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Respiratory function during wakefulness and sleep among survivors of respiratory and non-respiratory poliomyelitis

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Respiratory function during wakefulness and sleep among survivors of respiratory and non-respiratory poliomyelitis. T.E. Dolmage, M.A. Avendano, R.S. Goldstein.
ABSTRACT: The purpose of this study was to determine whether there is a difference in respiratory mechanics and gas exchange between polio survivors and healthy, age-matched controls during wakefulness and sleep.

Polio survivors were divided into four groups. The first group included those who had evidence of respiratory muscle involvement originally (P_{RM}) and the second group included those who had bulbar muscle involvement originally (P_{BM}). The third and fourth groups had only limb involvement originally but were separated by absence (P_{SL}) or presence of a scoliosis (P_{SS}) at the time of their evaluation.

Each subject completed baseline and one year follow-up measurements of lung volumes, diffusion, flow rates, respiratory muscle strength, central and peripheral chemoreflexes and arterial blood gases. Sleep measurements included a full respiratory polysomnographic study.

Fifty polio survivors and 13 controls completed the study. The P_{RM} and P_{SS} groups had an elevated arterial carbon dioxide tension ($Paco_2$) (mean±SE 6.0±0.4 and 6.0±0.3 kPa, respectively), reduced vital capacity (2.8±0.3 and 2.9±0.3*l*, respectively), reduced maximal inspiratory pressure (-5.9±0.7 and -5.4±0.8 kPa, respectively) and reduced maximal expiratory pressure (9.8±1.1 and 9.1±1.2 kPa, respectively), when compared with non-polio controls. During sleep P_{RM} and P_{SS} groups experienced a higher $Paco_2$ (6.5±0.5 and 6.7+0.4 kPa, respectively) and a lower arterial oxygen saturation (Sao_2) (89±4 and 86±3%, respectively). There were no differences among groups for diffusion, flow rates and chemoreflexes. All other polio survivors showed essentially normal respiratory function.

A respiratory evaluation is important in polio survivors if there is a clear history of initial respiratory muscle involvement or the finding of a kyphoscoliosis many years later. This study does not support previous reports suggesting respiratory dysfunction may occur among those who had only limb involvement at the time of their acute polio.
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Several authors have reported that new muscle weakness and fatigue occurs among polio survivors 30 yrs after the acute onset of poliomyelitis [1–3]. The exact cause of these symptoms remains unclear but they probably reflect dysfunction of enlarged motor units formed by axonal branching, a recognized part of the process of neuromuscular recovery from the anterior horn cell destruction [4]. Although new muscle weakness occurs predominantly in muscles affected during the acute illness, weakness of muscles not thought to be clinically involved has also been reported [4, 5]. This apparently new muscle weakness has been explained as being a consequence of asymptomatic infection, in which insufficient motor units were destroyed to manifest as acute weakness, but sufficient units were involved to render the muscle vulnerable and predisposed to the late effects of the disease. This explanation is supported by animal studies in which the polio virus was identified not only in sections of the spinal cord that supplied the weakened limb, but also in sections of the brain and spinal cord that supplied apparently healthy tissue [6]. Further support has come from human autopsy studies in which gliosis, inflammation and neuronal atrophy have been identified in apparently stable polio survivors dying of unrelated causes [7].

Involvement of the respiratory muscles or brain stem (bulbar polio) may impair respiratory mechanics and control of breathing [8–10]. Some polio survivors require ongoing mechanical ventilation [3, 11] whereas others in whom assisted ventilation was initially required, deteriorate after 30 yrs of independent respiration, necessitating the reintroduction of mechanical support [3, 12]. It is possible that a third group of polio survivors exists, in whom late respiratory dysfunction occurs in the absence of clinical involvement

of the respiratory system at the time of the acute illness. This is analogous to the described asymptomatic involvement of limb muscles. If polio affected the respiratory system subclinically resulting in slowly progressive dysfunction, worsening respiratory function may occur in polio survivors 30 yrs or more after recovery, despite the absence of any appreciable clinical respiratory impairment at the time of the acute event. Reports both in the scientific and the lay press that emphasize this possibility may cause considerable anxiety among polio survivors.

To date, the late effects of polio on the respiratory system have been reported only among patients attending clinics for regular respiratory care [9, 12, 13], or among those identified from the polio population as having symptoms and signs of respiratory failure [14, 15]. Measurements of respiratory function during wakefulness and sleep have not included age-matched controls or comparisons among polio survivors with differing clinical involvement at the time of their acute presentation. Furthermore, previous reports have not excluded the effects of age, smoking history and other diseases or trauma to the respiratory or central nervous system. In this study, respiratory function was measured among polio survivors with a history of acute respiratory muscle involvement, those with only limb involvement, those with bulbar involvement and healthy age-matched controls.

Methods

A request for healthy volunteers and for polio survivors was made by advertising on a local radio station and to members of an Ontario-based post-polio registry (Ontario March of Dimes). Interested individuals were screened by one of the investigators and, subsequently, all subjects were seen by one of the physicians associated with the study. Those accepted were all nonsmokers, between 30–60 yrs of age, and their weight was between 80–120% predicted. Participants had no known history of other disease or trauma to the ventilatory or central nervous system. Their history of polio was clear, with original hospital records being obtained whenever possible. Measurements were conducted following informed consent. A standard posterior anterior and lateral radiograph was taken to complement the clinical evaluation of scoliosis.

Pulmonary function tests included standard measurements of lung volumes, diffusion, flow rates [16–18] and respiratory muscle strength [19]. Central [20] and peripheral [21] chemoreflexes were measured in a standardized manner during wakefulness using the ventilatory responses to rebreathing and single breath CO_2 respectively. Arterial blood gases were measured with the subject in the supine position whilst breathing room air.

Each subject completed a full respiratory polysomnographic study. Standard sleep variables included an electroencephalogram (EEG), submental electromyogram (EMG), electro-oculogram (EOG) and electrocardiogram (ECG), with sleep stages being scored in 40 s epochs by standard criteria [22]. Respiratory movements were monitored by inductive plethysmography (Respitrace, Ambulatory Monitoring, Ardsley, New York, USA). An apnoea was defined as a tidal volume of <150 ml for more than 10 s. An hypopnoea was defined as a fall to 50% of the tidal volume from the preceding baseline for > 10 s. Arterial oxygen saturation (Sao_2) was estimated with an ear oximeter and arterial carbon dioxide tension ($Paco_2$) was measured transcutaneously. Measurements of Sao_2 were made on an epoch-by-epoch basis. During each epoch the highest and lowest values were recorded and the results were expressed as the mean, the mean high and the mean low values for each sleep stage. Measurements of $Paco_2$ were made on an epoch-by-epoch basis and were expressed as the mean and mean high value for each sleep stage.

The group designation for each subject was made on the following hierarchy. If there was involvement of the respiratory muscles during the acute polio the subject was classified P_{RM}. Respiratory involvement included those requiring assisted ventilation who subsequently became independent of mechanical ventilatory support. If there was no apparent respiratory muscle involvement but bulbar involvement was evident, then the subject was classified as P_{BM}. Bulbar polio was identified from a history of upper airway dysfunction such as hoarseness and stridor or a nasal quality of speech or difficulty swallowing. If the subject did not have respiratory muscle or bulbar involvement, he/she was classified as spinal polio. The spinal group was then subdivided into those with involvement of trunk musculature and subsequent scoliosis (P_{SS}) and those with only limb involvement (P_{SL}). The fifth group was comprised of healthy aged-matched controls.

All subjects were evaluated at baseline and again twelve months later. The study was a two factor design (5×2) with repeated measures on the factor "time". Analysis of variance was performed on each dependent variable, to determine whether there was a significant difference among groups between baseline and at one year of follow-up. If there was no significant interaction between group and time, the main effect of group was analysed. Post hoc analysis using Dunnett's test was used to determine if there was a significant difference between each polio group and the control group [23, 24].

All values are presented as mean and standard error. A probability value of 0.05 was used to determine significant differences for all tests. When required, analysis of covariance was used to compare groups. Means were also adjusted for age, height and gender for measurements of lung volume and age and gender for measurement of respiratory muscle strength.

Results

Fifty polio subjects and 13 age-matched control subjects completed the baseline evaluation (table 1). Subsequently, 40 polio subjects and all control subjects completed the follow-up evaluation at one year. Of

the 10 subjects not included in the follow-up results, two subjects from the P_{RM} group received mechanical ventilation before their follow-up measurements and eight (one P_{BM}, two P_{SL}, two P_{SS}, three P_{RM}) were unwilling to be re-evaluated. One subject from the P_{SS} group received mechanical ventilation subsequent to the completion of the study and another subject from the P_{RM} group will probably receive mechanical ventilation within the next year. Both of these subjects had marked respiratory dysfunction at baseline and follow-up.

Pulmonary function tests

All lung volumes were adjusted for age, gender and height with analysis of covariance. There was no significant interaction between the independent variables group and time for the dependent variables, vital capacity (VC), functional residual capacity (FRC), total lung capacity (TLC) and residual volume (RV). There was a significant difference in VC and TLC among the groups. As shown in table 2, Post hoc analysis showed that the VC of the P_{SS} group (adjusted mean 2.9±0.3 *l*) and P_{RM} group (adjusted mean 2.8±0.3 *l*) were significantly less than the control group (adjusted mean 4.3±0.3 *l*). The TLC of the P_{RM} group (adjusted mean 4.5±0.4 *l*) was significantly less than the control group (adjusted mean 6.0±0.4*l*). The TLC of the P_{SS} group (adjusted mean 4.9±0.4*l*) showed a trend to be less than the control group (p=0.06). The range of TLC of the P_{SS} group was 32–126% predicted whereas the TLC of the control group was 85–140% predicted. There were no significant differences among groups in diffusing capacity or flow rates (fraction of forced vital capacity expired in one second (FEV_1/FVC), maximum forced expiratory flow at 50% and 25% FVC ($\dot{V}max_{50}$, $\dot{V}max_{25}$)).

Respiratory muscle strength

Analysis of variance of the dependent variables, maximal inspiratory pressure from residual volume (MIP_{RV}) and from functional residual capacity (MIP_{FRC}), maximal expiratory pressure from TLC (MEP_{TLC}), as well as maximal voluntary ventilation, showed no significant interaction effect of the independent variables, group and time. There were significant differences among the groups for the dependent variables, MIP_{FRC}, MIP_{RV} and MEP_{TLC}. Table 2 shows adjusted means for MIP_{RV} and MEP_{TLC}. Post hoc analyses showed that the MIP_{FRC}, MIP_{RV} and

Table 1. – Mean and standard error of age, height and weight for each group

Group	n	Sex M/F	Age yrs	Height cm	Weight kg	Polio age* yrs
Respiratory Muscle (P_{RM})	12	4/8	48±2	165±3	62±2	39±1
Bulbar Muscle (P_{BM})	16	7/9	52±2	165±2	69±3	38±1
Spinal Scoliotic (P_{SS})	9	4/5	48±3	162±4	62±5	41±3
Spinal Limb (P_{SL})	13	1/12	47±2	164±2	68±4	42±2
Control	13	7/6	49±3	174±3	72±3	–

*: polio age equals the number of years since acute polio event.

Table 2. – Lung volumes and respiratory muscle strength in polio survivors

	Control	Spinal Limb	Bulbar Muscle	Spinal Scoliotic	Respiratory Muscle
VC *l*	4.7±0.3 (3.5–7.3)	3.8±0.2 (3.0–5.2)	3.9±0.3 (2.6–5.9)	2.7±0.5 (1.0–6.2)	2.7±0.4 (0.9–5.4)
Adjusted†	4.3±0.3	4.1±0.3	4.0±0.2	2.9±0.3*	2.8±0.3*
TLC *l*	6.5±0.4 (5.0–9.8)	5.4±0.3 (3.6–8.4)	5.6±0.4 (3.9–8.2)	4.8±0.7 (2.2–8.5)	4.4±0.4 (2.1–6.5)
Adjusted†	6.0±0.4	6.0±0.4	5.7±0.3	4.9±0.4	4.5±0.4*
MIP_{RV} kPa	-8.1±0.9 (-3.0– -12.8)	-7.5±0.7 (-4.2– -11.0)	-6.8±0.5 (-3.4– -9.8)	-5.5±1.0 (-1.4– -8.8)	-5.6±0.8 (-2.0– -11.8)
Adjusted††	-8.1±0.7	-8.1±0.8	-7.0±0.6	-5.4±0.8*	-5.9±0.7*
MEP_{TLC} kPa	14.8±1.3 (9.2–22.8)	11.4±0.7 (8.3–16.0)	10.8±1.1 (4.1–21.8)	9.5±1.7 (3.0–16.9)	9.0±1.6 (2.4–21.9)
Adjusted††	14.6±1.0	13.3±1.1	11.3±0.9*	9.1±1.2*	9.8±1.1*

Data are presented as mean±SE, with range in parenthesis. VC: vital capacity; TLC: total lung capacity; MIP_{RV}: maximal inspiratory pressure from residual volume; MEP_{TLC}: maximal expiratory pressure from TLC; *: significantly different from control group (p<0.05); †: adjusted for age, gender and height; ††: adjusted for age and gender.

MEP_{TLC}, adjusted for age and gender, of the P_{SS} and P_{RM} groups were significantly less than the control group. The MEP_{TLC} of P_{BM} group was significantly less than the MEP_{TLC} of the control group.

Arterial blood gases

Analysis of variance, of the dependent variables arterial carbon dioxide tension ($Paco_2$), arterial oxygen tension (Pao_2), pH and arterial oxygen saturation (Sao_2), showed no significant interaction of the independent variables, group and time. There was a significant difference among groups for the dependent variable, $Paco_2$. Post hoc analysis showed that $Paco_2$ of the P_{RM} group (6.0±0.4; range 4.8–8.1 kPa) was significantly greater than the $Paco_2$ of the control group (5.3±0.1; range 4.8–5.9 kPa). There was a trend for the P_{SS} group (6.0±0.3 kPa) to have a higher $Paco_2$ than the control group but the means were not significantly different. The range of $Paco_2$ for the P_{SS} group was 4.9–7.6 kPa, compared to the range of the control group of 4.8–5.9 kPa. There were no significant differences among groups or across time for the dependent variables, Pao_2, pH and Sao_2.

Carbon dioxide response

Analysis of variance of the central chemoreflex showed that there was no significant interaction between the independent variables, group and time. There was a significant difference for the central chemoreflex among groups. Post hoc analysis showed no significant difference between the polio groups and the control group. The P_{BM} and P_{RM} had a lower central chemoreflex (10.2±1.4 and 9.8±2.3 $l \cdot min^{-1} \cdot kPa^{-1}$, respectively) than the control group (13.1±1.6 $l \cdot min^{-1} \cdot kPa^{-1}$) but mean values were within the normal range. Only the P_{SS} group had a mean value (7.3±1.6 $l \cdot min^{-1} \cdot kPa^{-1}$) near the lower limit of the normal range [20]. Analysis of variance of the peripheral chemoreflex revealed no significant differences among groups. Mean values were within the normal range for all groups [21].

Sleep summary

Quantity and distribution of sleep. Analysis of variance of the main effect group for the dependent variables, total time asleep (TTA) and sleep efficiency, showed a significant difference among groups. Post hoc analysis revealed that the P_{RM} group had less TTA (263.3±19.4 min) than the control group (343.8±14.0 min). The sleep efficiency of the P_{RM} group (67.0±4.4%) was also significantly less than the control group (82.4±2.9%). There were no significant differences among groups in rapid eye movement (REM) latency, movement time or movement arousals.

Nocturnal blood gases. There was no significant interaction of the independent variables, group and time, for the dependent variable, Sao_2 during sleep. However, there was a significant difference among groups in Sao_2 during REM sleep and in mean Sao_2 for the total time asleep (fig. 1). Post hoc analysis showed: 1) the low Sao_2 during REM sleep for the P_{SS} group (81±6%) was significantly less than the control group (91±1%); 2) the mean Sao_2 during REM sleep for the P_{SS} group (86±4%) was significantly less than the control group (94±1%); 3) the overall mean Sao_2 for the total time asleep of the P_{RM} group (90±3%) was significantly less than the control group (94±1%).

There was a significant difference among groups in high and mean $Paco_2$ during wakefulness, stage 1 and REM sleep (fig. 1). There was also a significant difference among groups in mean $Paco_2$ during stage 2 and for the total time asleep. Post hoc analysis showed that the high and mean $Paco_2$ for the P_{SS} group was greater than the control group during wakefulness, Stage 1 and REM sleep. The mean $Paco_2$ of the P_{RM} group was greater than the control group during stage 2 and over the total time asleep.

When nocturnal blood gases were analysed with analysis of covariance and adjusted for daytime values, there was no significant difference among groups in $Paco_2$. However, there was a significant difference among groups in Sao_2 during REM sleep. When Sao_2 was adjusted for daytime Sao_2, Post hoc analysis showed that the P_{SS} group (adjusted mean 89±2%) had a lower Sao_2 during REM sleep when compared to the control group (adjusted mean 93±1%). There was a trend for the P_{RM} group (adjusted mean 89±2%) to have a lower Sao_2 than the control group during REM sleep.

Apnoeas and hypopnoeas. Analysis of variance of the independent variable group, showed a significant difference among groups in the number of apnoeas and the apnoea index during REM sleep. Post hoc analysis showed that during REM sleep: 1) the P_{SS} group (16±8 apnoeas) had significantly more apnoeas than the control group (5±2 apnoeas); 2) the apnoea index of the P_{SS} group (26±9 apnoeas$\cdot h^{-1}$) was significantly greater than the apnoea index of the control group (5±2 apnoeas$\cdot h^{-1}$) as shown in figure 2. There was no significant difference among groups for the number of apnoeas during the total time asleep. The apnoeas were predominately obstructive.

Analysis of variance of the independent variable group, showed a significant difference in the number of hypopnoeas and the hypo-pnoea index during REM sleep. Post hoc analysis showed that during REM sleep the P_{SS} group (11±3 hypopnoeas) had significantly more hypopnoeas than the control group (6±2 hypopnoeas) and, as shown in figure 2, the hypopnoea index of the P_{SS} group (22±7 hypopnoeas$\cdot h^{-1}$) was greater than the hypopnoea index of the control group (6±2 hypopnoeas$\cdot h^{-1}$).

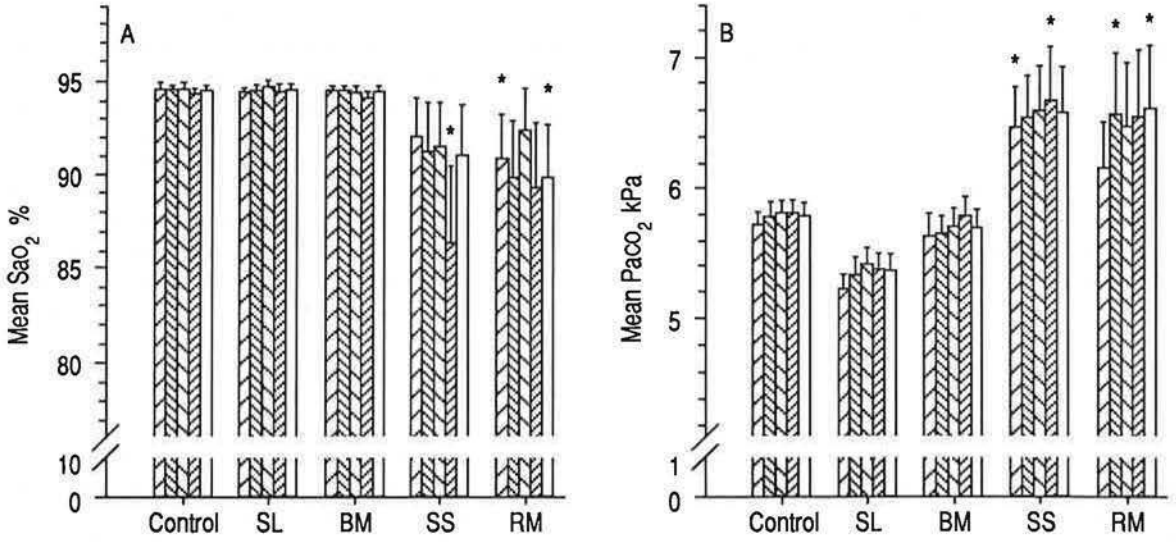

Fig. 1. – A) mean arterial saturation (Sao_2); and B) mean arterial carbon dioxide tension ($Paco_2$) with standard error for the control group, as well as the spinal limb (SL), bulbar muscle (BM), spinal scoliotic (SS) and respiratory muscle (RM) polio groups. ▨: Stage 1 sleep; ▧: Stage 2 sleep; ▨: slow wave sleep; ▨: rapid eye movement sleep; □: total time asleep. *: significantly different from control group ($p<0.05$).

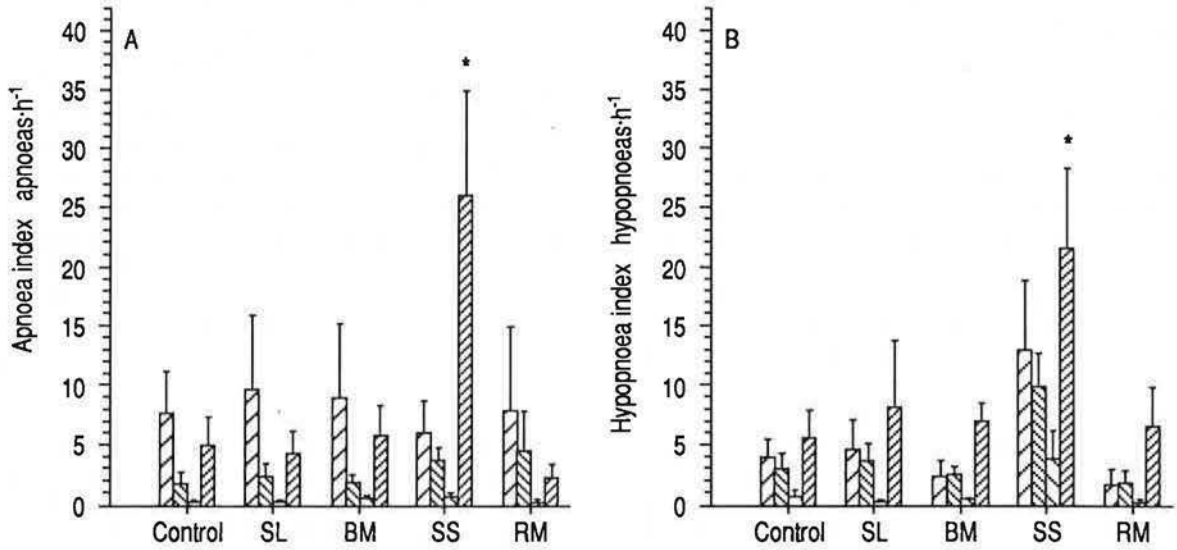

Fig. 2. – A) apnoea index; and B) hypopnoea index with standard error for the control group, as well as the spinal limb (SL), bulbar muscle (BM), spinal scoliotic (SS) and respiratory muscle (RM) polio groups. ▨: Stage 1 sleep; ▧: Stage 2 sleep; ▨: slow wave sleep; ▨: rapid eye movement sleep. *: significantly different from control group ($p<0.05$).

Discussion

It has been well established that 30 yrs after the acute event, many polio survivors experience generalized fatigue and muscle weakness in previously involved muscles. It might, therefore, be expected that survivors of respiratory polio (P_{RM}) would experience measurable respiratory muscle weakness and subsequent respiratory dysfunction 30 yrs after the acute event. In a group of P_{RM} subjects who required ventilatory support during the acute illness but who were subsequently weaned and lived without ventilatory support for many years, we observed respiratory muscle weakness (table 2), reduced lung volumes (table 2) and altered daytime and nocturnal arterial blood gases (fig. 1). Clearly the previous respiratory muscle involvement contributed to the present respiratory status of the P_{RM} group.

The present status of any muscle group will depend upon the degree of residual dysfunction at the time of maximal recovery and upon any subsequent deterioration. Within the two year time frame of this study, three subjects were ventilated. In each case, these subjects were observed to have had clinical evidence of respiratory failure at the time of their recruitment, confirmed by laboratory measurements of gas exchange during wakefulness and sleep. One subject deteriorated clinically and required emergency intubation. The other two were of sufficient severity that nocturnal ventilation was commenced electively. A

fourth subject was observed to have marked respiratory dysfunction at baseline, such that he will probably receive elective mechanical ventilatory support within the next year. It remains unresolved as to whether respiratory dysfunction in these polio survivors represents an accelerated deterioration in some way linked to the previous infection or "natural ageing" (age 30–60 yrs) superimposed upon an abnormal baseline. However, it would appear that those polio survivors with known initial respiratory muscle involvement or with subsequent scoliosis may be in jeopardy.

In the present study, respiratory dysfunction was generally limited to those with a history of acute respiratory impairment or those with subsequent scoliosis. The spinal scoliotic group presumably had normal respiratory function during the acute event but the subsequent development of scoliosis reflected upper trunk involvement with mechanical consequences measurable many years later (table 2), as well as an elevated daytime and nocturnal $Paco_2$ (fig. 1). Oxyhaemoglobin saturation decreased during the night in the P_{SS} group (fig. 1) in association with an increase in the apnoea and hypopnoea indices during REM sleep (fig. 2). Presumably, the higher apnoea and hypopnoea indices within the P_{SS} group during REM sleep occurred as the changes in ventilatory mechanics and drive, observed during sleep among healthy individuals, are imposed on a system that has underlying changes in respiratory function as a result of previous polio. However, we did not observe the same elevated apnoea and hypopnoea indices within the P_{RM} group.

The presence of subclinical respiratory involvement resulting in dysfunction either of ventilatory mechanics or of respiratory control has been implied both by animal [6] and postmortem studies [7] but remains speculative. The 13 subjects with isolated limb weakness (P_{SL}), who presumably had normal respiratory function during the acute event, did not differ from the 13 control individuals in any measurements of respiratory mechanics or control of breathing during wakefulness or sleep. This argues against a late progressive dysfunction, consequent upon earlier subclinical respiratory involvement, among these individuals.

It has been reported that some individuals with bulbar polio (P_{BM}) have measurable dysfunction of respiratory control and irregular breathing during sleep [8, 10]. However, our group of bulbar polio survivors had normal respiratory mechanics with the exception of a slight reduction in expiratory muscle strength. Their awake central chemoresponsiveness (carbon dioxide rebreathe) was within the normal range as were their apnoea and hypopnoea indices during sleep. Conceivably those with severe bulbar involvement might have been left with measurable bulbar dysfunction, but survival of such individuals was low [25] making it unlikely that many would be represented 30 yrs later.

A number of reports have evaluated respiratory function among polio survivors [9, 11–14, 26]. A report by ALCOCK *et al.* [11] suggested that 27% of respiratory polio survivors experienced a subjective deterioration but no quantitative pulmonary function data were presented. When quantitative data have been presented [9, 12, 13], polio survivors generally have reduced lung volumes and an elevated $Paco_2$. However, in the above studies, polio survivors were recruited to the study after presenting with respiratory complaints and therefore it is unlikely that these results can be generalized to all polio survivors. BORG and KAIJSER [14] recently reported on 20 consecutive out-patients referred on the basis of polio-related limb weakness. Some of these subjects had respiratory involvement at the time of their acute infection. Although the mean $Paco_2$ (5.5 kPa) for the group was normal, 10 of these individuals also complained of dyspnoea, two required intermittent mechanical ventilation and six required diuretics, presumably for cardiorespiratory failure. The present study expands these observations: firstly, by controlling for age, weight, smoking and other respiratory diseases; secondly, by attempting to group subjects according to their predominant involvement at the time of their acute infection; thirdly, by measuring all variables at baseline and again after one year. We also included a control group of age matched non-polio volunteers.

We deliberately excluded those individuals currently receiving mechanical ventilatory support, who clearly would have had measurable respiratory dysfunction. Therefore, our results apply to polio survivors between 30–60 yrs of age who have not received any respiratory intervention subsequent to the acute event. Among this population, a respiratory evaluation may be important, especially if there is a clear history of initial respiratory muscle involvement or the finding of a subsequent kyphoscoliosis many years later. Our current practice is to offer a full respiratory evaluation at the time of their referral with annual follow-up measurements if indicated. When these assessments have suggested severe respiratory dysfunction, elective mechanical support has been successful in improving gas exchange and level of function among these individuals [9, 13, 15, 27]. Finally, there is no evidence in this study to support late respiratory sequelae occurring among those with isolated limb polio.

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