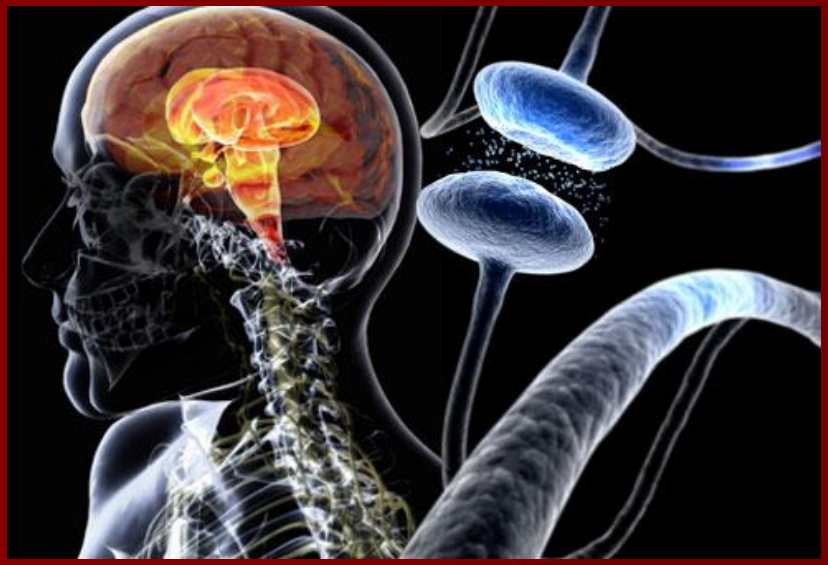
Acute and Chronic Neurological Diseases: Quick Notes



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Neurological Conditions

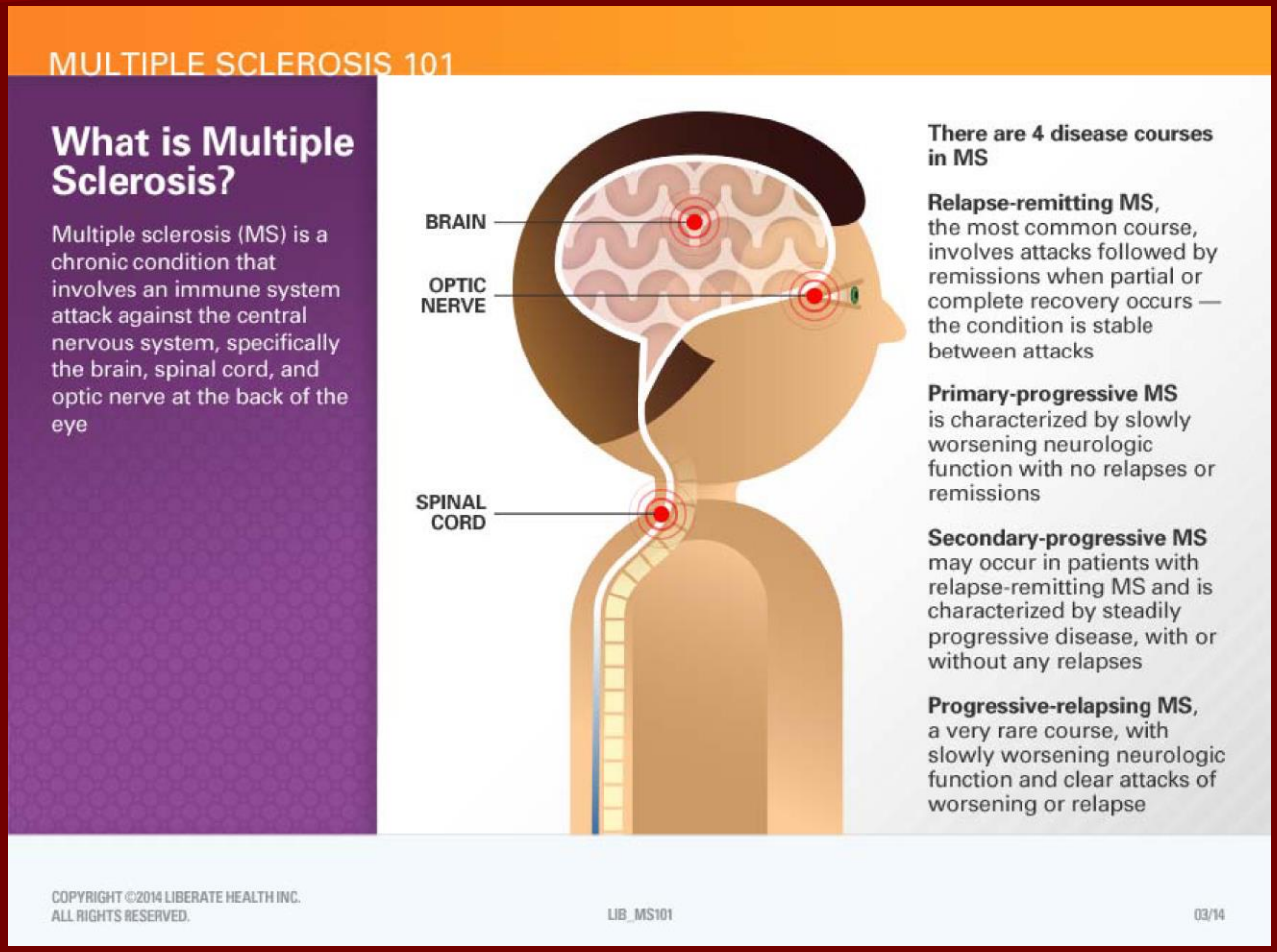
- Multiple Sclerosis
- Parkinson's Disease
- Guillain-Barré Syndrome
- Myasthenia Gravis
- Amyotrophic Lateral Sclerosis
- Postpolio Syndrome

Objectives

Participants will be able to:

- **Describe scope of each neurological disease/condition**
- **State Symptoms of each neurological disease/condition**
- **Identify the Goals of the Rehabilitation Nurse related the specific neurological disease/condition**
- **Describe the Nursing Interventions for each neurological condition**

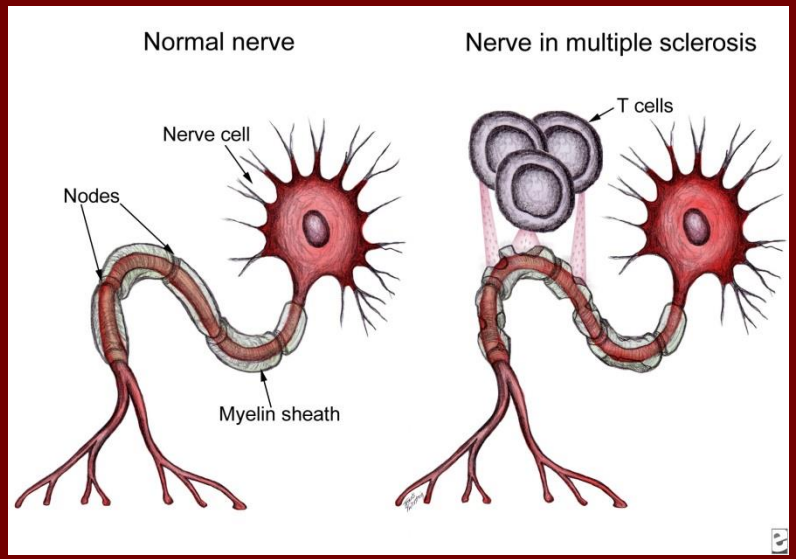
Multiple Sclerosis



Management Options

- Goal

  To decrease the number and frequency of relapses, enhance recovery from exacerbations, alleviate symptoms, maintain independence and ensure the highest quality of life.



Epidemiology

- Major cause of disability and economic hardship in young adults 20-50 years of age.
- Incidence
  - The average person in the United States has about one in 750 (.1%) chance of developing MS.
  - 2.3 million worldwide
- Approximately 200 people are diagnosed weekly
- More women than men
- MS occurs in most ethnic groups,
  - more common in Caucasians of N. European ancestry.

Epidemiology

- Occurs more often
  - In women than men
  - In people who live in colder northern latitudes
  - In people who have 1st degree relatives with MS
- Population of children with MS is increasing, statistics suggest that there may be 20,000 undiagnosed children in the U.S. because healthcare providers do not associate MS with children

Disease Progression

- Average age of onset- 30, most diagnosis between 15-50
- Onset after 40,
more likely is Primary Progressive

Etiology

- Specific cause remains unknown
- Factors that may be involved in causing MS are:
- Viral

  A latent viral infection may cause inflammation of white matter or trigger an autoimmune reaction that precipitates demyelination
- Immunologic

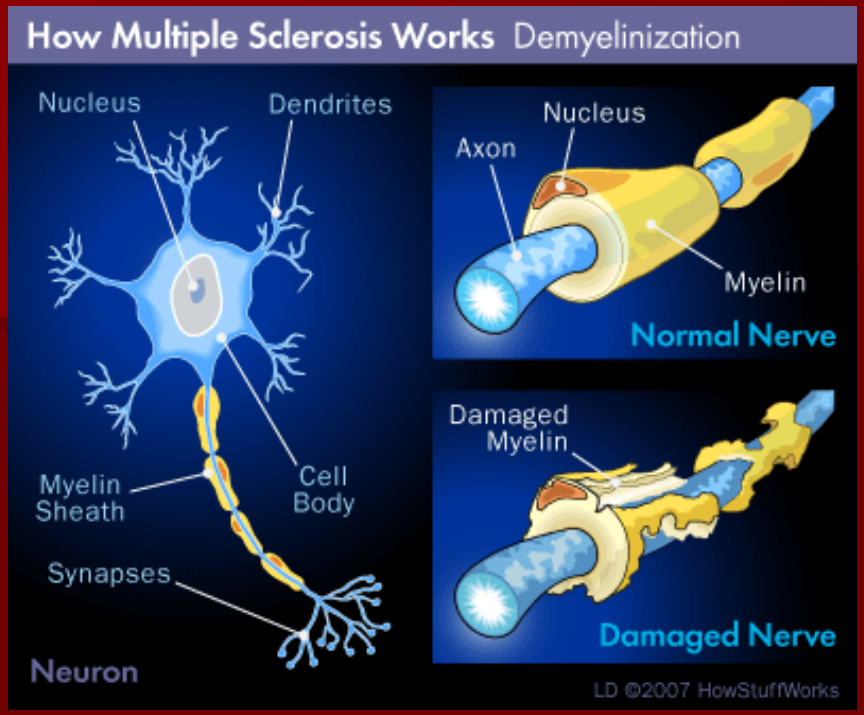
Etiology

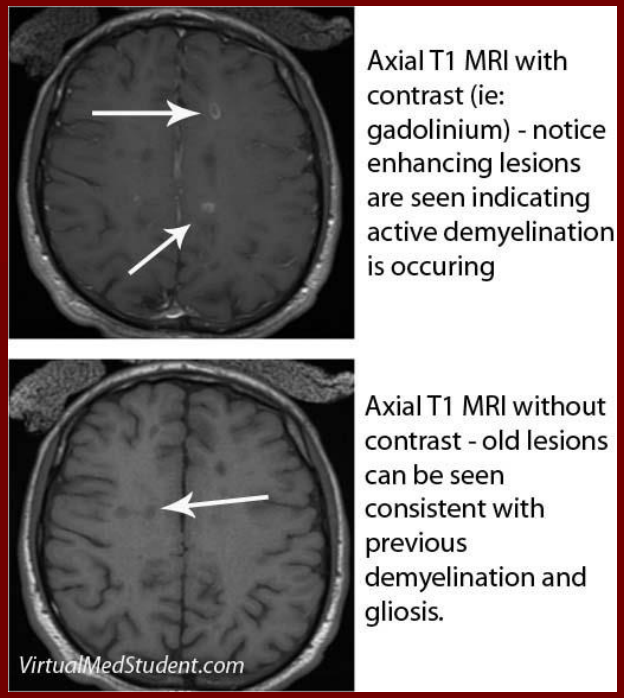
- **Genetic susceptibility**

  There is no specific genetic pattern of transmission for MS, researchers support a multigenic predisposition that makes certain people susceptible to MS

- **Stressors have been suggested as triggers**

  Emotional stress
  Fatigue
  Pregnancy
  Secondary illness
  Viral infection
  Trauma
  Extreme physical exertion





Normally the blood brain barrier protects the brain from immune-cell attack. In MS, activated T Cells migrate into the CNS- starting an antibody-antigen response leading to inflammation. The myelin is attacked, oligodendrocytes disappear and astrocytes remove damaged myelin forming scar tissue.

Multiple Sclerosis

Myelin is lost in multiple areas, leaving scar tissue

- **A chronic neuroimmunologic condition that affects the white matter of the central nervous system.**
- **Affects primarily adults in the prime years of life**
- **Characteristics**
  - **Numerous etiologic possibilities**
  - **Uncertain prognosis**
  - **Episodes of remission and relapse**

Multiple Sclerosis

- Unpredictable disease that results in diverse neurologic impairments, requires a collaborative approach to care
- Associated with Signs and Symptoms caused by the loss of myelin sheath integrity that interferes with the efficiency of nerve impulse conduction in the CNS

Multiple Sclerosis

- Involves partial or complete destruction of the myelin sheath followed by sclerotic plaques or scar tissue formation
- Lesions in the CNS are called Plaques.

  Plaques consist of complete and incomplete destruction of the myelin, lesser degree of damage to axis cylinders or neurons, proliferation of glial cells, changes in blood vessels. Older lesions become sclerotic.(ARN, 2002)

- In 1996 the United States National Multiple Sclerosis Society standardized the following four subtype definitions:
  - Relapsing-remitting-
  - Secondary progressive
  - Primary progressive
  - Progressive relapsing
  - Clinically-isolated syndrome (2001)

Clinically isolated syndrome (CIS)

- First episode of neurologic symptoms that lasts at least 24 hours and is caused by inflammation or demyelination in the central nervous system. The episode usually has no associated fever or infection and is followed by a complete or partial recovery.

CIS can be either monofocal or multifocal:

- **Monofocal episode**: The person experiences a single neurologic sign or symptom — for example, an attack of optic neuritis — that's caused by a single lesion.

- **Multifocal episode**: The person experiences more than one sign or symptom — for example, an attack of optic neuritis accompanied by numbness or tingling in the legs — caused by lesions in more than one place.

Clinically isolated syndrome (CIS)

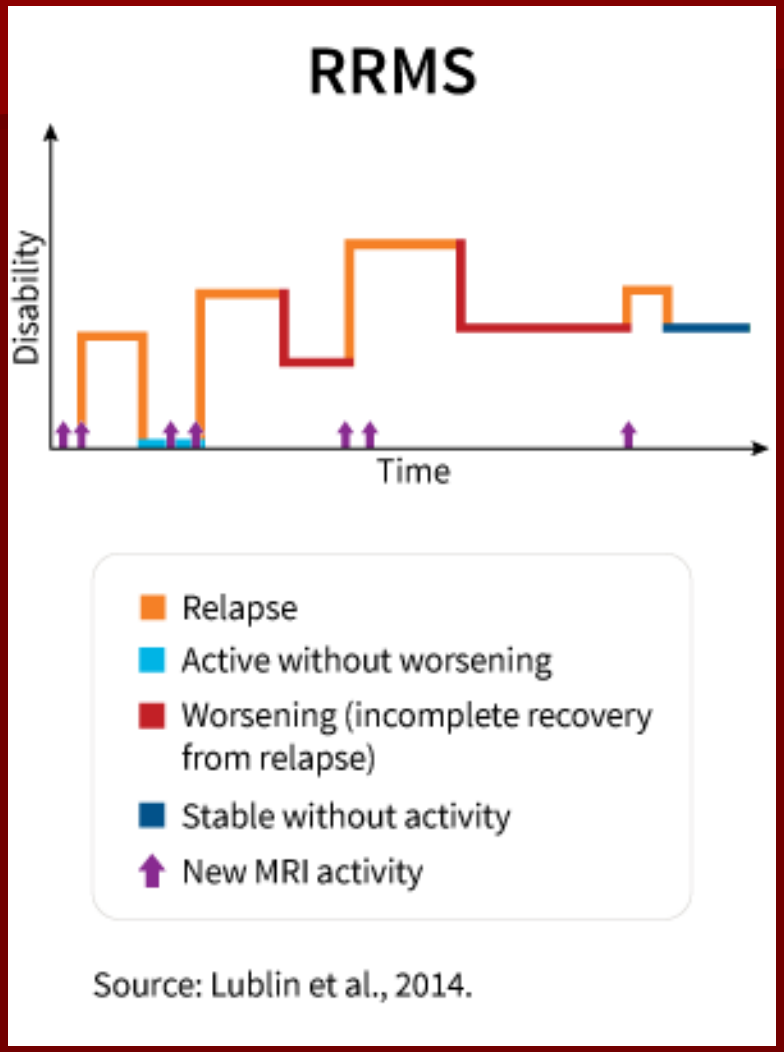
- **If MRI-detected brain lesions similar to those in MS are present then the risk of developing MS is High:** *the person has a 60 to 80 percent chance of a second neurologic event and diagnosis of MS within several years.*

- **If there is no MRI-detected brain lesions then the risk of developing MS is Low** : *the person has about a 20 percent chance of developing MS.*

Studies have shown that early treatment with disease-modifying medications may decrease the risk, or delay the occurrence, of a second exacerbation.

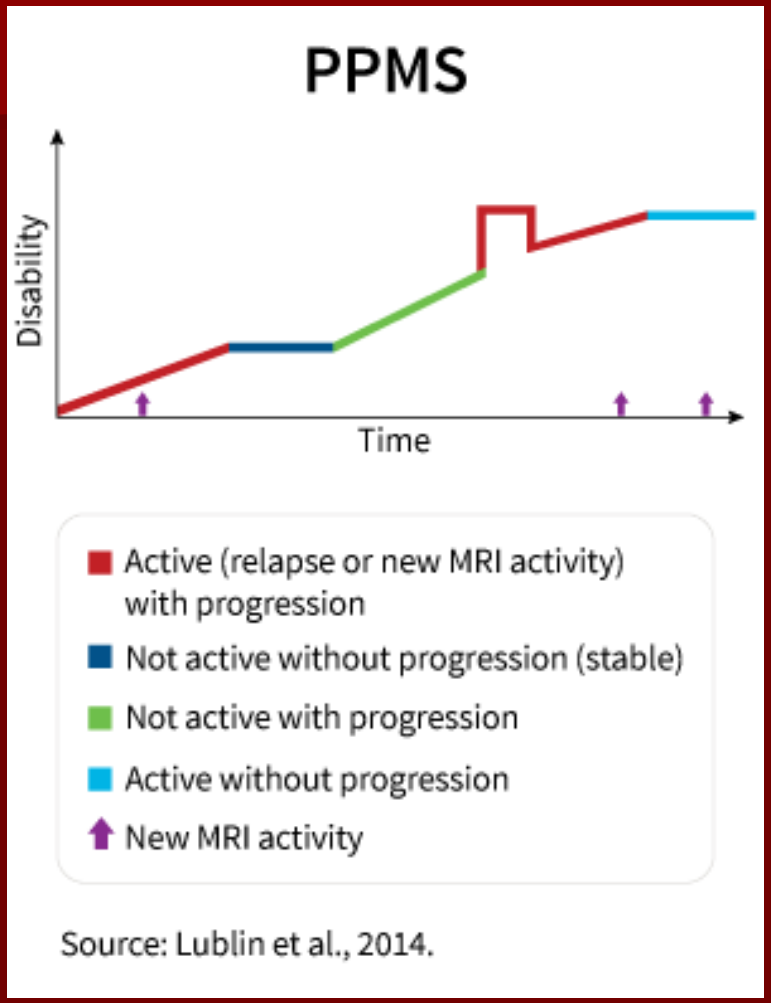
Results from these studies have led to FDA approval of several disease-modifying treatments to be used by people diagnosed with CIS.

Relapsing-remitting MS (RRMS).



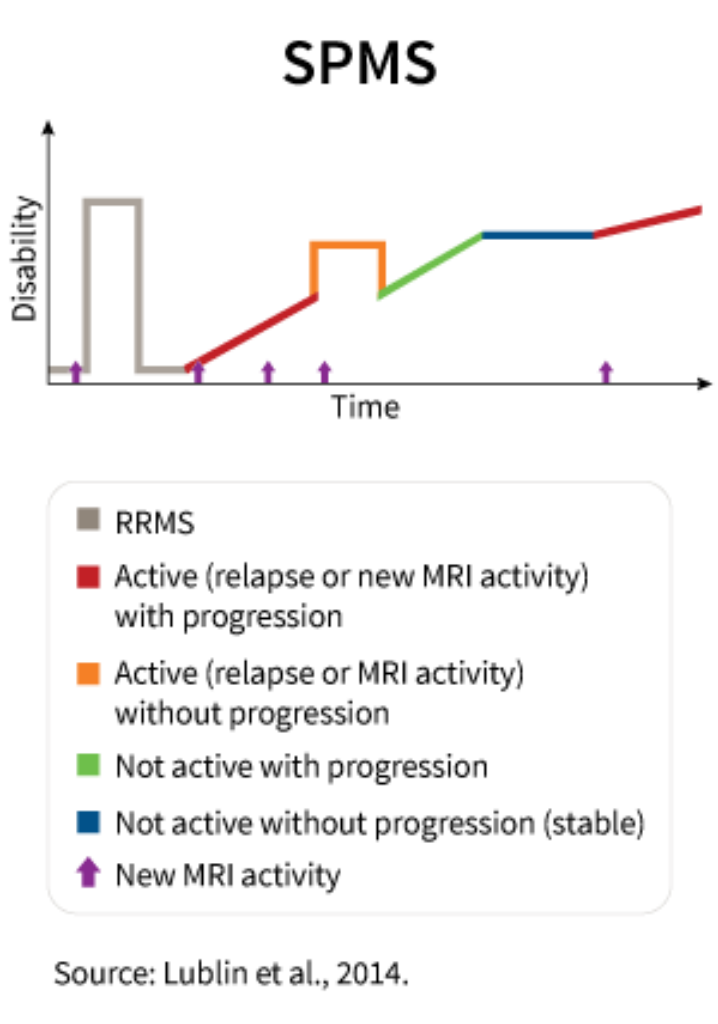
The most common MS course, RRMS may now also be described either as active—meaning the individual is experiencing a relapse and/or new MRI activity—or as not active, meaning that no disease activity is occurring. However, RRMS characterized as "not active" may still be worsening, if there is a confirmed increase in disability due to symptoms persisting after a relapse. Conversely, doctors may characterize a person's RRMS as active but not worsening if they see new MRI activity, but no increase in clinical symptoms.

Primary progressive MS (PPMS).



PPMS is characterized by steadily worsening neurologic function or disability from the onset of symptoms. A diagnosis of PPMS may be further modified at any point in time as active, with new MRI activity and/or relapses, or as not active. In addition, both active and not active PPMS may be further modified as with progression, meaning there is objective evidence of sustained worsening over time, or without progression. Active PPMS may still be described as "without progression" if there are new lesions on MRI, but no observable increase in disability

Secondary progressive MS (SPMS)



Like PPMS, SPMS is characterized by a progressive worsening of neurologic function; however, unlike PPMS, SPMS follows an initial relapsing-remitting course. It can be characterized at different points in time as active or not active, as well as with progression or without progression. As with active PPMS, people with active SPMS should discuss treatment with a DMT with their healthcare providers.

Management of MS uses a comprehensive, interdisciplinary approach that encompasses:

- relapse management
- disease modification
- symptom management
- rehabilitation
- psychosocial support
- wellness

Management

Clinical Manifestations

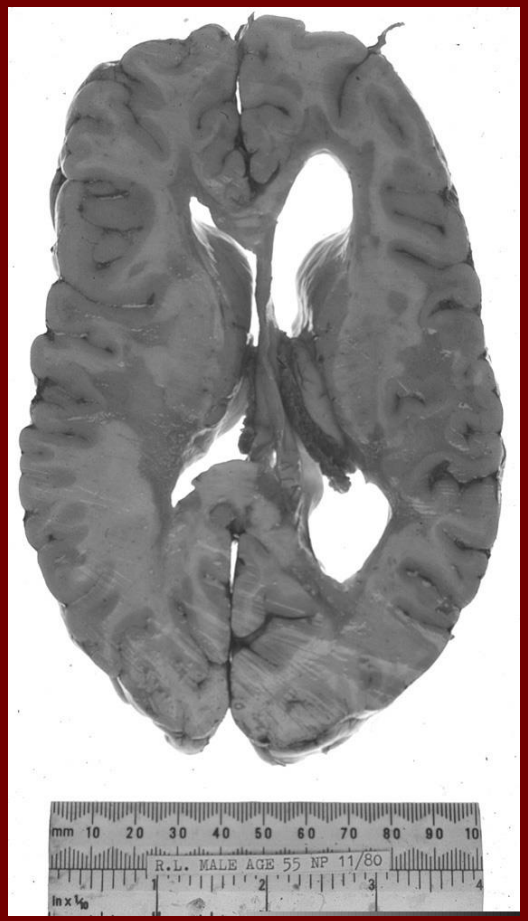
- **Primary Symptoms**
  - Occur as result of nerve conduction deficits
  - Reflect a specific area of dysfunction in the CNS
  - Range from mild to severe, unpredictable, vary from person to person, and time to time in the same person

Primary Signs & Symptoms

- Paresis/paralysis
- Spasticity
- Hyperreflexia
- Vertigo
- Nystagmus
- Dysarthria
- Seizures
- Heat intolerance
- Dysphagia
- Cognitive deficits
- Bladder dysfunction
- Mild to disabling fatigue
- Vision loss, diplopia
- Sensory loss, paresthesia
- Balance disturbances, ataxia
- Numbness, tingling, pain and tremors
- Euphoria, depression
- Hearing deficits, tinnitus

Clinical Manifestations

- **Secondary Symptoms**
  - Occur as a consequence of primary symptoms
  - Include problematic complications resulting from decreased neurologic function



Secondary Signs and Symptoms

- Injuries
  - Falls, skin breakdown, contractures, fractures
- Self Care Deficits
- Decreased Safety due to visual deficits
- Interruption in rest, disturbed sleep
- Decline in libido and orgasmic ability
- Urinary tract infections
- Bowel and bladder incontinence or retention
- Gait pattern deficits, communication deficits, swallowing deficits
- Marked decline in healthy and effective coping strategies

Consider Sexuality

Primary Signs & Symptoms

- Paresis/paralysis
- Spasticity
- Hyperreflexia
- Vertigo
- Dysarthria
- Seizures
- Heat intolerance
- Cognitive deficits
- Bladder dysfunction
- Mild to disabling fatigue
- Sensory loss, paresthesia
- Balance disturbances, ataxia
- Numbness, tingling, pain and tremors
- Euphoria, depression

Secondary Signs & Symptoms

- Injuries
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- Bowel and bladder incontinence or retention
- Communication deficits
- Marked decline in healthy and effective coping strategies

Clinical Manifestations

- **Tertiary Symptoms**
  - Evolve as cumulative and detrimental effects of the disease affect all aspects of the person's life

  Include:
  - Psychosocial
  - Vocational
  - Financial
  - Emotional problems

Tertiary Signs and Symptoms

- Loss of job
- Change in Roles
- Social Isolation
- Divorce
- Ineffective coping with anxiety, denial, anger, reactive depression, and suicide
- Loss of financial stability, self-esteem, and self-worth

Diagnosis

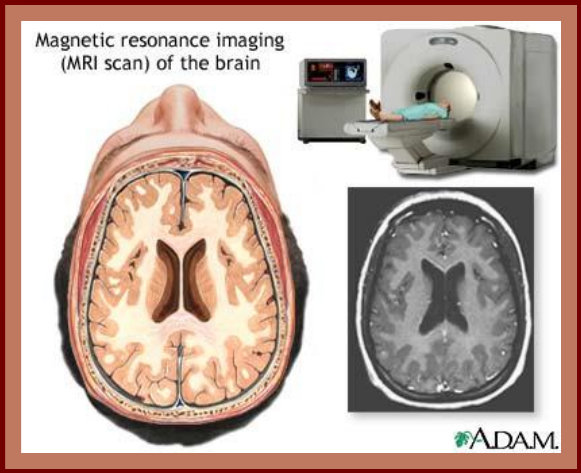
Schumacher Criteria

2012 McDonald Criteria

- History

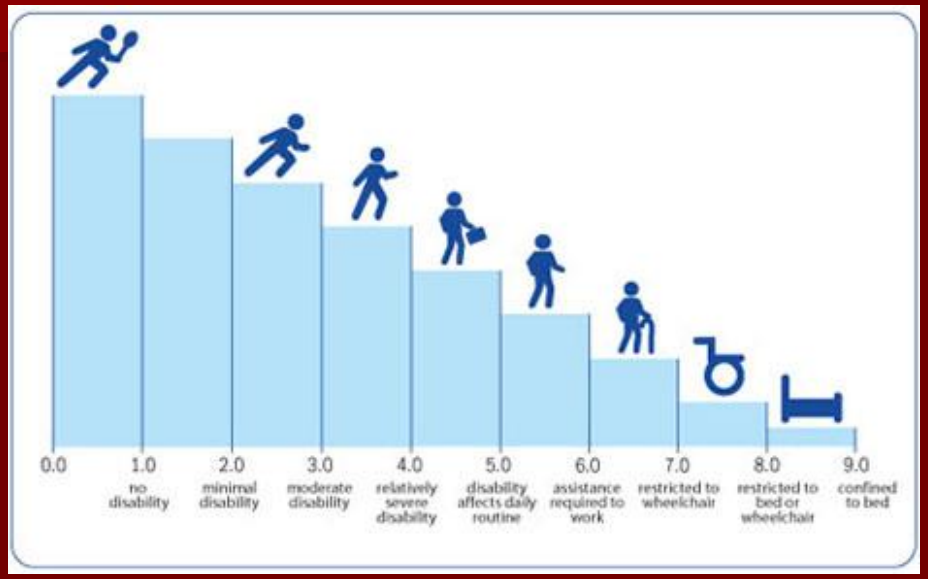
Exam

- No specific Test
  - MRI
  - Evoked Potentials
  - Lumbar Puncture



1: Evidence of damage in 2 or more separate areas of the CNS
(brain, spinal cord, and optic nerves)
+
2: Evidence that the damage happened 1 or more months apart
+
3: The damage did not happen because of another disease
=
Diagnosis of MS

Expanded Disability Status Scale



- The EDSS is a way of measuring physical disability. Two-thirds of people with MS do not progress past level 6, with treatment.

- http://www.nationalmssociety.org/Treating-MS/Medications

- http://www.nationalmssociety.org/NationalMSSociety/media/MSNationalFiles/Brochures/Brochure-The-MS-Disease-Modifying-Medications.pdf

Modifying the disease course

- Injectable medications
  - Avonex (interferon beta-1a)
  - Betaseron (interferon beta-1b)
  - Copaxone (glatiramer acetate)
  - Extavia (interferon beta-1b)
  - Glatopa (glatiramer acetate -- generic equivalent of Copaxone 20mg dose)
  - Plegridy (peginterferon beta-1a)
  - Rebif (interferon beta-1a)
  - Zinbryta (daclizumab)
- Oral medications
  - Aubagio (teriflunomide)
  - Gilenya (fingolimod)
  - Tecfidera (dimethyl fumarate)
- Infused medications
  - Lemtrada (alemtuzumab)
  - Novantrone (mitoxantrone)
  - Tysabri (natalizumab)

PML

- **Treatment of Disease Progression**
  Natalizumab is an effective drug for the treatment of relapsing-remitting multiple sclerosis (RRMS). However, its use is rarely associated with the development of *progressive multifocal leukoencephalopathy* (PML), a potentially fatal complication.

Tsabri

Works by blocking the WBC receptors that allow them to enter the brain and spinal cord , this leads to decreased inflammation

Managing relapses

- For severe relapses (involving loss of vision, severe weakness or poor balance, for example), which interfere with a person's mobility, safety or overall ability to function, most neurologists recommend treatment with corticosteroids.
- Corticosteroids are not believed to have any long-term benefit on the disease.

Medication options include:

- High-dose Intravenous Solu-Medrol® (methylprednisolone)
- High-dose Oral Deltasone® (prednisone)
- H.P. Acthar Gel (ACTH) is an option for those who are unable to cope with the side effects of high-dose corticosteroids, have been treated unsuccessfully with corticosteroids, do not have access to intravenous therapy, or have trouble receiving medication intravenously because of difficulty accessing the veins.

Managing symptoms

Bladder Problems

- **Dysfunction**
  - Botox (onabotulinumtoxin A)
  - DDAVP Nasal Spray (desmopressin)
  - Detrol (tolterodine)
  - Ditropan (oxybutynin), Ditropan XL
  - Enablex (darifenacin)
  - Flomax (tamsulosin)
  - Hytrin (terazosin)
  - Minipress (prazosin)
  - Myrbetriq (mirabegron)
  - Oxytrol (oxybutynin)
  - Pro-Banthine (propantheline)
  - Sanctura (trospium chloride)
  - Tofranil (imipramine)
  - Vesicare (solifenacin succinate)
- **Infection**
  - Bactrim; Septra (sulfamethoxazole)
  - Cipro (ciprofloxacin)
  - Macrodantim (nitrofurantoin)
  - Hiprex (methenamine)
  - Pyridium (phenazopyridine)

Bowel Dysfunction

- Colace (docusate)
- Dulcolax (bisacodyl)
- Enemeez (docusate stool softener laxative)
- Fleet Enema (sodium phosphate)
- Mineral Oil
- Metamucil (psyllium hydrophilic musilloid)
- Phillips Milk of Magnesia (magnesium hydroxide)
- Sani-Supp suppository (gylcerin)

Depression

- Cymbalta (duloxetine hydrochloride)
- Effexor (velafaxine)
- Paxil (paroxetine)
- Prozac (fluoxetine)
- Wellbutrin (bupropion)
- Zoloft (sertraline)

Dizziness and Vertigo

- Antivert (meclizine)

Emotional Changes

- Nuedexta (dextromethorphan + quinidine)

Managing symptoms

- **Fatigue**
  - Amantadine
  - Provigil (modafinil)
  - Prozac (fluoxetine)
- **Itching**
  - Atarax (hydroxyzine)
- **Pain**
  - Dilantin (phenytoin)
  - Elavil (amitriptyline)
  - Klonopin (clonazepam)
  - Neurontin (gabapentin)
  - Pamelor; Aventyl (nortriptyline)
  - Tegetrol (carbamazepine)
- **Tremors**
  - Laniazid - Nydrazid (isoniazid)
  - Klonopin - Rivotril - Syn-Clonazepam (clonazepam)
- **Spasticity**
  - Botox (onabotulinumtoxin A)
  - Dantrium (dantrolene)
  - Gablofen (baclofen [intrathecal])
  - Klonopin (clonazepam)
  - Lioresal (baclofen)
  - Valium (diazepam)
  - Zanaflex (tizanidine)
- **Walking (Gait) Difficulties**
  - Ampyra (dalfampridine)
- **Sexual Problems**
  - Cialis (tadalafil)
  - Levitra (vardenafil)
  - Papaverine
  - MUSE (alprostadil)
  - Prostin VR (alprostadil)
  - Viagra (sildenafil)

Integrative Medicine

- Tai chi
- Yoga
- Acupuncture
- Cooling Strategies
- Nutritional intervention
  - Prevent constipation
  - Healthy Diet
  - Vitamin D
  - Cannabis
    - Cognition- worse
    - Spasticity- better
    - Progression- More Research Needed

Nursing Process

- **Assessment**
- **Plan of Care**
- **Goals**
- **Nursing Interventions**

  Improve mobility and neuromuscular function

  Conserve energy

  Maintain independence in ADLs

  Improve Bladder function and prevent complications

  Improve knowledge

  Develop effective coping strategies to adjust to the illness

  Maintain visual functioning

  Promote comfort

Nursing Process

- **Assessment**
  - **Physical**
    - **Observe physical appearance**
      - **Spasticity**
      - **Weakness**
      - **Incontience**
      - **Visual impairments**
    - **Medication reconciliation**
      - **Prescription medications**
      - **Compliance**
      - **Alternative medications**

Nursing Process

- **History**
  - **Current symptoms**
  - **Time of onset**
  - **History of relapses**
  - **Recent or past viral infections**
  - **Stress**
  - **Pregnancy**
  - **Exposure to extreme temperatures**
  - **Self Care Deficits**

Plan of Care: Nursing Diagnosis

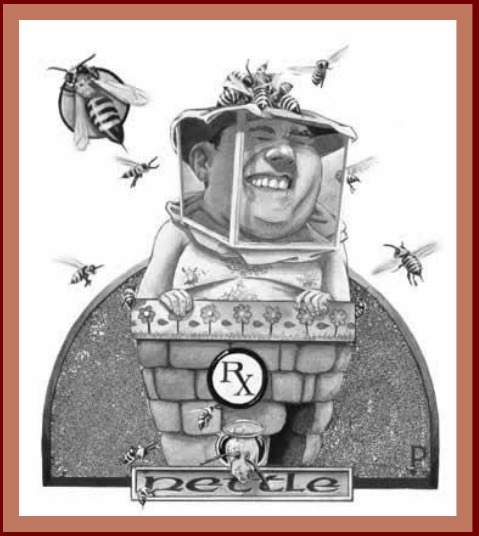
- Impaired physical mobility re: neuromuscular impairment
- Fatigue re: MS disease process
- Self-care deficits re: weakness, spasticity, and tremor
- Altered urinary elimination re: spinal cord involvement and decreased functional ability
- Knowledge deficit re: the variable nature of symptoms and multifaceted treatment options
- Ineffective individual coping re: the variability of the disease course, cognitive impairments, decreased independence and changes in family and vocational roles
- Sensory perception alterations: visual, re: optic nerve involvement
- Chronic pain re: neuropathy

Goals

- Maintain maximal level of mobility
- Demonstrate safety in mobility and recognize need for appropriate assistive devices
- Conserve energy and verbalize understanding of ways to integrate energy conservation principles into ADLs & daily activities
- Attain maximal level of function in ADLs
- Maintain continence and identify symptoms of UTI
- Verbalize understanding of the disease process, significant implications and prescribed regimens
- Verbalize appropriate plans for coping with stress
- Attain maximal visual functioning and demonstrate satisfactory use of compensatory measures when needed
- Verbalize satisfactory pain relief

Nursing Interventions

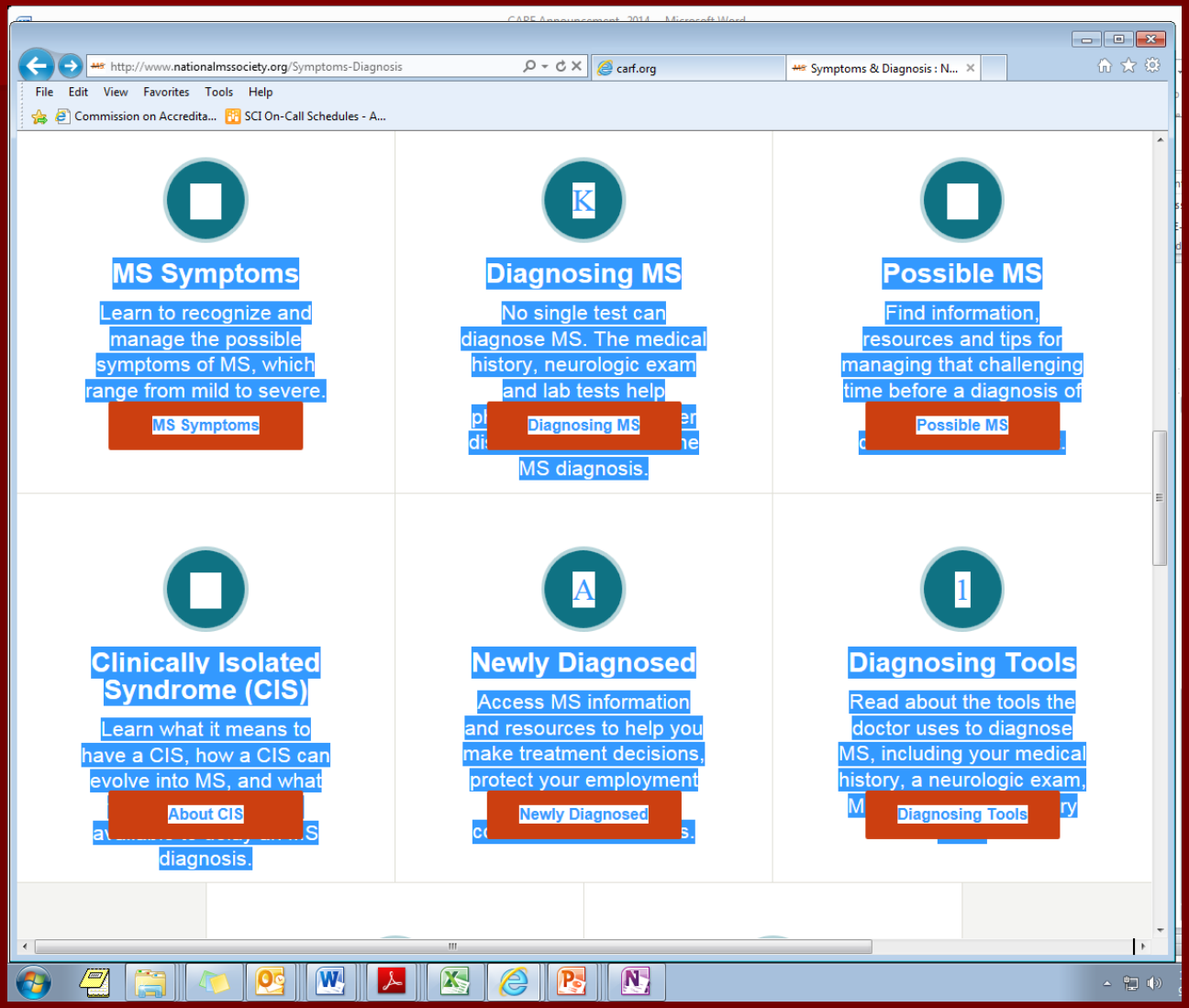
- Improve mobility and neuromuscular function.
- Conserve Energy
- Maintain independence in ADL's
- Improve bladder function and prevent complications
- Improve knowledge
- Develop effective coping strategies to adjust to the illness
- Maintain visual functioning
- Promote comfort



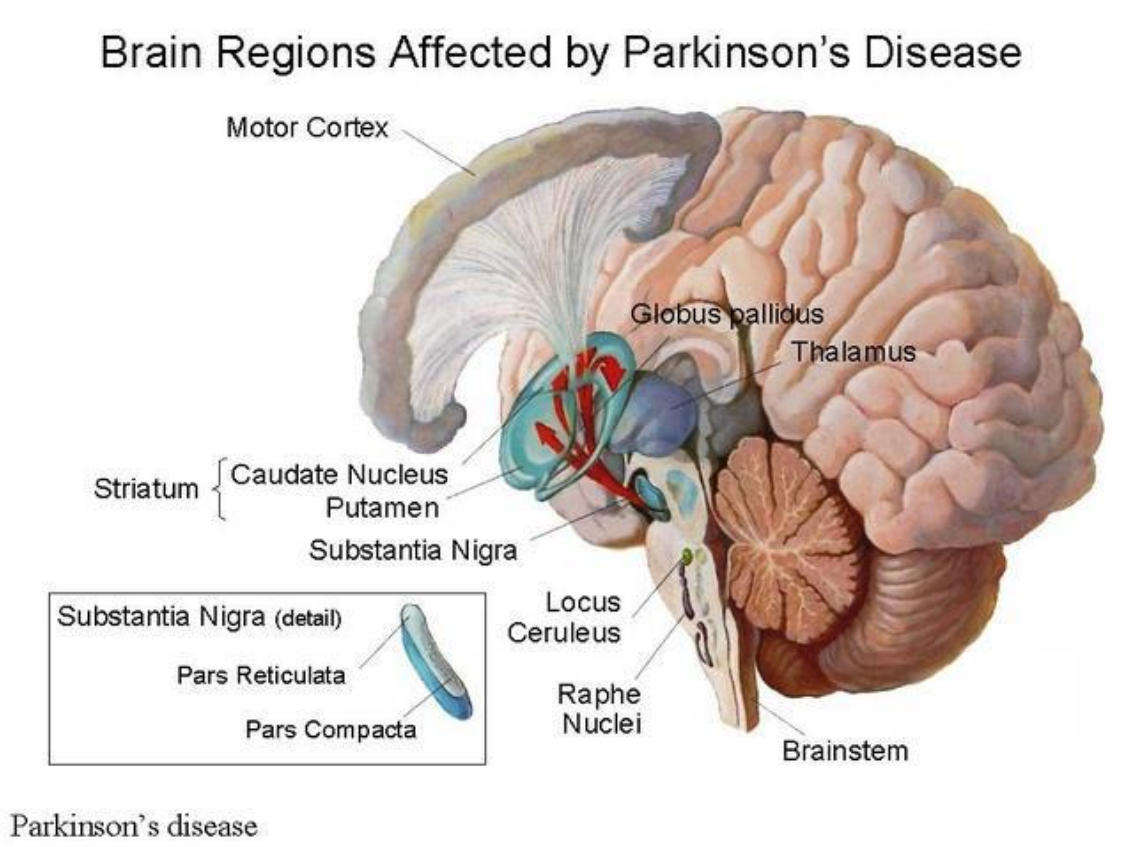
Case Study: Joanna, Nurse with new diagnosis of MS

- Questions
- What is the role of the APN in this case, as a colleague and as a rehabilitation specialist?
- What nursing interventions should take priority in this case?
- What can you anticipate will be the outcome of this acute episode?
- What safety issues can you identify, and how may they be addressed?
- What long term needs can you address at this stage in the course of Joanna's disease?
- How can you best support the staff in the ICU?
- What medications may be helpful for long-term management of Joanna's disease?

http://www.nationalmssociety.org/Symptoms-Diagnosis/MS-Symptoms



Parkinson's Disease



Parkinson's disease

Parkinson's Disease

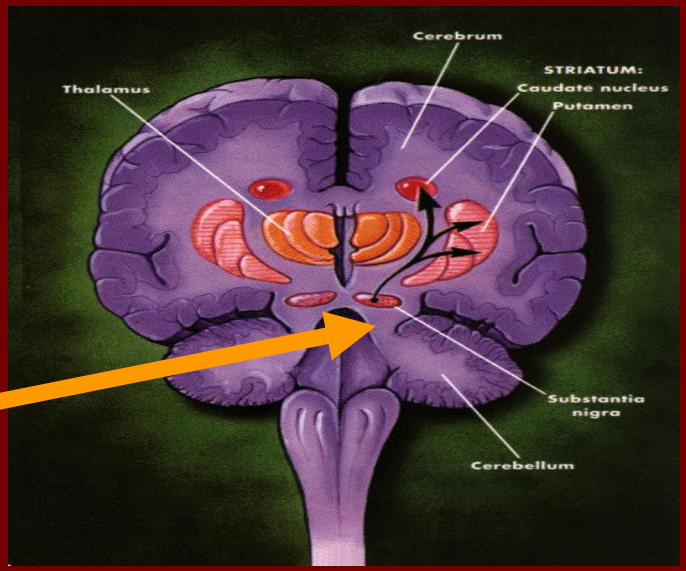
- Slowly progressive neurodegenerative disease of the brain
- Manifestations that occur when there is significant damage to or destruction of dopamine-producing neurons in the substantia nigra within the basal ganglia of the brain
- Begins insidiously has a prolonged course of illness
- Loss of dopamine causes neurons to fire out of control, leading to marked disability with the iitiation and execution of smooth coordinated voluntary movements and balance.
- There is no known way to stop or cure PD
- Common Chronic diseases of the nervous system

Parkinson's Disease

- **Primary Parkinson's Disease**

  Chronic debilitating deficiency caused by an idiopathic dopamine deficiency in the basal ganglia of the brain

- **Clinical syndrome with 3 cardinal features**
  - Tremor at rest
  - Rigidity
  - Akinesia/bradykinesia
  - Postural instability



- **Secondary Parkinsonism**

  Group of symptoms where there is a known cause of injury to the dopamine-producing cells

Epidemiology and Incidence

- 1 million Americans have Parkinson's Disease
- Approximately 60,000 new cases are diagnosed annually in the U.S.
- Approximately 4 million people world wide are living with PD
- Men slightly more often then women
- Most commonly after 55, likely to increase in an aging population
- Approximately 10% of people with Parkinson's Disease are younger than 40 years of age, incidence in younger persons is growing

Etiology

- **Primary Parkinson's Disease**

  idiopathic

- **Secondary Parkinsonism**

  Response to antipsychotic, antihypertensive or neuroleptic agents

  - Illicit drug use
  - Response to brain trauma
  - Tumors
  - Ischemia
  - Encephalitis infections
  - Arteriosclerosis

- Neurotoxins
  - Cyanide
  - Manganese
  - Carbon monoxide
  - Pesticides

Pathophysiology

- Braak hypothesis proposes that the earliest evidence of PD is found in the medulla and olfactory bulb and then progresses to the substantia nigra and cortex.
- Degenerative changes in several areas in the basal ganglia deplete the inhibitory neurotransmitter dopamine, normally provided to the basal ganglia by the neurons in the substantia nigra
- Dopamine is a neurotransmitter essential for the functioning of the extrapyramidal system, which includes control of upright posture, support and voluntary motion.

Pathophysiology

- Normal function is due to a balance between the neurotransmitters dopamine and acetylcholine, responsible for controlling and refining motor movements and have opposing effects
- An increase in the excitatory effects of Acetylcholine , caused by the depletion of dopamine causes the symptoms of PD
- As the disease progresses, dopamine receptors in the basal ganglia are reduced

Diagnosis: Parkinson's Disease

- First sign usually a resting tremor
  - Patient history and symptoms
- No lab test
- PET scan can detect low levels of dopamine but not usually done
- If Sinemet has a positive response + Parkinson's Disease
- Typical onset Men age 50-60

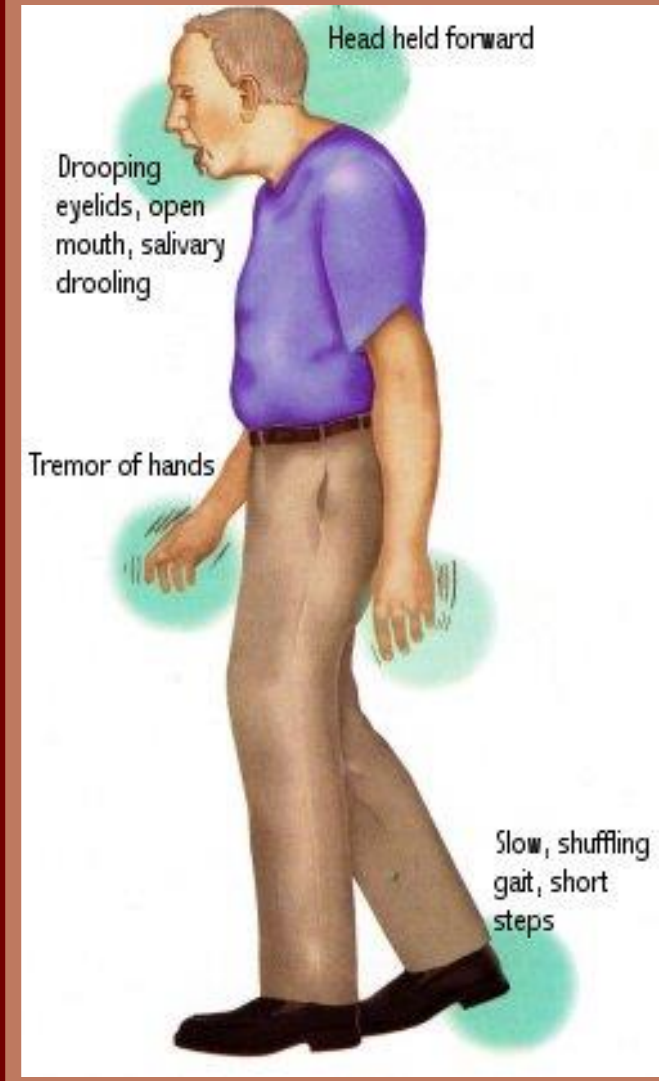
- Tremor
  - Due to instability of central feedback circuit
  - Resting Tremor- goes away with active movement
- Rigidity
  - Due to increased resting muscle activity
- Akinesia
  - Failure of system that plans complex movement
- Postural Instability
  - Impaired Balance

*Dementia
  - Cognitive difficulties part of general slowness
  - Apathy most common cognitive change
  - Can have cortical dementia
  - Subcortical dementia less common

"TRAP"

The stages of Parkinson's

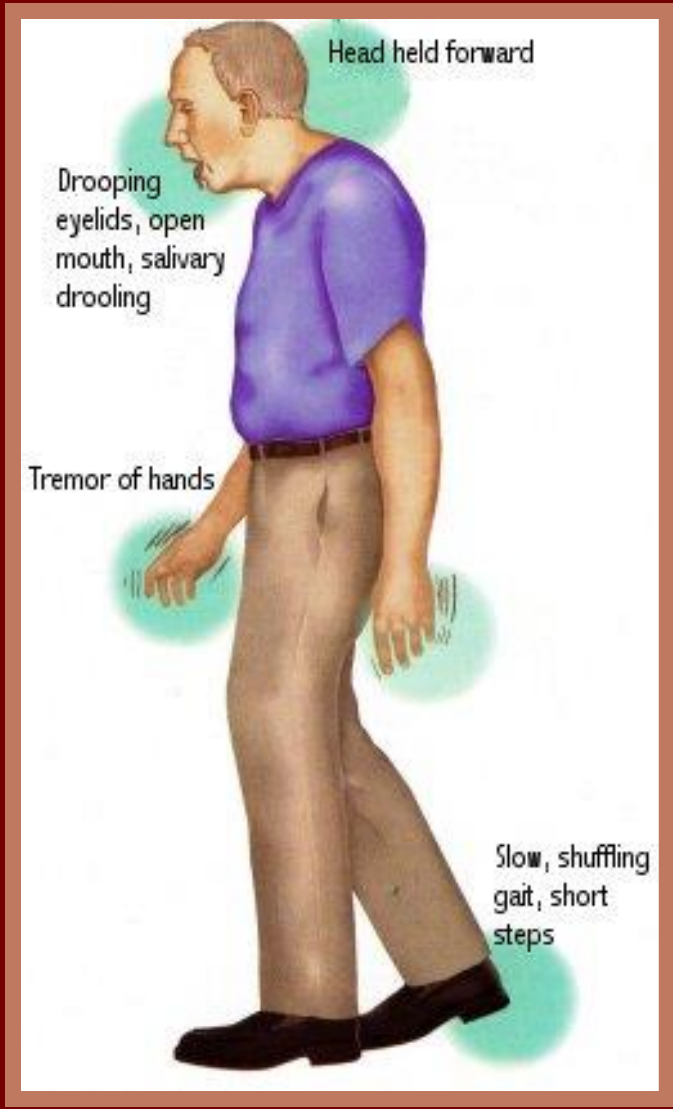
- **Stage One**
  1. Mild signs and symptoms on one side
  2. Symptoms inconvenient but not disabling
  3. Usually presents with tremor of one limb
  4. Friends have noticed changes in posture, locomotion, and facial expression



The stages of Parkinson's

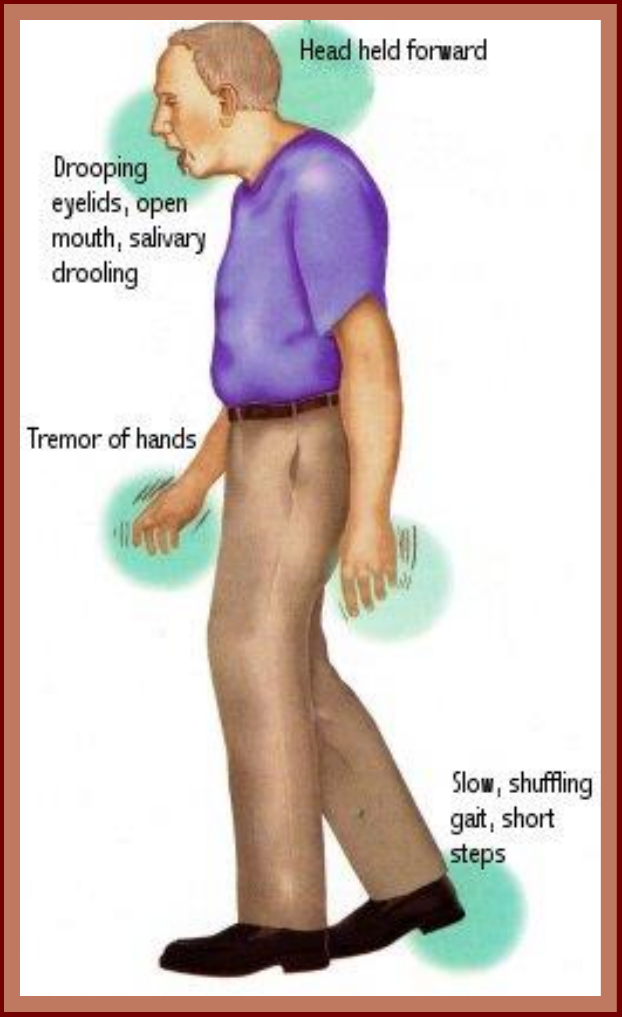
Stage Two

1. Symptoms are on both sides (bilateral)
2. Minimal disability
3. Posture and gait affected
4. Responds well to medication



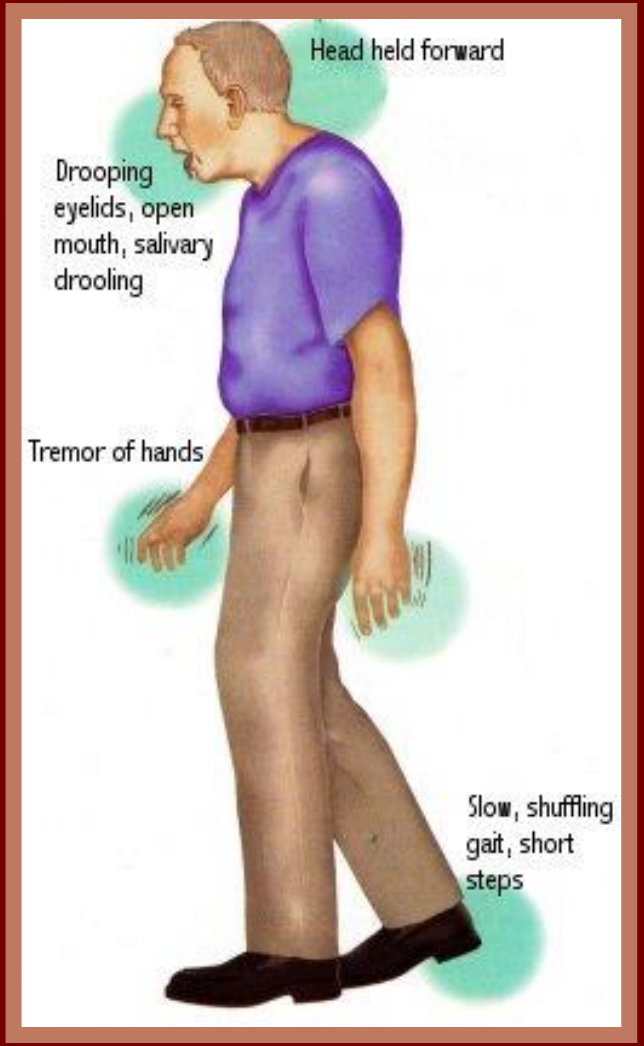
The stages of Parkinson's

- **Stage Three**
  1. Significant slowing of body movements
  2. Early impairment of balance on walking or standing
  3. Generalized disability; moderately severe
  4. Predictable "wearing off" effects of medication, on-off fluctuations, and dyskinesias



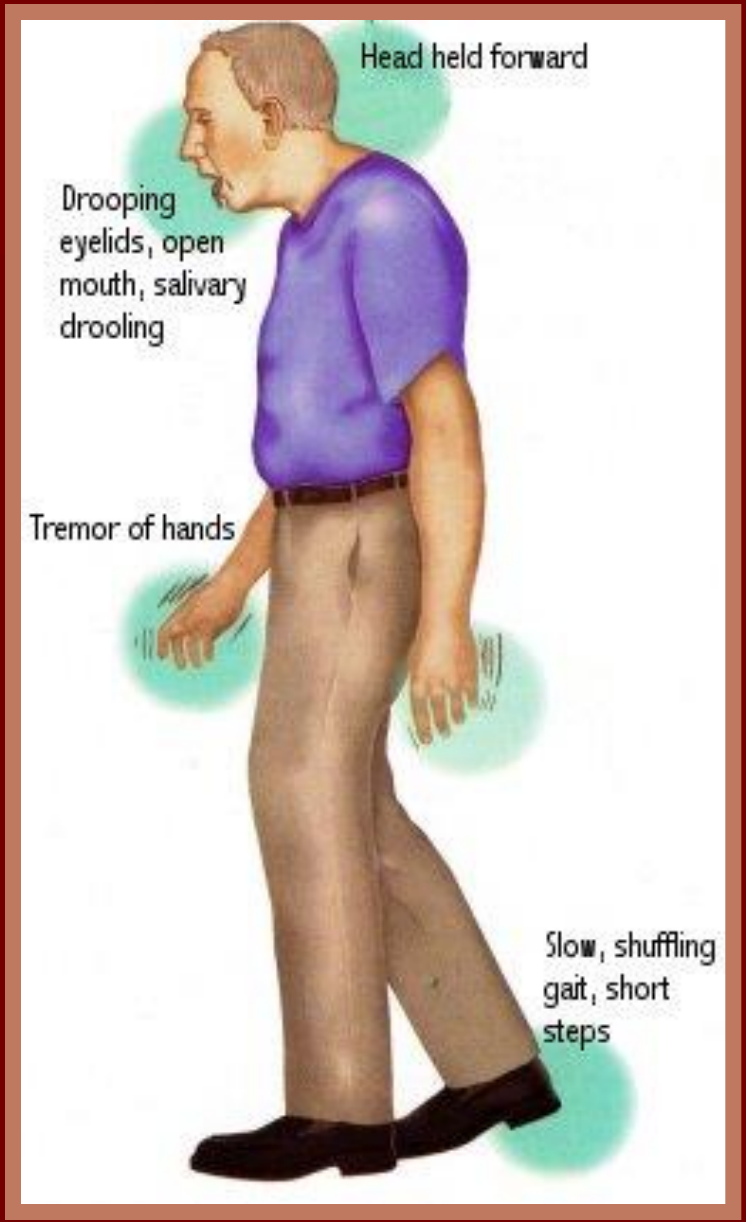
The stages of Parkinson's

- **Stage Four**
  1. Severe symptoms
  2. Can still walk to a limited extent
  3. Rigidity and bradykinesia
  4. No longer able to live alone
  5. Tremor may be less than earlier stages
  5. On-off fluctuations of medication with dyskinesias



The stages of Parkinson's

- **Stage Five**
  1. Cannot stand or walk
  2. Motor fluctuations and cognitive impairment
  3. Requires constant nursing care



Hoehn and Yahr Staging

- **Stage 1** -- symptoms are only on one side of the body
- **Stage 2** -- symptoms are on both sides of the body, but balance isn't impaired.
- **Stage 3** -- there is some balance impairment and disability.
- **Stage 4** -- disability is severe, but the person can still walk or stand without help.
- **Stage 5** -- the person cannot stand or walk, and is wheelchair-bound or bedridden.

Unified Parkinson's Disease Rating Scale

- **Intellectual impairment**, depression, motivation
- **Activities of daily living** (speech, swallowing, handwriting, cutting food, dressing, hygiene and walking)
- **Motor skills** (speech, facial expression, tremor, rigidity, posture and walk)

Each function is rated on a four-point scale, from normal (0) to significant problems (4).

There are a total of 199 points, with 0 being no disability and 199 being total disability

Medications-

- **Medications Approved for the Treatment of Parkinson's Disease in the USA**
  - **Handout**

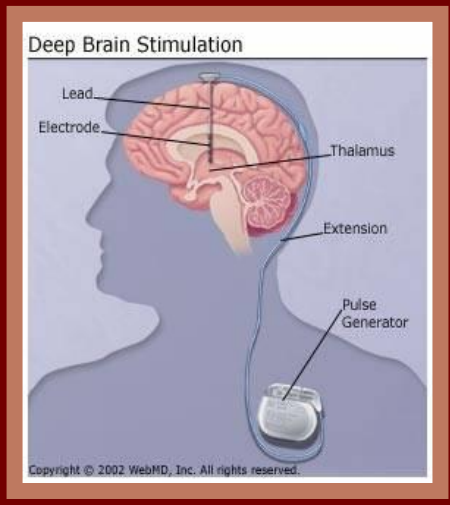
Medications-

- Sinemet (carbidopa levodopa) is made up of levodopa and another drug called carbidopa. Levodopa enters the brain and is converted to dopamine while carbidopa increases its effectiveness and prevents or lessens many of the side effects of levodopa, such as nausea, vomiting, and occasional heart rhythm disturbances.
- It is generally recommended that patients take Sinemet on an empty stomach, at least 30 minutes before, or one hour after meals.

Medications-

- **Optimize delivery of Levodopa to the brain by blocking COMT, which breaks down dopamine in the digestive system, allowing a steady supply of Levadopa to reach the bloodstream. This class of drug includes tolcapone (Tasmar) and entacapone (Comtan).**
- **Reduce the activity of the neurotransmitter acetylcholine. This class of drug normalizes the balance between dopamine and acetylcholine and includes trihexyphenidyl (Artane) and benztropine (Cogentin).**

- **Surgery**
  - **Deep brain stimulator implants** FDA approved for over a decade.
- **Integrative Medicine**
  - Nutritional diet
  - Exercise: include cardiorespiratory exercise, resistance exercises, flexibility exercises, and gait and balance training (**Tai chi)**
  - Yoga: among the leading alternative therapies in the United States.
  - Massage: to manage their pain and/or stress and provide stimuli to the legs, which is helpful in treating patients afflicted by Restless Leg Syndrome (RLS).
  - Natural medicines



Increased Risk

- **Melanoma**
- In addition to PD, other risk factors for developing melanoma are: male gender, Caucasian race, constant exposure to ultraviolet (UV) light and family history of melanoma.
- Use the melanoma ABCDE's to monitor irregularities in moles and beauty marks:
  - **A**symmetrical
  - **B**orders are uneven or irregular
  - **C**olors such as many shades of brown within the same mole, or even red or blue
  - **D**iameter bigger than the eraser on a pencil
  - **E**volution – grows over time

Increased Risk

- **Neurogenic Orthostatic Hypotension (nOH)**
  - a persistent drop in blood pressure that occurs within three minutes of standing. brings blood to the brain..
- **Pseudobulbar Affect (PBA)**
  - characterized by frequent, uncontrollable outbursts of crying or laughing. Outbursts can be intense and often do not match the situation or the way the person is actually feeling.

Nursing Plan of Care: Assessment

- Complete Health History
- Mental status
- How disease has affected Role, family
- Observe appearance, posture, gait pattern
- Determine level of extremity stiffness, tremors, and ability to move
- Investigate safe mobility, Self-care deficit

Nursing Diagnoses

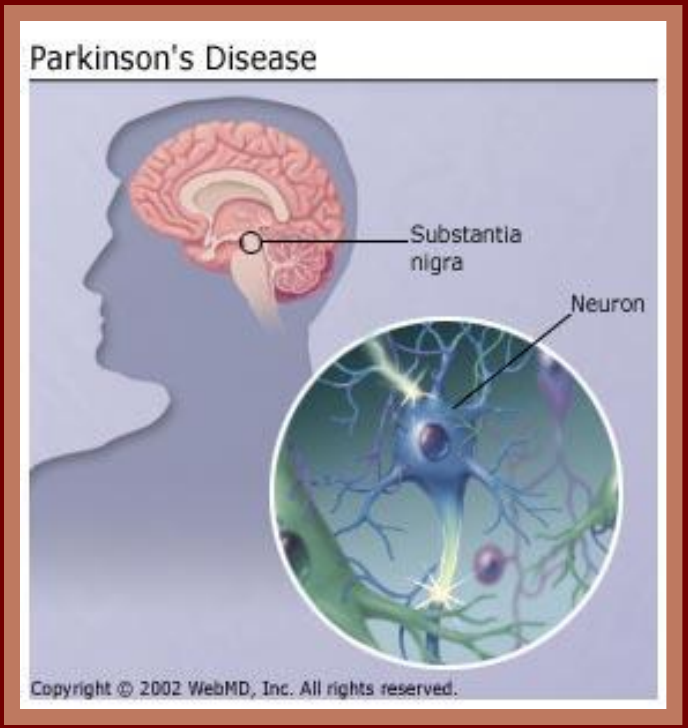
- Ineffective individual coping re: depression and increasingly severe physical limitations
- Knowledge deficit re: disease progression, treatment, ongoing adaptations, and availability of support systems.
- Impaired physical mobility re: tremor, rigidity, bradykinesia or akinesia, and postural instability.
- Self-care deficits re: tremor, rigidity, bradykinesia, and postural inability.
- Inadequate nutrition re: difficulty with chewing, swallowing, and drooling.
- Impaired verbal communication re: low voice, slow speech, and difficulty moving facial muscles.
- Risk of injury re: tremors, bradykinesia, and altered gait.

Interventions

- Develop positive coping mechanisms
- Develop a sound knowledge base about PD
- Improve mobility and maximize neuromuscular function
- Maintain independence in ADL's
- Achieve satisfactory hydration and nutritional status
- Improve verbal communication
- Maintain safety

PD: Summary

- **Rehabilitation Interventions**
  - **Activity**
  - **Freezing episodes intervention (pt. education)**
  - **Aspiration**
  - **Diet- Proteins separate from medications**
  - **Fall Risk**
  - **ADL Deficits**
  - **Body image concerns**
  - **Decreased blink**
  - **Social Isolation**
  - **Dysarthria**
  - **Increased risk for pneumonia**
  - **Family/ Caregiver Stresses**

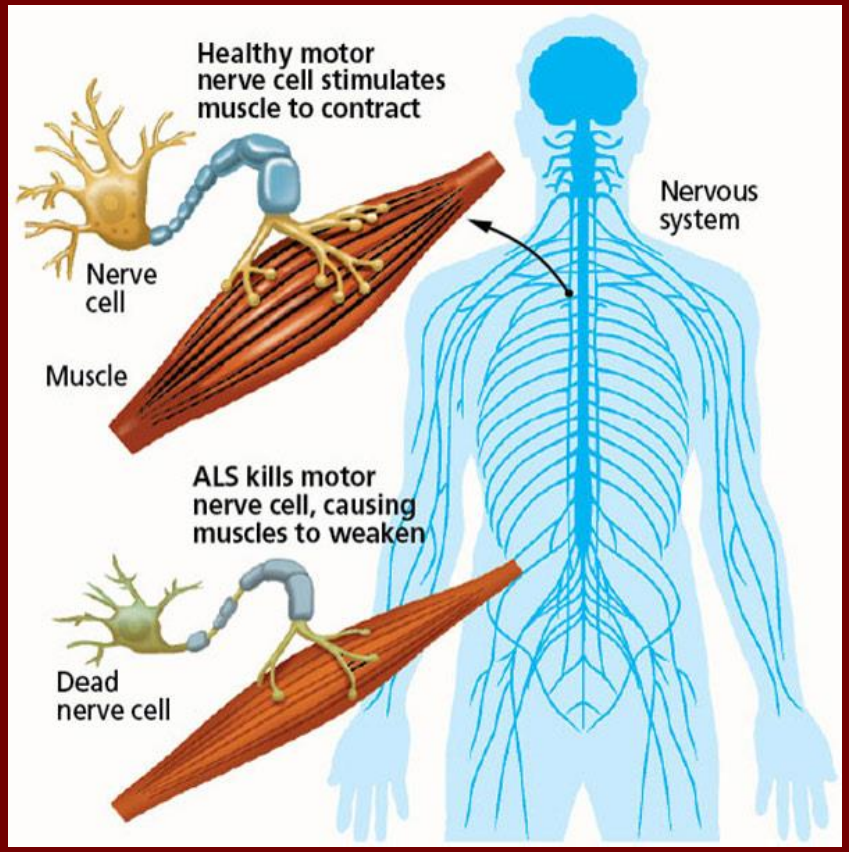


Freezing episodes-Interventions

- Stop trying to continue activity
- Call for help
- Change direction
- Use a sound or rhythm to stimulate movement
- Think of or sing a tune, try to move to beat
- Count silently or out loud, try to move to count
- Visualize an object and try to step over object
- Use floor tiles as stepping stone, try to step stone to stone
- Use flashlight to illuminate floor, try to step into light
- Draw an imaginary line, try to step over line
- If frequently freezes in same place, visualize beyond the obstacle

Amyotrophic Lateral Sclerosis

- Lou Gehrig's Disease



Amyotrophic Lateral Sclerosis

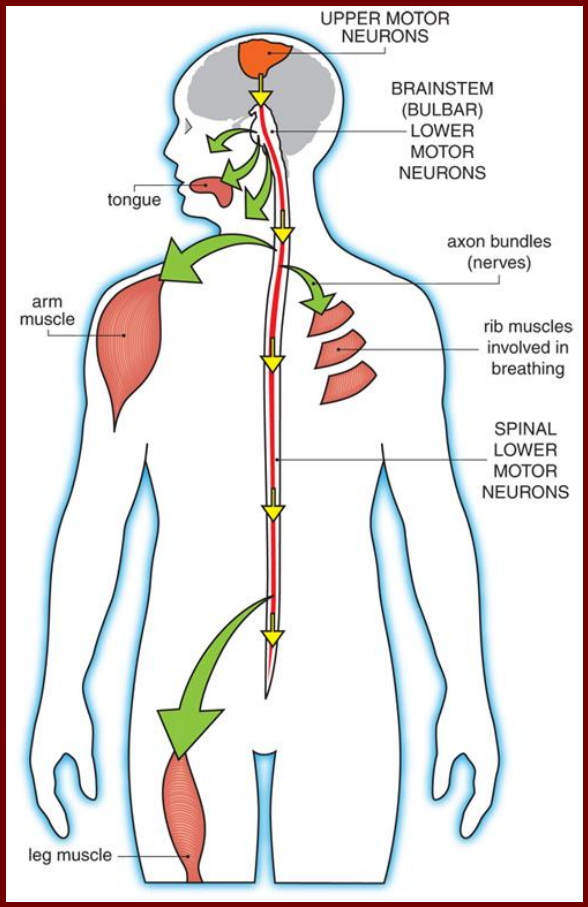
- Neurological disorder with progressive degeneration of skeletal muscle motor/nerve cells throughout the nervous system
- No cerebellar effects
- No sensory loss- posterior nerve roots not affected
- Anterior horn cells of S2 not affected, rare bowel & bladder deficits
- Movement disorder is profound

Variations in Disease Progression

- **Functional Loss may start with Upper-motor neurons, lower-motor neurons, bulbar symptoms only or a combination**
- **Onset is often subtle- first symptoms maybe disregarded**
- **No Single Diagnostic Test to diagnosis ALS**
- **No know Prevention and No known Cure**
- **Treatments to assist in slowing deterioration**

Amyotrophic Lateral Sclerosis

- **Symptoms-**
  - **Upper motor neuron disease**
    - **Weakness**
    - **spasticity**
    - **hyperactive reflexes**
    - **hypertonicity**
    - **Disuse atrophy**
  - **Lower motor neuron disease**
    - **Weakness**
    - **Flaccidity**
    - **Hypoactive reflexes**
    - **Hypotonicity and muscle atrophy**



- Intellectual ability, vision, hearing, and sensation are not generally affected
- Some people do experience cognitive deficits
  - Cognitive impairment can be
    - Frontotemporal dementia with cognitive decline and increased apathy
    - Mild cognitive impairments with no detectable progression

Amyotrophic Lateral Sclerosis

What is ALS?

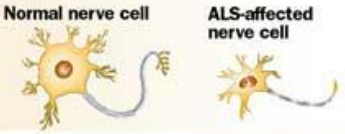
ALS (Amyotrophic Lateral Sclerosis), also known as Lou Gehrig's disease, is a fatal disease of the nervous system, characterized by progressive muscle weakness resulting in paralysis.

What are motor neurons?

Motor neurons are nerve cells in the brain and spinal cord that attach to muscles and control voluntary movement.

How does ALS progress?

When motor neurons gradually degenerate and die, the muscles no longer receive nerve impulses. As a result of the nerve death, the muscles shrink and waste away.



A closer look at a healthy nervous system

Nervous system

The basic unit of the nervous system is a highly specialized cell, known as a neuron. Its main purpose is to transport messages from one part of the body to another in the form of nerve impulses.

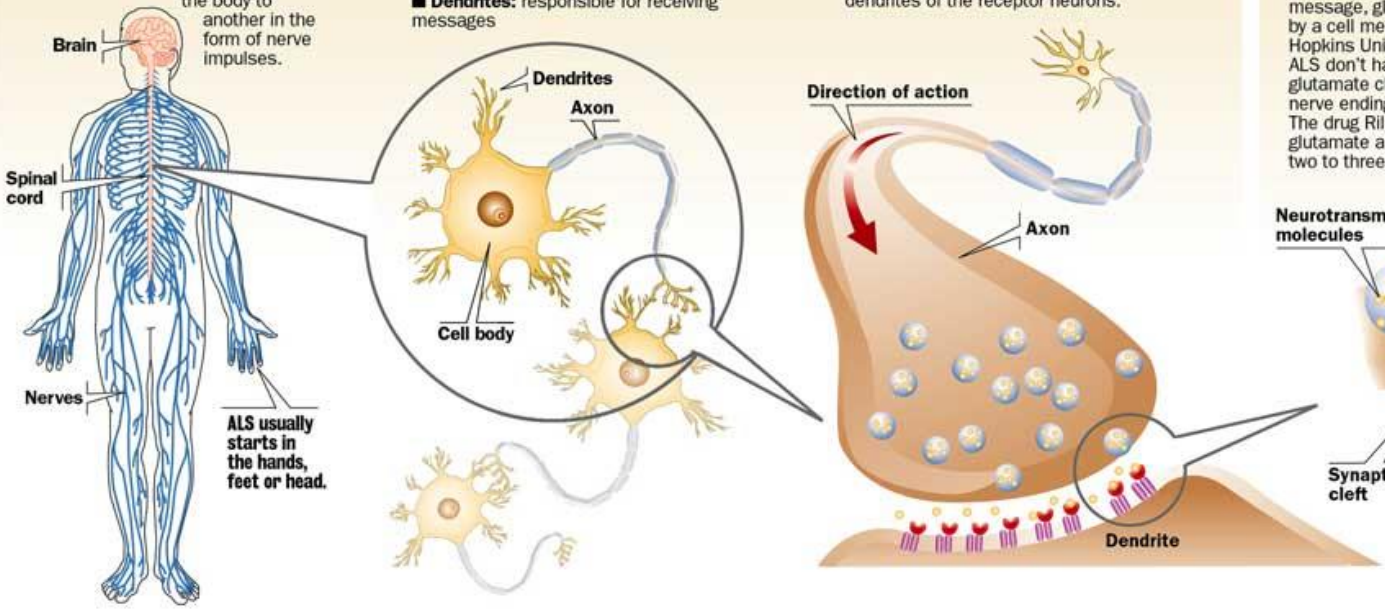
Motor neuron

A motor neuron is made up of three main functional parts.

- **Cell body:** biosynthetic center of the cell
- **Axon:** responsible for sending messages
- **Dendrites:** responsible for receiving messages

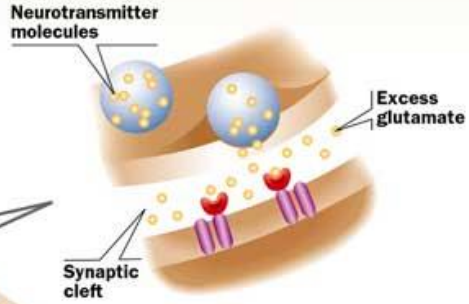
Nerve impulse

A nerve impulse is transmitted when the terminal fibers of one neuron's axon release chemicals called neurotransmitters that attach to dendrites of the receptor neurons.



A possible cause of ALS: Too much glutamate

Scientists aren't sure what causes ALS, but glutamate poisoning is a popular theory. Glutamate is an amino acid that acts as a neurotransmitter, allowing motor neurons to "talk" to one another. After transmitting a message, glutamate is supposed to be vacuumed up by a cell membrane protein. But researchers at Johns Hopkins University in Baltimore suggest people with ALS don't have enough of that protein. Over time, glutamate clogs the synaptic cleft, the space between nerve endings, and chokes motor neurons to death. The drug Rilutek slows the body's production of glutamate and keeps ALS patients alive for an extra two to three months.



Dulcie Teesateskie/Huntsville Times

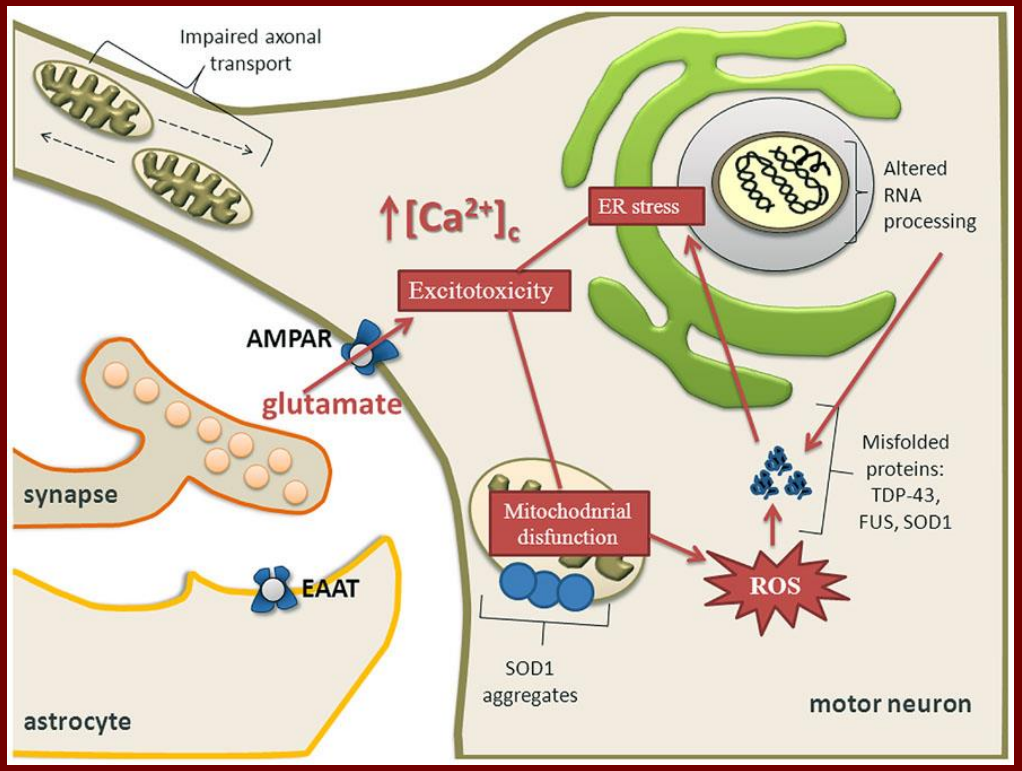
Amyotrophic Lateral Sclerosis

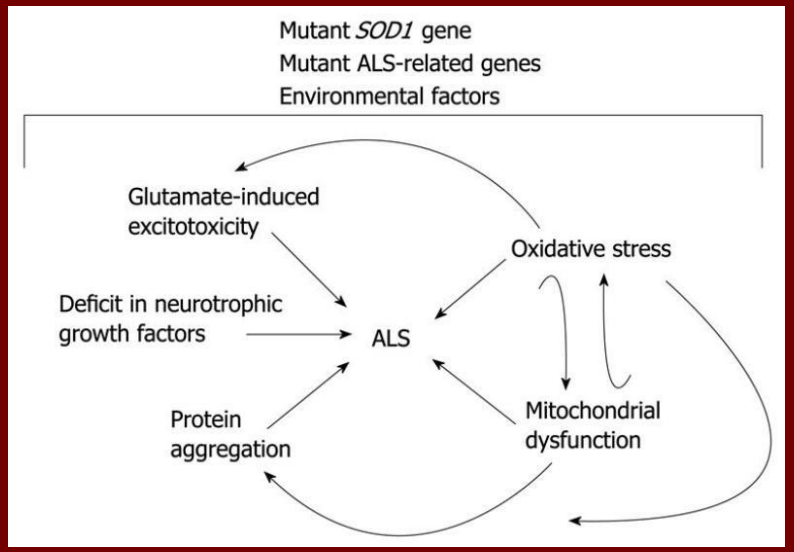
- Approx 30,000 people in the US have ALS at any given time
- Incidence 1.5-2.5 per 100,000
- Approx 15 new cases per day
- 60% Men
- Typically midlife, between 40-70 years- most common Caucasian
- Life Expectancy is 2-5 years, more than ½ live longer than 3 years

Etiology

- Sporadic , unkown cause
- Genetic in 5-10% of cases-

an inherited autosomal trait





Etiology

- Theoretical Models
  - Excitotoxicity- Glutamate defect in possibly metabolism, transport or storage
  - Oxidative stress caused by free radicals
  - Autoimmune-antibodies to calcium channels, activated T lymphocytes , Monoclonal paraproteinemia
  - Cytoskeletal defects or abnormalities
  - Neurofilament abnormalities- abnormal accumulation and damage to the structure

Rehabilitation Goals

- Prevent complications of immobility
- Strengthen unaffected muscles
- Saliva management
- Dysphasia
- Aspiration prevention, Assisted Cough
- Communication, plan early before need for ventilator
- Adaptive techniques and equipment
- Depression, coping with progressive loss
- Preparation for disease progression
- Preparation for death

Focus on Palliative Care

- Key Decisions must be discussed in advance of crisis
  - G-Tube
  - Mechanical Ventilation
  - Advance Directives
- Teaching Plan for Patient and Caregiver/Family
  - Symptom Management
  - Reduce Complications
- Assistive Devices to maintain independence
- Referrals for support groups and other resources

Nursing Process

- Assessment
  - Full Health History
  - Family Incidence of ALS
  - Onset of Symptoms and Progression
- Current Function & ADLs
  - Gait, strength and stability
  - Flaccidity, spasticity
  - Eval Swallowing and chewing
  - Eval Respiratory Status
  - Bowel and Bladder function
  - Skin Assessment
  - Observe Family Interactions

Plan of Care

- **Nursing Diagnoses**
  - Impaired physical mobility re: muscle wasting, weakness and spasticity
  - Self Care deficit
  - Impaired Communication re: impairment of muscles for speech
  - Ineffective breathing pattern re: impairment of diaphragm and accessory muscles

Plan of Care

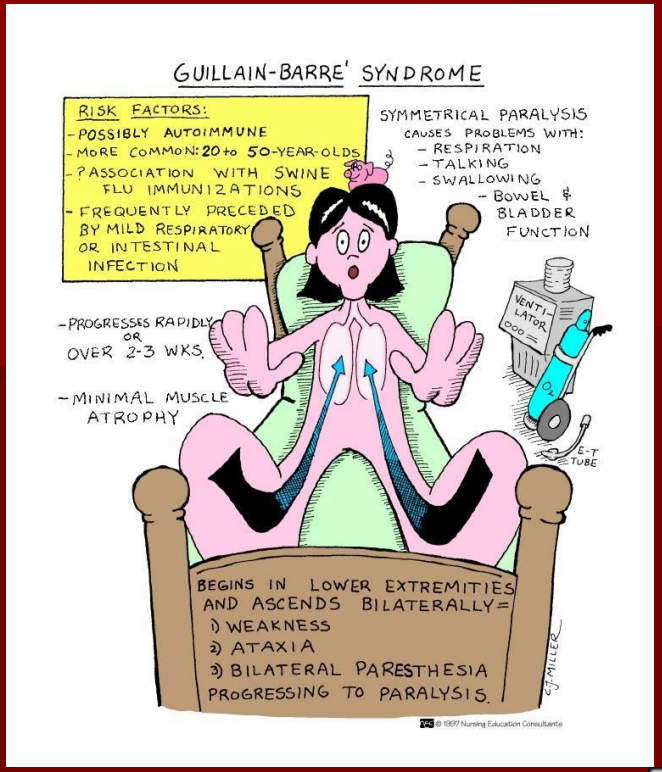
- **Nursing Diagnoses**
  - Altered nutrition: Less than required re: bulbar muscles
  - Potential for anxiety
  - Risk for ineffective coping
  - Interrupted family processes re: change in health of family member, modifications of family roles and foreseen loss of family member
  - Risk for caregiver strain

Interventions

- Maintain Independence
- Limit complications from progressive loss of function
  - Ineffective breathing
  - Impaired swallowing and decrease in nutritional intake
  - Impaired communication
  - Maintain skin integrity
  - Patient and Family Education and support in decision making
  - Pain management

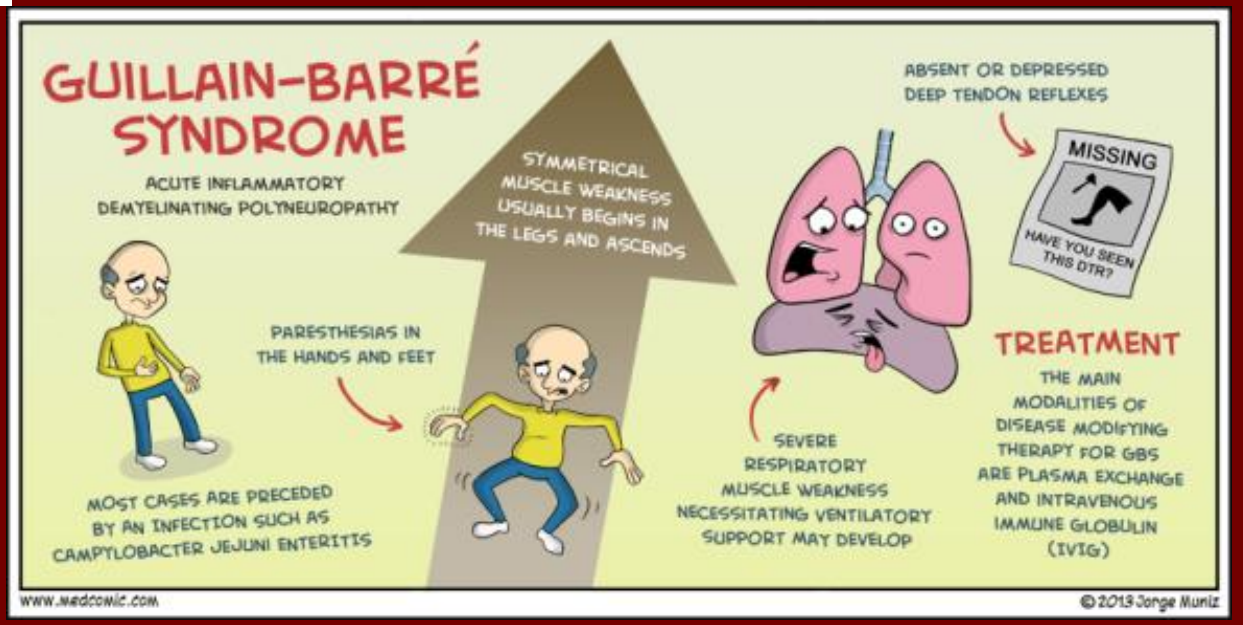
Treatment for ALS

- **the FDA has approved the first drug treatment for the disease—Riluzole. Riluzole is believed to reduce damage to motor neurons and prolongs survival by several months, mainly in those with difficulty swallowing**



Guillain Barre' Syndrome

Acute inflammatory polyneuropathy



Epidemiology

- Annual incidence is 1 per 100,000 per year
- GBS in children 15 years and younger between 0.34 and 1.34 per 100,000
- Both Genders, all ages and all ethnicities equally affected
- Incidence increases in adults 50 years and older
- Hospitalization decreased in recent years probably due to IV Immunoglobulin

Subtypes

- Acute inflammatory demyelination polyneuropathy (AIDIP)
  - Classic GBS, 90% cases in Western World
- Acute motor axonal neuropathy (AMAN)
  - Common in Chinese children
- Acute motor sensory axonal neuropathy (AMSAN)

Etiology

- Cause unknown
  - **Several triggers that seem to relate to an autoimmune attack on the body**
  - **Most often a Respiratory or Gastrointestinal Virus in the days to weeks prior to onset**

Respiratory or Gastrointestinal Virus	
Campylobacter jejuni	**Cytomegaloviris**
Mycoplasma pneumoniae	**Epstein-Barr virus**
	Zika virus

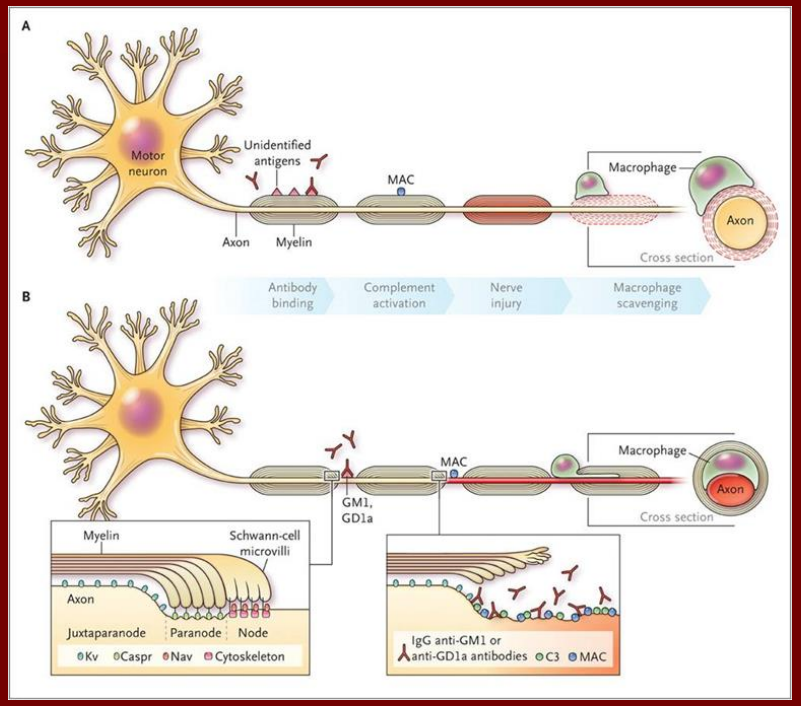
  - **Less often surgery or vaccine thought to be trigger. Vaccines associated with GBS are:**
    - **Rabies**
    - **Swine Flu**
    - **Polioviris**

Clinical Manifestations

- Ascending symmetric weakness occurs
- Ascending flaccid paralysis is typical
- Loss of neurologic function & deep tendon reflexes occurs
- Resp insufficiency and failure may result from weakness in diaphragm and intercostal muscles and mechanical failure
- 50% damage to facial nerve (CN VII) causes facial diplegia
- Damage to golssopharyngeal (CN IX) and Vagus (CN X) nerves will cause dysphagia and laryngeal paralysis
- Autonomic Dysfunction is highly likely; certain if Vagus nerve is involved
- Pain, numbness & hypersensitivity to touch

Autonomic Dysfunction in GBS

- Paroxysmal hypertension
- Orthostatic hypotension
- Cardiac arrhythmias
- Paralytic ileus
- Urinary Retention
- Syndrome of inappropriate antidiuretic hormone secretion



Pathology

- **Acute, inflammatory disease affecting the myelin of the nerves in the peripheral nervous system- in some cases axonal degeneration can occur**
- **Immune mediated cellular and humoral response→ triggers antibody production→ antimyelin antibody causes demyelination**
- **Remyelination occurs slowly**
- **Onset can be from hours to about 3 weeks**
- **May Improve for 3 years after onset**

Diagnosis- Based on Clinical Presentation

- **Progressive weakness in two or more limbs due to neuropathy**
  - **Areflexia**
  - **Disease course of less than 4 weeks**
  - **Exclusion of other causes of symptoms**
- **History of recent viral infection**
- **Electrophysiological study shows slowing of conduction or block in motor or sensory nerves**
- **Lumbar puncture shows increase in protein**

Management Options

- Medical management urgent due to rapid onset of disease
- Therapeutic plasma exchange may be performed every other day for 10-15 days
- IV Immunoglobulin administration
- Medical management of symptoms
- Corticosteriods appear to delay recovery
- The most critical part of the treatment is keeping the patient's body functioning during recovery of the nervous system.

Nursing Process

- Assessment
  - Obtain a full health history
  - List of current symptoms and onset (timeline)
  - Assess Resp function
  - Assess for pain, paresthesia, numbness or paralysis
  - Assess bowel and bladder function
  - Evaluate Cranial Nerve involvement
  - Evaluate Swallowing
  - Assess nutrition and weight
  - Observe patient and family interactions

Plan of Care: Nursing Diagnoses

- Impaired physical mobility re: disease process
- Ineffective breathing pattern re: neuro-muscular weakness of respiratory muscles
- Altered nutrition: Less than body requirements re: inability to swallow
- High risk for aspiration re: dysphagia/ cranial nerve involvement
- Imparied communication re: impairment of speech muscles /cranial nerve involvement
- Risk for impaired skin integrity

Plan of Care: Nursing Diagnoses

- Risk for DVT
- Risk for constipation
- Risk for Urinary retention
- Acute pain
- Self Care deficit re: loss of function
- Altered Sensory perception due to disease process
- Potential for anxiety re: lack of control within environment
- Risk for depression re: loss of function and independence

Interventions

- Provide Pain Management
- Maintain function in unaffected limbs
- Limit atrophy to affected limbs
- Maintain oxygenation and effective breathing patterns
- Manage autonomic dysfunction
- Provide nutritional support
- Provide means of effective communication

Interventions

- Prevent skin breakdown
- Prevent DVT
- Maintain Bowel and Bladder elimination
- Provide control and comfortable environment
- Provide psychological and emotional support for patient and family
  - Educate patient and family on disease course

Rehabilitation

- **Rehab Goals:**
  - **Help patient pace recovery to obtain maximum use of muscles**
  - **Aid patient in adapting to residual dysfunction**
- **Rehabilitation does not improve nerve regeneration and thus has no effect on return of nerve supply to muscle.**
  - **Strength returns in descending pattern**
  - **Most pts get better but severity, duration and course are variable**
  - **40% patients will need Rehab**
  - **Over fatigue may decrease recovery:**

  Pacing is Essential

Treatments

- Plasmapheresis (total plasma exhange)
- IV IG

Secondary complications

- May require ventilator support
- DVT/ PE common
- Unstable blood pressure
- Complications of Immobility
- Anemia

Variant of GBS

- **Miller Fisher Syndrome**
  - Abnormal muscle coordination
  - paralysis of eye muscles
  - absence of tendon reflexes

  Recovery
  - begins within 2-4 weeks
  - May complete within 6 month
  - Some people may have residual deficits

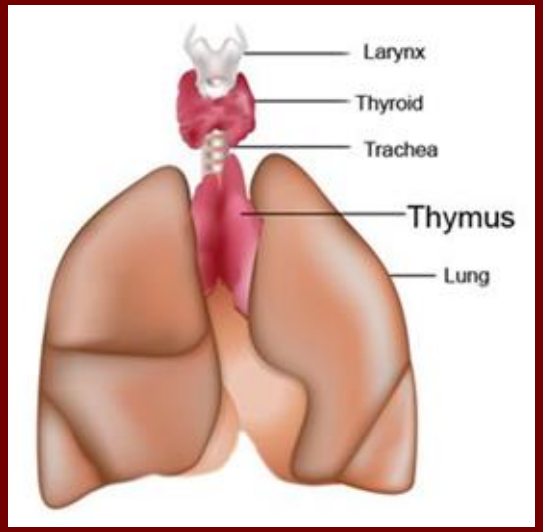
National Institute of Neurological Disorders and Stroke (NINDS).

Myasthenia Gravis

The thymus gland plays an important role in the development of the immune system in early life. Its cells form a part of the body's normal immune system. The gland is somewhat large in infants, grows gradually until puberty, and then gets smaller and is replaced by fat with age.
In adults with myasthenia gravis, the thymus gland remains large and is abnormal. It contains certain clusters of immune cells indicative of lymphoid hyperplasia—
a condition usually found only in the spleen and lymph nodes during an active immune response.
Some individuals with myasthenia gravis develop thymomas (tumors of the thymus gland). Thymomas are generally benign, but they can become malignant.

Epidemiology

- Approx 1 per 5000 people in the USA
- Can affect
  - All Ages
  - All Races
  - Both Sexes-
    - Highest Incidence
      - Female to male ratio 3:2 affected before 50yr
      - Men older than 60 yrs of age
- Most Severe Weakness in 1st 2 yrs



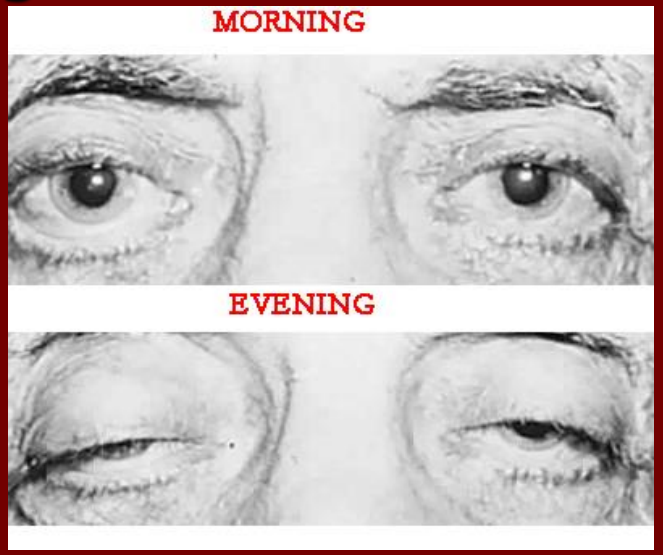
Variations

- Ocular
  - Eye and lid muscles are affected
- Generalized
  - Proximal muscles of both upper and lower extremities are involved, with ocular or bulbar involvement
- Bulbar
  - Muscles of speech, swallowing and breathing are affected
- Neonatal Transient

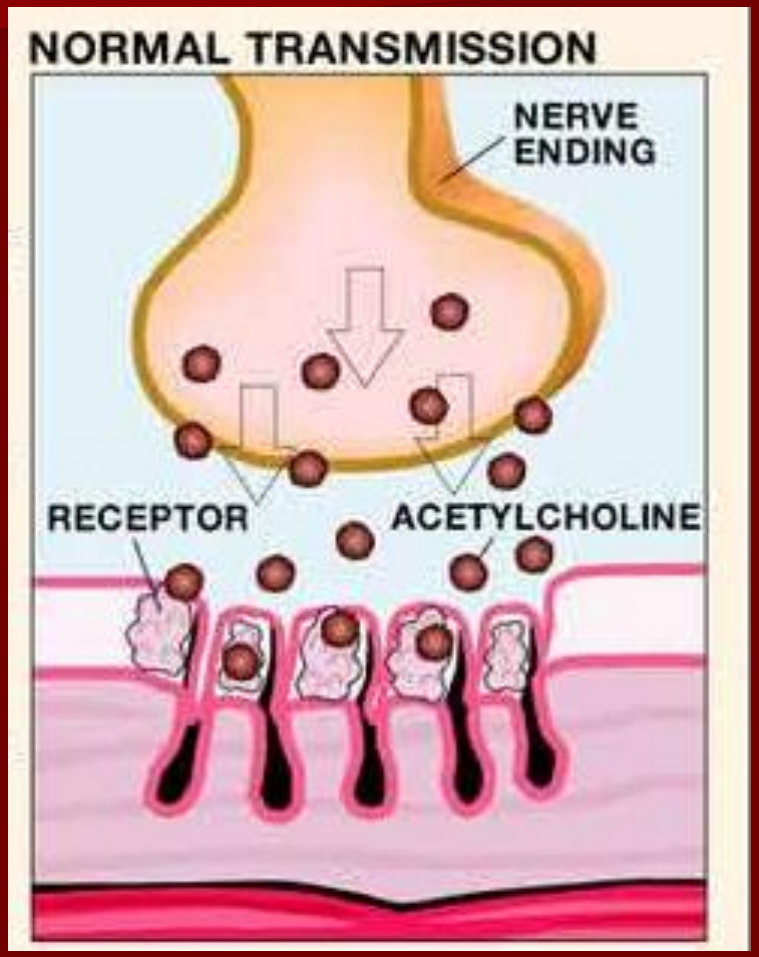
Etiology

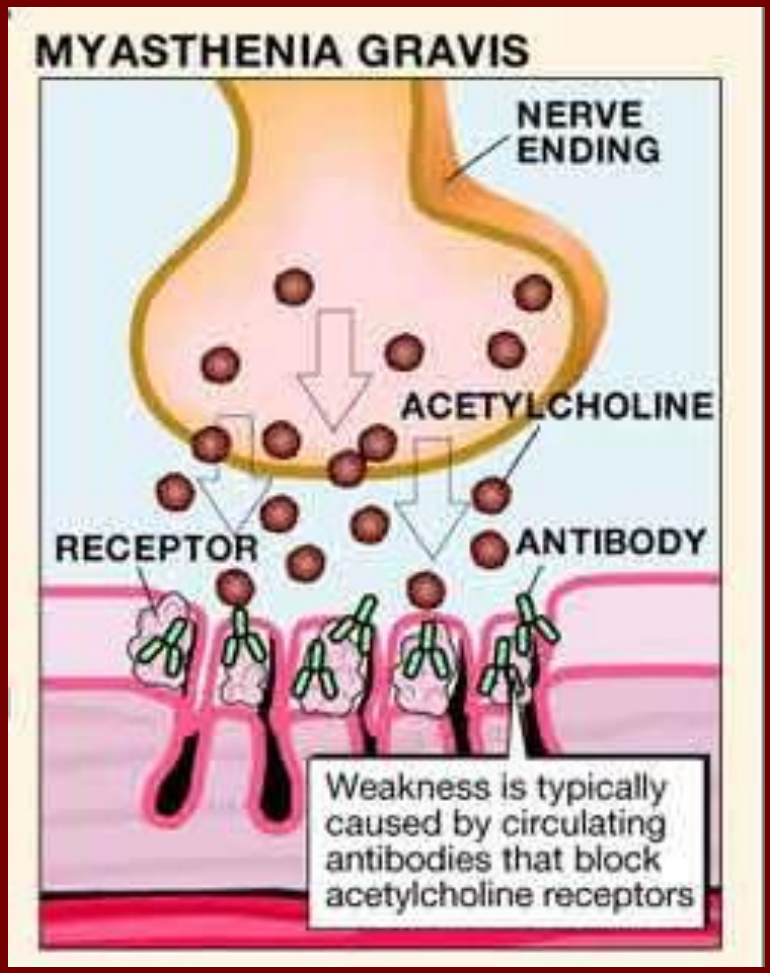
Common symptoms can include:

- A drooping eyelid
- Blurred or double vision
- Difficulty chewing and swallowing
- Slurred speech
- Weakness in the arms and legs
- Chronic muscle fatigue
- Difficulty breathing



http://www.ninds.nih.gov/disorders/myasthenia_gravis/myasthenia_gravis.htm





Diagnosis

- Review of medical history and physical and neurological examinations
- Blood Test- acetylcholine receptor antibodies (80-90% show elevated antibody titer elevation)
- 2nd blood test- anti-MuSK antibody—found in about 30 to 40 percent of people with myasthenia gravis who do not have acetylcholine receptor antibodies
- nerve conduction study which tests for specific muscle "fatigue" by repetitive nerve stimulation. This test records weakening muscle responses when the nerves are repetitively stimulated by small pulses of electricity

Diagnosis

- Edrophonium test uses intravenous administration of edrophonium chloride to very briefly relieve weakness in people with myasthenia gravis
- Electrophysiology- tests for specific muscle "fatigue" by repetitive nerve stimulation
- Single-fiber electromyography- impaired nerve-to-muscle transmission. Failure to transmit or delay indicates a positive test- for MG confirmation approx 99% sensitive
- MRI or CT- may be used to identify the presence of a thymoma

- **Myasthenia Crisis**
  - Involvement of respiratory and accessory muscles escalates to a crisis
  - Requires Respiratory support for Survival
- **Cholinergic crisis**
  - Results from excessive dosage of cholinergic treatment medications
  - Symptoms mimic organophosphate poisoning

  Salivation, lacrimation, urinary incont, GI upset, emesis, miosis

MGFA Classification System

- **Class I MG**
  - **Any ocular muscle weakness**
  - **May have weakness of eye closure**
  - **All other muscle strength is normal**
- **Class II MG**
  - **Mild weakness affecting other than ocular muscles**
  - **May also have ocular muscle weakness of any severity**
- **Class IIa MG**
  - **Predominantly affecting limb, axial muscles, or both**
  - **May also have lesser involvement of oropharyngeal muscles**
- **Class IIb MG**
  - **Predominantly affecting oropharyngeal, respiratory muscles, or both**
  - **May also have lesser or equal involvement of limb, axial muscles, or both**

- **Class III MG**
  - **Moderate weakness affecting other than ocular muscles**
  - **May also have ocular muscle weakness of any severity**
- **Class IIIa MG**
  - **Predominantly affecting limb, axial muscles, or both**
  - **May also have lesser involvement of oropharyngeal muscles**
- **Class IIIb MG**
  - **Predominantly affecting oropharyngeal, respiratory muscles, or both**
  - **May also have lesser or equal involvement of limb, axial muscles, or both**

- Class IV MG
  - Severe weakness affecting other than ocular muscles
  - May also have ocular muscle weakness of any severity
- Class IVa MG
  - Predominantly affecting limb, axial muscles, or both
  - May also have lesser involvement of oropharyngeal muscles
- Class IVb MG
  - Predominantly affecting oropharyngeal, respiratory muscles, or both
  - May also have lesser or equal involvement of limb, axial muscles, or both
- Class V MG
  - Defined by intubation, with or without mechanical ventilation, except when used during routine postoperative management
  - Use of a feeding tube without intubation places the patient in class IVb

Management Options

Medications

- anticholinesterase agents such as neostigmine and pyridostigmine, which help improve neuromuscular transmission and increase muscle strength.
- Immunosuppressive drugs such as prednisone, azathioprine, cyclosporin, mycophenolate mofetil, and tacrolimus may also be used. These medications improve muscle strength by suppressing the production of abnormal antibodies. They have major side effects

Management Options

- **Thymectomy**
  - recommended for individuals with thymoma
- Plasmapheresis, a procedure in which serum containing the abnormal antibodies is removed from the blood while cells are replaced
- High-dose intravenous immune globulin

Crisis Management

- Bilevel positive airway pressure and other respiratory support systems can prevent need to for intubation or decrease recovery time
- Safe Environment
- Energy Conservation
  - Therapy times
  - Rest periods
- Nutritional support
  - Soft diet to decrease fatigue in eating
  - Caloric support if needed

Management

- Monitor for crisis events, side effects of treatments, individual tolerance and weakness
- Communicate with medical team for treatment changes based on patient response
- Assist to remain as independent as possible with ADLs
- Patient and Family Education
  - Disease Process
  - Side Effects of meds and treatments
  - Medical emergency- Myasthenic crisis or Cholinergic Crisis
  - No over the counter meds w/o consult of Dr.

Research

- Technological advances have led to more timely and accurate diagnosis,
- New and enhanced therapies have improved management of the disorder.
- There is a greater understanding about the structure and function of the neuromuscular junction, the fundamental aspects of the thymus gland and of autoimmunity, and the disorder itself.
- Researchers are seeking to learn what causes the autoimmune response in myasthenia gravis, and to better define the relationship between the thymus gland and myasthenia gravis.

Nursing Process

- Assessment
  - Full Health History
  - Assess baseline to monitor for crisis- (strongest time of day)

- Resp function
- Cardiac Function
- Bowel and Bladder function
- GI Symptoms

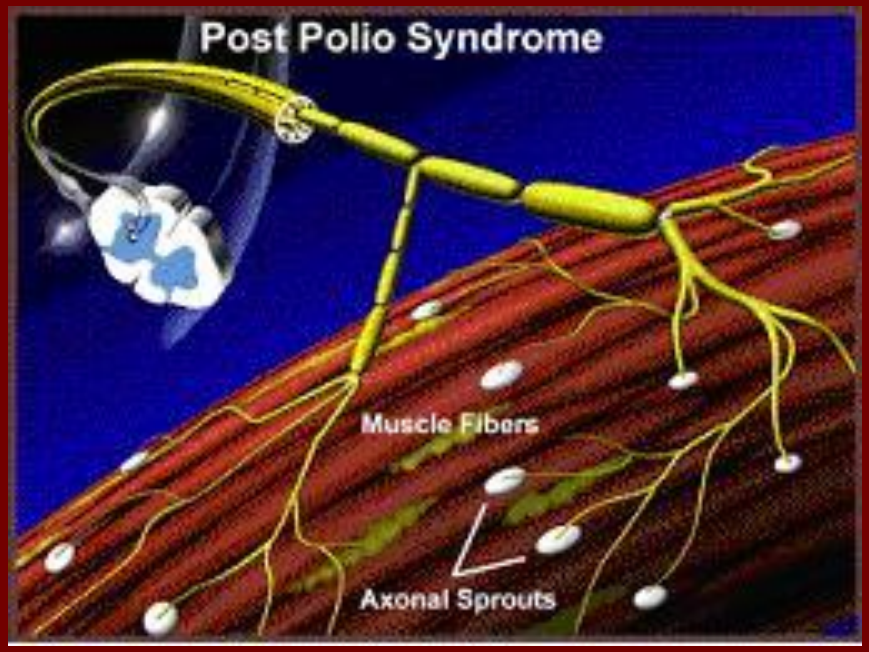
- Visual Acuity
- Strength and Mobility
- Swallowing
- Speech

Plan of Care

- Knowledge Deficit re: disease process and side effects of treatment
- Activity intolerance re: fatigue
- Risk for aspiration re: muscle weakness and increased secretions
- Risk for Falls
- Risk for Medical Crisis (Myasthenic or Cholinergic)

Post polio Syndrome

- http://www.ninds.nih.gov/disorders/post_polio/detail_post_polio.htm#223203172



ANESTHESIA WARNING!

I am a Polio Survivor...

- **EASILY SEDATED, difficult to wake;**
- **Difficulty BREATHING and SWALLOWING with anesthesia;**
- **HYPERSENSITIVE to PAIN and COLD.** Need heated blanket and increased pain medication post-op.

For more information, visit:
www.nj.gov/health/cd/postpolio/index.shtml

C1510

NEW JERSEY DEPARTMENT OF HEALTH AND SENIOR SERVICES

I am a Polio Survivor with
Post-Polio Sequelae (PPS)
unexpected midlife symptoms:

- **Overwhelming Fatigue**
- **Muscle Weakness**
- **Muscle and Joint Pain**
- **Sleep Disorders and Cold Intolerance**
- **Difficulty Swallowing and Breathing**
- **Heightened Sensitivity to Anesthesia**

(over)

Epidemiology

- Only Afghanistan, Nigeria, and Pakistan remain polio-endemic as of February 2012
- Accurate statistics do not exist today- the survey in 1987 asked questions related to Post Polio Syndrome.
- Researchers estimate that the condition affects 25 to 40 percent of polio survivors.

- Post polio affects survivors of polio decades after the acute illness
- Symptoms:
  - **Pain**
  - **Fatigue**
  - **Weakness- NEW weakness**
  - May have sleep, breathing and swallowing problems
  - May have muscle atrophy or wasting
  - Severity of initial acute polio predicts severity of Post Polio Syndrome

Major Symptoms

Etiology-Criteria from NINDS

- Prior Paralytic Poliomyelitis with lower motor neuron loss
- Period of partial or complete recovery for 15 years or more
- Gradual onset of progressive or persistent new muscle weakness or fatigue

  (Often after surgery or period of inactivity)
- Symptoms that persist for > 1 year
- Exclusion of other problems/diagnosis

Diagnosis

- There are no diagnostic tests for PPS
  - MRI
  - CT
  - Neuroimaging
  - Electophysiological studies
- High Risk for Fractures

Helpful to determine course of decline

Pathophysiology

- Wechler Theory
  - New "sprouts" of nerve cells reconnect the nerve cell to the muscles during recovery from acute polio
  - New "sprouts" trigger contraction of the muscles and supply more muscle fibers with intervention
  - New "sprouts" are not stable and degenerate over time through "overexertion" and no longer contract muscle fibers, leading to perception of new weakness or loss of function

Management Options

- Energy Conservation
- Lifestyle changes to reduce stress
- Avoiding inactivity and overuse
- Rehabilitation
- Bracing to support weak muscles
- Canes or crutches to improve safety and decrease weight on weak limbs
- Orthotics- leg length discrepancy & gait disturbances
- Weight Loss

Management Options

- Biphasic positive pressure ventilator at night to treat underventilation
- Treatment for Depression
  - Common in PPS
- Training Programs with Warm Water is helpful
- Training Programs with low-level aerobic and low level muscle strengthening
- Prevent pathological fractures
- Reduce Risk for Falls

Management Options

- Pain relief
  - Nociceptive pain more common than neuropathic pain
  - Worse in younger
  - Worse in females
  - Worse in persons with younger age onset of initial acute polio
- Support for Adjustment
- Education

Nursing Process

- Assessment
  - Full Health History
    - Initial Acute Polio illness and recovery
    - Functional level
    - Work history
    - Onset of PPS Symptoms
  - List of current symptoms and onset
  - Assess for Pain, paresthesia, numbness and paralysis
  - Assess Bowel and Bladder Function
  - Observe Patient and Family interactions

Plan of Care

- Impaired physical mobility re: disease process
- Decreased activity tolerance re: muscle weakness, pain and overuse syndrome
- Ineffective breathing pattern re: neuromuscular weakness of respiratory muscles
- Altered Nutrition: less than body requirements
- Risk for constipation

Plan of Care

- Risk for DVT re: change in gait and mobility
- Acute pain re: disease process
- Self-care deficit re: loss of function
- Altered sensory perception due to disease process
- Potential for anxiety re: loss of control within environment and change in lifestyle
- Risk for depression re: loss of function and independence

Interventions

- Teach Energy Conservation Strategies
- Schedule activities with rest periods
- Make environmental accommodations to decrease energy needs for ADLs
- Teach AROM to patient; PROM to caregivers
- Respiratory Hygiene and Management
- Nutritional Support
- Prevent DVT- teach signs & symptoms
- Bowel and Bladder Management
- Pain management
- Psychological and emotional support

Resources

- Jacelon, C.S., ed. (2011). *The Specialty Practice of Rehabilit Nursing: A Core Curriculum* (6th ed.) Association of Rehabiltation Nurses: Glenview,IL.
- Multiple Sclerosis
  - –http://www.ninds.nih.gov/disorders/multiple_
  - –sclerosis/multiple_sclerosis.htm
  - –http://www.nationalmssociety.org/about-multiple-sclerosis/index.aspx
- Guillain-Barré Syndrome Support Group
  - –**http://www.gbs.org.uk/index2.shtml**
  - –**www.ninds.nih.gov/disorders/gbs/gbs.htm**
- Amyotrophic Lateral Sclerosis Fact Sheet
  - –**http://www.ninds.nih.gov/disorders/amyotrophiclateralsclerosis/detail_amyotrophiclateralsclerosis.htm**
- NINDS Parkinson's Disease Information Page
  - –**http://www.ninds.nih.gov/disorders/**
  - – **parkinsons_disease/parkinsons_disease.htm**

Other topics

- Diabetes Mellitus
- Cancer
- HIV and AIDS
- Obesity

Too Quick? YES

- **Study well**
- **Use your Resources at your Facility**