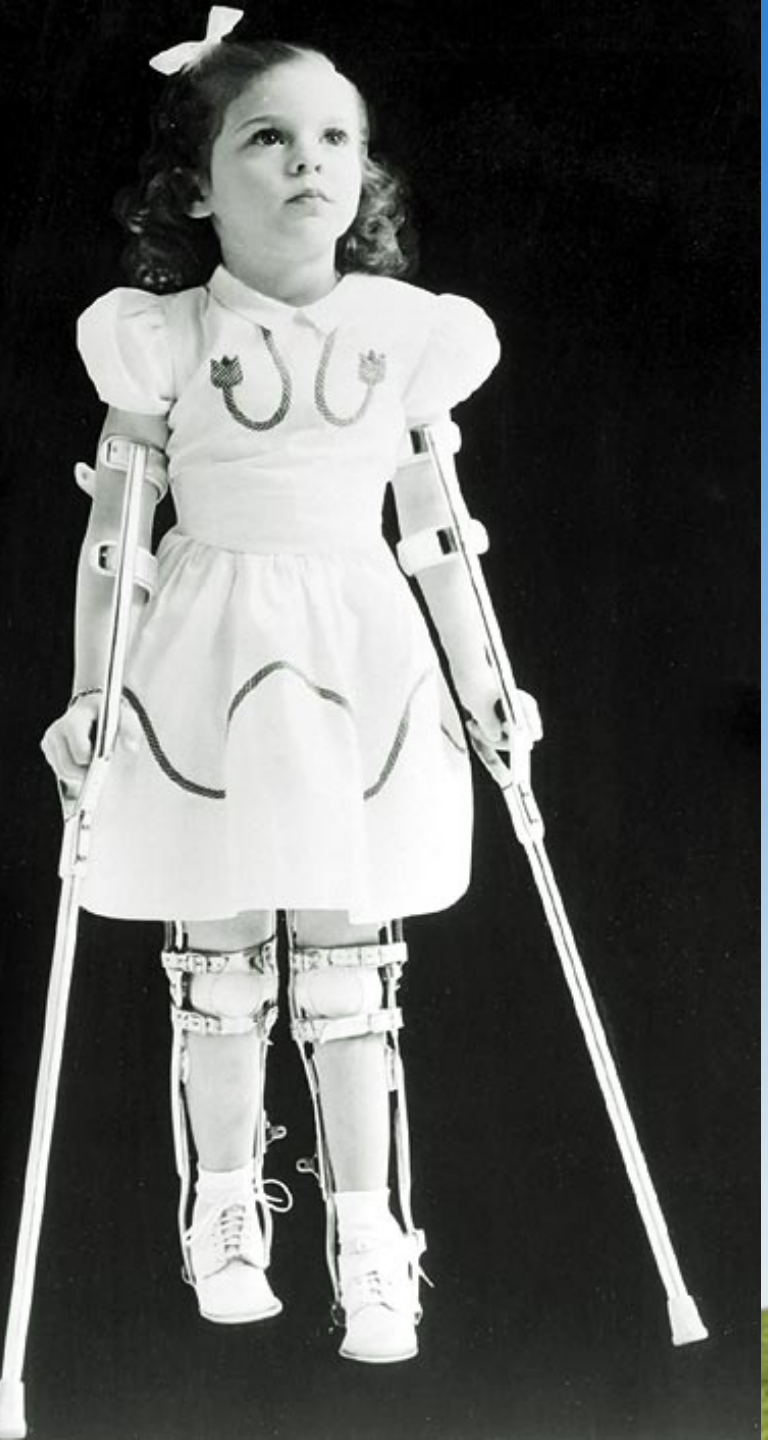


POLIOMYELITIS

ETIOLOGY

The polioviruses are nonenveloped, positive-stranded RNA viruses belonging to the Picornaviridae family, in the genus Enterovirus, and consist of 3 antigenically distinct .serotypes (types 1, 2, and 3)

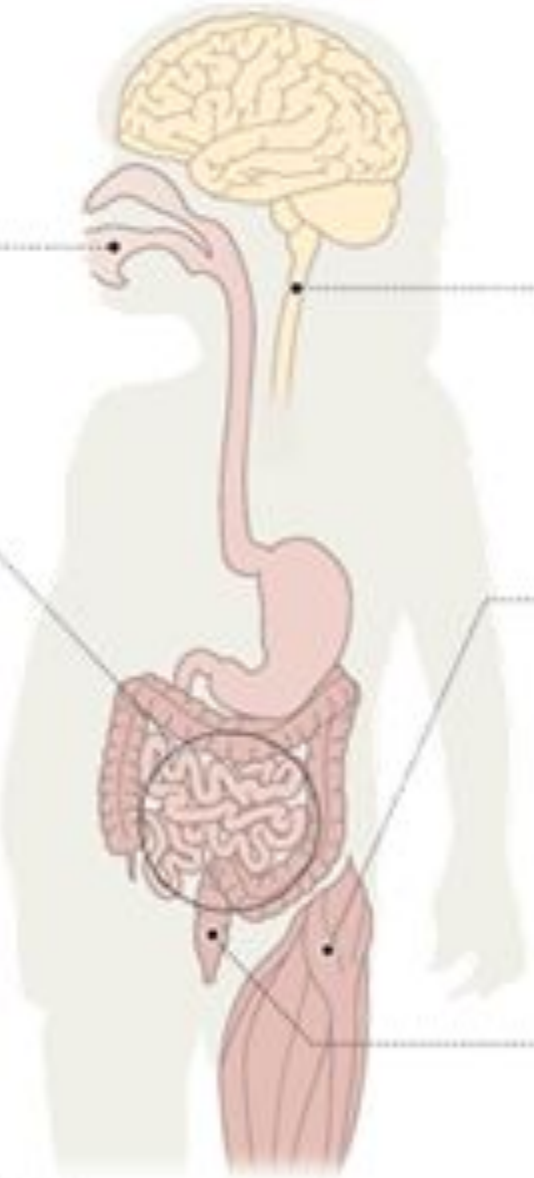
Polioviruses spread from the intestinal tract to the central nervous syste (CNS), where they cause aseptic meningitis .and poliomyelitis, or polio

The polioviruses are extremely hardy and can retain .infectivity for several days at room temperature

TRANSMISSION

Humans are the only known reservoir for the polioviruses, which are spread by the fecal-oral route. Poliovirus has been isolated from feces for longer than 2 wk before .paralysis to several weeks after the onset of symptoms

The Life Cycle of Polio



FINDING A VICTIM

The virus enters the body through contaminated food, dirty fingers or water tainted with sewage.

SETTLING IN

The virus attaches to receptors on the intestinal walls. (Oral polio vaccine attaches to the same ones.) From there it can get into the bloodstream.

THE ATTACK

In 99.5% of cases, the virus causes no symptoms, or mild flulike illness.

In less than 0.5% of cases, the virus attacks the central nervous system, destroying cells in the spinal cord.

THE DAMAGE

Nerve cell death can cause muscle paralysis, mostly in the legs.

MOVING ON

Even in people with no symptoms, the virus is excreted in feces that can contaminate food and water.

Source: World Health Organization

The New York Times

CLINICAL MANIFESTATIONS

The incubation period of poliovirus from contact to initial clinical symptoms is usually considered to be 8-12 days, with a range of 5-35 days. Poliovirus infections with wild-type virus may follow 1 of several courses: inapparent infection, which occurs in 90-95% of cases and causes no disease and no sequelae; abortive poliomyelitis;non paralytic poliomyelitis; or paralytic poliomyelitis. Paralysis, if it occurs, appears 3-8 days after the ..initial symptoms

Abortive Poliomyelitis

In approximately 5% of patients, a nonspecific influenza-like syndrome occurs 1-2 wk after infection, which is termed abortive .poliomyelitis

Fever, malaise, anorexia, and headache are prominent features, and there may be sore throat and abdominal or muscular pain. Vomiting occurs irregularly. The illness is short lived, lasting up to .2-3 days

The physical examination may be normal or may reveal nonspecific pharyngitis, abdominal or muscular tenderness, and .weakness

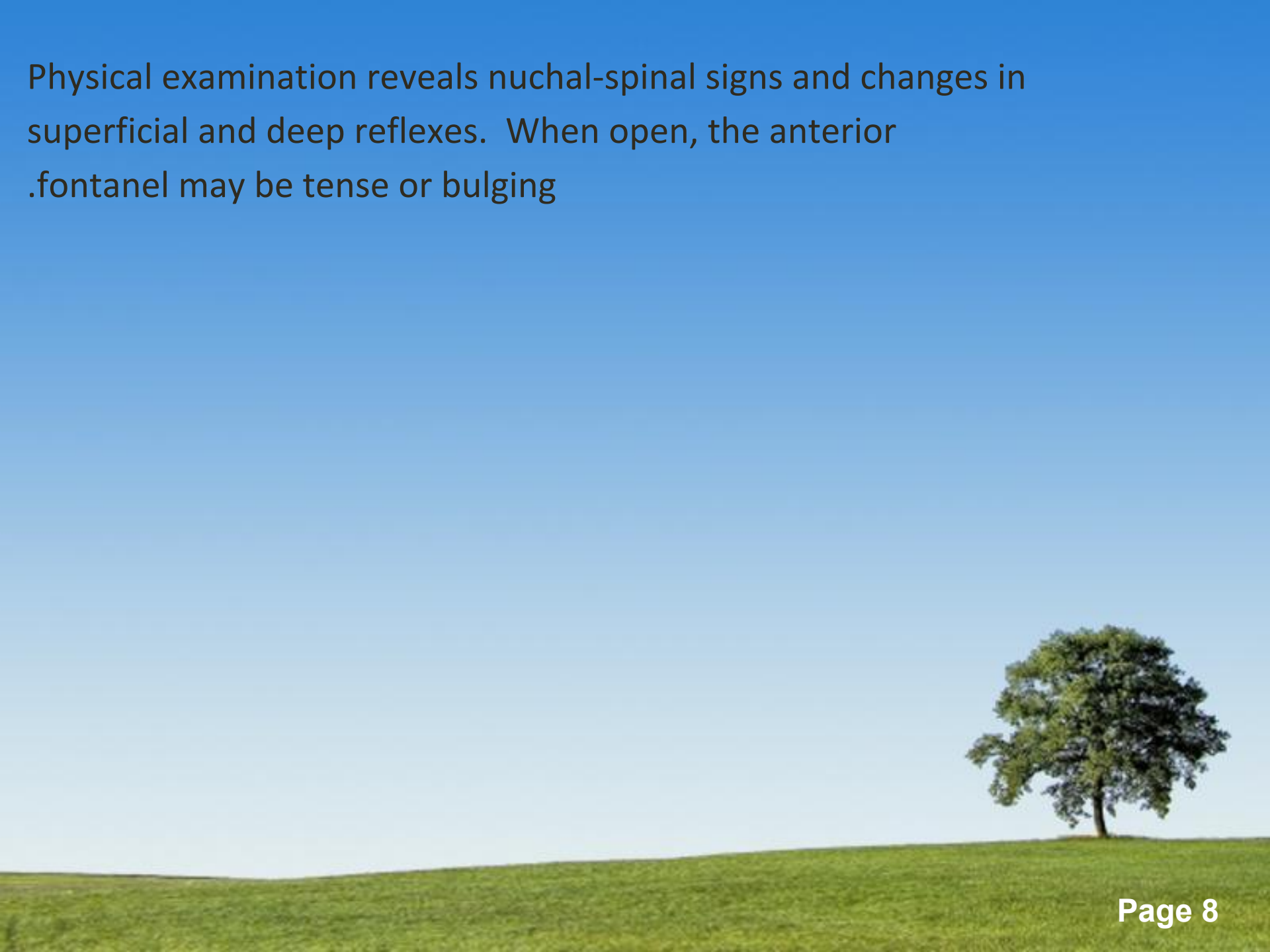
Recovery is complete, and no neurologic signs or sequelae .develop

Nonparalytic Poliomyelitis

In approximately 1% of patients infected with wild-type poliovirus, signs of abortive poliomyelitis are present, as are more intense headache, nausea, and vomiting, as well as soreness and stiffness of the posterior muscles of the neck, trunk, and limbs. Fleeting paralysis of the bladder and constipation are frequent. Approximately two thirds of these children have a short symptom-free interlude between the 1st phase (minor illness) and the 2nd phase (CNS disease or major .illness)

Nuchal rigidity and spinal rigidity are the basis for the diagnosis .of nonparalytic poliomyelitis during the second phase

Physical examination reveals nuchal-spinal signs and changes in superficial and deep reflexes. When open, the anterior .fontanel may be tense or bulging



In the early stages the reflexes are normally active and remain so unless paralysis supervenes. Changes in reflexes, either increased or decreased, may precede weakness by 12-24 hr. The superficial reflexes, the cremasteric and abdominal reflexes, and the reflexes of the spinal and gluteal muscles are usually the first to diminish. The spinal and gluteal reflexes may disappear before the abdominal and .cremasteric reflexes

Changes in the deep tendon reflexes generally occur 8-24 hr after the superficial reflexes are depressed and indicate impending .paresis of the extremities. Tendon reflexes are absent with paralysis .Sensory defects do not occur in poliomyelitis

Paralytic Poliomyelitis

- Paralytic poliomyelitis develops in approximately 0.1% of persons infected with poliovirus, causing 3 clinically recognizable syndromes that represent a continuum of infection differentiated only by the portions of the CNS most severely affected. These are spinal paralytic poliomyelitis, (2) bulbar poliomyelitis (1) .(3) polioencephalitis

Spinal paralytic poliomyelitis

severe headache and fever occur with exacerbation

of the previous systemic symptoms. Severe muscle pain is present, and sensory and motor phenomena (e.g., paresthesia, hyperesthesia, fasciculations,and spasms) may develop. On physical examination the distribution of paralysis is characteristically spotty. Single muscles, multiple muscles, or .groups of muscles may be involved in any pattern

.Within 1-2 days, asymmetric flaccid paralysis or paresis occurs

Involvement of one leg is most common, followed by .involvement of one arm

The proximal areas of the extremities tend to be involved to .a greater extent than the distal areas

Examination may reveal nuchal stiffness or rigidity, muscle tenderness, initially hyperactive deep tendon reflexes (for a short period) followed by absence or diminution of reflexes, and paresis or flaccid paralysis. In the spinal form,there is weakness of some of the muscles of the neck, abdomen, trunk,diaphragm, thorax, or extremities. Sensation is intact; sensory disturbances, .if present, suggest a disease other than poliomyelitis

The paralytic phase of poliomyelitis is extremely variable; paralysis occurs if >50% of the neurons supplying the muscles .are destroyed

;The extent of involvement is usually obvious within 2-3 days

Bowel and bladder dysfunction ranging from transient incontinence to paralysis with constipation and urinary retention often accompany paralysis of the lower limbs

The return of strength and reflexes is slow and may continue to improve for as long as 18 mo after the acute disease. Lack of improvement from paralysis within the 1st several weeks or months after onset is usually evidence of .permanent paralysis

Atrophy of the limb, failure of growth, and deformity are .common and are especially evident in the growing child

Bulbar poliomyelitis

The clinical findings seen with bulbar poliomyelitis with respiratory difficulty include (1) nasal twang to the voice or cry caused by palatal and pharyngeal weakness (2) inability to swallow smoothly, resulting in accumulation of saliva in the pharynx, indicating partial immobility (3) accumulated pharyngeal secretions, which may cause irregular respirations (4) absence of effective coughing, shown by constant fatiguing efforts to clear the throat; (5) nasal regurgitation of saliva and fluids as a result of palatal paralysis, with inability to separate the oropharynx from the nasopharynx during swallowing; (6)deviation of the palate, uvula, or tongue; (7) involvement of vital centers in the medulla, which manifest as irregularities in rate, depth, and rhythm of respiration; as cardiovascular alterations, including blood pressure changes (especially increased blood pressure), alternate flushing and mottling of the skin, and cardiac arrhythmias; and as rapid changes in body temperature; (8) paralysis of 1 or both vocal cords, causing hoarseness, aphonia, and, ultimately, asphyxia unless the problem is recognized on laryngoscopy and managed by immediate tracheostomy; and (9) the rope sign, an acute angulation between the chin and larynx caused by weakness of the hyoid muscles (the hyoid bone is pulled posteriorly, narrowing the hypopharyngeal inlet).

polioencephalitis

is a rare form of the disease in which higher centers of the brain are severely involved. Seizures, coma, and spastic paralysis with increased reflexes may be observed. Irritability, disorientation,drowsiness, and coarse tremors are often present with peripheral or cranial nerve paralysis that coexists or ensues. Hypoxia and hypercapnia caused by inadequate ventilation due to respiratory insufficiency may produce disorientation without true encephalitis. The manifestations are common to encephalitis of any cause and can be attributed to polioviruses only with specific viral diagnosis or if .accompanied by flaccid paralysis

The clinical findings associated with involvement of the respiratory muscles include (1) anxious expression; (2) inability to speak without frequent pauses, resulting in short, jerky, “breathless” sentences; (3)increased respiratory rate; (4) movement of the ala nasi and of the accessory ;muscles of respiration

inability to cough or sniff with full depth (5)

paradoxical abdominal movements caused by (6) diaphragmatic immobility caused by spasm or weakness of 1 or both leaves; and (7)relative immobility of the intercostal spaces, which may be segmental,unilateral, or bilateral. When the arms are weak, and especially when deltoid paralysis occurs, there may be impending respiratory paralysis because the phrenic nerve nuclei are in adjacent areas of the .spinal cord

DIAGNOSIS

.History and examination*

the laboratory diagnosis of poliomyelitis be confirmed by* isolation and identification of poliovirus in the stool, with specific identification of wild-type and vaccine-type strains. In suspected cases of acute flaccid paralysis, 2 stool specimens should be collected 24-48 hr apart as soon as possible after the .diagnosis of poliomyelitis is suspected

Poliovirus concentrations are high in the stool in the 1st wk after the onset of paralysis, which is the optimal time for .collection of stool specimens

Polioviruses may be isolated from 80-90% of specimens from acutely ill patients, whereas <20% of specimens from such .patients may yield virus within 3-4 wk after onset of paralysis

The CSF is often normal during the minor illness and typically* contains a pleocytosis with 20-300 cells/µL with CNS involvement. The cells in the CSF may be polymorphonuclear early during the course of the disease but shift to mononuclear cells soon afterward. By the 2nd wk of major illness, the CSF cell count falls to .near-normal values

In contrast, the CSF protein content is normal or only slightly elevated at the outset of CNS disease but usually rises to 50-100 mg/dL by the 2nd wk of illness.In polioencephalitis, the CSF may .remain normal or show minor changes

Serologic testing demonstrates seroconversion or a 4-fold or* greater increase in antibody titers from the acute phase of .illness to 3-6 wk later

MRI of spine and brain*

Nerve and muscle biopsy*

DIFFERENTIAL DIAGNOSIS

Non.paralytic poliomyelitis(other causes of meningeal.1 irritation)

bacterial,viral,T.B meningitis *meningismus(neck rigidity* accompany pneumococal pneumonia or typhoid)

Paralytic poliomyelitis(other causes of acute flaccid paralysis).2

guillain-barre syndrome/symmetricalparalysis,abscent CSF* .pleocytosis

Other infections;*enterovirus,*herpes zoster virus,*rabies.3 .virus,*diphtheria,*botulism

Spinal cord disorders;*transverse myelitis *cord compression.4

Periphral motor neuropathy*acute intermittent porphyria.5

mononeuritis multiplex*

Disorders of neuromuscular transmission.6

Inflammatory myopathy(symmetrical weakness,increased.7 serum CK level

COMPLICATIONS and causes of death

Respiratory failure

Hypostatic pneumonia

Acute gastric dilitation

Malena due to intestinal erosion

Hypertention and heart failure

Acute urinary retention and UTI(stasis)

Hypercalcemia

Retarded motor development

Muscle atrophy,contractureand deformities

TREATMENT

Abortive Poliomyelitis

Supportive treatment with analgesics, sedatives, an attractive diet, and bed rest until the child's temperature is normal for several days is ,usually sufficient. Avoidance of exertion for the ensuing 2 wk is desirable and careful neurologic and musculoskeletal examinations should .be performed 2 mo later to detect any minor involvement

Nonparalytic Poliomyelitis

relief is indicated for the discomfort of muscle tightness and spasm of the neck, trunk, and extremities. Analgesics are more effective when they are combined with the application of hot .packs for 15-30 min every 2-4 hr. Hot tub baths are sometimes useful A firm bed is desirable and can be improvised at home by placing table leaves or a sheet of plywood beneath the mattress. Patients with nonparalytic poliomyelitis should also be carefully examined 2 mo after apparent recovery to detect minor residual effects that might cause postural problems in later years

Paralytic Poliomyelitis

Most patients with the paralytic form of poliomyelitis require* hospitalization with complete physical rest in a calm atmosphere for the .1st 2-3 wk

A neutral position with the feet at right angles to the legs, the knees* slightly flexed, and the hips and spine straight is achieved by use of .boards, sandbags, and, occasionally, light splint shells

.The position should be changed every 3-6hrs•

Constipation is common, and fecal impaction should be prevented. *• *When bladder paralysis occurs, a parasympathetic stimulant such as .bethanechol may induce voiding in 15-30 min

Adequate dietary and fluid intake can be maintained by placement of a* .central venous catheter

for Bulber poliomyelitis

monitoring of vital sign*

Airway maintenance*

N.G tube feeding*

decreased FVC indicated for mechanical ventilation*

Chronic case

Physiotherapy

Plastic splint and walking aids

Orthopedic operation

PREVENTION

Vaccination is the only effective method of .preventing poliomyelitis

inactivated polio virus vaccine(IPV,salk)*

oral attenuated poliovirus vaccine(OPV,sabin)*

THANK YOU