Recovery from Acute Paralytic Polio

Mortality from acute paralytic polio is usually the result of respiratory or bulbar involvement.[3] In those who survive the acute illness and recover, paralysis remains static for several days or weeks before a slow recovery occurs over several months to years.[7] Muscle strength in partially denervated muscles increases to a maximum over a two year period, with 60 percent of the muscle strength recovery occurring in the first three months after onset and 80 percent in the first six months. Further improvement may continue over the next two years.[6]

Muscle strength recovery and increase in functional ability occur by several physiological processes. These include:

- Terminal sprouting;
- Myofibre hypertrophy;
- Fibre type transformation; and
- Ongoing denervation and reinnervation.

Terminal Sprouting

During the recovery process following acute paralytic polio, remaining brainstem and spinal cord motor neurons can elaborate new branches, or axonal sprouts. The physiological basis for axonal sprouting is believed to be related to the expression of neural-cell adhesion molecules by the denervated myofibres.[14] These molecules seem to provide a chemotactic stimulus to the terminal axons of neighbouring, surviving motor neurons, causing these neurons to send axonal sprouts to denervated muscle fibres.[15] These sprouts can reinnervate orphaned muscle fibres that have been denervated by the acute polio infection. Sprouting (or collateral innervation) can restore the capacity of voluntary muscle fibres to contract and thus improve clinical strength.[8]

There is considerable electrophysiological evidence, including single fibre and macro-electromyography (EMG) and muscle morphological data to support this concept of reinnervation.[16-20] Muscle biopsy studies have shown that the remaining motor neurons may innervate up to eight times or more the normal number of muscle fibres.[21] Survivors of acute polio may be left with a few, significantly enlarged motor units doing the work previously performed by many units.[5] Figure 1 provides a schematic illustration of this process.

Myofibre Hypertrophy

In addition to sprouting, the remaining innervated muscle fibres hypertrophy through exercise and activity during the rehabilitation phase after the acute illness. Muscle fibre hypertrophy contributes further to the recovery of strength after paralytic polio. Because this mechanism of neurophysiological compensation is so effective, a muscle can retain normal strength even after 50 percent of the original motor neurons have been lost. Several studies have documented that the fibre area of type I and type IIA myofibres in the quadriceps of post-polio men are, on average, twice the control value.[22,23]

Further studies have supported these results. In a study by Borg and colleagues (1988)[24] marked hypertrophy of the muscle fibres, as determined by surface electromyography during gait, within the tibialis anterior muscle, was observed in post-polio subjects who excessively overused that muscle. Grimby and colleagues (1989)[22] reported a significant negative correlation between muscle strength and mean fibre area in post-polio male subjects, i.e. the weakest subjects had the largest muscle fibres

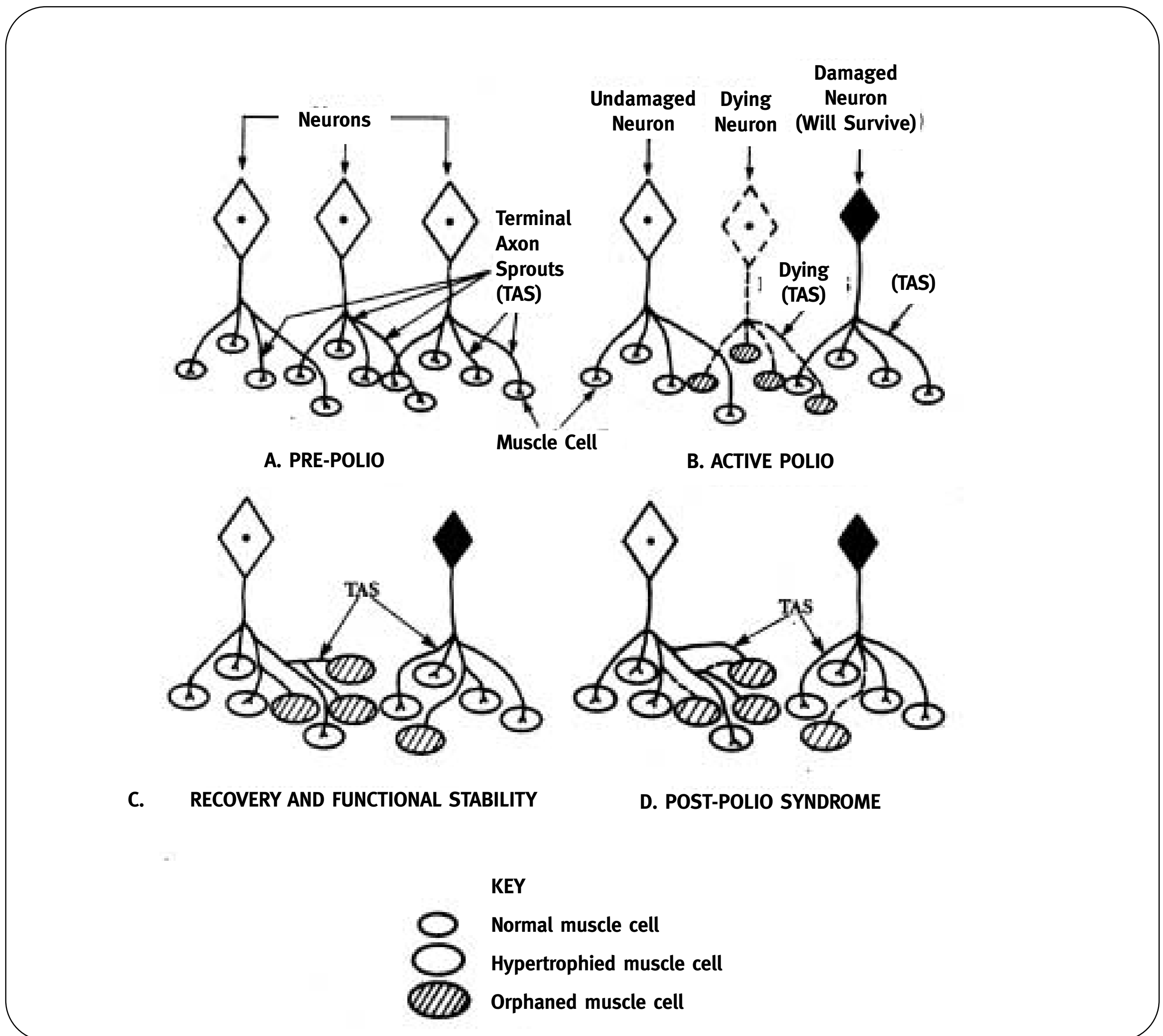

Figure 1: The Motor Neuron Unit Before and After Polio
(Adapted from Halstead, 1998)[25]

Fibre Type Transformation

It has been speculated that fibre transformation from type II fibres (fast twitch, glycolytic) to type I (slow twitch, oxidative) fibres is another neuromuscular adaptive mechanism. A study by Grimby and colleagues (1989)[22] demonstrated a significant negative correlation between the relative occurrence of type I fibres and muscle strength in the quadricep muscles of women with a history of polio. They suggested that this may be due to transformation of type II to type I fibres in subjects with the most marked reduction in the number of muscle fibres and strength. Muscle biopsies conducted on the anterior tibialis muscle of ambulating post-polio subjects exhibited almost exclusively type I muscle fibres, while subjects who used

wheelchairs did not demonstrate type I fibre dominance. This suggested that this type I fibre dominance in some of the subjects was due to excessive use of remaining muscle fibres causing a transformation of type II to type I fibres.[24]

Ongoing Denervation and Reinnervation

The process of denervation and reinnervation is ongoing in post-polio patients with and without complaints of new weakness. Reinnervation of recently denervated muscle fibres appears to be another adaptive neuromuscular mechanism to maintain function. Histologic[26] and electrophysiologic[16,27,28] evidence supports this concept.

Such extensive compensatory physiological processes mask the profound neurological deficits caused by the disease. In addition to these physiological processes, the body possesses a number of compensatory mechanisms to maintain function in the presence of residual paralysis. These compensations include:

- Use of weak muscles at a higher level of capacity;
- Substitution of strong muscles with increased energy expenditure for the tasks; and
- Use of ligaments for stability with resulting hypermobility.