Late Effects of Polio:

Introduction to Clinical Practice

Second Edition

Published by Polio Australia Incorporated

PO Box 2799, North Parramatta, New South Wales, 1750, Australia

www.polioaustralia.org.au

www.poliohealth.org.au

© Polio Australia Incorporated 2012, 2022

ISBN 978-0-9874392-2-2

TABLE OF CONTENTS

1. Introduction: What are Late Effects of Polio (LEoP) and Post-Polio Syndrome (PPS)?

Acute poliomyelitis ('polio', 'infantile paralysis') is increasingly a disease of history as a result of the adoption in 1988 at the forty-first World Health Assembly of a resolution for the worldwide eradication of polio, and the subsequent Global Polio Eradication Initiative.

Despite this progress towards the eradication of polio, in recent years it has become increasingly apparent that new muscle weakness, atrophy and fasciculations can develop decades after a patient endured an episode of acute paralytic poliomyelitis. The nomenclature in Australia used to describe the new health problems being experienced by those previously exposed to acute poliomyelitis, are Late Effects of Polio (LEoP) and Post-Polio Syndrome (PPS).

For the purposes of this document, the term LEoP is used to capture:

- Symptoms that are attributable to damage caused by the original acute poliomyelitis, including such aspects as residual weakness, musculoskeletal imbalance etc.;
- Symptoms attributable to a failure to maintain the level of function achieved following the original acute infection - eg, new weakness and fatigue (PPS); and
- Secondary effects of chronic neuromuscular dysfunction, such as degenerative arthritis of overused joints, soft tissue conditions etc.[1,2]

The most common symptoms of LEoP include a triad of fatigue, deterioration in muscle strength, and pain.[2,3]

Post-Polio Syndrome is generally considered a sub-category of LEoP, and is considered a neurological disorder characterised by increased weakness and/or abnormal muscle[4] fatigability occurring many years after the initial polio infection.[1,2] Symptoms are thought to manifest when the compensatory neuronal processes which sprouted following nerve damage from the initial polio infection can no longer effectively innervate the muscles within their motor unit territory, resulting in an unmasking of the neurological deficit caused by the original polio infection.[4]

The risk factors for LEoP have not been thoroughly elucidated, although some factors more predictive of a patient's risk of LEoP have been identified. Patients who originally presented with paralytic polio appear to have a higher risk of developing LEoP than those who had non-paralytic polio.[5] The extent of functional recovery during acute rehabilitation is also a risk factor for development of PPS, with those achieving greater recovery during the rehabilitation phase being more predisposed to develop post-polio syndrome.[6] The age of the initial polio infection also appears to be a risk factor, with a younger age correlated with a higher risk of developing LEoP.[7] Though, contrary to this, the reverse has also been reported, with older age when contracting polio identified as a risk factor. This has been attributed to the fact that acute polio is typically more severe in adolescents and adults than in infants and young children.[8] Other risk factors that have been observed in studies include the polio to post-polio interval and female gender.[9,10] In addition, an inactive lifestyle can increase the risk of muscle pain and fatigue in individuals previously hospitalised with acute poliomyelitis.[10]

It is estimated that between 20,000 to 40,000 people were diagnosed with paralytic polio in Australia between 1930 and 1988[11], with the highest rate reported in 1938 (39.1 per 100,000 population).[12] This figure must be increased 100-fold to obtain the estimated number of infected cases during the same period (up to 4 million people)[11-14] and it does not include people who contracted polio overseas and who have since come to Australia.

Polio Australia's 2020 position statement clarifies that "there are tens of thousands of polio survivors living in Australia who may be experiencing late effects of polio".[15] A main limitation in obtaining an accurate prevalence is that cases of LEoP can go undiagnosed - LEoP can emerge in those who experienced non-paralytic polio (i.e. varied

symptomatic presentations of polio virus infection present a LEoP risk).[16]

LEoP can be identified and multidisciplinary treatment initiated by a General Practitioner (GP). Under Chronic Disease Management guidelines, a GP may establish a General Practitioner Management Plan (GPMP) and a Team Care Arrangement (TCA) for a polio survivor client in order to manage complex care needs.[17,18]

Diagnosis of PPS is performed by a neurologist or rehabilitation specialist via a process of elimination and is a solely clinical assessment. While there are no specific tests for the diagnosis of PPS, recently developed MRI techniques (measuring spinal cord grey matter atrophy) show promise.[19] Criteria for the diagnosis of PPS were agreed upon at the March of Dimes international conference on PPS in 2001.[11,13,14,20] Prof Frans Nollet declared these to be validated at the European Conference on Post Polio Syndrome in Copenhagen, 2011. The March of Dimes criteria include:

1. Prior paralytic poliomyelitis with evidence of motor neuron loss, as confirmed by history of the acute paralytic illness, signs of residual weakness and atrophy of muscles on neurologic examination, and signs of denervation on electromyography (EMG).
2. A period of partial or complete functional recovery after acute paralytic poliomyelitis, followed by an interval (usually 15 years or more) of stable neurologic function.
3. Gradual or sudden onset of progressive and persistent of new muscle weakness or abnormal muscle fatigability (decreased endurance), with or without generalised fatigue, muscle atrophy, or muscle and joint pain. Sudden onset may follow a period of inactivity, or trauma or surgery. Less commonly, symptoms attributed to PPS include new problems with breathing or swallowing.
4. Symptoms persist for at least a year.
5. Exclusion of other neurologic, medical and orthopaedic problems as causes of symptoms.

About half of the polio survivors responding to a Polio Australia survey in 2020 had received a diagnosis of PPS[21]. The diagnosis provides clinicians and patients with confidence in the source of and approach to symptom management, and (where available) enables access to chronic condition support and services.

LEoP and PPS are the two post-polio conditions recognised by the medical and health sector in Australia. Post-polio conditions are not considered rare compared to other neuromuscular conditions.[22,23] However, they are inconsistently recognised and understood by clinicians within and outside Australia.[24]

This document serves two educational purposes for clinicians:

a) To present the major symptoms of LEoP and strategies for their clinical management; and

b) To highlight interventional risks and considerations when working with LEoP clients.

"Due to efficient vaccination programs, acute poliomyelitis is no longer common. Nevertheless, polio survivors are still common worldwide. As PPS is prevalent in these survivors, it is prudent to raise the awareness for this condition. Ignoring this significant morbidity can lead to unnecessary tests, delayed diagnosis and mistreatment with potential harm." [25]

2. Muscle weakness and atrophy

Key messages

- New muscle weakness can involve muscles known to have been affected by the original acute polio, as well as muscles that appeared to have been unaffected by the original acute polio.
- Even when muscle strength appears normal on clinical assessment, there may be significant denervation of muscle fibres that contributes to functional weakness.
- The management of new muscle weakness is primarily focussed on selective, appropriate physical activity; such activity should be well paced and allow time for rest.
- Further progression of weakness may be managed by use of assistive technology (eg. walking sticks, crutches, wheelchairs, orthoses) and home modifications (eg. ramps, rails, stair lifts).

Incidence and impact

In surveys of post-polio subjects, reports of muscular symptoms have included[26]

- New weakness in previously affected muscles (60% - 87%)
- New weakness in previously unaffected muscles (37% - 77%)
- New muscle atrophy (17% - 28%)
- Twitching or fasciculations (39% - 41%)
- Muscle cramps (43% - 48%)

Recent research on the change in muscle strength over time in the normal population over the age of 50 has shown an average decline of approximately one percent per year. For post-polio individuals this rate is reported to be higher, at approximately two percent per year.[26]

In addition, polio survivors often have a significant reduction in motor innervations and as such do not have an adequate "reserve" to sustain the same level of function as normal individuals as they continue to age.[27,28]

Increasing muscle weakness can have a substantial effect on a person's ability to function and participate. In addition to creating difficulties with activities of daily living, increasing weakness can lead to reduced balance and a higher number of falls.[3] Overuse of weak muscles, or of other muscle groups compensating for weakened polio-affected muscles, can lead to muscle pain significantly impacting a person's quality of life.[29]

Clinical characteristics

The degeneration and loss of alpha motor neurones in response to acute polio infection leaves scattered and variable muscle groups with altered neuromuscular physiology and anatomy, including:

- Terminal (collateral) sprouting of remaining motor neurones, creating giant motor units[30]
- Muscle fibre hypertrophy enabling gross functional contractions[31,32]
- Reduced capillarisation negatively affecting muscle fibre metabolism[31,32]
- Predominance of Type I fibres (slow twitch; in ambulatory survivors)[32,33]
- Increased motor neurone metabolic demands in catering to the additional (adopted) terminals[26]

Later life new-onset muscle weakness, with or without associated muscle atrophy, can involve both previously affected muscles (which were partially or fully recovered) and muscles that *appeared to be* unaffected by the original polio infection. The new weakness or atrophy is usually asymmetric.

Decreased muscle endurance and increased muscle fatigue is also common, and may precede the development of weakness. The first subtle sign may be that the time needed for recovery after muscle activity increases.[29]

Assessment

It is important to realise that people with a history of polio may have significant denervation of muscle fibres even when muscle strength appears normal on routine clinical assessment.[29] This was highlighted by Beasley's work in 1961 showing the number of functioning polio muscle fibres on autopsy related to antemortem muscle grades.[34] Notably, a grade of 5 (normal) may have as few as 53% muscle fibres *functioning*, a grade of 4 as few as 42%, and a grade of 3 as few as 9%.

Loss of motor units and a decrease in muscle strength and endurance is not necessarily associated with impaired physical function, physical activity, or social participation. This non-linear association between denervation and function is possibly because of compensatory neuromuscular mechanisms.[3]

Diagnosis for PPS can be supported by electrophysiological examination including electromyography (EMG), which usually reveals longstanding neurogenic signs (giant motor units, partial interference patterns).[3] An EMG may confirm damage from acute polio, but it does not diagnostically indicate LEoP or PPS.

Management

Carefully monitored physical activity forms the basis of management of patients with LEoP. Most patients will benefit from appropriate physical activity and a large proportion benefit from individually chosen, specific muscle exercise.[3] It is, however, important that these activities are well-paced and allow sufficient rest time to help minimise local muscle fatigue and facilitate strength recovery.[35]

In Polio survivors, the number of functioning muscle fibres within various muscle groups may be significantly less than in normal individuals. Recommendations for exercise and activity must be tailored to the capacity of each muscle group.[3] Increasing muscle weakness may be caused by recent overuse, habitual disuse or a combination of both. The underlying cause of muscle weakness should hence be determined so that appropriate management strategies can be implemented. There is no evidence that overuse weakness permanently damages muscles affected by poliomyelitis.[36,37] Conversely, muscles affected by polio readily atrophy with disuse.[3]

Managing new weakness may include:[26]

- Education regarding energy conservation techniques: pacing, rest, activity reduction and understanding muscle capacity.
- Strengthening exercise (isometric, isotonic, isokinetic).
- Aerobic exercise.
- Stretching exercises to decrease or prevent contractures.
- Weight loss.
- Prescription of assistive technology.

Prescribed orthoses may also help a polio survivor compensate for muscular weakness.[38]

Polio survivor activity and exercise should be judiciously prescribed and monitored to identify signs of muscle weakness and muscle pain in order to respond to dose-induced weakness of muscles.[39] This approach should include the monitoring of muscles appearing normal but symptomatically found (clinically or by use of EMG) to have impaired neuromuscular function. Polio survivors have reliable muscle fatigue perception, hence any report of fatigue in response to activity or exercise can guide modifications in prescription.[40]

Status quo exercise prescription leading to excessive fatigue, muscle pain or joint pain should be avoided.[41] To reduce prescription frustration and patient disengagement, referral to an exercise professional may be indicated to ensure an appropriate exercise program is developed for this population.[42,43]

Many patients will find that modifying their work environment or load, or early retirement may be necessary to manage weakness. These patients may also need modify their physical pursuits. As polio muscles can take longer to recover from physical activity, pacing physical activity and including deliberate rest will increase patients' activity tolerance.[35]

3. Fatigue

Key messages

- Fatigue can be the most disabling symptom in the cluster of LEoP symptoms.
- LEoP fatigue typically occurs every day and increases toward the evening.
- An active yet balanced lifestyle is recommended in order to minimise the impact of fatigue.

Incidence and impact

Typically, fatigue related to LEoP will begin after a period of functional and neurological stability of at least 15 years following the initial episode of acute poliomyelitis.[3] In a survey of polio survivors, new or increased fatigue was reported by 91% of respondents. Respondents also reported that fatigue was interfering with self-care (25%) and with completing or performing work (41%).[44]

Clinical characteristics

Current understanding suggests the aetiology of fatigue lies in the overuse or overwork of polio damaged nerves and muscles and can be categorised in two forms.[2,26]

General Fatigue: otherwise known as central fatigue will often occur every day and progresses during the day. General fatigue can occur following only minimal activity and is described as an overwhelming exhaustion with flu-like aching and can include hot and cold flushes and sweating in conjunction with a marked change in the level of energy, physical and mental endurance.

Muscle Fatigue: otherwise known as peripheral fatigue or muscle weakness, is reported as a decline in muscle strength upon exertion, which may be best described as muscle fatigability or lack of endurance. Post-polio individuals have described muscle fatigue as “a heavy sensation in the muscles", “increased physical weakness", and an “increased loss of strength during exercise”.[45] Muscle strength often returns after a period of rest.

A 2001 study found that physical fatigue in polio survivors, more than cognitive fatigue, caused the most functional interference.[46]

Assessment

Whilst fatigue is a primary clinical symptom of LEoP, it is non-specific with a variety of possible aetiologies.[46] As a result, before a diagnosis of fatigue is made, it is necessary to exclude other conditions that could cause fatigue such as anaemia, cancer, chronic infections, disrupted sleep, hypoxaemia, heart failure, diabetes, mood disorders, medications, lupus, and renal or thyroid disease.[26]

A 2019 Review identified the following putative factors in the etiology of PPS fatigue:[47]

- Decreased endurance
- Muscle pathology
- Weight gain
- Depression and anxiety
- Poor sleep quality
- Polypharmacy
- Possible cerebral alterations and focal atrophy
- Inflammatory central nervous system changes
- Subclinical respiratory compromise

Severity of fatigue is important to assess. Polio survivors with severe fatigue in a 2002 study had significantly higher scores than survivors with less fatigue, and controls, on a test measuring obsessive-compulsive behaviour, depression and anxiety.[48] The psychological impact of the (usual) presence of comorbidities should be considered when assessing fatigue.

Survivors reporting severe fatigue may demonstrate significant deficits on tests of attention, concentration or information processing speed; no impairments of cognitive ability or verbal memory should be expected.[49]

LEoP fatigue is often associated with onset later in the day and with increasing severity toward the evening.[45] Polio survivors in Australia report varied temporal fluctuations in fatigue, and it's effect on participation (see Figure 1).[50]

If fatigue is diagnosed, assessment by a multidisciplinary team is recommended to enable the well-considered prescription of physical interventions - particularly mobility devices (due to psychosocial factors).

Management

Fatigue management in patients with LEoP and physical and psychological co-morbidities is a persistent challenge. LEoP related fatigue may not improve over time without intervention - there are modifiable variables that can be addressed by a multidisciplinary team.[51]

Patients with PPS should be advised to avoid both inactivity and over-capacity activity.[3,29] Inactive polio survivors have a higher risk for late poliomyelitis-related symptoms compared with active patients.[10] Although some risk factors for fatigue (eg, age and time since the acute poliomyelitis) are non-modifiable, others (eg, stress and physical activity) are modifiable.

Exercise therapy (ET) and cognitive behavioural therapy (CBT) were compared to normal care in a 2016 study of polio survivors with severe fatigue.[52] No significant effect was observed for either intervention, however, the exercise intervention may have exceeded participants' anaerobic capacities (thus negating fatigue reduction).[53] Participants reported other benefits from engaging in these interventions.[43] Polio survivors with less severe fatigue may benefit from CBT, as anecdotally in Australia, and by survey in a British study, survivors report that behavioural modifications and education help them to manage their fatigue.[54]

The individualised management of polio survivor fatigue should take a multidisciplinary approach. Exercise professionals for specific training, enabling active but balanced lifestyles (work smarter, not harder), in combination with minimising any interrelated psychological stress is recommended.[29,51,55] Occupational therapists can intervene to address fatigue management, assistive technology use, lifestyle modification, fall reduction and behavioural change.[56,57] Mobility inefficiency may be reduced with assessment by orthotists and pedorthists who fabricate protections and supports, help compensate for limb weakness, accommodate and prevent deformity, and reduce pressure and limb malalignments.[38] In fatigued polio survivors referred for specialist sleep evaluation, sleep disordered breathing was common, this primarily being from obstructive hypopnea (86%). [58]

Figure 1. Fatigue characteristics of Australian polio survivors responding to a survey in 2020.[50]

My post-polio related FATIGUE currently (last 3 months) affects me doing these activities, this often (n=177)

FATIGUE LIMITATIONS	Dressing, bathing, hygiene	Chores, cook, shop, clean	Exercise routine	Hobbies	Care of other people or pets	Visiting people or places	Work or responsible to others	Volunteer or community activities
A few times a month	11%	19%	19%	18%	12%	17%	8%	10%
A few times a week	8%	19%	16%	14%	7%	12%	6%	8%
Daily	21%	27%	26%	14%	14%	13%	12%	11%
NA - due to COVID	1%	1%	3%	2%	1%	11%	6%	7%
NA - I don't do this	8%	10%	20%	13%	39%	7%	40%	33%
Rarely or never	51%	25%	16%	40%	27%	41%	29%	30%

Fatigue Incidence key: 10 to 19% >20%

4. Pain

Key messages

- Muscle and joint pain is a major symptom for people experiencing LEoP - it is typically the first or second most common symptom reported to health professionals.
- Improvement in the assessment, evaluation and treatment of pain can significantly improve comfort and reduce pain's impact on function.
- Successful management strategies focus on improving asymmetrical body mechanics and postures, supporting weakened muscles with bracing, targeted exercises, and promoting lifestyle changes that improve health and wellness and prevent further episodes of pain.

Incidence and impact

A 2013 study of polio survivors found that 68% experienced pain during examination, and that there was a significant impact of pain on the vitality and general health subdomains of the SF-36.[59] An earlier study found a similar incidence of pain (67%), with women and younger survivors reporting pain more often, and half of the subjects experiencing pain in more than one body region.[60]

Pain is widely spread across the body of those polio survivors reporting pain. One study reported an average of 10 body areas had pain, and another had found an average of 17 body areas were pain affected.[61,62] It is important to contextualise such findings against any co-diagnosis of fibromyalgia.[20]

Predominantly, pain is experienced in the shoulders (77%), low back (75%), legs (75%) and hips (67%), to a lesser incidence in many other areas, and, infrequently in the head (14%) and chest (16%).[61]

Clinical characteristics

Three different types of pain are reported by polio survivors:

- *Nociceptive* - pain from damaged tissues
- *Neuropathic* - pain from nerve dysfunction
- *Post-polio* - a muscular deep ache and cramping pain

Nociceptive pain is a pain raising an alarm to tissue injury or disease. Although nociceptive pain has been deemed the most frequent type of pain (58%) in polio survivors, it should be clearly contrasted to post-polio pain during assessment.[60]

When neuropathic pain is present, another neurological disorder likely exists as a comorbidity, hence it is important to consider the pain profiles and influence of co-existing or undiagnosed health conditions.[3,26,60]

Ten percent of patients with LEoP have neuropathic pain, mainly caused by secondary disorders such as nerve compression or disc hernia.[60] Neuropathic sensory symptoms (such as twinges, radiating pain or pins and needles) have been reported in 36% of polio survivor subjects.[63] This pain is non-motor, and may be related to compressive neuropathies or acute polio sub-clinical sensory damage emerging.[63]

When investigating the further characterisation of post-polio pain, a 2022 study concluded that "Muscle pain in patients with post-polio syndrome does not fulfil the criteria for either nociceptive or neuropathic pain".[64] Further, post-polio pain intensity was characterised as:

- Being more intense while moving
- Not affecting physical function
- Being separate from post-polio fatigue

Muscle pain is very common and is thought to be due to an over exertion of weak muscles, or an over exertion of adjacent muscle groups compensating for weakened polio-affected muscles.[29]

Joint pain can also be significant, typically caused by joint instability, anisomelia (unequal length of limbs), disuse of muscles, abnormal

biomechanical movements, and degenerative joint diseases.[29]

Assessment

It is essential to capture the character of the complex pain polio survivors may experience by utilising a combination of qualitative and quantitative measures, such as but not limited to:

- Body diagrams of pain location
- Pain quality descriptions
- Temporal patterns of pain
- Pain impact on function and participation
- Pain management history
- Visual analogue or numerical rating scales
- The SF-36
- The SF-McGill Pain Questionnaire
- Brief Pain Inventory

To facilitate the diagnosis and treatment of pain, a classification that divides the pain syndromes into three classes has been developed[65]:

- Biomechanical pain
- Overuse pain
- Post-polio muscle pain

Biomechanical pain

Pain that results from various aspects of chronic impaired posture is the most common type reported by polio survivors. It is associated with chronic microtrauma or fall injuries.[26] This pain may present as relatively stable.

Weakness in polio-affected muscles (particularly the legs) often leads to muscular imbalance and skeletal malalignment.[65] Years of walking on unstable joints with asymmetrical weightbearing makes polio survivors more likely to develop degenerative joint disease.[26,65] Polio survivors often experience shoulder pain due to compensations for weakened muscles or because of weight gain.[66] Late effect spinal deformity (primarily scoliosis) has been reported in nearly 30% of polio survivors.[67]

Nerve compression syndromes such as carpal tunnel syndrome or stenosis may develop from years of altered body alignment, compensatory movement patterns, or postural fatigue, but may be considered as superimposed neurologic disorders.[26]

Overuse pain

The second most common type of pain reported by polio survivors is due to overuse (i.e. beyond capacity use; functional strain) of soft tissue. This pain may fluctuate in relation to function and be mistaken for fibromyalgia.[20,26]

Muscles, tendons, bursa and ligaments are all vulnerable to overuse pain. These structures are susceptible when accommodating weakened polio muscles overlying altered body biomechanics – a combination causing chronic dysfunctional movement patterns. This results in strains, sprains and inflammation. Tendinitis, bursitis and myofascial pain are examples of pain inducing overuse conditions.[65]

Post-polio muscle pain

This particular pain type seems to be specific to those who have experienced polio.[64] Compensatory changes in capillarisation and the contractile properties in polio-affected muscles cause pain.[3] Conceptually, aspects of this pain may be comparable to claudication pain.

Survivors describe post-polio muscle pain (a myalgia) as burning, cramping or a deep ache. This type of pain is usually associated with physical activity, so the pattern of this pain varies according to the individual's lifestyle and habits. It is usually exacerbated by activity, exercise, stress or cold temperatures.[26]

Post-polio myalgias typically occur at night or at the end of the day. Muscle cramps and/or fasciculations are indications of this activity-related pain in polio muscles.[65]

Management

While there is no established specific treatment for pain associated with LEoP.[3] The primary clinical management is based on non-pharmacologic intervention[29], although pharmacology can be and is used for managing neuropathic and nociceptive pains.[3] Non-pharmalogic alternatives include: improving abnormal body mechanics and postures,

supporting weakened muscles with orthoses, prescribing individualised exercise programs, utilising physical modalities, weight loss, and lifestyle modification.[3,20,26,47,68]

Adaptations recommended by a qualified health care provider, such as a physiotherapist, may include appropriate bracing, adaptive devices (walking sticks, crutches, corsets), special seating and postural modification.[65] The prescription of exercise should be alert to and seek to minimise the exacerbation of pain.[20,40,41]

Muscle weakness and muscle pain may be improved or attenuated with specific training programs - training in warm water seems to be particularly helpful.[29] Rest, localised heat and stretching may alleviate post-polio myalgias.[26]

Lifestyle modifications can be effective in reducing overuse symptoms. Occupational interventions, weight-control programmes, group therapy (eg, water exercise) and increased use of assistive devices should be considered by the multi-professional rehabilitation team.[3]

Polio survivors report trying a wide variety of pain management strategies; the continued utilisation of each being variable and individual-specific.[61] The most persisted-with pain-management strategies (which over 50% of the subjects in the 2008 study had tried) were reported as:[61]

- Heat 47%*
- Aspirin/Ibuprofen 44%*
- Stretching 39%
- Acetaminophen 35%
- Narcotics 32%*
- Strength exercise 28%
- Ice 25%*
- Massage 19%*
- Physiotherapy 11%

(*Provided 5.0 or greater average relief based on a numerical rating scale, using the strategy.)

Importantly, this study noted that for polio survivors pain problems are chronic - men for 20 years on average, and women for 33 years on average.[61] Numerous pain management strategies will have been tried over several decades, suggesting polio survivors continue to seek and are willing to try interventions which improve their pain profile. Chronic pain is complex and is benefited by coordinated care.[18]

It is important to remember that an interplay often exists between the primary LEoP symptoms of pain, weakness and fatigue for each individual polio survivor.

5. Respiratory complications/insufficiency

Key messages

- The most common respiratory presentation is shortness of breath.
- Other symptoms may be consistent with sleep disordered breathing (see Section 6).
- Complications should be evaluated and managed by a respiratory physician specialising in neuromuscular disorders.

Incidence and impact

Studies in Australia and overseas suggest the incidence of respiratory symptoms amongst polio survivors lies between 27% and 58%.[69,70]

Late respiratory symptoms may be experienced by any survivor of polio, but are thought to occur more commonly in those who required artificial ventilation (not limited to iron lung use) during the acute phase of poliomyelitis.[71] The incidence of later respiratory symptoms has also been reported to be higher in patients who experienced acute poliomyelitis beyond the age of 10, and in those whose acute infection occurred more than 35 years ago.[72,73]

A comorbidity of obesity has a negative correlation with polio survivors' pulmonary function.[74] Many of those with a history of poliomyelitis are obese according to DEXA body composition standards (96%) and by percentage body fat (81%); using BMI clearly underestimates obesity in this population.[75,76]

Respiratory symptoms are primarily hypo-ventilatory in character. A common reason for respiratory function decline is nocturnal alveolar hypo-ventilation.[71] Symptoms are caused by a combination of respiratory muscle weakness and deformity to the chest cavity, and are characterised by a progressive loss of vital capacity and an inability to clear pulmonary secretions, particularly during infection.[26] Complications can include ventilation/perfusion imbalance, pneumonias and pulmonary scarring.[77]

Clinical characteristics

The most obvious respiratory symptoms of LEoP are dyspnoea on exertion and/or at rest and difficulty clearing respiratory secretions. Other symptoms including fatigue and daytime sleepiness, impaired intellectual function (including poor concentration), morning headaches, speech difficulties, snoring and anxiety may be present and may be indicative of nocturnal hypoventilation.[78]

Assessment

An evaluation of patients should begin with comprehensive patient history, in particular a history of previous ventilation requirements, other respiratory illness and tobacco use, as well as symptoms consistent with nocturnal respiratory dysfunction such as snoring, excessive daytime sleepiness, headache, etc.[26]

Other causes for respiratory symptoms should also be borne in mind as, whilst a common symptom of respiratory muscular weakness, shortness of breath may also be a manifestation of non-respiratory pathologies, such as cardiac dysfunction, primary respiratory disease, scoliosis, smoking, and obesity.[26,79,80]

Investigations should include spirometry, evaluating vital capacity (both sitting and lying), where a decline of more than 24% in supine indicates diaphragm impairment.[81] In addition, inspiratory and expiratory pressure and maximum ventilatory volume should be measured. Arterial blood gas may also be indicated to evaluate lung function.[79,82]

It should be noted that patients who have a history of polio may have symptoms but show normal respiratory function test results - testing may not reveal ventilatory muscle endurance impairment.[73,81] A maximal respiratory pressure test and measuring sEMG may identify those with PPS with fatigue of respiratory muscles.[83]

Dyspnoea may have a root in muscle endurance impairment (neuromuscular fatigue) - this should be considered and may be challenged with appropriate exercise testing in suspiciously false negative presentations, i.e. symptomatology may reflect the respiratory pump being near but not always crossing an insufficiency threshold.[73]

Patients experiencing LEoP are also at risk of nocturnal hypoventilation caused by weakness of the respiratory muscles, chest wall deformity and sleep disordered breathing (eg, obstructive apnoea, central apnoea, or a mixed dyspnoea). For those presenting non-specific symptoms such as daytime somnolence, morning headache and fatigue, or markedly reduced lung capacity, consideration should be given to polysomnography.[3,79]

Complications such as restrictive lung disease and sleep disordered breathing should be evaluated and managed by a respiratory physician with experience in neuromuscular disease (refer to the section on *Sleep Disturbance* in this module for more information).

Management

There is some evidence from controlled trials that early recognition of respiratory impairment and introduction of non-invasive ventilator aids (eg, intermittent positive pressure ventilation (IPPV) or Bi-Level Positive Air Pressure (BIPAP) may delay further decline in respiratory function and the need for invasive ventilator intervention.[1] The use of ventilator aids may be appropriate in some cases but not necessarily all cases, their use should be discussed between the patient and their respiratory specialist.[84,85]

Respiratory muscle training is recommended for patients at risk for or receiving ventilator treatment to assist pulmonary function.[1,83] Stopping smoking, mobilisation of secretions and cough assistance are also recommended.[1]

For polio survivors with obesity, a weight reduction programme can improve respiratory symptoms.[3] In appropriate patients, weight loss and aerobic exercise, postural correction or treatment of sleep disordered breathing may be beneficial.[26,74]

If invasive ventilator aids are needed, home ventilation in tracheotomised patients is effective, though disabling.[3]

Serial follow-up should be planned and performed to monitor any respiratory deterioration, in order to avert respiratory crises.[81]

All persons with impaired pulmonary function and/or a history of respiratory disease should receive influenza and pneumococcal vaccines at recommended intervals according to National Health and Medical Research Council (NHMRC) guidelines.[86]

6. Sleep disturbance

Key messages

- Sleep disturbance in those with LEoP is usually the result of a primary sleep disorder (including obstructive sleep apnoea, central sleep apnoea and hypoventilation) or muscle twitching.
- Residual polio physical factors - anatomical, biomechanical or neuromuscular – may be significant to sleep comfort and disturbances.
- Referral to a respiratory specialist for overnight oximetry and sleep studies is recommended to help identify the cause of sleep disturbance.

Incidence and impact

Sleep disturbance is common in patients experiencing LEoP and may be due to a number of factors ranging from a primary sleep disorder to disturbance from muscle twitching.[11,87,88]

A 2020 systematic review of 41 studies on sleep disturbances in those with a history of polio noted disturbance prevalences of:[89]

- Sleep apnoea syndrome (7%-65%)
- Nocturnal alveolar hypoventilation (15%-20%)
- Restless legs syndrome (28%-63%)

The incidence of primary sleep disorders appears to be higher in post-polio patients compared to the general population[88,89], and this may be due to chest and spinal deformities, weakened respiratory muscles and damage to the respiratory control centre following the primary poliomyelitis infection.[3,26,29,88]

Primary sleep disorders include obstructive sleep apnoea (OSA), central sleep apnoea (CSA) and hypoventilation.[3,26,87,88]

OSA results in the interruption of airflow (apnoea) and occurs when the upper airway collapses.[26] Apnoeas are resolved upon waking and can occur many times per night, which in turn leads to sleep disruption.[11,26,88] One study indicated that 65% of fatigued polio survivors have sleep apnoea, with the majority of these cases (86%) classified as OSA.[58] OSA also increases the risk of hypertension, myocardial infarction, congestive heart failure and stroke in those with LEoP.[26,88]

CSA occurs when the bulbar reflexes signalling breathing during sleep fail. As a result, patients have trouble falling asleep as the transition from wakefulness to sleep is disrupted by frequent central apnoeas.[26] Although less common, CSA apnoeas last longer and occur more frequently than apnoeas with OSA; the body does not instigate a breath during CSA pauses.[90]

Random muscle twitching at night can also disrupt sleep and can be related to conditions such as restless legs syndrome (RLS), periodic movement (PLM) in sleep, and generalised random myoclonus (muscle contractions involving muscles throughout the body).[26,87] A survey in LEoP patients found that two thirds of those surveyed reported muscle twitching or jumping and 33% reported disrupted sleep due to twitching.[91]

Clinical characteristics

Common clinical signs of sleep disturbance are the result of primary sleep disorders. These signs include:[3,11,26,87,88]

- Tiredness upon waking
- Daytime tiredness and/or sleepiness
- Loud snoring (associated with OSA)
- Headache upon wakening
- Irritability
- Impaired intellectual function
- Poor concentration and fatigue

Dysfunction of surviving motor neurons within the brainstem appear to be the cause of new or emerging sleep dysfunction in polio survivors.[92]

Chronic sleep dysfunctions may be related to anatomical, biomechanical or neuromuscular factors.

In a study of 99 polio survivors with PPS, those with PLM did not have sleep measures significantly different to those without PLM.[93] Findings included:

- Total time of sleep was low in both groups and not affected by PLM; subjects were awake longer, not more frequently.
- Efficiency of sleep mean was low at 71%-73% for participants (good >85%).
- The awaking index (AI) was high in both groups but it was not worsened by PLM.
- The apnea-hypopnea index (AHI) was high in both groups showing widespread reduced sleep quality.

RLS and PLM movements occur primarily in Stage II of sleep.[94] Some polio survivors are not aware of the abnormal sleep movements detected during sleep testing.[94] Although the aetiology is uncertain, a close relationship exists between PPS and PLM.[93]

Assessment and Management

To improve polio survivors' long-term survival and quality of life, and appreciating the wide clinical spectrum of presentations, sleep disorder evaluation and management should be a priority.[89]

Assessment of sleep disturbances should always involve a thorough clinical and physical examination.[26,88] As sleep disturbances may be associated with a number of aetiologies, the cause of the sleep disturbance must be identified and patients should be referred for overnight oximetry and sleep studies.[26]

Polio-related physical aetiologies may include:

- Chest and spinal deformities
- Weak respiratory muscles
- Hemi-diaphragm
- Secretion retention

Referral to a respiratory specialist should be considered for the assessment of OSA, CSA or hypoventilation.[3,11,26,88]

If sleep disturbance is associated with restless legs syndrome then medication to treat this condition may be indicated.[3] It is important to ask both polio survivors and their partners about any incidences of PLM, RLS; polio survivors may be unaware of their RLS.[93,94]

Other considerations may include weight loss in OSA and hypoventilation related to obesity,[3,87] as well as other causes of sleep disturbance including pain and stress.[26]

It is also important to note that when assessing patients for fatigue it is necessary to exclude sleep apnoea as a cause for this LEoP symptom.[26]

7. Swallowing difficulties (dysphagia) and speech difficulties (dysarthria)

Key messages

- Clinical and sub-clinical damage to the bulbar region during acute poliomyelitis may result in the subsequent development of difficulties in swallowing and speech.
- Assessment through clinical history alone may not detect milder manifestations of disease.
- Patients with LEOP should be referred to speech pathologists for evaluation and treatment to reduce the potential negative consequences of dysphagia and dysarthria.

Incidence and impact

Daily problems with swallowing or voice production have been reported in 29% of those with a history of polio.[95]

The majority of polio survivors who experience dysphagia and/or dysarthria confirm a history of swallowing problems in their acute phase of polio. However, even those with no such history may have sustained sub-clinical damage to the bulbar nerves during the original infection.[96]

The incidence of new swallowing problems is between 6% and 22% of those experiencing LEoP.[69,97] This may be an underestimate as laryngeal penetration (passage of materials into the larynx) and loss of cough reflex can occur without obvious symptoms. In one study, testing suggested weakness in tongue/palate and laryngeal abnormalities are present in 80% and 57% of polio survivors respectively.[96]

The severity of swallowing impairment can vary substantially between patients, who often develop strategies to compensate for symptoms such as tilting or turning their heads during swallowing, eating slowly with smaller mouthfuls, and avoiding foods that are difficult to swallow.[95,98] Aspiration is reported to be rare.[99] There is evidence of a slow progression of these symptoms.[100]

The impact on those whose occupations are voice dependent should not be overlooked.[101,102]

Clinical characteristics

Intermittent or constant swallowing symptoms related to decreased pharyngeal transit, bilateral pharyngeal weakness, and decreased bolus control.[100]

Speech problems including voice fatigue and hoarseness, or difficulties co-ordinating breathing with vocalisations (i.e. singing).[95,102]

Assessment

Clinical history will help to determine whether patients are aware of bulbar or other symptoms that may be consistent with LEoP.

Nutritional habits should be reviewed.[103] Temporal aspects of dysphagia and dysarthria may expose neuromuscular capacity breaches:

- Broad food type, volume and texture choices early, but narrower food choices later in the day (unrelated to appetite).[104]
- Weaker voice projection or clarity late in the day, affecting conversations.[102]
- Periods (days, weeks) of aphonia.[101]

An assessment by a speech and language pathologist may be very helpful to the primary care physician in the evaluation of the cause of dysphagia, as may be imaging such as video fluoroscopy.[96,99] The differential diagnosis includes structural abnormalities from mouth to stomach and any disease involving muscles of swallowing (eg, motor neurone disease).

Management

Modifications in swallowing position, a change in diet[100] and exercises (phonotherapy, core, ventilatory) to improve swallow or voice effectiveness may be beneficial.[26,101] Physiotherapists may have a role to play.[102]

Polio survivors with dysphagia should have their swallowing assessed at regular intervals to monitor progressive changes as well as to determine whether compensatory techniques continue to be effective.[105]

8. Impaired thermoregulation

Key messages

- Cold intolerance due to poor peripheral thermoregulation is a common symptom of LEoP.
- Patients may not recognise that their limb is cold until they feel it is cold when touched.
- Management of cold intolerance is focused on prevention of heat loss and managing symptoms.

Incidence and impact

Cold intolerance (particularly of the extremities) is a common symptom of LEoP, and is attributed to nerve injury and muscle atrophy.[106]

Between 46-62% of responders in post-polio surveys report cold intolerance.[26]

Sensitivity to cold occurs when the external environment is cold, for example in winter or in air-conditioned environments.[107] The symptoms can present even at mild temperatures and in warm indoor surroundings.[106]

Cold intolerance may worsen or amplify other LEoP symptoms, such as pain and fatigue, affecting activity motivation and participation.[26,108] Muscle strength in polio survivors can also be affected by a cold environment; a 75% reduction in strength has been observed when a room cooled from 29°C to 18°C.[77]

Heat intolerance is reported by some polio survivors, experienced as a delayed, abrupt and widespread flushing or sweating in response to activity or hot environments.

Clinical characteristics

Cold intolerance due to vasomotor circulatory disturbance can be attributed directly to damage caused by the poliovirus.[26]

This symptom has been described clinically as inadequate muscular support for vasoconstriction,[103] a circulatory disturbance,[26] and sympathetic vasoconstrictor outflow impairment allowing passive dilatation.[77]

Generally patients do not recognise that their limb is cold until they touch it and feel it is cold.[107] Hence, this symptom may be detected locally (asymmetrically) but the consequence affects the person broadly.

Virtually all patients reporting cold intolerance will have a normal core body temperature, however limbs with significant atrophy may exhibit coolness to touch, a bluish discoloration and variable degrees of swelling.[26]

Symmetrical age-related factors causing poor cold defence such as skeletal muscle mass decline, reduced metabolism, reduced brown adipose tissue capacity, and diminished reactive tone of the cutaneous vasculature, compound the polio-related vasomotor impairments.[109]

Cold limb(s) due to LEoP is unlikely to delay wound healing unless the patient has comorbidities (such as diabetic vascular disease).[110]

Being cold is an inconvenience for the individual.[26] In some cases, these symptoms can become very uncomfortable and even painful.[106]

Heat intolerance in polio survivors is recognised but less studied and understood than cold intolerance; similar aetiology is assumed, from the vasomotor disturbances already described.

Treatment

There are no medications or other types of clinical interventions to treat the root cause of cold intolerance in polio survivors.

The treatment of cold intolerance is focused on prevention, where possible, and actively mitigating the environment and body to reduce heat loss.

Cold intolerance is a difficult symptom to address.[54]

Prevention of heat loss may include practical strategies such as:

- Avoiding cold environments
- Staying alert to weather changes
- Reducing stasis and blood pooling postures
- Wearing in heat-retaining textiles
- Having heat retaining measures at hand (e.g. blankets, extra clothes, wind shields)
- Maintaining dryness of clothing
- Transitioning astutely between activities or environments (i.e. after hydrotherapy or bathing, and after exercise)
- Using and requesting individualised climate control indoors, and in hospitals

Mitigating heat loss includes strategies such as:

- Multiple layers of clothing, massage, and localised heat[26]
- Moving out of the cold environment
- Using radiant heat (sunlight, space heater, fireplace)
- Heat modalities (heat packs, hot water bottles, lamps, electric blankets)

Biofeedback, relaxation and visualization have also been recommended for cold intolerance.[103,111]

Mild exercise can generate metabolic heat, and consuming warm foods and fluids can supplement core temperature.

As highlighted in Pain (*Section 3 of this document)*, polio survivors are inclined to utilise heat modalities for pain management.

9. Bladder dysfunction

This section is a summary of a presentation given by Dr Lise Kay at the Living with Polio in the 21st Century Conference, in 2009.[112] Other sources are included as cited.

Key messages

- Symptoms of bladder dysfunction can have a significant impact on a person's quality of life.
- Potential causes of bladder dysfunction in survivors of polio include impaired detrusor muscles and nerves, oedema in the legs, restricted mobility and problematic voiding habits.
- The choice of treatment of urinary dysfunction in polio survivors varies depending upon the underlying pathology.

Incidence and impact

During the epidemics of acute polio, bladder dysfunction was reported in approximately 20% of polio cases, with a greater prevalence among adults. The majority of patients experienced urinary retention, although incontinence, urinary stasis and urinary stones were also reported. Symptoms of bladder dysfunction in acute polio generally lasted one week, however approximately 15% of cases resulted in permanent damage and ongoing sequelae.

In a post-polio survey with 272 respondents, 87% reported at least one bladder symptom, this being twice the incidence seen in the background population. Of those having bladder symptoms 76% were bothered by it.[113]

Changes in toileting ability were reported by 32% of Australian polio survivors in a survey; the youngest case being 38 years (Figure 2.).[50]

Urinary incontinence can have a significant impact on a person's quality of life. Patients can feel embarrassed about their symptoms and consequently restrict their involvement in social activities outside of their home.

Clinical characteristics

There are a number of potential causes of bladder dysfunction in survivors of polio, including impaired detrusor muscles and nerves, oedema in the legs, restricted mobility and problematic voiding habits.

A weak detrusor muscle may cause incomplete voiding and consequently voiding becomes more frequent and overflow incontinence may result. Polio survivors may also have a weak sphincter/pelvic floor leading to stress incontinence (leakage of urine), or an imbalance in the autonomic nervous system giving rise to urge incontinence (difficulty inhibiting the desire to void) or difficulties in initiation of voiding.

The most common voiding bladder symptom reported by polio survivors is incomplete emptying. Males with bladder symptoms predominantly report straining and weak stream, while females predominantly report urge incontinence.[113]

Polio survivors with restricted mobility due to weak leg and arm muscles may experience functional incontinence because they are unable to make it to the toilet in time to avoid leakage of urine.

Polio survivors with paralysed legs may experience urinary incontinence as a result of oedema in the legs - fluid which accumulates in the legs during the day becomes mobilised when these patients lay down in bed, resulting in a larger urine production at night.

Another factor contributing to bladder dysfunction in polio survivors is problematic voiding habits, in particular suppressing the need to void leading to an overstretched detrusor muscle.

Assessment

Generally, three simple tests and a screen for other diseases are required for a diagnosis of bladder dysfunction:

- A drinking/voiding chart for 3 days to be completed by the patient.
- Measurement of velocity of urine flow via a flowmeter.
- Measurement of residual urine via ultrasound.
- Screening for other diseases by urinary stick, vaginal-rectal examination and ultrasound.

A 2017 study reported assessing somatic and autonomic nervous function in a polio survivor experiencing bladder dysfunction, They utilised neurophysiological diagnostic procedures (electromyography, nerve conduction velocity, sacral reflex evaluation) to discern central and peripheral nervous system damage, and pelvic floor and detrusor paralysis.[114]

If further work-up is required to reach a diagnosis, the patient should be referred to a urologist for full urodynamic investigation.

Treatment

The choice of treatment of urinary dysfunction in polio survivors varies depending upon the individual's underlying cause of symptoms.

Multidisciplinary referrals may be required; rehabilitation programmes should also focus on bladder symptoms given their incidence.[113]

Stress incontinence may be treated by strengthening the sphincter/pelvic floor with specific exercises, however urge incontinence may require pharmacotherapy.

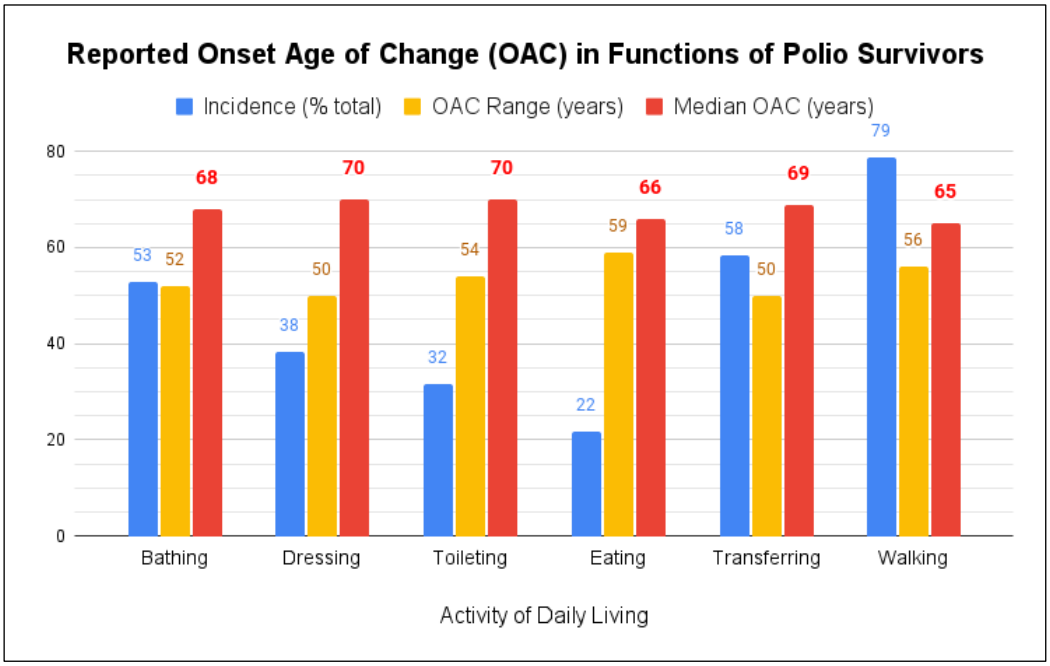
Patients who experience incomplete voiding (residual urine > 100ml) may find it beneficial to trial double voiding (i.e. void once again at the same toilet visit). If this is unsuccessful, clean intermittent self-catheterisation should be considered, with permanent catheterisation reserved as a final treatment option.

If urinary flow is less than 15ml per second or if the patient is unable to obtain voiding volumes greater than 100ml, they should be referred for a full urodynamic investigation.

In patients presenting with oedema of the legs and a large urine production at night, daytime fluid retention should be prevented by elevation of the legs and/or elastic stockings, supplemented with a mild diuretic in the evening (about 5pm) when necessary.

Where an idiopathic overactive neurogenic bladder is diagnosed, selective botulinum toxin infiltration of the detrusor may be considered.[115]

Figure 2. LEoP affected activities of daily living (ADL) reported by Australian polio survivors in 2020.[50]



10. Surgical considerations

Key messages

- When a polio survivor presents for surgery, special precautions are necessary as these patients may face complications during and post-surgery as a consequence of their LEoP.
- The choice of anaesthesia requires special consideration and lower doses (titrating up from 50%) are generally recommended for general anaesthesia.
- In dental surgeries, higher doses (double) of local anaesthesia may be required to control pain.
- Intensive monitoring may be required in the post-operative period; recovery may be prolonged.

Surgery and anaesthesia is one of the four primary interventional risk areas for LEoP

Polio survivors and clinicians across their health team should obtain (free online) and be guided by the highly recommended document *Surgery and Anaesthesia: A guide for people with a history of surgery.*[116]

Utilising the document's *Polio History Form* reduces hospitalisation risks for polio survivors by ensuring a thorough polio history is obtained, clinicians are alerted to LEoP and assumptions are minimised, and proactive and pre-emptive action can be taken.

A conservative, collaborative multidisciplinary approach for surgeries with this patient population will enable more success than approaches using 'fast track' pathways.[116]

This section is non-specific to the type of surgery that is being performed.

Pre-operative assessment

Patients with LEoP should have the following assessments prior to surgery:[117]

- Assessment for contractures or spinal deformities to establish a baseline condition and predict positioning requirements during surgery.
- A detailed respiratory evaluation should be conducted, regardless of whether the patient experiences respiratory symptoms related to LEoP or not. Any patient with symptoms suggestive of a decreased respiratory reserve should be referred for a baseline chest radiograph and spirometry.
- Assessment for a history of sleep apnoea or hypoventilation syndrome as these patients are at a higher risk of cardiac dysfunction.

Peri-operative considerations

The following are important factors for health care providers to consider when a patient experiencing LEoP undergoes surgery:

- Regional anaesthesia is preferable to general anaesthesia in LEoP patients as it is associated with fewer side effects.[26]
- The choice of general anaesthetics requires special consideration. Generally, a selection of shorter-acting agents with titration to desired effects is preferred in patients with LEoP.[117]
- Patients with LEoP may have increased sensitivity to the effects of induction drugs, maintenance drugs, muscle relaxants and opioids. A lower dose of any such mediation is recommended in this patient population.[117]
- Baseline twitch response to peripheral nerve stimulation should be measured before administration of neuromuscular blocking agent, as this response may be abnormally small in some muscles in post-polio patients.[117]
- Patients should be comfortably positioned with consideration given to limbs with contractures.[117] Sustained muscular stretch or pressure can sabotage the post-operative mobility and safety of those with LEoP.

- Blankets or warming devices may be needed during surgery for patients with LEoP cold intolerance.[117] A loss of body heat can be challenging to regain and can amplify other LEoP symptoms (see *Section 7*. of this document), delaying the recovery pathway.
- Prophylactic anti-emetic medication may be required as some patients with LEoP have bulbar dysfunction and an increased risk of aspiration. It is crucial to carefully suction the laryngopharynx prior to emergence from anaesthesia.[117]

Post-operative management

Important considerations for health care providers during the convalescent period include[26]:

- It should be recognised that being outside of one's comfort zone may lead to the need for more assistance (i.e. utilise the aforementioned Polio History Form).
- Recovery from surgery in patients with LEoP may be prolonged by 2 or 3 times beyond the expected duration for the general population.
- Some patients may require intensive monitoring in the post-operative period, particularly in order to monitor their pulmonary function.
- LEoP patients with sleep apnoea may experience a worsening of their symptoms following general anaesthesia.
- Polio affected muscles may be temporarily weaker after general anaesthesia and patients may require mobility aids.
- Delayed wound healing is uncommon on donor sites from polio affected limbs.[118]

Dental surgery

Although polio survivors with LEoP are more sensitive to general anaesthesia, they seem to require about twice the typical dose of local anaesthetic for dental surgery because of their increased sensitivity to pain. A problem is that the increased dosage may cause paralysis of facial, tongue and pharyngeal muscles and impair the ability to swallow saliva or breathe.[119]

Swallowing and breathing may be further compromised in those who had bulbar polio or paralysis of the respiratory muscles simply by reclining in a dental chair. A safe and comfortable reclined position should be identified before any dental procedure begins.[119]

For these reasons, polio survivors need to communicate their polio histories and any requirement for physical assistance in transferring to and from the dental chair in the pre-operative period. Their medical history should include swallowing difficulties and the need to use ventilatory support. They should also inform the dentist of all medications taken since high doses of painkillers such as aspirin can cause excessive bleeding of the gums and anti-cholesterol drugs can further weaken muscles.[119]

Polio survivors with osteoporosis should inform their dentist if they are being treated with bisphosphonate (Fosamax) because of the risk, albeit no more than 1/10,000, of developing osteonecrosis of the jaw; especially if the bisphosphonate therapy has been continued for more than 3 years.[120]

Recovery from dental surgery with local anaesthesia in LEoP patients may also be prolonged by 2 or 3 times beyond the expected duration for the general population. This is a major reason why same-day oral surgery for complicated dental procedures in polio survivors with LEoP is not advisable. Further, sedation-impaired coordination after dental surgery makes falling more likely and the need for physical assistance or mobility aids much greater.[119]

11. Falls considerations

Key messages

- Tailored exercise programs that include exercises to challenge balance should be implemented for polio survivors with LEoP to reduce their high fall rate.
- Osteoporosis is common in the hips of polio survivors, especially those with polio-affected lower limbs, so bone mineral density of both hips should be determined in males and females in this population and treatment instituted, if indicated.
- The removal or modification of environmental hazards in the home and the avoidance of risky behaviour and external hazards can prevent falls.
- Appropriate and consistent use of braces and walking aids contributes to the prevention of falls.

Fall mitigation is one of the four primary interventional risk areas for LEoP

Polio survivors and clinicians across their health team should be aware of the high incidence of serious falls annually and their nature, and the impactful consequences of falling on function and participation.

A main theme revealed across polio survivors in one study was: *Everyday life is a challenge to avoid the consequences of falls.*[121]

The risk calculations and attention demands for polio survivors on - what may appear to be benign - health interventions and environments, should not be overlooked.

Incidence and Impact

A Cochrane Review of the medical literature on falls in people over 65 years of age living in the community asserts that approximately 30% fall each year.[122] In contrast, the frequency of falls in polio survivors is significantly higher.

An early article on falls in polio survivors from the United States in 2002 reported a fall rate of 64% in the previous year, with 61% of the falls requiring medical attention and 35% resulting in at least one bone fracture.[123]

A report from Ireland in 2009 recorded a polio survivor fall rate of 64% over the previous six months; 38% had a fracture as the result of a fall over the previous five years.[124]

A 2010 article from The Netherlands reported that 74% polio survivors who visited a rehabilitation department between 2003 and 2009 had sustained at least one fall in the previous year. More than one fall was reported by 60% of those.[125] This study stands as a benchmark for the considerable subsequent fall research in this population.

A survey of Australian community-dwelling polio survivors revealed a serious fall rate of 58% amongst respondents in the previous year; a further 9% reported nearly falling in the same period.[50]

Assessment

Each individual's risk assessment, and any subsequent interventions, should be based on their unique presentation and history.[125] Internal and external factors must be evaluated to precisely predict fall risk.[126]

The reasons identified in the literature for falling are numerous. Falls may occur in context of:

- Walking in a familiar environment, in the afternoon.[125]
- A fear of falls, and impaired balance.[125]
- Walking variability.[127]
- Reduced walking performance, and decreased dynamic balance.[128]
- Depression.[129]
- Being a slower walker, having a high FES-I score, and high orthosis use.[126]
- Walking outside, slipping while walking, and leg length difference.[130]

- Quadriceps weakness of the most polio-affected leg. [125,126,128]

Measures utilised across these studies included:

- Activities-specific Balance Confidence scale (ABC)[127]
- Surveys,[123,125,129] and interviews[130]
- Timed Up and Go (TUG)[127,128]
- Fall Efficacy Scale International (FES-I)[126,128]
- Gait characteristics[126-128]
- Muscle strength[126,128]

Reduced muscle strength, relatively rapid muscle fatigability, and the impaired balance and gait associated with LEoP are risk factors for falling. Other risk factors to be assessed in this population are visual impairment, dizziness on standing (eg, due to low blood pressure), and the taking of certain medications (e.g. psychotropic drugs).[122]

Environmental hazards in the home and risky behaviour by the occupants should be assessed, preferably by an occupational therapist, where frequent falls have occurred in the home.[57,122]

Osteoporosis is common in the hips of post-polio populations, especially in the hip that is associated with a polio-affected lower limb.[124,131,132] Bone mineral density should be measured in both hips in males and females in such populations and appropriate treatment instituted, if indicated.

Amongst Irish post-polio study participants, 56% were diagnosed with osteoporosis and 40 with osteopaenia. Only 32% of those who had already sustained a fracture were receiving anti-resorptive treatment for their diagnosed low bone mineral density.[124]

Intervention and Prevention

Polio survivors report being greatly affected by falls, and, in order to reduce falls and their consequences, they utilise problem-focused and emotion-focused strategies.[121]

Referral to a fall reduction clinic or multifaceted falls management programme is recommended.[121]

Reviews of the medical literature provide strong evidence that exercise programs can reduce fall rates in older populations but the exercises that are more likely to be effective are those that challenge balance.[122,133]

The exercise program chosen should be tailored to the capabilities of the individual polio survivor with LEoP, under the direction of a physiotherapist who has been trained or is experienced in devising appropriate exercise programs for polio survivors.

The removal or modification of environmental hazards in the home under the direction of an occupational therapist has been shown to prevent falls among older people who are at increased risk of falling. In addition, the home visits may also lead to changes in behaviour that enable older people to move more safely in the home and in the external environment.[57]

Rough terrain, sloped surfaces, wind and crowds in the external environment increase the risk of falling for those with LEoP and are best avoided, if possible.

Well-fitting and maintained bracing that prevents foot drop or stabilises weightbearing joints also contributes to the prevention of falls. Utilising prescribed assistive technology will enable continued and safer walking (i.e. the use of a wheeled walker instead of a cane when leg muscles lose capacity).

Polio survivors who live alone should be encouraged to wear personal alarms as they may be unable to recover from a fall, even in the absence of injury or fracture. A coded key safe box containing house keys should be installed on the outside of the house near the front door to ensure prompt access when help arrives.

12. Exercise considerations

Key messages

- Exercise helps to reduce the risk and symptomology of numerous health conditions.
- Exercise professionals who prescribe exercise should be well informed on LEoP.
- Responses to exercise should be closely monitored to not exacerbate primary symptoms.
- A reduced capacity for exercise indicates conservative FITT-VP dosing of exercise.
- Professionally prescribed and monitored LEoP exercise programs have very low risks.

Activity and exercise is one of the four primary interventional risk areas for LEoP

Polio survivors are likely to ask clinicians across their health team about choosing and dosing activity and exercise. Survivors have valid concerns about tasks which can exacerbate LEoP symptoms, challenge their body capacity, interfere with reliability of function, or disrupt patterns of participation.[134,135]

Overview

In polio survivor populations, in the literature, and amongst clinicians there has been uncertainty regarding LEoP symptom exacerbation and progression, and what influence activity and exercise may have on it.[36,40,41,136]

Current research emphasises several key considerations for exercise by polio survivors:

- Most patients with LEoP benefit from appropriate dosed physical activity and a large proportion benefit from individually customised muscle training.[3]
- Strength exercise is suitable for polio survivors - a variety of muscle groups have been strengthened both effectively and safely across many studies.[41]
- However, it is crucial to monitor and respond to person-specific limits and adverse events during interventions, with individualised modifications.[137,138]
- Periodic muscle strength assessments enable monitoring of LEoP progression during long-term non-fatiguing muscle strengthening.[139]
- Barriers to improving cardiovascular fitness include severe fatigue, muscle weakness, use of assistive devices, activity mode choices, and risk of falls.[137]

Prescription of exercise

The primary goal is to have the polio survivor client performing reliable, consequence-free exercise long-term to obtain both the physical and mental health benefits.

The FITT-VP principle should be applied conservatively, starting with deliberately low parameters and titrating carefully towards a 'sweet spot' just short of LEoP symptom exacerbation. This approach avoids frustration for both the polio survivor and clinician, and reduces risk of disengagement by the survivor.

Further research is needed to better inform clinicians on recommendations for the prescription of specific types of exercise, and the post-polio individualisation of exercise.[41]

Body system capacity limitations

Exercise tolerance can be influenced by the primary LEoP symptoms, but also by:

- Disease stage (stable or unstable).[140]
- Anaerobic thresholds <50% of HRR.[141]
- Ventilation – impaired volumes, anatomy or diaphragm may impair oxygen delivery.
- Circulatory – lower muscle capillary density, and impaired blood flow control may affect muscle performance.[32,77]
- Thermoregulation – impaired responses to cooling or heating may interrupt exercise sessions or programming.

13. Pharmacological considerations

In 2011 the Cochrane Collaboration undertook a review of the treatments for symptoms associated with LEoP.[20,142] This section briefly summarises that review. Other sources are included as cited.

Key messages

- Due to the lack of both good quality data and randomised controlled studies, definite conclusions on the efficacy of various treatment options for LEoP could not be drawn.
- Results showed that intravenous immunoglobulin and lamotrigine may help with the pharmacological management of LEoP, however, they require further investigation.

Medications is one of the four primary interventional risk areas for LEoP

Polio survivors and clinicians across their medical team should be aware of the need for greater scrutiny and titration of medications where polypharmacy exists, appreciating the muscle mass (atrophy) and symptom sensitivity factors unique to LEoP.[143]

The following therapeutic drugs may worsen the symptoms of LEoP and should be avoided or used with caution.[143]

- These may amplify weakness and fatigue:
  - Central nervous system depressants
  - Anti-histamines
  - Anti-depressants
  - Anti-anxiety agents
  - Cholesterol-reducing drugs; statins
  - Beta-blockers
  - Calcium channel blockers
  - Muscle relaxants
  - Local anaesthetics
- These may cause mineral depletion affecting neuromotor function:
  - Diuretics
  - Laxatives
- These may contribute to nerve damage:
  - Chemotherapy
  - Antibiotics
  - High dose vitamins

The decision whether or not to take any drug always has to weigh up the benefits and the possible side effects (including risk of falls).

Background and objective

The objective of the Cochrane review was to systematically review the efficacy of a range of potential LEoP treatments, both pharmacological and non-pharmacological (not included here), compared to placebo, usual care or no treatment.

The efficacy and optimum use of pharmacological treatments to manage LEoP is yet to be definitively established.

Pharmacological treatments

The pharmacological treatments included in the Cochrane review were:

- Amantadine and modafinil: these drugs act on various areas of the brain, being used to address fatigue.
- High-dose prednisone and intravenous immunoglobulin (IVIG): muscle strength, fatigue and pain may be improved by the immunosuppressive and immunomodulating properties of these drugs.
- Pyridostigmine: inhibits the breakdown of acetylcholine in the neuromuscular synapse and may have a positive effect on fatigue and other LEoP symptoms.
- Lamotrigine: its postulated neuroprotective effects may reduce fatigue and pain.

Interventions not included in the review due to ineffectiveness or risk of adverse events, were: bromocriptine, IGF-I, human growth hormone, coenzyme Q10 and selegiline.

Summary of main results

Commentary was provided for the following pharmacological treatments:

- Amantidine: 200mg/day for six weeks did not reduce fatigue compared to placebo. There is very low quality of evidence on the benefits or otherwise of amantidine.
- Modafinil: 400mg/day did not reduce activity limitations, fatigue or pain compared to placebo. The studies investigating modafinil were considered to be of high quality, hence the authors concluded there are no beneficial effects of modafinil.
- IVIG: Two infusions of 90g or one of 2g/kg body weight did not show improvements in activity limitations or fatigue. The effects of IVIG on muscle strength and pain were inconsistent. The evidence is considered of moderate quality and further investigation is required. A recent review of IVIG on LEoP pain made indefinite conclusions of its effectiveness.[144]
- Prednisone: 80mg/day for four weeks followed by a 20 week tapering scheme showed no beneficial effects on fatigue, and was based on evidence of very low quality.
- Pyridostigmine: There is moderate quality evidence that 180mg or 240mg/day showed no effect on activity limitations, muscle function, fatigue or pain. Higher daily doses are used with myasthenia gravis treatement; plasma concentrations vary significantly between individuals. Further investigation is warranted.
- Lamotrigine: 50mg to 100mg/day for four weeks was shown to improve activity limitations and pain. The evidence is of very low quality; further investigation is required to establish its efficacy.

Cochrane Review authors' conclusion

It was concluded that it was impossible to draw definite conclusions on the efficacy of the various treatment options for LEoP due to the lack of both good quality data and randomised controlled studies.

Results showed that intravenous immunoglobulin and lamotrigine may be beneficial however all require further investigation.

14. Psychological considerations

Key messages

- Psychological and emotional factors may be significant contributors to the severity of symptoms associated with LEoP.
- Conversely the severity of symptoms may in itself have an impact on psychological and emotional well- being.
- Psychological state is inextricably linked to physical wellbeing and recovery.

Incidence and impact

Polio survivors experience considerable impact with the new and increased impairments on their life situation presented by LEoP, and are generally not prepared for the impact LEoP will have on their daily life.[145] Survivors report fearing a late onset health deterioration.[135]

The overall incidence of psychological diagnoses amongst polio survivors has not been reported.

A 2007 Danish study of psychiatric hospital admissions calculated polio survivors had a 40% increased risk of being hospitalised for a psychiatric disorder.[146] The study noted that this risk was characterised as being:

- Likely related to early traumatic experience of acute polio, the subsequent parental and social attitudes, and the early life struggle for social integration
- Related to milder, organic (brain tissue disease or injury), or non-psychotic disorders (depression, anxiety, panic, obsessive compulsive disorder, phobias)
- Among those hospitalised for polio before 7 years of age (the majority of polio cases)
- More likely to occur before 45 years of age

The authors noted that psychiatric disorders may be misinterpreted as part of PPS - and vice versa – this leading to diagnostic ambiguity.[146]

Reports of the Type-A personality (hard driving over-achievers) being characteristic of polio survivors exist; survivors frequently demand perfection of themselves in all aspects of their lives and it can be confronting to face new, progressive disability.[44] The unique circumstances surrounding the development of an unexpected second disability are thought to result in particular psychosocial difficulties.[11]

Central to the aetiology of later psychological symptoms may be polio survivors reliving many of the realities, emotional and physical, of their *acute* paralytic poliomyelitis.[145] Polio survivors frequently report that the onset of post-polio symptoms have forced them, often for the first time, to recall and examine their acute polio experience.[91]

Psychological symptoms and their impact on polio survivors vary greatly with the incidence and severity of LEoP symptoms. The intertwined triad of LEoP (fatigue, pain and muscle weakness) often results in social isolation and strained interpersonal relationships which puts marriages and friendships at risk.[11,147]

Most studies do not report an increased incidence of mood disorders or depressive events amongst LEoP patients.[135]

Depressed, anxious or stressed patients with LEoP report a more severe physical deterioration, more pain with a higher rate of somatic complaints, poorer coping mechanisms, a lesser quality of life and more social exclusion.[91]

Clinical characteristics

As the physical causes and optimum treatment for post-polio symptoms are being clarified, psychological symptoms including chronic stress, anxiety, depression, and compulsive behaviour are becoming evident in polio survivors.[44]

It is important for clinicians to consider the psychological impact of a chronic illness like polio; psychological symptoms are commonly missed.[147] Psychological symptoms not only exacerbate LEoP, but they often prevent patients from making the lifestyle changes necessary to achieve a benefit from treatment programs.[20,91]

Psychological issues which may affect polio survivors were identified in a clinic review article, and listed as:[148]

- Separation, fear of abandonment
- Detachment form environment, self, others
- Body image shame, stigma, discrimination
- Self-image and identity dysfunction
- Claustrophobia
- Amnesia of early polio experiences
- Anxiety or dissociation when polio-reminiscent stimuli present
- Anxiety or panic attacks
- Trust issues
- Perfectionism
- Adjustment to new disability
- Ambivalence accepting help; feelings of uselessness
- Lack of empathy from others
- Grief related to loss of ability, role or functioning
- Social isolation
- Depression
- Post-traumatic stress disorder (PTSD)

Individuals who lose abilities which they previously re-gained through strenuous rehabilitation, may experience a deep feeling of bereavement resulting in social withdrawal, isolation, relationship hardships and a change in self perception. Polio survivors will often respond to these new symptoms with anger, fear and confusion.[91]

Due to their experiences during the acute illness, many post-polio individuals fear hospitals and are wary of health professionals. As a result, faith in the medical profession has often been lost.[11,26,135]

Assessment

It can often be difficult to separate the symptoms of LEoP due to the interwoven connectivity of physical and emotional states. In order to provide the best therapeutic advice, it is essential to have a good knowledge of the symptoms but also take the time and listen to patients in order to differentiate the underlying physical and psychological components.[135]

The most appropriate method of providing a comprehensive and coordinated evaluation that detects and addresses the polio survivor's medical, functional, psychosocial and vocational needs, is through the use of an interdisciplinary team including physician, physiotherapist, occupational therapist, psychologist and social worker. [11,26]

Management

Little has been written about psychotherapy treatment with the post-polio population.[148] However, existing literature and polio survivors' accounts reveal positive responses to psychotherapy and psychologically informed care for issues related to acute polio and rehabilitation experiences, as well as PPS.[148]

The main aims of addressing the psychological needs of the post-polio patient should be to:[11,26]

- Increase and expand the patient's personal and external resources
- Provide education and support (to both the patient and family)
- Reinforce the need for the patient to have control over their lives

Trauma Aspects

Trauma-informed treatment is strongly recommended, regardless of performance on post-traumatic stress disorder (PTSD) measures.[148] In a survey of 215 Australian polio survivors' response to COVID 19 Pandemic lockdowns, 40% scored as *probable PTSD* and *PTSD supressing immune function* on the Impact of Event Scale-Revised (IES-R) instrument (using McCabe strata).[149] Polio

survivors appear to experience trauma in response to non-health events similarly to those living with Parkinson's Disease, but to a lesser degree than those with other chronic neuromuscular diseases.[149]

Polio survivors may react defensively to questions about their psychological profile – particularly to any questions interpreted as a challenge of intelligence. This can be tied to their experiences of growing up with disability in an era when disability was narrowly understood. Hence, it is helpful for clinicians to clarify the aspect and context of mental health being investigated, some examples being:

- Coping ability, given LEoP body changes
- Depression, affecting quality of life
- Anxiety or phobias, affecting relationships
- Attention and concentration, because of severe fatigue

Psychological support can also assist the patient in their evaluation of therapeutic choices (rehabilitation, orthotics, mobility devices, medications, speech pathology or surgery), as well as the choice between treatment or an abstention of treatment.[135] In particular, the concept of relying on mobility devices such as walking sticks and wheelchairs can be extremely traumatic for polio survivors. It is suggested that patients first trial these devices to help enhance their enjoyment of an activity (eg, visiting an art gallery or at an airport).

Importantly, historic approaches to polio treatment have been to ignore pain and fatigue and to exercise as much as possible; these therapeutic strategies are now being regarded as possible contributors to the post-polio symptoms.[11,26] Now, post-polio patients and their families are being challenged by being told to act conservatively - dramatically changing their approach to managing their symptoms. This presents a major upheaval for many individuals and often results in higher levels of non-compliance in treatment programs. [91]

Interpersonal Aspects

Psychologists can provide counselling, education and support with regards to emotional difficulties the individual and close family members may experience. Intimate relationships between partners may also be affected due to the symptoms of pain, fatigue and weakness which affect the individual's self-image and their sexuality.[11,26]

Unfortunately, the support of family and friends is often not sought by polio survivors and, even when it is, may not prove to be adequate to the patient. Newly LEoP-diagnosed patients frequently experience problems in communicating effectively about LEoP with their families and friends, and in obtaining help from them. It has been reported that 39% of polio survivors request help from their family, but of these only 52% found it "very helpful" to do so. While 75% had talked with family about LEoP, only 40% rated this as "very helpful". In addition, this study found that 74% had talked with friends about LEoP but only 23% of these found this "very helpful".

Personal coping strategies (such as becoming more involved in interests they can still pursue, developing their philosophy of life, reading more about LEoP) were more frequently reported as being adopted than were interpersonal coping strategies and were more frequently rated as "very helpful". The length of time required to get a diagnosis results in many survivors' symptoms being discounted by their families and often health practitioners.[70,150]

Health Team and Support

As with the assessment of patients, the most successful approach to managing the complex psychological components of LEoP will involve a comprehensive interdisciplinary treatment program comprising physicians, physiotherapists, social workers and psychologists.[11,26,91] Each team member brings with them specific skills and knowledge that could assist the patient to address some of the aspects of their condition.

In addition, patients should be encouraged to connect with post-polio support groups to facilitate communication and awareness about their illness, and to allow access to an additional support network.[26] Together they can address LEoP as a whole.

15. Comorbidity considerations

This section provides a summary of a presentation given by Irene Tersteeg at the European Conference on Post polio Syndrome in 2011.[91,151] Other sources are included as cited.

Key messages

- A higher level of comorbidity has been shown to be associated with a lower level of physical functioning, particularly where obesity is present.
- Polio survivors have more cardiac disease, respiratory disease, endocrine and metabolic disease, and more bone disease than the non-polio-affected population.
- Screening tests and early vigilance is essential, and should be done for diabetes, hyperlipidemia, hypothyroidism, osteoporosis, and lung function.

Incidence and Impact

Polio survivors have paresis, are less able to lead an active lifestyle and are therefore more prone to certain types of comorbidity.

A higher level of comorbidity is associated with a lower level of physical functioning and a faster decline in physical functioning in polio survivors. Lifestyle-related factors, physical inactivity and excess weight are also associated with a lower level of functioning in polio survivors.

The most common comorbidities (those with a prevalence of 40% or greater in at least one study) include:[152]

- Fatigue
- Pain
- Depression
- Respiratory and sleep disorders
- Falls
- Bone disorders
- Bladder dysfunction.

Obesity may be underestimated when utilising BMI with polio survivors; a vast difference in potential diagnosis may result when percentage body fat is measured (39% versus 81%).[76]

Longitudinal research examining the course of health conditions in people aging with LEoP is limited. Disentangling typical ageing from ageing with LEoP to help determine secondary condition frequency and severity, requires further investigation.[152]

Clinical Characteristics

Compared with the non-polio-affected population, polio survivors have more disease of the heart and blood vessels, such as heart attacks, hypertension and cardiac arrhythmias.

They also have more respiratory disease such as chronic pulmonary disease and asthma. When polio survivors age, they may encounter late onset respiratory failure due to weakening of respiratory muscles and deformities of the chest.

The incidence of endocrine and metabolic disease such as diabetes, hyperlipidaemia, and hypothyroidism are also higher in polio survivors.

Due to an imbalance in muscle load, polio survivors have more musculoskeletal disease, such as arthrosis and osteoporosis. Increased muscle weakness can lead to reduced balance and combined with osteoporosis can increase the risk of fractures after a fall.[130,132]

Assessment & Management

Clinical vigilance is essential for identifying any early signs of secondary health conditions in polio survivors.[152]

Many of the comorbidities associated with LEoP are asymptomatic in their early stages, highlighting the importance of regular screening tests.

While fatigue is a common symptom associated with LEoP, it is important to rule out fatigue from other causes such as hypothyroidism.

Screening should be done for diabetes, hyperlipidaemia, hypothyroidism, osteoporosis, and lung function.

To reduce the risk of acute respiratory failure and hospitalisation, non-invasive inspiratory and expiratory muscle aids should be implemented where chronic alveolar hypoventilation is evident.[153]

Walking aids may be needed in patients with increased PPS muscle weakness in order to reduce the risk of falls and related bone fractures.[154]

Advice should also be offered regarding ways to manage diet and weight.

Physical activity should be encouraged, with activities prioritised to lowering (while not exacerbating) symptoms of fatigue and pain and to minimising the decline in physical functioning.[41]

Training in warm water is recommended, as it reduces the stress on joints and muscles and warm water may have an analgesic effect. Dynamic water exercises, not necessarily swimming, are a good alternative to weight-bearing exercises.

The challenges of experiencing a clinical or sub-clinical lifelong disability, followed by the emergence of a progressive and chronic health condition (LEoP), with the confounding effects of secondary health conditions should not be underestimated. Several factors have been identified that generate improved quality of life and higher life satisfaction for polio survivors, these being:

- Perceived participation[155]
- Purpose in life[156]
- Future-oriented coping strategies[157]

Limited Capacity: An analogy

Polio survivors can be easily frustrated by clinicians not understanding the post-polio presentation of *limited capacity across body systems*. This manifests through clinicians having status-quo expectations about survivors' physical ability and underestimating the weight of the mostly invisible symptoms on their function and participation. The following analogy serves to provide clarity for both clinicians evaluating a polio survivor, and polio survivors communicating their limited capacity.

Imagine a polio survivor's body as a vehicle with a 5-speed manual gearbox. With 5 gears, they could travel locally, intrastate (to the next city) and interstate (crossing borders) without much trouble. But acute polio caused permanent scattered damage to numerous systems in their body, so their maximum potential function – 5th gear – is unavailable. Sub-clinical damage that the survivor may not be aware of also limits the dependability of higher functioning – 4th gear is unreliable and won't stick. Without 4th and 5th gears, driving interstate would cause significant strain on the car, risking a breakdown. Intrastate trips would be avoided unless critically required.

LEoP emerges, and what was useful of the limited 4th gear is now unavailable. In addition, the 3rd gear has now become unreliable. Local driving is the only reasonable function the car now has, because no mechanic (clinician) can un-damage or replace these particular 3rd through 5th gears. The driver (survivor) would like to drive more and further, but they know the breakdown risk to their car is not worth it. Further, the headlight bulbs now dim after an hour's use, and the windshield wipers rapid setting fails in heavy rain. It takes a lot of effort and attention to drive this car.

All these things a mechanic *would not see on a usual inspection*. Only careful assessment and listening very astutely to the driver's complaints will reveal the car's actual functioning capacities.

16. The post-polio health team

- Due to the numerous and varied considerations for the diagnosis and management of patients experiencing LEoP, a broad team of healthcare professionals may be required to make up the post-polio health team.
- A list of experts that could make up this team is provided below.

- **Rehabilitation Specialist:** To support the management of pain and energy levels.
- **Neurologist:** To provide definitive diagnoses and comprehensive treatment plans for the neurological sequelae in polio survivors.
- **General Practitioner:** To develop a management program to address post-polio patients' specific symptoms, ensure continuity of care, educate the patient and promote a healthy lifestyle.
- **Physiotherapist:** To support polio survivors in improving/maintaining mobility, function and provide relief from pain. https://australian.physio/
- **Exercise Physiologist:** To improve the health of patients using exercise interventions and health education. www.essa.org.au
- **Occupational Therapist:** To assist polio survivors to employ strategies to help maintain overall health and encourage the highest level of lifestyle independence. www.otaus.com.au
- **Speech Pathologist:** To support the management of patients with speech or swallowing disorders. www.speechpathologyaustralia.org.au
- **Orthotist:** To fit appropriate and well-fitting orthoses for patients who require them. www.aopa.org.au
- **Podiatrist:** For assessment and treatment of foot conditions (commonly present in patients with a history of polio). www.apodc.com.au
- **Pedorthist:** To manage and treat conditions related to the foot, ankle and lower extrem ities. Assists with fitting and fabrication of medical grade footwear and pedorthic appliances. www.pedorthics.org.au
- **Respiratory Specialist / Sleep Specialist:** For management of respiratory dysfunction in patients with LEoP including preventative measures, ventilator assistance and treatment of sleep apnoea.
- **Psychologist / Social worker:** To provide counselling, education and support. www.psychology.org.au
- **Dietitian / Nutritionist:** To provide education and management strategies regarding weight management. www.daa.asn.au
- **Massage Therapist:** Can assist in the management of cold intolerance and pain control. www.massagemyotherapy.com.au
- **Registered Nurse / Nurse Practitioner:** To provide client advocacy, coordinate care plans across disciplines, provide chronic disease management and wound care in various settings including home, general practice and hospitals. www.nursingmidwiferyboard.gov.au
- **Osteopath:** To provide support for the management of postural / biomechanical issues in patients with LEoP. www.osteopathy.org.au

17. Summary

Post-polio conditions are neither contagious or fatal, but are chronic and progressive for those who have been exposed to polio. The damage incurred (scattered neurone death) during acute polio is irreversible and unique across neuromuscular conditions.

Clinically, post-polio conditions present as variable within and between individuals, and are managed primarily by careful assessment and avoiding exceeding the capacity of body systems whose capacity is limited.

- LEoP is a set of sequelae in polio survivors characterised by the chronic impairments caused by the original polio infection, the secondary traumatic effects of those impairments, and from PPS, a neurologic disorder characterised by increased weakness and/or abnormal muscle fatigability and pain occurring many years after the initial polio infection.
- New muscle weakness can involve previously affected muscles, as well as muscles that appeared to be originally unaffected. Management of this new muscle weakness requires appropriately assessed and paced physical activity.
- Central and peripheral fatigue is one of the most common and disabling symptoms of LEoP; a balanced, active lifestyle is recommended to minimise this symptom.
- Muscle and joint pain is a major and common issue in LEoP. Successful management focuses on improving abnormal body mechanics and posture, supporting weakened muscles with bracing and mobility devices, exercise, and lifestyle changes such as weight loss.
- The most common presentation of respiratory complications is shortness of breath; other symptoms may relate to sleep disordered breathing. These should be evaluated by a respiratory physician.
- Swallowing and speech difficulties are caused by damage to the bulbar region during the acute phase of polio, and may not be detected by clinical history assessment alone. Referral to a speech pathologist for full evaluation and management is recommended.
- A common symptom associated with LEoP is cold intolerance, which is managed by preventing or mitigating the symptoms of impaired thermoregulation, rather than influencing the cause.
- Disturbance to sleep is common and may be due to chest and spinal deformities, and weakened respiratory muscles following primary polio infection. Assessment should always involve a thorough clinical and physical examination.
- Bladder dysfunction - potentially caused by impaired detrusor muscles and nerves, leg oedema or restricted mobility - can have a significant impact on quality of life. Treatments vary depending upon the underlying cause of symptoms.
- Special precautions are necessary for surgeries (including dental surgery) due to the higher risk of complications as a result of LEoP, and a potentially longer recovery period. The anaesthesia choice and dose requires special consideration. More intensive monitoring during the post-operative period may be required.
- The modification of environmental hazards and individualised exercise programs to challenge balance should be implemented to help reduce falls. If appropriate, mobility aids can also help reduce fall risk.
- Exercise is recommended for its broad physical and mental health benefits, but it must be prescribed and monitored.
- Psychological and emotional factors may be significant contributors to the severity of symptoms associated with LEoP. Physical wellbeing and management of symptoms can be linked to psychological state.
- The efficacy of pharmacological treatments to manage LEoP are not firmly established. Definite conclusions cannot be drawn on the efficacy of various treatments due to the lack of good quality data and randomised controlled studies.

18. Useful resources for patients

- The following is a list of resources that may be useful for patients with post-polio conditions.
- Clinicians are encouraged to use the post-polio information curated and available on www.poliohealth.org.au

Australian Polio Register

The register collects information on Australian survivors. Registrants can opt to keep their details private.

www.australianpolioregister.org.au

Community Information Sessions

In-person information sessions for polio survivors, their family and carers on LEoP.

www.polioaustralia.org.au/community-information-sessions/

State Polio Networks

State-based polio support and services.

www.polioaustralia.org.au/support-and-services/

Polio Australia's YouTube Channel

Educational videos for polio survivors.

www.youtube.com/user/PolioAustraliaInc

Topic-Based Resources

Fact sheets about living with polio.

www.polioaustralia.org.au/living-with-polio/

Post-Polio Health International (PHI)

Care considerations for families and friends.

post-polio.org/post-polio-health-care-considerations-for-families-and-friends/

19. References

1. Farbu E GN, Barnes MP et al. . Post-polio syndrome. In: Gilhus NE BM, Brainin M, ed. *European Handbook of Neurological Management*. 2 ed. Wiley-Blackwell; 2011:311-319.
2. Halstead LS. A brief history of postpolio syndrome in the United States. *Arch Phys Med Rehabil*. Aug 2011;92(8):1344-9. doi:10.1016/j.apmr.2011.03.002
3. Gonzalez H, Olsson T, Borg K. Management of postpolio syndrome. *Lancet Neurol*. Jun 2010;9(6):634-42. doi:10.1016/S1474-4422(10)70095-8
4. Dalakas MC. The post-polio syndrome as an evolved clinical entity. Definition and clinical description. *Ann N Y Acad Sci*. May 25 1995;753:68-80. doi:10.1111/j.1749-6632.1995.tb27532.x
5. Rekand T, Albrektsen G, Langeland N, JA A. Risk of symptoms related to late effects of poliomyelitis. *Acta Neurol Scand*. Mar 2000;101(3):153-8. doi:10.1034/j.1600-0404.2000.101003153.x
6. Klingman J, Chui H, Corgiat M, Perry J. Functional recovery. A major risk factor for the development of postpoliomyelitis muscular atrophy. *Arch Neurol*. Jun 1988;45(6):645-7. doi:10.1001/archneur.1988.00520300065020
7. Ragonese P, Fierro B, Salemi G, et al. Prevalence and risk factors of post-polio syndrome in a cohort of polio survivors. *J Neurol Sci*. Sep 15 2005;236(1-2):31-5. doi:10.1016/j.jns.2005.04.012
8. Jubelt B, Cashman NR. Neurological manifestations of the post-polio syndrome. *Crit Rev Neurobiol*. 1987;3(3):199-220.
9. Bertolasi L, Acler M, dall'Ora E, et al. Risk factors for post-polio syndrome among an Italian population: a case-control study. *Neurol Sci*. Dec 2012;33(6):1271-5. doi:10.1007/s10072-012-0931-2
10. Rekand T KJ, Farbu E, Gilhus NE, Barnes MP, Roose M, Gilhus NE, Langeland N, Aarli JA. Lifestyle and late effects after poliomyelitis. A risk factor study of two populations. *Acta Neurol Scand*. Feb 2004;109(2):120-5. doi:10.1034/j.1600-0404.2003.00186.x
11. The Late Effects of Polio: Information for Healthcare Providers (Woodville: Commonwealth Department of Community Services and Health) (1991).
12. Notifiable diseases surveillance, 1917 to 1991 (Communicable Disease Intelligence) 226-236 (1993).
13. Epidemiology and Prevention of Vaccine-Preventable Diseases (CDC) (2012).
14. Polio and Prevention. The Global Polio Eradication Initiative. 2012.
15. Numbers of Polio Survivors Living in Australia. 2020.
16. P B. *Aspects of the Post-Polio Syndrome*. Polio Selbsthilfe e.V.; 2011.
17. Health Do. Chronic Disease Management (formerly Enhanced Primary Care or EPC) — GP services. Australian Department of Health. Updated 2 April 2014. Accessed 17 May, 2022.
18. Self-managing chronic pain. Pain Australia; 2016. Accessed 14 June 2022. www.painaustralia.org.au/static/uploads/files/painaust-factsheet10-final-wfvojakjczhi.pdf
19. Wendebourg MJ, Weigel M, Richter L, et al. Spinal cord gray matter atrophy is associated with functional decline in post-polio syndrome. *Eur J Neurol*. May 2022;29(5):1435-1445. doi:10.1111/ene.15261
20. Dimes. Mo. Identifying best practice and care. presented at: March of Dimes International Conference on Post Polio Syndrome; May 19-20 2000; Warm Springs, Ga.
21. Australia P. Polio Survivor Survey on Medical Professional Interactions Regarding the Late Effects of Polio (n=734). Polio Australia Inc; 2020.
22. Deenen JC, Horlings CG, Verschuuren JJ, Verbeek AL, van Engelen BG. The Epidemiology of Neuromuscular Disorders: A Comprehensive Overview of the Literature. *J Neuromuscul Dis*. 2015;2(1):73-85.
23. Ahlström G GL, LeissnerP, Sjödén PO Epidemiology of neuromuscular diseases, including the postpolio sequelae, in a Swedish county. . *Neuroepidemiology*. 1993;12(5):262–269.
24. de Lira CAB, Santos DAT, Viana RB, et al. Knowledge of healthcare professionals about poliomyelitis and postpoliomyelitis: a cross-sectional study. *Sao Paulo Med J*. Aug-Sep 2021;139(5):464-475. doi:10.1590/1516-3180.2020.0617.16032021
25. Enghelberg S, Love IY, Rapoport M. Post-Polio Syndrome Causing Late Onset Respiratory Failure. *Isr Med Assoc J*. Jun 2020;22(6):395-396.
26. The Late Effects of Polio: Information for GPs (Queensland Health) (2001).
27. WA. L. *Travelling without a spare: a survivors guide to navigating the post-polio journey*. WAL Publishing; 2011.
28. JK S. Sustaining Strength. In: K S, ed. *Post-polio: A guide for polio survivors and their families*. New Haven: Yale University Press; 2001:62-68:chap 8.
29. Farbu E. Update on current and emerging treatment options for post-polio syndrome. *Ther Clin Risk Manag*. Jul 21 2010;6:307-13. doi:10.2147/tcrm.s4440
30. Senol MG, Kaplan C, Ozdag F, Saracoglu M. How Long does Denervation Take in Poliomyelitis? Or is it a Lifetime?". *J Neurosci Rural Pract*. Oct-Dec 2017;8(4):511-515. doi:10.4103/jnrp.jnrp_173_17
31. Grimby G, Stalberg E. Dynamic changes in muscle structure and electrophysiology in late polio with aspects on muscular trainability. *Scand J Rehabil Med Suppl*. 1994;30:33-44.
32. Borg K, Henriksson J. Prior poliomyelitis-reduced capillary supply and metabolic enzyme content in hypertrophic slow-twitch (type I) muscle fibres. *J Neurol Neurosurg Psychiatry*. Mar 1991;54(3):236-40. doi:10.1136/jnnp.54.3.236
33. Borg K, Borg J, Edstrom L, Grimby L. Effects of excessive use of remaining muscle fibers in prior polio and LV lesion. *Muscle Nerve*. Dec 1988;11(12):1219-30. doi:10.1002/mus.880111206
34. Beasley WC. Quantitative muscle testing: principles and applications to research and clinical services. *Arch Phys Med Rehabil*. Jun 1961;42:398-425.
35. JC A. Exercise in the treatment of postpolio syndrome. In: Silver JK GA, ed. *Postpolio syndrome*. Hanley and Belfus; 2004:117-144.
36. Willen C, Hou L, Stibrant Sunnerhagen K. A very long-term longitudinal follow-up of persons with late effects of polio. *Eur J Phys Rehabil Med*. Apr 2020;56(2):155-159. doi:10.23736/S1973-9087.20.05918-3
37. Bickerstaffe A, Beelen A, Nollet F. Change in physical mobility over 10 years in post-polio syndrome. *Neuromuscul Disord*. Mar 2015;25(3):225-30. doi:10.1016/j.nmd.2014.11.015
38. Orthoses to support people with neuromuscular conditions. The Australian Orthotic Prosthetic Association; 2016.
39. Agre JC. The role of exercise in the patient with post-polio syndrome. *Ann N Y Acad Sci*. May 25 1995;753:321-34. doi:10.1111/j.1749-6632.1995.tb27558.x
40. Agre JC, Rodriquez AA. Muscular function in late polio and the role of exercise in post-polio patients.

NeuroRehabilitation. 1997;8(2):107-18. doi:10.3233/NRE-1997-8205
41. Ramachandran AK, Goodman SPJ, Jackson MJ, Lathlean TJH. Effects of muscle strengthening and cardiovascular fitness activities for poliomyelitis survivors: A systematic review and meta-analysis. *J Rehabil Med*. Apr 27 2021;53(4):jrm00184. doi:10.2340/16501977-2832
42. Exercise Guide for People with Polio: A guide for Physiotherapists. Polio Services Victoria; 2005.
43. Bakker M, Schipper K, Koopman FS, Nollet F, Abma TA. Experiences and perspectives of patients with post-polio syndrome and therapists with exercise and cognitive behavioural therapy. *BMC Neurol*. Feb 10 2016;16:23. doi:10.1186/s12883-016-0544-0
44. Bruno RL, Frick NM. Stress and "type A" behavior as precipitants of post-polio sequelae: the Felician/Columbia Survey. *Birth Defects Orig Artic Ser*. 1987;23(4):145-55.
45. Berlly MH, Strauser WW, Hall KM. Fatigue in postpolio syndrome. *Arch Phys Med Rehabil*. Feb 1991;72(2):115-8.
46. Schanke AK, Stanghelle JK. Fatigue in polio survivors. *Spinal Cord*. May 2001;39(5):243-51. doi:10.1038/sj.sc.3101147
47. Li Hi Shing S, Chipika RH, Finegan E, Murray D, Hardiman O, Bede P. Post-polio Syndrome: More Than Just a Lower Motor Neuron Disease. *Front Neurol*. 2019;10:773. doi:10.3389/fneur.2019.00773
48. Schanke AK, Stanghelle JK, Andersson S, Opheim A, Strom V, Solbakk AK. Mild versus severe fatigue in polio survivors: special characteristics. *J Rehabil Med*. May 2002;34(3):134-40. doi:10.1080/165019702753714165
49. Bruno RL, Galski T, DeLuca J. The neuropsychology of post-polio fatigue. *Arch Phys Med Rehabil*. Oct 1993;74(10):1061-5. doi:10.1016/0003-9993(93)90062-f
50. Australia P. Polio Survivors Health and Health Professional Use (n=177). Polio Australia Inc; 2020.
51. Tersteeg IM, Koopman FS, Stolwijk-Swuste JM, Beelen A, Nollet F, Group CS. A 5-year longitudinal study of fatigue in patients with late-onset sequelae of poliomyelitis. *Arch Phys Med Rehabil*. Jun 2011;92(6):899-904. doi:10.1016/j.apmr.2011.01.005
52. Koopman FS, Voorn EL, Beelen A, et al. No Reduction of Severe Fatigue in Patients With Postpolio Syndrome by Exercise Therapy or Cognitive Behavioral Therapy: Results of an RCT. *Neurorehabil Neural Repair*. Jun 2016;30(5):402-10. doi:10.1177/1545968315600271
53. Voorn EL, Koopman FS, Nollet F, Brehm MA. Individualized Aerobic Exercise in Neuromuscular Diseases: A Pilot Study on the Feasibility and Preliminary Effectiveness to Improve Physical Fitness. *Phys Ther*. Mar 3 2021;101(3)doi:10.1093/ptj/pzaa213
54. Curtis A LJ, Kaltsakas G, Auyeung V, Shaw S, Hart N, Steier J. The value of a post-polio syndrome self-management programme. *J Thorac Dis*. Oct 2020;12(Suppl 2):S153-S162. doi:10.21037/jtd-cus-2020-009
55. Tiffreau V, Rapin A, Serafi R, et al. Post-polio syndrome and rehabilitation. *Ann Phys Rehabil Med*. Feb 2010;53(1):42-50. doi:10.1016/j.rehab.2009.11.007
56. Young G. Occupational Therapy and the Postpolio Syndrome. *American Journal of Occupational Therapy*. 1989;43(2):97-103.
57. Cumming RG, Thomas M, Szonyi G, et al. Home visits by an occupational therapist for assessment and modification of environmental hazards: a randomized trial of falls prevention. *J Am Geriatr Soc*. Dec 1999;47(12):1397-402. doi:10.1111/j.1532-5415.1999.tb01556.x
58. Dahan V, Kimoff RJ, Petrof BJ, Benedetti A, Diorio D, Trojan DA. Sleep-disordered breathing in fatigued postpoliomyelitis clinic patients. *Arch Phys Med Rehabil*. Oct 2006;87(10):1352-6. doi:10.1016/j.apmr.2006.07.256
59. Werhagen L, Borg K. Impact of pain on quality of life in patients with post-polio syndrome. *J Rehabil Med*. Feb 2013;45(2):161-3. doi:10.2340/16501977-1096
60. Werhagen L, Borg K. Analysis of long-standing nociceptive and neuropathic pain in patients with post-polio syndrome. *J Neurol*. Jun 2010;257(6):1027-31. doi:10.1007/s00415-010-5456-0
61. Stoelb BL, Carter GT, Abresch RT, Purekal S, McDonald CM, Jensen MP. Pain in persons with postpolio syndrome: frequency, intensity, and impact. *Arch Phys Med Rehabil*. Oct 2008;89(10):1933-40. doi:10.1016/j.apmr.2008.03.018
62. Lygren H, Jones K, Grenstad T, Dreyer V, Farbu E, Rekand T. Perceived disability, fatigue, pain and measured isometric muscle strength in patients with post-polio symptoms. *Physiother Res Int*. Mar 2007;12(1):39-49. doi:10.1002/pri.352
63. Kay L, Nielsen NM, Wanscher B, Jennum P. Neurological Symptoms in Danes with a History of Poliomyelitis: Lifelong Follow-Up of Late Symptoms, their Association with Initial Symptoms of Polio, and Presence of Postpolio Syndrome. *Eur Neurol*. 2018;80(5-6):295-303. doi:10.1159/000497483
64. Boshuis EC, Melin E, Borg K. Pain in Post-Polio Syndrome: A Separate Pain Entity? *J Rehabil Med Clin Commun*. 2022;5:1000077. doi:10.2340/20030711-1000077
65. International P-pH. *Post polio Health Care*. Post-polio Health International; 2012.
66. Save our Shoulders: A guide for Polio survivors. Moss Rehabilitation Research Institute; 2003.
67. Dalakas MC, Elder G, Hallett M, et al. A long-term follow-up study of patients with post-poliomyelitis neuromuscular symptoms. *N Engl J Med*. Apr 10 1986;314(15):959-63. doi:10.1056/NEJM198604103141505
68. Olkin R. What Psychotherapists Should Know About Disability. Microtraining Associates; 2006.
69. Halstead LS, Rossi CD. New problems in old polio patients: results of a survey of 539 polio survivors. *Orthopedics*. Jul 1985;8(7):845-50. doi:10.3928/0147-7447-19850701-06
70. M. W. A survey of post-poliomyelitis sequelea: Manifestations, effects on people's lives and responses to treatment. *Aust J Physio*. 1991;37(2):89-102.
71. Howard RS, Wiles CM, Spencer GT. The late sequelae of poliomyelitis. *Q J Med*. Mar 1988;66(251):219-32.
72. Dean E, Ross J, Road JD, Courtenay L, Madill KJ. Pulmonary function in individuals with a history of poliomyelitis. *Chest*. Jul 1991;100(1):118-23. doi:10.1378/chest.100.1.118
73. Knobil K, Becker FS, Harper P, Graf LB, Wolf GT, Martinez FJ. Dyspnea in a patient years after severe poliomyelitis. The role of cardiopulmonary exercise testing. *Chest*. Mar 1994;105(3):777-81. doi:10.1378/chest.105.3.777
74. Han SJ, Lim JY, Suh JH. Obesity and Pulmonary Function in Polio Survivors. *Ann Rehabil Med*. Dec 2015;39(6):888-96. doi:10.5535/arm.2015.39.6.888
75. Chang KH, Lai CH, Chen SC, Hsiao WT, Liou TH, Lee CM. Body composition assessment in Taiwanese individuals with poliomyelitis. *Arch Phys Med Rehabil*. Jul 2011;92(7):1092-7. doi:10.1016/j.apmr.2011.01.019

76. Seo KH, Lee JH, Lee SY, Lee JY, Lim JY. Prevalence and Effect of Obesity on Mobility According to Different Criteria in Polio Survivors. *Am J Phys Med Rehabil*. Mar 1 2021;100(3):250-258. doi:10.1097/PHM.0000000000001556
77. Bruno RL, Johnson JC, Berman WS. Vasomotor abnormalities as post-polio sequelae: functional and clinical implications. *Orthopedics*. Jul 1985;8(7):865-9. doi:10.3928/0147-7447-19850701-09
78. Borg J WJ. Respiratory management in late post-polio. In: Halstead LS GG, ed. *Post Polio Syndrome*. Hanley and Belfus Inc; 1995:113-124.
79. Farbu E RT, Gilhus NE. Post-polio syndrome and total health status in a prospective hospital study. *Eur J Neurol*. Jul 2003;10(4):407-13. doi:10.1046/j.1468-1331.2003.00613.x
80. Stanghelle JK, Festvag L, Aksnes AK. Pulmonary function and symptom-limited exercise stress testing in subjects with late sequelae of poliomyelitis. *Scand J Rehabil Med*. Sep 1993;25(3):125-9.
81. Ward NS, Hill NS. Pulmonary function testing in neuromuscular disease. *Clin Chest Med*. Dec 2001;22(4):769-81. doi:10.1016/s0272-5231(05)70065-4
82. International P-pH. *Handbook on the late effects of poliomyelitis for physicians and polio survivors: Pulmonary function tests*. Post-polio Health International; 2012.
83. Shoseyov D, Cohen-Kaufman T, Schwartz I, Portnoy S. Comparison of activity and fatigue of the respiratory muscles and pulmonary characteristics between post-polio patients and controls: A pilot study. *PLoS One*. 2017;12(7):e0182036. doi:10.1371/journal.pone.0182036
84. H C. Living and Breathing with Weak Respiratory Muscles. Post Polio Network (NSW) Network News Post Polio Network (NSW); 2004. p. 5-8.
85. I N. Sleep Apnoea Revisted. Post Polio Network (NSW) Network News Post Polio Network (NSW); 2004. p. 9-11.
86. Roundtable on Late Effects of Polio/Post-Polio Syndrome Report of the House Standing Committee on Health and Ageing Committee. (Australian Government) (2012).
87. Farbu E, Gilhus NE, Barnes MP, et al. EFNS guideline on diagnosis and management of post-polio syndrome. Report of an EFNS task force. *Eur J Neurol*. Aug 2006;13(8):795-801. doi:10.1111/j.1468-1331.2006.01385.x
88. Bach JR, Alba AS. Pulmonary dysfunction and sleep disordered breathing as post-polio sequelae: evaluation and management. *Orthopedics*. Dec 1991;14(12):1329-37. doi:10.3928/0147-7447-19911201-07
89. Leotard A, Levy J, Hartley S, et al. Sleep disorders in aging polio survivors: A systematic review. *Ann Phys Rehabil Med*. Nov 2020;63(6):543-553. doi:10.1016/j.rehab.2019.10.007
90. Central Sleep Apnoea. Sleep Health Foundation; 2011.
91. Bruno RL, Frick NM. The psychology of polio as prelude to post-polio sequelae: behavior modification and psychotherapy. *Orthopedics*. Nov 1991;14(11):1185-93. doi:10.3928/0147-7447-19911101-06
92. Silva TM, Moreira GA, Quadros AA, Pradella-Hallinan M, Tufik S, Oliveira AS. Analysis of sleep characteristics in post-polio syndrome patients. *Arq Neuropsiquiatr*. Aug 2010;68(4):535-40. doi:10.1590/s0004-282x2010000400011
93. Araujo MA, Silva TM, Moreira GA, Pradella-Hallinan M, Tufik S, Oliveira AS. Sleep disorders frequency in post-polio syndrome patients caused by periodic limb movements. *Arq Neuropsiquiatr*. Feb 2010;68(1):35-8. doi:10.1590/s0004-282x2010000100008
94. Bruno RL. Abnormal movements in sleep as a post-polio sequelae. *Am J Phys Med Rehabil*. Jul-Aug 1998;77(4):339-43. doi:10.1097/00002060-199807000-00015
95. Soderholm S, Lehtinen A, Valtonen K, Ylinen A. Dysphagia and dysphonia among persons with post-polio syndrome - a challenge in neurorehabilitation. *Acta Neurol Scand*. Nov 2010;122(5):343-9. doi:10.1111/j.1600-0404.2009.01315.x
96. Silbergleit AK, Waring WP, Sullivan MJ, Maynard FM. Evaluation, treatment, and follow-up results of post polio patients with dysphagia. *Otolaryngol Head Neck Surg*. Mar 1991;104(3):333-8. doi:10.1177/019459989110400308
97. F L. Late onset polio sequelae in Denmark. Results of a nationwide survey of 3,607 polio survivors. *Scand J Rehabil Med Suppl*. 1993;28:1-32.
98. Sonies BC. Dysphagia and post-polio syndrome: past, present, and future. *Semin Neurol*. Dec 1996;16(4):365-70. doi:10.1055/s-2008-1040995
99. Sonies BC, Dalakas MC. Dysphagia in patients with the post-polio syndrome. *N Engl J Med*. Apr 25 1991;324(17):1162-7. doi:10.1056/NEJM199104253241703
100. Sonies BC, Dalakas MC. Progression of oral-motor and swallowing symptoms in the post-polio syndrome. *Ann N Y Acad Sci*. May 25 1995;753:87-95. doi:10.1111/j.1749-6632.1995.tb27534.x
101. Biase NGD MB, Leb MDA, Korn GP, Padovani M, Moraes M, Madazio G. Pharyngolaryngeal manifestations of post-polio syndrome. *Int Arch Otorhinolaryngol* 2011;15(4):529-533.
102. Ference T CJ. Dysphonia as the initial presenting symptom in post-polio syndrome: a case report. *Research on Chronic Diseases*. 2017;1(1)
103. Oluwasanmi OJ, McKenzie DA, Adewole IO, et al. Postpolio Syndrome: A Review of Lived Experiences of Patients. *Int J Appl Basic Med Res*. Jul-Sep 2019;9(3):129-134. doi:10.4103/ijabmr.IJABMR_333_18
104. Trojan DA, Finch L. Management of post-polio syndrome. *NeuroRehabilitation*. 1997;8(2):93-105. doi:10.3233/NRE-1997-8204
105. International P-pH. *Handbook on the late effects of poliomyelitis for physicians and polio survivors: Swallowing*. Post-polio Health International; 2012.
106. J S. *Post-Polio Syndrome: A guide for polio survivors and their families*. Yale University Press; 2001.
107. MK E. *What having had polio causes, might cause and does not cause*. vol 28. Post Polio Health; 2012.
108. Winberg C, Carlsson G, Brogardh C, Lexell J. The Perception of Physical Activity in Ambulatory Persons with Late Effects of Polio: A Qualitative Study. *J Aging Phys Act*. Jan 2017;25(1):65-72. doi:10.1123/japa.2015-0282
109. Florez-Duquet M, McDonald RB. Cold-induced thermoregulation and biological aging. *Physiol Rev*. Apr 1998;78(2):339-58. doi:10.1152/physrev.1998.78.2.339
110. FM M. Slow Healing. Medical Help. 2. Post-Polio Health International; 2013;29(Spring):1. Accessed 23-06-2022.
111. Dietvorst TF, Eulberg MK. Self-regulation treatment of post-polio cold limb. *Biofeedback Self Regul*. Jun 1986;11(2):157-61. doi:10.1007/BF00999983
112. L K. Bladder Inconveniences. presented at: Living with polio in the 21st century at RWSIR; 2009; Warm Springs, GA.
113. Kay L, Bertelsen M. Bladder symptoms among polio survivors. *J Rehabil Med*. Jun 2013;45(6):560-4. doi:10.2340/16501977-1153
114. Fizzotti G BM, Giambelluca E, Nardone A. Pelvic Floor Dysfunction and Poliomyelitis: A Case Report with Neurophysiological Evidence. *Med Case Rep*. 2017;4(S1:006)

115. D'Alfonso A TC, Serva A, Galica V, Bruno M, Tabacco S, Guido M Botulinum toxin in incontinent women with neurogenic bladder after failure with anticholinergic therapy, long-term results: a pilot study. *Italian JOG*. 2022;34(2)
116. Victoria PS. *Surgery and Anaesthesia: A guide for people with a history of surgery*. St Vincent's Hospital; 2022:12.
117. Lambert DA, Giannouli E, Schmidt BJ. Postpolio syndrome and anesthesia. *Anesthesiology*. Sep 2005;103(3):638-44. doi:10.1097/00000542-200509000-00029
118. Changchien CH, Chen WC, Su YM. Free anterolateral thigh flap harvesting from paralytic limbs in post-polio syndrome. *Case Reports Plast Surg Hand Surg*. 2016;3(1):50-5. doi:10.1080/23320885.2016.1200978
119. RL B. *Preventing complications in polio survivors undergoing dental procedures*. vol 6. Post-Polio Sequelea Monograph Series. 1996:1-8.
120. Khosla S, Burr D, Cauley J, et al. Bisphosphonate-associated osteonecrosis of the jaw: report of a task force of the American Society for Bone and Mineral Research. *J Bone Miner Res*. Oct 2007;22(10):1479-91. doi:10.1359/jbmr.0707onj
121. Brogardh C, Lexell J, Hammarlund CS. Experiences of falls and strategies to manage the consequences of falls in persons with late effects of polio: A qualitative study. *J Rehabil Med*. Aug 31 2017;49(8):652-658. doi:10.2340/16501977-2262
122. Gillespie LD, Robertson MC, Gillespie WJ, et al. Interventions for preventing falls in older people living in the community. *Cochrane Database Syst Rev*. Sep 12 2012;(9):CD007146. doi:10.1002/14651858.CD007146.pub3
123. Silver JK AD. Polio survivors: falls and subsequent injuries. *Am J Phys Med Rehabil*. Aug 2002;81(8):567-70. doi:10.1097/00002060-200208000-00002
124. Mohammad AF, Khan KA, Galvin L, Hardiman O, O'Connell PG. High incidence of osteoporosis and fractures in an aging post-polio population. *Eur Neurol*. 2009;62(6):369-74. doi:10.1159/000242444
125. Bickerstaffe A, Beelen A, Nollet F. Circumstances and consequences of falls in polio survivors. *J Rehabil Med*. Nov 2010;42(10):908-15. doi:10.2340/16501977-0620
126. Imoto D, Sawada K, Horii M, et al. Factors associated with falls in Japanese polio survivors. *Disabil Rehabil*. Jun 2020;42(13):1814-1818. doi:10.1080/09638288.2018.1537381
127. Ofran Y, Schwartz I, Shabat S, Seyres M, Karniel N, Portnoy S. Falls in Post-Polio Patients: Prevalence and Risk Factors. *Biology (Basel)*. Oct 28 2021;10(11)doi:10.3390/biology10111110
128. Brogardh C, Flansbjer UB, Lexell J. Determinants of Falls and Fear of Falling in Ambulatory Persons With Late Effects of Polio. *PM R*. May 2017;9(5):455-463. doi:10.1016/j.pmrj.2016.08.006
129. Da Silva CPPDN, Zuckerman BD, Olkin RP. Relationship of depression and medications on incidence of falls among people with late effects of polio. *Physiother Theory Pract*. May 2017;33(5):370-375. doi:10.1080/09593985.2017.1307889
130. Nam KY, Lee S, Yang EJ, et al. Falls in Korean Polio Survivors: Incidence, Consequences, and Risk Factors. *J Korean Med Sci*. Feb 2016;31(2):301-9. doi:10.3346/jkms.2016.31.2.301
131. Haziza M, Kremer R, Benedetti A, Trojan DA. Osteoporosis in a postpolio clinic population. *Arch Phys Med Rehabil*. Aug 2007;88(8):1030-5. doi:10.1016/j.apmr.2007.05.010
132. Lo JK, Robinson LR. Postpolio syndrome and the late effects of poliomyelitis. Part 1. pathogenesis, biomechanical considerations, diagnosis, and investigations. *Muscle Nerve*. Dec 2018;58(6):751-759. doi:10.1002/mus.26168
133. Sherrington C, Whitney JC, Lord SR, Herbert RD, Cumming RG, Close JC. Effective exercise for the prevention of falls: a systematic review and meta-analysis. *J Am Geriatr Soc*. Dec 2008;56(12):2234-43. doi:10.1111/j.1532-5415.2008.02014.x
134. Hammarlund CS, Lexell J, Brogardh C. Self-reported impairments among people with late effects of polio: a mixed-methods study. *J Rehabil Med*. Jul 31 2020;52(7):jrm00084. doi:10.2340/16501977-2706
135. Yelnik A, Laffont I. The psychological aspects of polio survivors through their life experience. *Ann Phys Rehabil Med*. Feb 2010;53(1):60-7. doi:10.1016/j.rehab.2009.11.006
136. Oorschot S, Brehm MA, van Groenestijn AC, et al. Efficacy of a physical activity programme combining individualized aerobic exercise and coaching to improve physical fitness in neuromuscular diseases (I'M FINE): study protocol of a randomized controlled trial. *BMC Neurol*. May 13 2020;20(1):184. doi:10.1186/s12883-020-01725-0
137. Davidson AC, Auyeung V, Luff R, Holland M, Hodgkiss A, Weinman J. Prolonged benefit in post-polio syndrome from comprehensive rehabilitation: a pilot study. *Disabil Rehabil*. 2009;31(4):309-17. doi:10.1080/09638280801973206
138. Koopman FS, Beelen A, Gilhus NE, de Visser M, Nollet F. Treatment for postpolio syndrome. *Cochrane Database Syst Rev*. May 18 2015;(5):CD007818. doi:10.1002/14651858.CD007818.pub3
139. Fillyaw MJ, Badger GJ, Goodwin GD, Bradley WG, Fries TJ, Shukla A. The effects of long-term non-fatiguing resistance exercise in subjects with post-polio syndrome. *Orthopedics*. Nov 1991;14(11):1253-6. doi:10.3928/0147-7447-19911101-14
140. Rodriquez AA, Agre JC, Franke TM. Electromyographic and neuromuscular variables in unstable postpolio subjects, stable postpolio subjects, and control subjects. *Arch Phys Med Rehabil*. Sep 1997;78(9):986-91. doi:10.1016/s0003-9993(97)90062-9
141. Voorn EL, Gerrits KH, Koopman FS, Nollet F, Beelen A. Determining the anaerobic threshold in postpolio syndrome: comparison with current guidelines for training intensity prescription. *Arch Phys Med Rehabil*. May 2014;95(5):935-40. doi:10.1016/j.apmr.2014.01.015
142. Koopman FS, Uegaki K, Gilhus NE, Beelen A, de Visser M, Nollet F. Treatment for postpolio syndrome. *Cochrane Database Syst Rev*. Feb 16 2011;(2):CD007818. doi:10.1002/14651858.CD007818.pub2
143. S P. Use of Medication in People with Post-Polio Syndrome. *Polio Network News*. 1. International Polio Network. Saint Louis, Missouri USA; 1999;15(Winter 1999):1-3. Accessed 24-06-2022.
144. Chang MC, Park JS, Hwang JM, Park D. Effectiveness of Intravenous Immunoglobulin for Management of Pain in Patients with Postpolio Syndrome. *Pain Res Manag*. 2021;2021:6637705. doi:10.1155/2021/6637705
145. Sjodahl Hammarlund C, Lexell J, Brogardh C. Perceived consequences of ageing with late effects of polio and strategies for managing daily life: a qualitative study. *BMC Geriatr*. Aug 9 2017;17(1):179. doi:10.1186/s12877-017-0563-8
146. Nielsen NM, Rostgaard K, Hjalgrim H, Askgaard D, Skinhoj P, Aaby P. Psychiatric hospitalizations in a cohort of Danish polio patients. *Am J Epidemiol*. Feb 1 2007;165(3):319-24. doi:10.1093/aje/kwk003
147. Olkin R. *What psychotherapists should know about disability*. Guilford Press; 1999:xvi, 368 pages.

148. Machell ST. Psychiatric Approaches and Outcomes. *Phys Med Rehabil Clin N Am*. Aug 2021;32(3):557-568. doi:10.1016/j.pmr.2021.03.002
149. M J. Lockdown Impact On Polio Survivors. September 2021 ed. Polio Oz News: Polio Australia Inc.; 2021.
150. M W. Living with the late effects of disability: A five-year follow-up survey of coping among post-polio survivors. *Australian Occupational Therapy Journal*. 1996;43:60-71.
151. Tersteeg I SJ, Beelen A, Nollet F Impact of comorbidity, aging and lifestyle-related factors in polio survivors. presented at: 1st European Conference on Post Polio Syndrome; 2011; Copenhagen.
152. McNalley TE, Yorkston KM, Jensen MP, et al. Review of secondary health conditions in postpolio syndrome: prevalence and effects of aging. *Am J Phys Med Rehabil*. Feb 2015;94(2):139-45. doi:10.1097/PHM.0000000000000166
153. Bach JR, Tilton M. Pulmonary dysfunction and its management in post-polio patients. *NeuroRehabilitation*. 1997;8(2):139-53. doi:10.3233/NRE-1997-8207
154. Bertolasi L, Danese A, Monaco S, Turri M, Borg K, Werhagen L. Polio Patients in Northern Italy, a 50 Year Follow-up. *Open Neurol J*. 2016;10:77-82. doi:10.2174/1874205X01610010077
155. Lund ML, Lexell J. Relationship between participation in life situations and life satisfaction in persons with late effects of polio. *Disabil Rehabil*. 2009;31(19):1592-7. doi:10.1080/09638280802638881
156. Harrison TC, Stuifbergen AK. Life purpose: effect on functional decline and quality of life in polio survivors. *Rehabil Nurs*. Jul-Aug 2006;31(4):149-54. doi:10.1002/j.2048-7940.2006.tb00379.x
157. Shiri S, Wexler ID, Feintuch U, Meiner Z, Schwartz I. Post-polio syndrome: impact of hope on quality of life. *Disabil Rehabil*. 2012;34(10):824-30. doi:10.3109/09638288.2011.623755

20. List of Abbreviations

BIPAP	Bi-Level Positive Airway Pressure
CSA	Central Sleep Apnoea
EMG	Electromyography
FITT-VP	Frequency Intensity Time Type – Volume Progression
IGF-1	Insulin-like growth factor 1
IPPV	Intermittent Positive Pressure Ventilation
IVIG	Intravenous Immunoglobulin
LEoP	Late Effects of Polio
NHRMC	National Health and Medical Research Council
OSA	Obstructive Sleep Apnoea
PPS	Post-Polio Syndrome

21. Credits

Commissioned by:

Polio Australia Incorporated

Written by:

Members of the GlaxoSmithKline Australia Vaccines Medical Department as part of a volunteering program: Iris Depaz, Claire Borg, David Crump, Christopher Clarke, Pooi Eu Nham, Chris Vearing, Gabrielle Formosa, Helen Ho and Jutta Richter

Dr Margaret Peel, Committee of Management Member (Qld), Polio Australia

Dr Mary Westbrook, Clinical Advisory Group Member, Polio Australia

First Edition (2012) Reviewed by:

Polio Australia's Clinical Advisory Group Members: Ann Duncan (Respiratory Nurse), Gnanalechumy Jegasothy (Physiotherapist), Natasha Layton (Occupational Therapist), and Dr Mary Westbrook (Psychologist)

Blaise Doran, Physiotherapist and Coordinator, Polio Services Victoria, St Vincent's Hospital, Melbourne

Tessa Jupp, CEO/Clinic Nurse, Post Polio Network of WA

Mary-ann Liethof, National Program Manager, Polio Australia

Elizabeth Telford, President, Post Polio Victoria

Gillian Thomas, President, Polio Australia

Second Edition Reviewed by:

Michael Jackson, Clinical Health Educator, Polio Australia

Mary-ann Liethof, Board Member, Polio Australia

Gillian Thomas, President, Polio Australia

Dr Timothy Lathlean, Post-Doctoral Research Fellow, South Australian Health and Medical Research Institute

About Polio Australia

Polio Australia is a community based, not-for-profit, national peak consumer body committed to standardising quality polio information and service provision across Australia for polio survivors.

Polio Australia's vision is that all polio survivors in Australia have access to appropriate health care and the support required to maintain independence and make informed lifestyle choices.

For more information: www.polioaustralia.org.au and www.poliohealth.org.au

ISBN 978-0-9874392-2-2