Differentiating Post-Polio Syndrome from Aging

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Post-polio syndrome demonstrates the difficulty of separating disease from normal aging. When individuals first presented with symptoms of post-polio syndrome, many were told they were just getting older. Some are still given this explanation. To a certain extent this is true, but experience and thoughtful observation reveal a process above and beyond normal aging. There is a slow multisystem decline in aging that interacts with the injury sustained during acute poliomyelitis. In the life of post-polio survivors, a degree of disability emerges that places them on a different trajectory from the slow accumulation of disability experienced in normal aging.

ETIOLOGY OF AGING

The overall cause of aging itself is not known. There are probably several mechanisms operating simultaneously that produce age-related decline in organ and tissue function. In some cells, reproduction is limited to a certain number of generations; distant organ failure may change the systemic milieu in a way that negatively alters cell function. Subtle conformational changes in critical proteins of cell metabolism may damage some cell types. In neurons, the accumulation of byproducts of cellular metabolism during the lifetime of the cell may be injurious.

CHANGES OF NORMAL AGING

In normal aging there is a slow multisystem decline. The onset of decline and rate of change vary from organ to organ.

The heart is fairly resistant to aging. The size of the heart is similar, however the thickness of the heart wall is slightly increased. Early diastolic filling is reduced. There is age-related decrease in maximum heart rate but a compensatory increase in volume per beat. Maximum oxygen consumption is reduced with age but it is uncertain if this is due to decreased cardiac output or decreased peripheral uptake of oxygen.

The forced vital capacity of ***the lungs*** decreases after age 27 by approximately 25ml/year (1). The surface area of the air sacs in the lungs decreases by 4% per decade after age 30 (2,3,4) These changes resemble those in emphysema.

Renal (kidney) blood flow is decreased from 1200ml/min in youth to 600ml/min at age 80.

How well the kidney removes waste products from the blood is stable until the middle of the fourth decade then declines.

Immune system changes also occur with aging (5):

- the involution of thymus gland;
- antibody response to vaccination decreases;
- autoantibodies (antibodies to self structures) increases;
- T- cell function diminishes;
- T supressor cell function increases.

Infectious diseases such as pneumonia and influenza rise exponentially after the age of 25 along with an increased incidence of cancer and autoimmune disease (6,7).

Changes in ***the brain*** with normal aging include:

- A decrease in weight;
- A decrease in cortex nerve cell size;
- A decrease in the speed of central processing;

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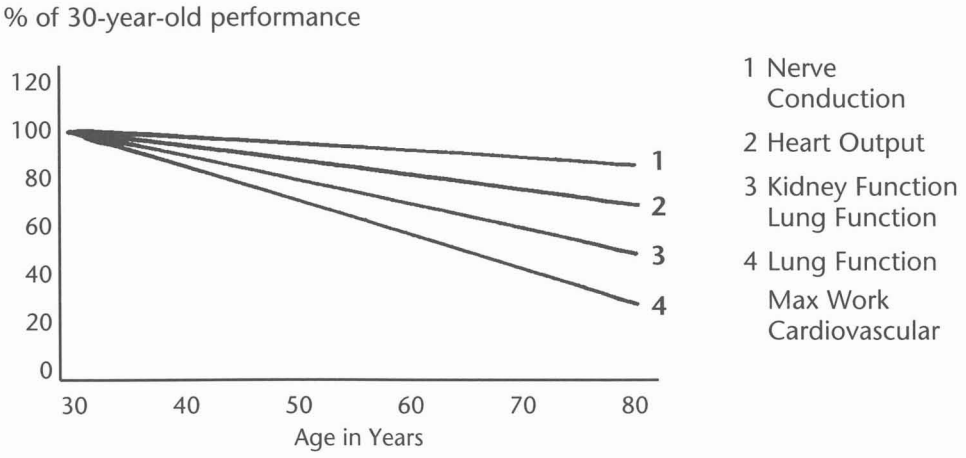
Age Decrements in Physiologic Performance



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♦ A significant decline in long-term memory (delayed recall) by age 50 (8);

♦ A loss of substantia nigra neurons, (400k @ birth, 200k @ age 80). These are the cells of paramount importance in Parkinson's disease. When the count drops to between 200k and 100k, individuals become symptomatic with Parkinson's disease;

♦ Aging of an important population of brain motor neurons (basal ganglia).

Large samples of the population studied in the course of standardization of the Wechsler Adult Intelligence Scale (1955) indicated that there is a steady decline in cognitive function, starting at 30 years of age and progressing into the senium. All forms of cognitive function demonstrated decline although, certain elements of the verbal scale (vocabulary, fund of information, and comprehension) withstood the effect of aging better than those of the performance scale (block design, reversal of digits, picture arrangement, object assembly, and the digit symbol task) (9).

The aging ***neuromuscular system*** is of most interest in the post-polio syndrome. Tomlinson and Irving have provided evidence that the motor neuron pool is stable until approximately age 60. Thereafter the motor neuron population diminishes. In some cases motor neuron numbers may decrease to 50% of the middle life count (10). There is also reduced terminal sprouting (11,12). In individuals over 65 it is not uncommon to see fiber type grouping (unpublished observations) which supports the observation of motor neuron dropout.

In addition to the alterations in the motor neuron there is change in muscle. It is observed that there is loss of muscle mass with aging (13). This loss will cause increased use of the remaining muscle for activities of daily living and, subsequently, may further stress those motor units already at the threshold of maintaining performance.

The Motor Unit in Post-Polio Syndrome

In people who had acute paralytic poliomyelitis there is electrophysiologic evidence that the motor unit is unstable. Fibrillation potentials, postive sharp waves, and fasciculations are observed in muscles of post-polio individuals who have no new complaints (14). These findings imply that the motor neuron is not performing normally. This instability can be demonstrated throughout the life of the polio survivor and worsens as the individual ages (15). These findings represent a continuous remodeling of the motor unit occuring at the level of the terminal nerve. As some terminal nerve/muscle connections are lost, the orphaned muscle may be reconnected to a terminal nerve from another motor neuron. A time comes when the disconnection rate overtakes the reconnection rate. Subsequently muscle fibers are lost and new weakness begins. This critical threshold is more related to the time since the acute poliomyelitis rather than absolute chronologic age. New weakness that is noted at age 45 is a significant divergence from normal aging on two counts. First as noted, age-related motor neuron and terminal nerve loss are deferred until age 60. Second, in normal aging the weakness that accrues is subclinical.

It is the change in the motor unit which is at the heart of the post-polio syndrome and, in light of the observed electrophysiologic alterations in the motor unit, it is evident that the post-polio motor unit is not behaving as a normal aging motor unit. There are three possible causes for this altered performance.

1. The motor unit is expanded. Many motor neurons are carrying a greatly increased load of muscle fibers to compensate for those motor neurons that were lost during the acute poliomyelitis. It is uncertain if the cellular metabolic machinery can carry this increased load for a lifetime. Those motor neurons that escaped from acute poliomyelitis uninjured may be injured later by this chronic increased load.

2. Not all surviving motor neurons escaped uninjured. Many neurons showed evidence of injury but subsequently recovered (16). These motor neurons may have an unpredictable lifetime performance and especially be unable to support an expanded motor unit.

3. Motor units are stressed by an increased demand for firing. It has been demonstrated that the select muscle groups are greatly overused (17). The motor neurons controlling the motor units within these muscle groups are also overfiring. This may have a long-term damaging effect.

Aging and Post-Polio Syndrome

I do not wish to imply that aging is not a factor in post-polio problems. Certainly the length of time a motor neuron carries an increased burden is critical. But it is the aging process itself that underlies the tendency of cell function to be less effective over time. There is something inherent in the youth of the neuron, or something lost with cell aging, that allows a young motor neuron, injured or uninjured, to be capable of handling an expanded muscle fiber population.

Immunologic findings in post-polio syndrome may or may not be related to aging.These findings may include: an increased CD4/CD8 (helper/suppressor) ratio (18), an immune activation where unexpected, a MHC class I expression in muscle, and an infiltration of muscle with lymphocytes and macrophages. Spinal cords of survivors who had poliomyelitis examined years after the original injury revealed mild perivascular and intraparenchymal inflammation. Some have also revealed oligoclonal bands in the cerebral-spinal fluid (16,19). These observations are consistent with an upregulation of immunologic function, possibly an autoimmune action. If an autoimmune process is present, this is in keeping with

the observation of increased autoimmunity with aging.

There is a slow multisystem decline in aging which becomes a factor but is not the cause of post-polio syndrome. For example, increased cardiopulmonary demands that have always been present due to a sub-optimal gait or body mechanics become more critical as age encroaches on cardiac and pulmonary reserve. Decreased pulmonary reserve due to scoliosis crosses the threshold and becomes functionally limiting due to loss of lung elasticity and diminished ventilatory capacity of aging. These changes of aging would have been silent in a similar aged individual who never had paralytic poliomyelitis.

Table 1

	Aging	Post-Polio Syndrome
1	No polio history	Old paralytic poliomyelitis
2	No residual biomechanical disadvantage	Residual biomechanical deficit
3	Mild diffuse loss of muscle mass, little functional impact	Focal moderate to severe loss of muscle strength and muscle mass with significant functional impact
4	Mild to moderate nuisance fatigue	Moderate to severe disabling fatigue
5	Cardiovascular, pulmonary, or cerebrovascular diseases most prominent	Neuromuscular complaint most common
6	Slow imperceptible decline in multiple systems	Slow to moderate decline in neuromuscular performance
7	Symmetric osteoarthritis	Assymetric osteoarthritis

In summary the physiology of normal aging is a slow multisystem decline. Post-polio syndrome is a more rapid oligosystem decline (neuromuscular). The divergence in performance of the post-polio syndrome individual from the course of normal aging represents a distinct pathophysiology. However, the pathology of aging likely plays a role in the emergence of the post-polio syndrome.

Post-Polio Syndrome and Aging: Clinical Features

In normal aging there is loss of muscle mass (13); some may be due to disuse. The loss of strength does not usually become functionally meaningful in the healthy elderly. Modest osteoarthritis produces only minor disability. The cause of greatest decline in performance is due to change in central motor control. The average individual also complains of some loss of productivity and decreased stamina. In disease-free aging, there is a gradual, almost imperceptible decline in function due to the combined effects of declining cardiopulmonary capacity, muscle strength, central motor control, and accumulating osteoarthritis.

The post-polio person also experiences these changes, which may be noted at a much earlier age (45 vs 60). Change is also much more dramatic then seen in normal aging. Loss of muscle strength is focal; if multifocal, it may lead to marked disability. The fatigue and loss of stamina is profound and disabling as opposed to a nuisance in normal aging. The osteoarthritis seen in the hips and knees of an individual with abnormal gait may also be profound.

In practice, the post-polio person stands out from the average geriatric center individual on several counts (Table 1). Polio survivors are usually 10 years younger. Their symptoms are more constrained to new weakness and fatigue indicating an oligosystem (1 or 2 system) failure vs multisystem failure. A typical geriatric patient has one or more medical problems. For example, a prior pneumonia and cardiac disease is a common combination. A modest fatigue is sometime present, but is accounted for by a clear medical problem. Profound fatigue is uncommon. A complaint of specific focal new weakness is even more uncommon. Thus the post-polio survivor with

Table 2. Post-Polio Syndrome vs Aging: Interventions

Goal	Aging	Post-Polio Syndrome
Maintain muscle strength	Strength training	Modified non-fatiguing, paced strength training of affected extremities
Cardiovascular fitness	Aerobic exercise	Modified non-fatiguing aerobic exercise
Increased stamina	Exercise and activity	Carefully meter physical activity
Optimum physical performance	Physical therapy to involved areas. Orthotic and assist devices	Physical therapy in the form of muscle training only in special situations. Orthotic and assist devices as needed.

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their defining features usually stands out on this basis alone.

Post-Polio Syndrome vs Aging: Interventions

Interventions to promote neuromuscular and cardiovascular fitness are different in the post-polio and general population (Table 2). Increasing muscle strength may be accomplished by conventional techniques of muscle training constrained only by the cardiac and orthopedic status of the individual. An otherwise healthy elderly individual may enter graduated weight training. As their training progresses they are able to increase their limit to produce a modest degree of soreness and fatigue. This approach would be deleterious to a post-polio survivor. In short, the training principles for the geriatric population are parallel to those of the younger adult population with allowences made for baseline cardiovascular and joint condition. In the post-polio individual there must be no residual pain and minimal fatigue after training. Some post-polio people are unable to pursue cardiovascular or strength training due to extensive motor neuron and attendent muscle loss. ❐

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"I probably am your oldest subscriber.

I had infantile paralysis in 1923 when I was 18 years old. I was traveling with my parents (camping, of course). Doctors had no idea why I had such a high temperature, was so miserable, and could not move my arms or legs without help. One suggested they put me in the "Fischer's Hot Mineral Springs," a rack-constructed tank about the size of a small swimming pool and help me move. It was in Eastern Oregon, near Haines, and only a couple of miles from my uncle's place, where we had expected to spend a month. This they did almost every day and although I improved, I had to have help to get up, dress, walk, and even to turn in bed.

Several months later we learned of a death from infantile paralysis in Missoula, Montana, where we had spent time prior to my illness. The doctor who later examined my still partially paralyzed legs at home in California said no doubt I had infantile paralysis. It was years before I discovered how lucky I was to receive the right treatment at the right time.

Now the weakness, paralysis, and cramps have returned, and x-rays show degenerative osteoarthritis of the left hip joint, although I have no other indication of arthritis in any other part of my body. I am 91 years old. I still walk with a cane or walker, handle my own business, and ask for no special help at the retirement home where I live.

This is why I want to be informed of current developments which hopefully will be discovered about post-polio syndrome. Thanks for your help."

Irene, California