■ REVIEW ARTICLE

Postpolio Syndrome and Anesthesia

David A. Lambert, M.D.,* Eleni Giannouli, M.D.,† Brian J. Schmidt, M.D.‡

The development of polio vaccines 50 yr ago essentially halted childhood polio epidemics in the industrialized world. During the past quarter century, a constellation of delayed neuromuscular symptoms, called *postpolio syndrome*, became recognized among the aging polio survivors. The prevalence of postpolio syndrome in the U.S. population is estimated to be in the hundreds of thousands. The most common symptoms are fatigue, pain, and new onset weakness thought to be related to delayed deterioration of motor neuron function. When a patient with postpolio syndrome presents for surgery, special precautions are warranted, because these patients may have respiratory impairment, sleep apnea, swallowing difficulties, and cold intolerance. This article first reviews clinical features and some pathoetiologic theories of postpolio syndrome and then focuses on anesthetic considerations including the use of common anesthetics, neuromuscular blockade, regional anesthesia, and general anesthetic management strategies.

POLIO survivors of the past century's epidemics are now entering their fifth to seventh decades of life. Some survivors have developed a constellation of signs and symptoms referred to as *postpolio syndrome* (PPS). With advancing age, patients with PPS are now presenting for surgical procedures, both elective and urgent. Anesthesiologists will be better prepared to provide safe care for these patients if equipped with a solid understanding of PPS. The following review summarizes the history of polio and PPS and suggests special anesthetic considerations when approaching patients with PPS. Patients with PPS may display altered respiratory function, chronic pain syndromes, cold intolerance, risk of aspiration, and altered sensitivity to anesthetic agents (induction agents, inhaled anesthetics, neuromuscular agents, and opioids).

* Anesthesiology Resident, Department of Anesthesia, † Assistant Professor, Section of Respirology and Critical Care, Department of Internal Medicine, ‡ Professor, Section of Neurology, Department of Internal Medicine, Faculty of Medicine, University of Manitoba.

Address reprint requests to Dr. Schmidt: Section of Neurology, Department of Internal Medicine, University of Manitoba, Room F-543, Health Sciences Centre, 820 Sherbrook Street, Winnipeg, Manitoba, Canada R3A 1R9. Address electronic mail to: brian@scrc.umanitoba.ca. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

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Poliomyelitis: Acute Illness and Recovery

Although cases of poliomyelitis had been described as early as 1600 BC, it was the 20th century that witnessed regular childhood polio epidemics.[1] In North America, these epidemics peaked in 1952–1953, with more than 57,000 reported new cases in the United States[2] and 17,000 new cases in Canada.[3] Thanks to the introduction of the Salk injectable vaccine in 1955 and the Sabin oral polio vaccine in 1961, these epidemics were essentially brought to a halt.

Currently, new cases of polio are mostly restricted to Africa, South East Asia, and the Middle East. One goal of the World Health Organization is the worldwide eradication of the polio virus by 2005. The few acute poliomyelitis cases occurring in the Western world today are associated with the low risk of transmission (1 in 2.5 million) of the disease from the oral polio vaccine itself, usually to an immunocompromised host.[2]§

Poliomyelitis results from infection by one of three subtypes of this single-stranded RNA enterovirus. It is transmitted by fecal–oral spread and is extremely infectious. Virus is replicated in the gut and lymphoid tissue. Most (95%) infected individuals have no symptoms or may report mild flu-like symptoms. In the presence of viremia, the central nervous system is susceptible to invasion through an unknown mechanism. Symptoms become more pronounced as fever and meningismus develop. These individuals may later demonstrate neurologic symptoms and signs of acute poliomyelitis infection, asymmetric flaccid paralysis in particular. The overall risk of paralytic polio in infected persons is 1–2%.

The polio virus ultimately causes destruction of anterior horn motor neurons, resulting in limb paralysis (figs. 1A and B). However, Bodian[4] and others in the late 1940s showed that, histopathologically, all cases had some "encephalitic" changes in addition to the typical anterior horn cell destruction. The centers most severely affected were in the brainstem and cerebellum and include the reticular formation, vestibular nuclei, and roof nuclei of the cerebellum. In his series, Bodian reported that "of twenty four human autopsies there was hardly an individual who did not have lesions, sometimes of a fairly severe degree, of most of the motor nuclei of the cranial nerves as well as in the surrounding reticular formation." He also commented that these findings were
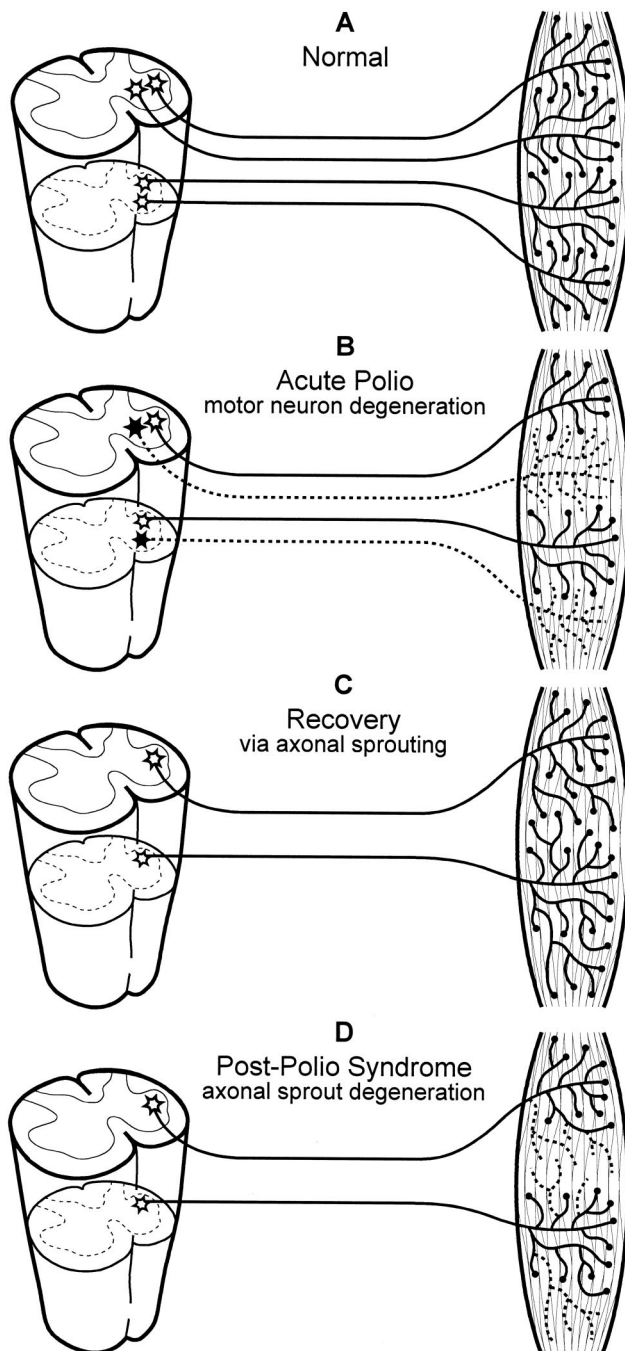
Fig. 1. Postulated mechanism of postpolio syndrome. (*A*) Under normal conditions, a healthy lower motor unit is composed of a motor neuron cell body (located in the anterior horn of the spinal cord gray matter), the motor axon, and muscle cells (fibers) innervated by the axon. (*B*) The polio virus infects some motor neuron cell bodies, which subsequently die, while others survive (or were never infected). The loss of lower motor units results in muscle fiber denervation and the weakness that occurs as a result of acute poliomyelitis. (*C*) Motor axon terminal sprouting reinnervates previously denervated muscle fibers, creating a "giant" motor unit. This is associated with improvement in strength in the weeks and months after an acute attack of polio. (*D*) However, after many years, abnormally enlarged motor units are no longer able to maintain the extensive sprout pattern. Sprouts start to degenerate, producing new denervation and muscle weakness (postpolio syndrome).

found in individuals with acute poliomyelitis who died from other causes (*e.g.*, appendicitis).

The clinical manifestations of poliomyelitis are variable. Patients may report weakness in only one limb or may have rapid progression of complete paralysis and loss of respiratory function due to weakness.[4] Paralysis is more often found in the legs than the arms.[5] Children are less likely to have paralysis (1 in 1,000) with acute infection than adults (1 in 75).[6] Brainstem symptoms (bulbar poliomyelitis) occur in 10–15% of patients, manifesting as involvement of any of the cranial nerve nerves; facial weakness, swallowing, and phonation difficulties are noted in particular.[7] Reticular formation involvement produces difficulty in swallowing, impaired respiratory control, and cardiovascular instability.[8] Occasionally, patients have cerebellar ataxia or, in the preparalytic stage, become agitated, obtunded, or display upper motor neuron signs.[8]

Recovery begins after 2–3 weeks and ranges from complete resolution to major residual dysfunction (*e.g.*, permanent respiratory difficulties, paralysis). Younger patients who have paralytic poliomyelitis have better recovery than older patients. Recovery is said to plateau at approximately 7–10 months.[9] Treatment is mainly supportive, ranging from ventilatory assistance to splints and crutches.[10] Three factors contribute to recovery: (1) number of recovered motor units that resume function, (2) number of motor units that develop "sprouts" to reinnervate "orphaned" muscle fibers (becoming giant motor units fig. 1C), and (3) muscle hypertrophy.[6]

Postpolio Syndrome

As early as 1875, Charcot and others reported the recurrence of muscle weakness years after acute infection of poliomyelitis.[6,10] It was, however, the large cohort of polio patients affected during the epidemics of the past century that really brought attention to this phenomenon. Polio survivors began to report new weakness in the 1970s.[10] Dalakas *et al.*[11] labeled the late development of progressive weakness and atrophy *postpoliomyelitis progressive muscular atrophy* in 1986. Various authors later began to use the term *postpolio syndrome*, a term originally developed by patients themselves, to include other symptoms in polio survivors.[12,13]

In 1987, it was estimated that there were 1.6 million survivors of poliomyelitis in the United States,[14] of which 640,000 had symptoms of PPS.[15] The prevalence of PPS based on a population of 250 million, at that time, would be approximately 1 in 390 persons. By comparison, the prevalence of multiple sclerosis in 1990 was 1 in 1,000.[16] However, the actual current prevalence of PPS is unknown because more recent statistics on the prevalence of PPS are unavailable. These estimates may be conservative. Given social fear and stigma surrounding

the disease during the era of epidemics, reporting of mild to moderate cases may have been suboptimal.

Criteria and Nomenclature

Postpolio syndrome symptoms include weakness, fatigue (generalized and muscular), atrophy, and pain. Because these are nonspecific symptoms, diagnostic criteria for PPS, based on those originally described by Mulder, have been set[6,10,17]:‖

- history of paralytic poliomyelitis with residual motor neuron loss (confirmed by history, neurologic examination, and if needed, an electrodiagnostic examination);
- a period of neurologic recovery followed by an interval (usually 15 yr or more) of neurologic and functional stability;
- a gradual or abrupt onset of new weakness or abnormal muscle fatigue (decreased endurance), muscle atrophy, or generalized fatigue; and
- exclusion of medical, orthopedic, and neurologic conditions that may cause the above symptoms.

Nomenclature in PPS can sometimes be confusing. Some authors define *postpolio muscular atrophy* or *postpolio sequelae* as conditions distinct from PPS.[6,10] The former terms are used to describe specific signs or symptoms related to previous polio infection, whereas the label *PPS* is used only when all criteria are met. These distinctions are of little practical importance in the assessment of a poliomyelitis survivor presenting for preanesthetic evaluation. The terms do, however, highlight the nonspecific nature of symptoms in PPS, and the difficulties that can arise in making clear diagnoses. The remainder of this article will use the term *PPS* only.

Pathogenesis

There has been considerable debate over the underlying cause of PPS. The most commonly accepted explanation for the late effects of polio is that of "overuse or premature aging of polio-affected motor units."[6,10] The so-called giant motor units that develop on recovery are presumed to be unable to sustain the increased metabolic demands, DNA/RNA repair, or protein synthesis.[18,19] As such, the sprouts begin to fall off, and motor unit function deteriorates (fig. 1D). Electrophysiologic and muscle biopsy data support this theory.[20] They suggest disintegration of function of the motor units and the terminal sprouts themselves 30–40 yr after the acute poliomyelitis infection. There have been no conclusive studies to prove this, however. Other explanations include musculoskeletal disuse, normal age-related loss of the residual motor units, and vascular or glial changes

predisposing polio survivors to premature motor neuron degeneration.[18] Persistent "low-grade" poliovirus infection or reactivation has been proposed by a few studies using histochemical, polymerase chain reaction, and probe techniques to try to isolate virus, genetic material, or humoral evidence in the cerebral spinal fluid of PPS patients.[6,10,18] However, there are at least as many studies providing evidence against the persistent virus theory.[6,21,22] The natural age-related decline in growth hormone concentrations, thereby no longer helping to support cellular maintenance of giant motor neurons, has also been implicated as a contributor to PPS.[23]

Symptoms

The most common symptoms reported by PPS patients include fatigue and weakness, joint and muscle pain, respiratory difficulties, cold intolerance, and dysphagia. These will now be addressed individually, with focus on areas of concern and interest to anesthesia.

Fatigue and Weakness. Fatigue is the most commonly reported symptom in PPS.[6,10,24] This includes central fatigue (somnolence, difficulty concentrating) and peripheral fatigue (muscular weakness).

Central fatigue is a nonspecific symptom given the numerous potential causes, such as sleep apnea or depression, in addition to PPS. Because previous reports described extensive lesions in the reticular activating system in acute poliomyelitis patients, Bruno *et al.*[25] performed *in vivo* magnetic resonance imaging of 22 subjects with PPS who had no other conditions, medications, or treatment that might contribute to fatigue. These investigators found hyperintense T1- and T2-weighted signals in the reticular activating system of 8 subjects (55%) in the high-fatigue group and in none in the low-fatigue group.

Peripheral (muscular) fatigue has also been examined using functional studies, electromyography, and muscle biopsies.[10] All of these methods suggest that there is constant remodeling of the giant motor units, with sprouts "dropping off" and reinnervating.[26,27] The most notable symptom of PPS is progressive muscular weakness. The progression is slow and may occur in muscles previously affected by polio or, less commonly, in muscles previously assumed to be unaffected by the original polio attack.[28] The extent of new weakness seems to correlate with the severity of the acute polio infection and with the amount of recovery, *i.e.*, individuals with greater recovery seem to have a greater chance of developing new weakness.[29]

Treatment of fatigue, both central and peripheral, primarily involves lifestyle changes.[15,30] These include regular rest scheduling as well as specifically tailored exercise programs, depending on the current functional level of the patient. Several investigators have examined the role of anticholinesterase medication in the treatment of fatigue.[31-33] Oral and intravenous preparations have

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‖ From Jubelt and Drucker,[10] based on a consensus of the Post Polio Task Force. Adapted with permission.

been used. Some of these studies showed improvement of electrodiagnostic features[31]; however, randomized, controlled trials have demonstrated no clinical benefit in PPS patients.[6,10,32,33]

Pain. Pain is almost as common as fatigue in PPS patients.[6,10,24,34] Therefore, anesthesiologists sometimes see PPS patients referred to chronic pain clinics. Rehabilitation medicine specialists have proposed three types of pain in PPS patients.[6] Type I pain is postpolio muscle pain. It is an aching, deep or superficial muscle pain described as similar to the pain experienced during the acute polio infection. It can be precipitated by strenuous activity, stress, or cold temperatures. Type II pain is part of an "overuse" syndrome, which includes bursitis, tendonitis, myofascial, and soft tissue injuries, secondary to poor biomechanics or posture. Type III pain includes degenerative joint disease, low back pain, and nerve compression syndromes. It is the result of chronic overuse, unequal loading of joints, and asymmetric muscle function secondary to weakness. For example, wrist and shoulder pain may develop in some patients secondary to long-standing use of crutches.[35]

Treatment of pain in PPS is similar to the management of chronic pain in general. Lifestyle modifications, physiotherapy, assist devices, analgesics, and joint or trigger point injections are the most commonly used options. A review of the treatment of muscle pain by Cohen *et al.*[36] is available in a recent issue of this journal.

Respiratory Dysfunction. One of the hallmarks of the treatment of acute poliomyelitis was the negative-pressure ventilator, or iron lung. Respiratory failure secondary to polio, when present, was the major cause of morbidity and mortality. Respiratory symptoms occur in up to 40% of PPS patients.[37] Symptoms range from mildly decreased pulmonary function to frank respiratory failure and the need for assisted ventilation. Contributing to these symptoms are restrictive chest wall changes (scoliosis, kyphosis), altered chest wall strength (decreased maximum inspiratory/expiratory pressures), recurrent infections, and sleep-related disordered breathing (SRDB).

Anesthesiologists are familiar with caring for patients with respiratory problems. However, patients with SRDB, including obstructive and central sleep apnea as well as hypoventilation syndromes, can be particularly challenging. Theoretically, PPS patients are at higher risk of SRDB because of previous damage to the reticular activating system, decreased strength and tone in upper airway musculature, and increased rates of obesity due to decreased mobility. Whether PPS patients have an increased incidence of SRDB relative to healthy individuals is not clear. However, in one study of 155 postpolio patients by Fischer,[38] the symptoms of frequent waking, snoring, and daytime fatigue were reported at least five times more often than in healthy controls. Daytime sleepiness, tiredness, morning headache, and restless leg

symptoms were reported more often in PPS subjects than in healthy controls.[39] Another study retrospectively examined 35 subjects who fit the criteria for PPS and had symptoms of SRDB.[40] These investigators found three patterns of sleep disturbance: obstructive sleep apnea (n = 19), hypoventilation (n = 7), and a combination of both (n = 9). Interestingly, compared with non-PPS patients evaluated for obstructive sleep apnea by the same laboratory, PPS patients were of similar age (55 *vs.* 56 yr) but weighed less (176% *vs.* 144% ideal body weight).

Laryngeal function was examined in nine subjects with PPS.[41] Subjects who reported severe swallowing problems were the same individuals who had impaired voice and laryngeal function, based on videostroboscopic evaluation, acoustical analysis, and, in three patients, laryngeal electromyography. Half of these patients were found to have unilateral vocal chord paralysis. These same patients gave a history of bulbar symptoms with their initial polio illness.

Cold Intolerance. Cold intolerance is not uncommonly reported in PPS patients. One 5-yr follow-up study of 68 PPS patients found that 65% of patients reported symptoms of cold intolerance.[24] Whether this is the result of altered perfusion to limbs secondary to vascular changes in atrophied muscles or from changes in vasoconstrictor tone due to damaged sympathetic pathways is not clear.[42] Treatment is symptomatic.

Dysphagia. Dysphagia is reported by 10–20% of PPS patients.[43] Symptoms range from mild "sticking" of food in the esophagus to frequent choking and symptoms of reflux disease. In one study, ultrasound and videofluoroscopy were used to evaluate swallowing function of 32 PPS patients.[44] Fourteen had symptoms of dysphagia, and 12 had a history of bulbar involvement. Interestingly, swallowing abnormalities were revealed in 31 patients, regardless of the presence or absence of dysphagia. These studies suggest that PPS patients, even if asymptomatic, may be at increased risk of both overt and silent aspiration.[40,41,43,44]

Anesthesia and Postpolio Syndrome

Only four case reports discuss anesthesia and PPS.[45–48] Two of these describe complications. The first report describes a 79-yr-old patient with unanticipated ventilatory failure postoperatively, which, on investigation, was thought to be the result of undiagnosed PPS.[48] The second and most recent report is that of a 51-yr-old patient presenting for foot surgery related to her previous childhood polio illness.[46] The patient experienced acute cardiopulmonary arrest in her hospital room approximately 1 h postoperatively and did not recover from the resulting cerebral injury. The arrest was presumed to be the result of oversedation secondary to opioid administration in the presence of possible ob-

structive sleep apnea. The other reports describe PPS patients who underwent anesthesia without incident (spinal anesthesia in one patient and anesthesia for electroconvulsive therapy in the other patient).[45,47]

Preoperative Assessment

Preanesthetic evaluation of a PPS patient should begin with an assessment of the history of the patient's previous poliomyelitis illness. The patient's age at the time of illness, severity (including the presence or absence of bulbar symptoms), and amount of recovery are all helpful in anticipating the likelihood of developing PPS. Documenting the extent of residual deficits is important to understand the patient's baseline function. If the patient reports symptoms suggesting PPS, one should consider referral to a specialist with experience with PPS patients, such as a neurologist, if this has not already been done. In some cases, the surgical service may be unaware of the fact that their patient has PPS. Communication of this information should enhance overall patient management.

Often, chronic pain syndromes are present in these patients. Evaluation of contractures or spinal deformities is important to establish a baseline and anticipate positioning issues that might arise intraoperatively. Although patients may already be taking oral opioid medications, many are "opioid naive." Some patients may report excessive sedation with opioid or sedative hypnotic drugs, as prescribed for dental procedures, for example.

A detailed respiratory evaluation is very important in this patient population. Anesthesiologists may encounter PPS patients with no respiratory symptoms at all, or conversely, the PPS patient may have a mature tracheostomy site and may be dependent on overnight positive-pressure ventilation. Any symptoms suggestive of decreased respiratory reserve should be thoroughly evaluated with a baseline chest radiograph and spirometry. Vital capacity of less than 50% of the predicted value, or 1,500 ml, warrants complete pulmonary function testing, including maximum inspiratory/expiratory pressures. Special attention should be made not to overlook a history consistent with sleep apnea or hypoventilation syndrome. This includes symptoms of morning headache, excessive daytime somnolence, and episodes of snoring or apnea during sleep. A positive history should prompt arterial blood gas sampling and consideration of referral to a respirologist and a formal sleep study.[49] Patients with SRDB syndromes are at higher risk of cardiac dysfunction, including cor pulmonale and pulmonary hypertension.

Finally, preoperative evaluation should include an inquiry into symptoms of dysphagia and reflux disease.

Perioperative Considerations

An important consideration in the anesthetic management of patients with PPS is whether regional anesthesia is safe. Many anesthesiologists are hesitant to use regional anesthesia in patients with preexisting neuromuscular deficits, because of the concern of exacerbating existing disease or difficulty evaluating complications. There have been no reports of adverse effects due to regional anesthesia in PPS patients,[45] but this does not necessarily mean that regional techniques are without risk.

Animal studies have determined specific intrathecal concentrations of local anesthetics that are lethal for neurons.[50] It is not possible to know the number of healthy *versus* damaged motor neurons in an individual PPS patient. However, patients with PPS have fewer motor neurons than normal, at least some of which are likely to have residual dysfunction, or increased metabolic demand because they supply enlarged motor units (fig. 1C). These motor neurons may be more sensitive to drug effects. Therefore, theoretically, the toxic intrathecal concentration of a local anesthetic may be lower in PPS patients. However, to date, there are no direct experimental data to confirm or refute this concept. There is also no evidence as to whether there is an increased risk of adverse effects using peripheral nerve blocks or indwelling catheters in PPS patients.

Ultimately, the decision to use general or regional anesthesia should be made on an individual patient basis weighing the risks and benefits. If a spinal anesthetic is selected, a medication with a long history of safety, such as hyperbaric bupivacaine, should be used.

There are several considerations when administering general anesthesia to patients with PPS. Initially, it is important to ensure that the patient is comfortably positioned and that attention is given to limbs with contracture. Blankets or warming devices are particularly appropriate because of cold intolerance. Baseline twitch response to peripheral nerve stimulation should be measured before administering neuromuscular blockade, because this response might be abnormally small in some muscles. Traditionally in patients with neuromuscular disease, succinylcholine is used cautiously to avoid precipitating hyperkalemia. However, there are no specific data on the use of succinylcholine in patients with PPS. One study suggested that patients with a remote history of polio have increased sensitivity to nondepolarizing muscle blockers.[51] For this reason, selection of shorter-acting agents, such as rocuronium and mivacurium, along with careful titration of doses to desired effect, is important in patients with PPS. In some cases, completely avoiding neuromuscular blockade may be appropriate.

The fact that patients with PPS often have residual lesions involving the reticular activating system[4,25] may be of particular relevance to anesthesia. The reticular activating system is a theoretical site of action for most anesthetic agents, and patients with PPS may show altered sensitivity to anesthetic drugs. No data describing

the dose–response characteristics of induction agents in patients with PPS exists, nor is it known whether there are differences in minimum alveolar concentration when patients with PPS are compared with the normal population. Considering possible altered sensitivity to induction drugs, maintenance agents, muscle relaxants, and opioids, caution in the selection of dose of virtually any medication administered for general anesthesia should be used in this patient group.

Emergence from anesthesia should be preceded by ensuring complete reversal of neuromuscular blockade. The risk of aspiration is greater in at least some PPS patients. As such, selected patients may benefit from prophylactic antiemetic medication. Careful suctioning of the hypopharynx before emergence is essential. Vital capacity "big breaths" before extubation may help to recruit a maximal number of alveoli. Doses of opioids should initially be low and carefully titrated to effect, and long-acting medications should be used cautiously. Other coanalgesics, such as nonsteroidal antiinflammatory agents, should be used when possible.

Postoperative Management

As noted in the first paragraph of the Anesthesia and Postpolio Syndrome section, two reports have described postoperative complications, one resulting in death, in patients with PPS.[46,48] In these two cases, postoperative respiratory failure, associated with weakness, oversedation, or both, was deemed contributory. Therefore, the most serious anesthesia-related risk for PPS patients may be in the postoperative period. Just as it is now recognized that more intensive postoperative monitoring may be needed for patients with a history of obstructive sleep apnea, it would be similarly appropriate to increase one's vigilance during postoperative monitoring of patients with PPS. Ambulatory surgery in this population should be considered only in select patients. It would seem prudent to avoid "fast-tracking" the transfer from the operating room immediately to the ward in patients with PPS. Finally, coughing should be encouraged. Incentive spirometry and humidification of inspired gases should be considered for PPS patients in the recovery room.

Conclusion

Survivors of the poliomyelitis epidemics are now more than ever presenting for a variety of surgical procedures requiring anesthesia. Some of these survivors have developed PPS. In reviewing the pathology of acute poliomyelitis and PPS itself, multiple considerations for anesthesia become apparent. These include compromised respiratory function, SRDB issues, chronic pain syndromes, aspiration risks, and cold intolerance. In addition, postpolio patients may display altered sensitivity to

any of the medications commonly used for regional and general anesthesia. Once aware of these considerations, anesthesiologists are better prepared to provide safe care, not only to patients with PPS, but to any patient with a history of poliomyelitis.

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