clin Orthop* 233: 145-162, 1988.
14. Read, D. J. C.: A clinical method for assessing ventilatory response to carbon dioxide. *Austr Ann Med* 16: 20-32, 1967.
15. Serisier, D. E., Mastaglia, F. L. & Gibson, J.: Respiratory muscle function and ventilatory control I in patients with motor neurone disease. II. In patients with myotonic dystrophy. *Quart J Med, New series LI*, No 202: 205-226, 1982.
16. Tufvesson, A. K.: Polioskadade—finns de? (A social medicine research of polio patients in Malmöhus county.) Lund-Malmö: SVCR Serie. Swedish Central Comitté for Rehabilitation, 1984.

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