Anesthetic Implications of Postpolio Syndrome: New Concerns for an Old Disease

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Poliomyelitis was pandemic in the United States and much of the world in the first half of the 20th century. The uses of polio vaccines have essentially eradicated the disease in the United States today. But poliovirus infection survivors who had experienced a paralytic attack can see a return of some symptoms, which is a syndrome called postpolio syndrome (PPS).

The anesthetist must preoperatively assess reported amounts of patient physical activity and patient age, which can indicate the amount of muscle degeneration that may have already occurred. Patients with PPS demonstrate altered respiratory function, cold intolerance, a risk for aspiration, and experience chronic pain in muscles and joints. Patients with PPS display an increased sensitivity to some anesthetic agents such as long-acting narcotics and potent inhaled anesthetic gases with a high blood-gas partition coefficient, along with report of increased fatigue, weakness, and somnolence after anesthesia. Anesthesia care must center on the preservation of muscle function postoperatively. The anesthetist should consider the use of short-acting anesthetic agents, increased doses of analgesics, the use of warming devices, and careful attention to patient positioning. Prolonged postoperative care and hospital admission after surgery are possible.

Keywords: Anesthesia, anesthesia delivery, poliomyelitis, polio virus, postpolio syndrome.

Poliomyelitis was pandemic in the United States and much of the world in the first half of the 20th century. The uses of the Salk or Sabin polio vaccines have essentially eradicated the disease in the United States today. But 25% to 40% of poliovirus infection survivors who had experienced a paralytic attack, and who later recovered from the attack some 10 to 40 years later, can see symptoms of fatigue, muscle weakness, pain, and muscular atrophy; a syndrome called postpolio syndrome (PPS).

Before surgery, the anesthetist must assess and consider the reported amounts of physical activity the patient typically experiences during his or her daily activities. This physical assessment, along with patient age, can be an indication of the amount of muscle degeneration that may have already occurred. Patients with PPS demonstrate altered respiratory function, cold intolerance, a risk for aspiration, and experience chronic pain in muscles and joints. Patients with PPS display an increased sensitivity to some anesthetic agents such as long-acting narcotics (eg, morphine, hydromorphone) and potent inhaled anesthetic gases with a high blood-gas partition coefficient (eg, isoflurane). Patients with PPS report increased fatigue, weakness, and somnolence after anesthesia. This article discusses the disease poliomyelitis, theorized causes of PPS, and appropriate perioperative anesthetic considerations and management.

Poliomyelitis: The Nature of the Disease

Poliomyelitis is from the Greek *polios* meaning gray, *myelos* meaning marrow, of the organism's spinal cord, and *itis*, which refers to inflammation.[1,2] Poliomyelitis is widely regarded as an eradicated disease of the past. The last wild type or typically seen poliovirus infection in the United States was reported in 1979. The wild type virus is still prevalent today in India, Pakistan, and parts of Africa.[2] Since the early 1970s, renewed interest and research of polio has grown due to the recurrence, some 10 to 40 years later, of some symptoms of paralytic polio that are being called PPS.[2-8]

Poliovirus is an infectious *Enterovirus* of the Picornaviridae family.[2] There are 3 poliovirus serotypes (*PV1*, *PV2*, *PV3*), each with differing antigenicity and cellular receptor affinity.[6] Each serotype is highly contagious and contracted through the oral-fecal gastrointestinal route. Ingested poliovirus replicates along the alimentary tract and is excreted in the saliva for 2 to 3 days and in the feces for 2 to 3 weeks.[2,9,10] The virus continues to multiply in the pharynx and gastrointestinal tract for 1 to 3 weeks after initial infection.[2,10] Viral multiplication spreads to local lymphatic vessels, the bloodstream, and can then invade the central nervous system causing damage to nerve fibers and motor neurons.[2]

Ninety-five percent of people infected with poliovirus are not aware they are infected and display no outward symptoms of the infection. Four percent to 8% of patients present with abortive poliomyelitis, which displays vague, minor, nonspecific symptoms such as sore throat, fever, nausea, vomiting, abdominal pain, constipation (rarely diarrhea), much like the symptoms of influenza.

1. Prior paralytic poliomyelitis with evidence of motor neuron loss
2. A period of partial or complete functional recovery after acute paralytic poliomyelitis, followed by an interval of stable neurological function (stability of 15 years or longer)
3. Gradual or sudden onset of progressive and persistent new muscle weakness or abnormal muscle fatigue (The patient may also have generalized fatigue and muscle atrophy, along with muscle and joint pain.)
4. Symptoms persisting for at least 1 year
5. Exclusion of all other possible medical, neurological and orthopedic etiologies that include the following: hypothyroidism, polymyalgia rheumatica, mitochondrial or metabolic myopathy, fibromyalgia, adult spinal muscular atrophy, amyotrophic lateral sclerosis, cauda equina syndrome, cervical stenosis, chronic inflammatory demyelinating polyneuropathy, diabetic neuropathy, entrapment neuropathy, heavy metal toxicity, inflammatory myopathy, multifocal motor conduction block, multiple sclerosis, myasthenia gravis, Parkinson disease, dysphagia, respiratory insufficiency, sleep disordered breathing, daytime somnolence, and depression. Patient testing can include electromyography, cerebrospinal fluid examination, and magnetic resonance imaging of the brain and spinal cord.[12]

Table 1. The Five Diagnostic Criteria for Postpolio Syndrome[3,7,8,10,11,15,16]

These symptoms are similar to those of other viral infections and can last about a week with complete recovery. One percent to 2% of patients who have contracted poliovirus infection show symptoms of nonparalytic aseptic meningitis. Patients first present with symptoms similar to a nonspecific viral infection of minor illness, which can then progress to symptoms of stiff neck, stiff back, and/or stiff legs. These symptoms can last from 2 to10 days with complete recovery. Less than 1% of poliovirus infections result in a flaccid paralysis called *paralytic polio*. Patients usually experience a prodromal phase with symptoms similar to a nonspecific viral infection, along with temperature elevation, and a progression toward neuronal dysfunction, resulting in a loss of superficial reflexes leading to flaccid paralysis and diminished deep tendon reflexes. Patients do not experience any cognitive or sensory losses.[2,3,9,11,12] Most patients experiencing paralytic polio recover fairly well after several weeks and may recover fully. Any weakness or paralysis lasting 12 months after onset is considered permanent.[2]

There are 3 types of paralytic polio: spinal polio, bulbar polio, or bulbospinal polio. Spinal paralytic polio is the most common type of paralytic polio and accounts for 79% of cases. The poliovirus primarily infects one of the anterior horns of the spinal cord, causing an asymmetric flaccid paralysis, usually of one of the legs. An anterior horn of the spinal cord becomes overwhelmed with white blood cell monocytes and neutrophils, which can then destroy the anterior horn neurons.[9] Anterior horn neurons of the spinal cord gray matter are generally motor neurons that innervate skeletal muscle fibers.[13]

Bulbar paralytic polio accounts for 2% of paralytic polio cases.[2] *Bulbar* refers to an obsolete term for the medulla oblongata of the brainstem.[1,13] The medulla oblongata, along with other areas of the brainstem, controls the subconscious activities of respiration, arterial blood pressure, equilibrium, feeding reflexes, as well as the primal emotional patterns of anger and excitement.[5] Respiratory failure from bulbar paralytic polio requires long-term ventilatory support.[6] Iron lung ventilatory support was necessary in the past for patients affected by the virus. Patients can also experience weakness of muscles innervated by the cranial nerves.[2]

Bulbospinal paralytic polio is a combination of spinal and bulbar paralytic polio, which accounts for 18% of paralytic polio cases.[2]

Historical death rates from spinal paralytic polio were 2% to 5% for children and up to 15% to 30% for adults, with older patients faring worse than younger adult patients. The death rate increased from 25% to 75% with bulbar involvement.[2]

Immunization against poliovirus began in 1955 with subcutaneous or intramuscular injection of an inactivated poliovirus vaccine containing all 3 poliovirus serotypes developed by Dr Jonas Salk. In 1961, an oral polio vaccine was developed by Dr Albert Sabin.[2,9,10,12] The oral polio vaccine (OPV) contains attenuated strains of all 3 poliovirus serotypes and was initially used worldwide until OPV-associated paralytic poliomyelitis was recognized in nonimmunized individuals who came in contact with the excretion of live attenuated virus from patients who had been immunized with OPV.[2,10] Inactivated poliovirus vaccine still remains in use in the United States.[14] The use of the OPV was discontinued in the United States in the year 2000 and the United Kingdom in 2004. The last reported OPV-associated paralytic polio case was reported in 1999.[2] Both vaccines remain in use today in other countries.

Postpolio Syndrome

There are approximately 1.63 million poliomyelitis survivors in the United States, and up to 641,000 Americans suffered from a paralytic form.[12,15,16] The aftereffects of poliomyelitis infection were documented in the literature as early as 1875, but they have only been widely recognized in the healthcare community since the mid-1980s. It is estimated that 25% to 40% of paralytic polio survivors, or between 200,000 to 400,000 people, may present with PPS.[11,16]

PPS was first defined in 1972 and was originally thought to be part of the aging process of older paralytic polio survivors. PPS presents with 5 diagnostic criteria

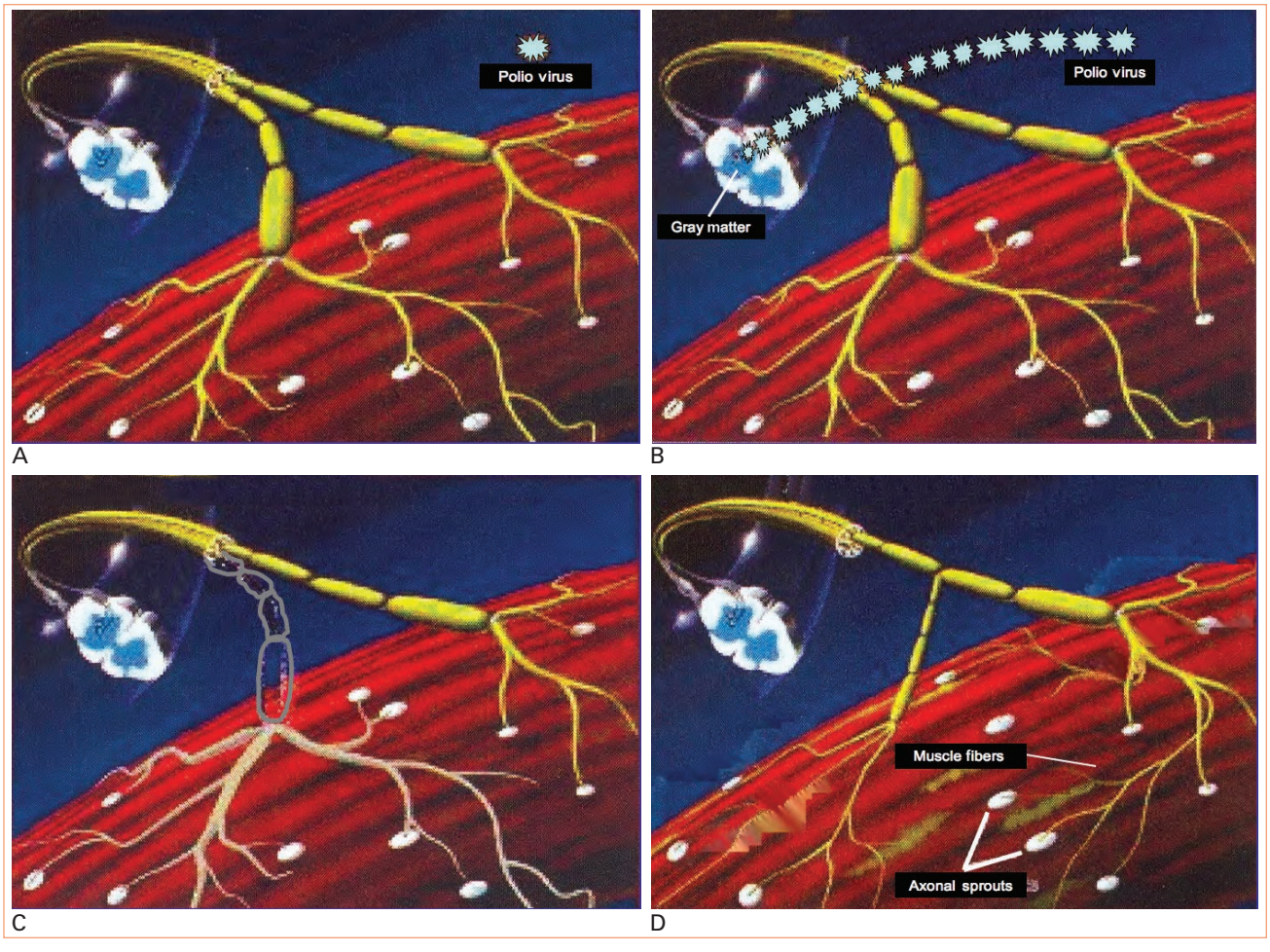

Figure. Anatomy of Postpolio Syndrome

A. Normal noninfected neuron and muscle: The typical motor neuron arises from the gray matter of the spinal cord and synapses with muscle fibers. (Notice the infectious poliovirus entering and attacking the gray matter of the spinal cord.) **B.** Neuron infected with poliovirus: Less than 1% of neurons infected with the poliovirus can lead to neuronal dysfunction. **C.** Polio infected nerve degeneration: Progressive neuronal poliovirus infection leads to flaccid paralysis and diminished deep tendon reflexes. **D.** Regenerative axonal sprouts: Terminal axons became enlarged and sprout to reinnervate skeletal muscle following poliovirus infection.
(Reprinted with permission from Post-Polio Health International.)

from patients with a history of paralytic polio (Table 1).[17] Research has promoted further understanding of the syndrome and is aiding in its treatment.[9-11,15,17]

It is theorized and now accepted that PPS develops because of the permanent denervation damage caused by the initial poliovirus infection. During recovery from the acute episode, axonal terminals of the damaged motor neurons enlarge, sprout, and grow to reinnervate affected skeletal muscle fibers, which allows near normal strength to return to the affected muscles (Figure).

Inherent weakness can recur years later in reinnervated skeletal muscles previously afflicted by acute paralytic polio. These enlarged and sprouted motor neurons degenerate in the normal process of aging, causing the symptoms of PPS.[6,11,15,16] PPS is *not* due to reinfection of the poliovirus, and patients do not shed poliovirus.[2] New research has also found signs of terminal axonal sprouting in muscles that had initially seemed unaffected by the initial acute poliovirus infection, which could account for additional patient weakness.[16]

PPS can first present in patients some 8 to 70 years after the acute paralytic polio attack. Most patients have had stable motor neuron function for the past 15 to 30 years.[11,15,16] These patients had experienced severe symptoms of acute poliovirus infection and had also experienced great recovery, with large quantities of nerve/muscle motor unit sprouting and motor neuron enlargement. PPS symptoms begin to present with weakness in limb muscles, signs of general or central fatigue, respiratory insufficiency, and dysphagia.[10,11,16] PPS is also seen in patients who had become infected with poliovirus later in life, have experienced recent weight gain, and have overuse or disuse of muscles. PPS is more prevalent in females. Debate continues as to whether PPS will continually progress until death or whether stabilization occurs.

Continued study and research show that with ad-

Musculoskeletal system
Joint pain
Muscle pain
Muscle weakness
Twitching and contraction of muscles
Nervous system
Anxiety
Fatigue
Headaches
Nightmares
Restless sleep
Memory problems
Swallowing difficulties
Difficulty speaking loudly
Use of analgesics
Effectiveness of analgesics
Respiratory system
Breathlessness
Airway secretions
Respiratory difficulties
Use of supplemental oxygen
Frequent respiratory infections
Urinary system
Urinary difficulties

Table 2. Anesthesia Assessments for Patients with Postpolio Syndrome[4,11,15-19]

equate knowledge and proper care, along with interdisciplinary management, prevention of muscle deterioration can stabilize the symptoms of PPS with possible improvement.[11,14,16] Management of PPS in patients requires an interdisciplinary approach, utilizing primary care physicians, nurses and nurse specialists, physical and occupational therapists, orthopedists, respiratory therapists, psychiatrists, dentists, registered dental hygienists, and others who are able to address and manage specific symptoms.[11,15]

Anesthesia Considerations for Patients With PPS

The mainstay of caring for the patient with PPS is to reduce the physical stress placed on the muscles that were previously affected by poliovirus infection.[11,17-19] Therefore, the anesthetist must be mindful of the typical physical activity the patient with PPS performs in his or her activities of daily living. Another consideration is the age of the patient with PPS. Muscle function can decrease with age; therefore, an older patient who stresses his or her muscles with exercise could indicate probable muscle degeneration, which could lead the anesthetist to expect a decrease in muscular function during and after anesthesia care. Table 2 outlines anesthesia assessments for patients with PPS. The patient with PPS may appear to have normal muscle function during the anesthetic assessment, but the anesthetist must tailor the entire anesthetic toward preservation of muscle function and protection of the patient with weakened muscle function, so the patient with PPS has strength and function after the anesthetic.

Special considerations must be made in how anesthesia is delivered and what kind of anesthesia technique is used. Patients with PPS may display sensitivity to inhaled or intravenous anesthetics. The anesthetist should consider selecting short-acting anesthetics whenever possible. Local anesthesia is a viable alternative to inhaled or intravenous anesthetic agents. Local anesthetic dose requirements are *increased* over typical doses, and patients can report temporary paralysis after injection.

Patients with PPS report cold intolerance, so the use of warming devices such as blankets, forced air warming blankets, head covers, and warmer ambient air in the surgical suite are necessary. Chronic pain and a history of chronic opioid use may be present; therefore, tolerance to opioid analgesics along with increased rates of liver metabolism can dictate more frequent and higher than usual doses of opioid analgesics. When positioning the patient with PPS, be mindful of padding and supporting the limbs, taking special care while moving limbs and extremities. Comfort is best ensured if positioning is performed before sedation or induction for general anesthesia. Postoperative anesthesia care could be prolonged because of the inherent weakness of muscles previously affected by polio, along with patient reports of increased fatigue and somnolence. This leads to extended times for postanesthesia care and the possibility for hospital admission to more closely monitor and observe the patient prior to hospital discharge.[3] Table 3 presents a summary of anesthesia delivery considerations for the patient with PPS.

In conclusion, PPS is not a reinfection with the poliovirus, but is a result of an initial poliovirus infection, which produced paralytic poliomyelitis with evidence of motor neuron loss, resulting in permanent denervation damage.

The patient experiences a period of partial or complete functional recovery after experiencing acute paralytic poliomyelitis, followed later by an interval of stable neurological function. The patient can then experience a gradual or sudden onset of progressive and persistent new muscle weakness or abnormal muscle fatigue, generalized fatigue, and muscle atrophy, along with muscle and joint pain.

The anesthetist must preoperatively assess the patient and be aware of the need to select short-acting anesthetic agents whenever possible and protect the patient because of inherent muscle weakness, painful joints and muscles, cold intolerance, and reduced protective reflexes.

Patients may experience chronic pain.[3,4,7,12]

- Positioning considerations for surgery due to limb weakness and joint pain
  - Joint pain–hip pain, knee pain, ankle pain
  - Back pain, scoliosis
  - Muscle pain

Patients can display altered respiratory function.

- Humidification of anesthetic gases for long surgical cases
- Consider use of cough and deep breathing as well as incentive spirometry postoperatively.[3,4]
  - Respiratory weakness
  - Sensitivity to narcotics
  - Carbon dioxide retention

Risk of aspiration/dysphagia/vomiting

- Follow nothing by mouth protocols.
- Consider the possibility of aspiration.
- Consider metoclopramide and histamine type 2 blockers such as famotidine or ranitidine.
- Cold intolerance
  - Prepare to keep the patient at normal body temperature.
  - Keep the patient continually warm from before the procedure begins to recovery from anesthesia.

Neurological effects

- Fatigue
- Generalized weakness
- Somnolence
- Difficulty concentrating
  - Modafinil, a schedule IV cerebral stimulant used in narcolepsy and fatigue, has *not* been shown to be effective for patients with PPS.[20,21]

Increased sensitivity to anesthetic agents[3,4,12]

- Induction medications
- Potent inhaled agents and nitrous oxide
  - Consider the use of potent inhaled agents with a low blood-gas partition coefficient (eg, sevoflurane, desflurane), along with the use of nitrous oxide.
  - Avoid the use of potent inhaled agents with a high blood-gas partition coefficient (eg, isoflurane).

Neuromuscular paralytics

- Succinylcholine is *not* contraindicated (based on 1 case report).[22]
  - Although unreported, fasciculation from the administration of a depolarizing neuromuscular paralytic might lead to increased muscle weakness postoperatively because of strenuous muscle stimulation.
  - Consider eliminating the use of paralytics if warranted by the dictates of the surgical procedure.
  - Consider selection of shorter acting paralytic medications.
  - Carefully monitor train-of-four, tetanic, and dual burst twitches.

Narcotics

- Patients may demonstrate excessive narcosis and/or sedation.
- Carefully titrate narcotic medications to effect.
- Avoid the use of long-acting narcotics (eg, morphine, hydromorphone).
- Consider use of narcotics with shorter half-life or nonnarcotic medications.

Local and/or regional anesthesia is considered acceptable for patients with PPS.

- No toxic effects of intrathecal anesthetics have been proven clinically.[3,12]

Table 3. Anesthesia Delivery Considerations for the Patient with Postpolio Syndrome (PPS)

More information on support with PPS is available on the Post-Polo Health International website at www.post-polio.org.

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