Department of
Physical Medicine and Rehabilitation

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The 13 Rehab Diagnoses

1. Stroke
2. Spinal Cord Injury
3. Congenital Deformity
4. Major Multiple Trauma
5. Hip Fracture/Femur Fracture
6. Brain Injury
7. Neurological Disorders
8. Burns
9. Active, polyarticular RA, Psoriatic Arthritis, or Seronegative Spondyloarthropathy
10. Amputations
11. Systemic vasculidities with joint inflammation
12. Severe or advanced osteoarthritis involving 2 or more major joints (elbow, shoulders, hips, knees)
13. Knee or hip replacement

Important Grading Systems

Muscle Stretch Reflexes:

0 – no response

1+ - requires distraction to illicit

2+ lower half of normal

3+ upper half of normal

4+ hyper reflexic, very brisk

Clonus is noted separately in beats → 2-3 beats of clonus may be physiologic, more than 3 is pathologic

Manual Muscle Testing (MMT)

Grade 0: no contractile activity can be felt in the gravity eliminated position

Grade 1: The muscle contraction can be palpated without joint movement

Grade 2: Full or partial range of motion with gravity eliminated

Grade 3: Full range of motion against gravity

Grade 4: Full range of motion against some resistance

Grade 5: Full range of motion against full resistance

Spasticity: a velocity dependent increase in tonic stretch reflex.

Modified Ashworth Scale for Grading Spasticity

Grade 0: no increase in muscle tone

Grade 1: Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension

Grade 1+: Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (<50%) of the ROM

Grade 2: More marked increase in muscle tone through most of ROM (>50%), but affected part(s) easily moved

Grade 3: Considerable increase in muscle tone, passive difficult

Grade 4: Affected part(s) rigid in flexion and extension

Traumatic Brain Injury (TBI)

Epidemiology:

- Causes: MVA, 2nd is violence

Mechanism of Injury:

- Primary (direct result of trauma):
    - Acceleration/Deceleration injuries – contusions
    - Diffuse axonal injury: damage to brain tissue, not seen on imaging
- Secondary (cascade of biochemical, cellular, molecular events causing local or systemic changes)
    - Ischemia, excitotoxicity, apoptosis
    - Brain swelling (increase in intravascular blood)
    - Brain edema (increase in water content)

Recovery Mechanisms:

- Plasticity – damaged brain "repairs" itself by morphologic and physiologic responses
    - Neuronal regeneration/neuronal (collateral) sprouting – intact axons make synaptic connections through dendritic and axonal sprouting in areas where damage occurred
    - Functional reorganization/unmasking neural reorganization – neural structures that were not used for a specific function will now go the lesioned area and perform that function
- Synaptic Alterations
    - Diaschisis – damage to one region of brain can produce altered function in other areas that were not severed, therefore initially there will be loss of injured and intact brain
- Functional substitution/Behavioral Substitution – techniques/new strategies are learned to compensate for deficits and to achieve a particular task
- Redundancy – recovery of function based on activity of uninjured brain areas that normally would contribute to that function
- Vicariation – functions taken over by brain areas not originally managing that function

Disorders of Consciousness:

- Consciousness is a function of the connections and interconnectedness of the midbrain and the cerebral cortex
- Types:
    - Coma: No self or environmental awareness, no sleep-wave cycles on EEG, no purposeful movement, can't open eyes
    - Vegetative State: Can open eyes, but can't track with eyes, can't interact with environment, there are sleep-wave cycles on EEG,
    - Minimally conscious state: minimal self and environmental awareness, inconsistent but reproducible purposeful behaviors, opening eyes and tracking
- Tx:

- Preventive therapeutic interventions:
  - Managing bladder and bowel, skin integrity, preventing contractures, controlling spasticity
- Meds:
  - Stop unnecessary meds that affect cognition (histamine 2 blockers, metoclopramide, pain meds)
  - Add stimulant medications (methylphenidate, amantadine, dopamine agonists)

Prognosis after TBI:

TABLE 38-2
Glasgow Coma Scale

BEHAVIOR	RESPONSE	SCORE
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place, and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1
Total score:	*Best response*	15
	Comatose client	8 or less
	Totally unresponsive	3

- Glascow Coma Scale(GCS):
  - Severe TBI (coma): GCS score <8
  - Moderate TBI: GCS 9-12
  - Mild TBI: GCS >12
- Coma duration:
  - Poor recovery if coma lasts >4 wks
  - Less severe disability if coma lasts <2 wks
- Posttraumatic Amnesia (PTA):
  - Longer duration of PTA → worse outcomes
  - <1 hr – mild
  - >1 hr, <24 hr – moderate
  - >7 days – severe
  - Use Galveston Orientation and Amnesia Test (GOAT) – end of PTA is when pt scores >/= 75 points
    - Period of PTA is the # of days beginning at the end of coma to the time the pt gets first of two successive GOAT scores >/= 75
  - Use Orientation Log (O-Log) – focuses on orientation of place, time, and circumstance
    - Two consecutive scores of 25 or higher means you are out of PTA
- Disability Rating Scale (DRS) – quantitative index of disability from TBI
- Rancho Los Amigos Levels of Cognitive Function Scale (Rancho scale)
- JFK Coma Recovery Scale (CRS-R):
  - Changes on this scale showed stronger correlations with outcomes

Acute Management of TBI:

- ABCs
- Manage ICP
- May need surgical intervention (emergent craniotomy, burr hole)

Complications of TBI:

- Posttraumatic seizures – no more than 1 week of seizure prophylaxis
- Autonomic instability (HTN, tachycardia, hyperthermia, spasticity, perspiration)
  - Tx: B-blockers for HTN, tachycardia,
- Posttraumatic Hydrocephalus – usually normal-pressure type

    - Incontinence, ataxia, dementia
    - Tx: LP, shunt placement
- Cranial nerve injuries: CNI, CNVII, CNVIII
- Posttraumatic agitation/impulsivity
    - Usually lasts 1-14 days but can last longer
    - reduce stimulation, may need a sitter, double lock belt, enclosure bed, meds [antipsychotics, propranolol, valproic acid, antidepressants,
- Heterotopic ossification – in hips, seen on bone scan
- DVT/PE: prophylaxis w/ subq heparin (5000U q8-12hr) or SCDs
- Urinary dysfunction
- Spasticity
- Nutrition – may need PEG tube for tube feeds
- Neuroendocrine Disorders
    - Hyponatremia due to SIADH (isovolemia) or cerebral salt wasting (hypovolemia)

Stroke

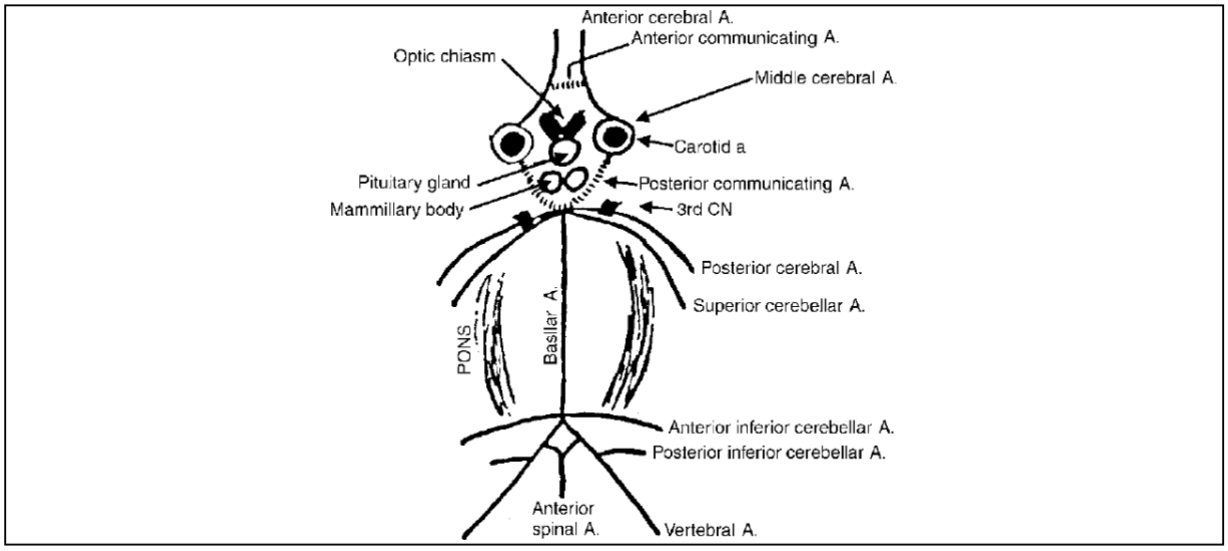
Definition: a CVA with clinical signs of focal or global disturbances of cerebral function with signs lasting more than 24hrs

Epidemiology:

- 3rd leading cause of death after heart disease and cancer
- Risk factors:
    - Nonmodifiable: age, gender (M>F), race (AA > Caucasians > Asians), family history of stroke
    - Modifiable: HTN, hx of TIA/prior stroke, heart disease (CHF, CAD, valvular heart disease, arrhythmia), diabetes, smoking, hypercoagulable states (cancers, sickle cell anemia, protein C and S deficiencies), HLD, sleep apnea, obesity

Main Arteries involved in a CVA:

- Anterior cerebral artery – contralateral (opposite) lower extremities > upper extremities
- Middle cerebral artery – contralateral face, upper extremities, aphasia > lower extremities
- Posterior cerebral artery – visual deficits, prosopagnosia (can't read faces), alexia (can't read), vertigo, nystagmus,



Type of CVAs:

- Ischemic (87%)
    - Etiology: Thrombotic, Embolic, Lacunar
    - Anterior circulation:
        - Internal carotid artery – ocular infarction, transient monocular blindness (amaurosis fugax), can have a mixture of ACA and MCA infarcts
        - MCA

- Superior division: contralateral sensory and motor deficits of face and arm > leg. If on left side (dominant)→ global aphasia initially then turns into Broca's aphasia. If on right side (nondominant) → spatial perception deficits, hemineglect, dressing apraxia
- Inferior division: superior quadrantanopia or homonymous hemianopsia. If left side → Wernicke's aphasia. If right side → left visual neglect

▪ ACA

- Contralateral sensory and motor deficits of leg. Gait apraxia. If left side → transcortical motor aphasia

o Posterior Circulation:

▪ PCA

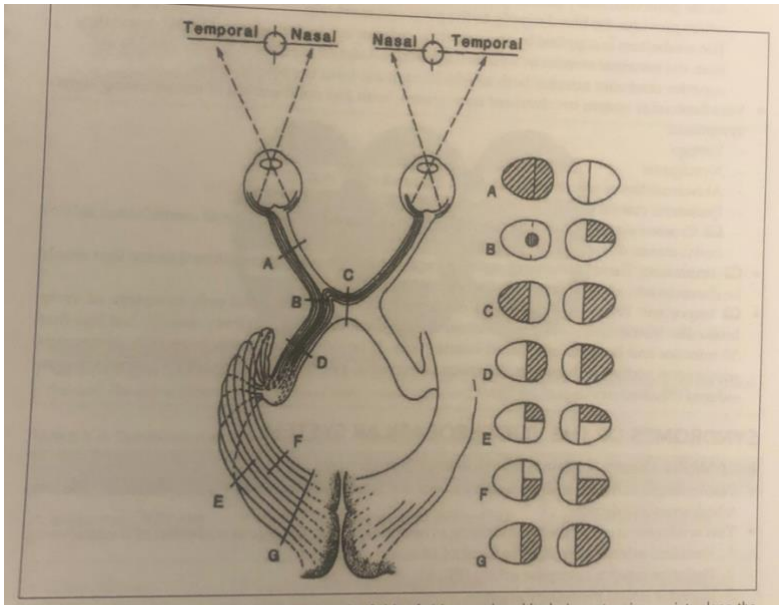


FIGURE 1–11 Diagram showing the effects on the fields of vision produced by lesions at various points along the optic pathway: (A) complete blindness in the left eye from an optic nerve lesion; (B) the usual effect is a left junction scotoma in association with a right upper quandrantanopia. The latter results from interruption of right retinal nasal fibers that project into the base of the left optic nerve (Wilbrand's knee). A left nasal hemianopia could occur from a lesion at this point but is exceedingly rare; (C) chiasmatic lesion causing bitemporal hemianopia; (D) right homonymous hemianopia from optic tract lesion; (E and F) right superior and inferior quadrant hemianopia from interruption of visual radiations; and (G) right homonymous hemianopia due to lesion of striate cortex. (From Ropper & Samuels, 2012, with permission.)

▪

o Vertebrobasilar System Syndromes

▪ Wallenberg (Lateral Medullary Syndrome) AKA PICA syndrome, vertebral artery syndrome – ipsilateral Horner's syndrome, decrease in pain and temp on face, ataxia (falling toward side of lesion), Contralateral decreased pain and temp on body, dysphagia, dysarthria, hoarseness, vocal cord paralysis, vertigo, N/V, hiccups, nystagmus

▪ Medial Medullary Syndrome – ipsilateral hypoglossal palsy, contralateral hemiparesis, contralateral proprioception and position sense loss

▪ Basilar artery occlusion – locked in syndrome

- Hemorrhagic (13%)

o Intracerebral hemorrhage – hypertensive

▪ HA, LOC, N/V,

        - Locations:
            - Putamen – afasia, hemiplegia, eyes deviate away
            - Thalamus – hemiparesis if internal capsule, contralateral sensory deficits. Aphasia if dominant side, contralateral hemineglect if nondominant side. Ocular symptoms if subthalamus
            - Pons – total paralysis
            - Cerebellum, - unusual vomiting, occipital HÁ, vertigo, inability to sit, stand or walk, eyes deviate to opposite side, dysarthria, dysphagia
            - Lobar (cerebral)
    - Subarachnoid hemorrhage – ruptured saccular/berry aneurysm – CNIII compression (ipsilateral deviation to lateral eye, ptosis, mydriasis)

Anticoagulation

- No added benefit for immediate anticoagulation in acute ischemic stroke
- Indications: cardiac emboli (Afib, mural thrombus from MI), TIA,
    - Start 24-36hrs post stroke so no risk for conversion into hemorrhagic CVA
- Agents: Xarelto (rivaroxaban), Eliquis (apixiban) more common, less side effects than warfarin

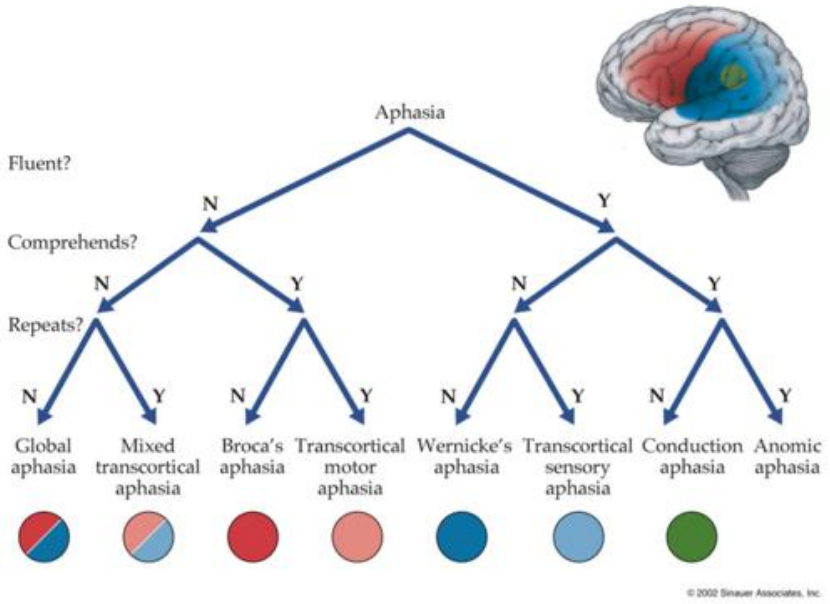
Stroke Rehab:

- Immediately following hemiplegia, there is a loss of voluntary movement and loss or decrease reflexes
- w/in 48hrs, there are increased DTRs
- There will be tone developing, which can lead to spasticity
- Recovery of movement: 6-33 days after onset of hemiplegia, the first intentional movements appear. Recovery starts from the proximal muscle group (shoulders and hips) and then progresses more distally (hands and feet)
- Most recovery occurs in the first 3 months and only minor additional recovery occurs after 6 months post-CVA
- If pt has some motor recovery of hand by 4 wks, there is up to 70% chance of making a full or good recovery
- Poor prognosis if: no grasp strength by 4 wks, severe proximal spasticity, prolonged “flaccidity”
- While we wait for muscle recovery, we must prevent contractures by positioning the patient, doing range of motion (ROM) exercises, strengthening, mobilization, endurance training,

Post-Stroke Complications:

- Post-stroke shoulder pain – very common, usually due to complex regional pain syndrome (CRPS) type 1
- Shoulder subluxation
- Bicipital tendinitis
- Rotator Cuff tear, shoulder impingement syndrome, adhesive capsulitis
- Brachial plexus/peripheral nerve injury
- Heterotopic ossification (HO) – in elbow or shoulder

- Spasticity
- Post-stroke depression
- Sexual dysfunction
- Seizures
- Bladder & Bowel dysfunction
- Dysphagia (difficulty swallowing) – Speech therapy, bedside swallow study, barium swallow study,
    - Oral feeding – change diet to what patient can tolerate without aspiration
    - Non oral feeding – using percutaneous endoscopic gastrostomy (PEG) tubes for tube feeding
- Aphasia (difficulty understanding and speaking)
    - Expressive Aphasia: You can't speak but you can understand
    - Receptive Aphasia: You can speak but you can't understand (word salad)



Return to Work

- Positive predictors: High score on the Barthel index, shorter IPR stay, no aphasia,

Spinal Cord Injury (SCI)

Epidemiology:

- Causes: MVA > falls > violence

Types of SCI:

- Nontraumatic: spinal stenosis, spinal cord compression from cancer, multiple sclerosis, transverse myelitis, infection,
- Traumatic: fractures of the spine

SCI Classification:

- Quadriplegia: impairment of both upper and lower extremities
- Paraplegia: impairment of both lower extremities, arms are spared
- Dermatome: area of skin innervated by sensory axons within each segmental nerve
- Myotome: collection of muscle fibers innervated by the motor axons within each segmental nerve
- ASIA Classification of SCI (see next page)

Incomplete SCI syndromes

- Central cord syndrome: seen with hyperextension to c-spine. Sacral sensory sparing, greater motor weakness in upper extremities than lower extremities
- Brown-Sequard syndrome: ipsilateral loss of sensory at level of lesion, flaccid paralysis, loss of position sense and vibration below lesion, contralateral loss of pain and temp below lesion
- Anterior cord syndrome: flexion injuries, loss of motor function, pain and temp, preserved proprioception and light touch and deep pressure sensation
- Posterior cord syndrome: loss of proprioception, sparing of muscle strength, pain, and temp
- Conus medullaris syndrome: at L1-2, areflexic bladder and bowel
- Cauda Equina syndrome: below L1-2, LMN injury, bladder and bowel dysfunction, impotence, sexual dysfunction, areflexia of ankle and plantar reflexes

UMN vs LMN Injury

- UMN: Lesion above L1-2, results in hyperreflexia, Babinski, detrusor sphincter dyssynergia
- LMN: Lesion below L1-2, results in hyporeflexia, flaccid weakness, muscle atrophy, areflexic/hypotonic bladder

Complications of SCI

- Seen at T6 injuries or above:
    - Orthostatic hypotension: lightheadedness, dizziness, pre-syncope, paleness
    - Autonomic dysreflexia: HTN, bradycardia, sweating, flushing, headache due to pain, distended bladder, fecal impaction, acute abdomen (pancreatitis, cholecystitis, appendicitis), urinary tract infections,

- Neurogenic Bladder Dysfunction management: timed intermittent catheterization (IC), crede maneuver (suprapubic pressure), medications (bethanecol, anticholinergics [oxybutynin])
    - UMN Neurogenic bladder: failure to store – spastic bladder
        - Lesion above S2 (sacral micturition center)
        - Tx: oxybutynin or alpha agonists (ephedrine)
    - LMN Neurogenic Bladder: Failure to empty – flaccid bladder
        - Lesion involving S2-4
        - Tx: IC, crede maneuver, valsalva maneuver, bethanecol, alpha blockers to relax sphincter
- Neurogenic Bowel Dysfunction management:
    - Bowel program: for daily bowel movements, same time every time
        - Medications:
            - Stool softeners: Colace
            - Stool bulking agents: fiber, Metamucil
            - Osmotics: miralax, go-lytely, lactulose, fleets enema, magnesium citrate
            - Bowel stimulants: senna, Dulcolax,
        - May need digital stimulation
    - UMN Neurogenic bowel: lesions above L1-2
        - Decreased propulsion, will need bowel stimulants with a combo of osmotics and stool softeners
    - LMN Neurogenic Bowel: lesions below L1-2
        - Flaccid external anal sphincter, need more stool bulking agents
- Spasticity: abnormal, velocity-dependent increase in muscle tone (tightness)
    - Seen in stroke, cerebral palsy, TBI, SCI, multiple sclerosis, etc
    - Modified Ashworth Scale
    - Medication treatment: oral or intrathecal baclofen, diazepam, dantrolene, clonidine, tizanidine
- Sexual Dysfunction
- UTIs
    - +UTI in SCI vs Asymptomatic bacteriuria
        - Atypical symptoms – no dysuria, will have foul-smelling, cloudy urine, more incontinence
        - +UA is $>10^{5v}$wbc → get urine culture and start on abx (cipro, Bactrim, Macrobid, cefepime if febrile) adjust abx with urine culture susceptibilities
- Hypercalcemia
- Heterotopic ossification in hips
- DVT/PE: subq lovenox or heparin.
    - Duration: for incomplete and ambulators – just during IPR stay. For complete SCI – 8 wks
- Pain: nociceptive (MSK, visceral), neuropathic
- Pressure Ulcers: patients need to be repositioned every 2 hours

INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY (ISNCSCI)

ASIA — American Spinal Injury Association

ISCOS — International Spinal Cord Society

Patient Name _____ Date/Time of Exam _____

Examiner Name _____ Signature _____

RIGHT

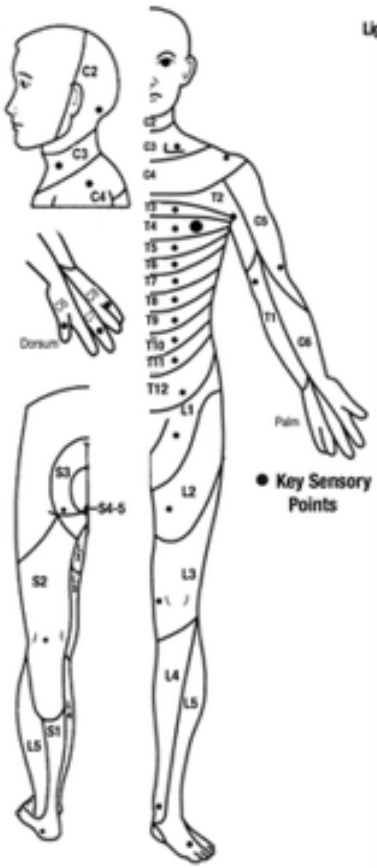
MOTOR KEY MUSCLES		SENSORY KEY SENSORY POINTS Light Touch (LTR)	Pin Prick (PPR)
	C2		
	C3		
	C4		
Elbow flexors	C5		
Wrist extensors	C6		
Elbow extensors	C7		
Finger flexors	C8		
Finger abductors (little finger)	T1		
	T2		
	T3		
	T4		
	T5		
	T6		
	T7		
	T8		
	T9		
	T10		
	T11		
	T12		
Hip flexors	L1		
Knee extensors	L2		
Ankle dorsiflexors	L3		
Long toe extensors	L4		
Ankle plantar flexors	L5		
	S1		
	S2		
	S3		
	S4-5		
RIGHT TOTALS (MAXIMUM)		(50)	(56) (56)

UER (Upper Extremity Right)

LER (Lower Extremity Right)

Comments (Non-key Muscle? Reason for NT? Pain?):

(VAC) Voluntary Anal Contraction (Yes/No) ☐



● Key Sensory Points

SENSORY KEY SENSORY POINTS Light Touch (LTL)	Pin Prick (PPL)	MOTOR KEY MUSCLES	
		C2	
		C3	
		C4	
		C5	Elbow flexors
		C6	Wrist extensors
		C7	Elbow extensors
		C8	Finger flexors
		T1	Finger abductors (little finger)
		T2	
		T3	
		T4	
		T5	
		T6	
		T7	
		T8	
		T9	
		T10	
		T11	
		T12	
		L1	
		L2	Hip flexors
		L3	Knee extensors
		L4	Ankle dorsiflexors
		L5	Long toe extensors
		S1	Ankle plantar flexors
		S2	
		S3	
		S4-5	
		LEFT TOTALS (MAXIMUM)	
(56)	(56)	(50)	

LEFT

UEL (Upper Extremity Left)

LEL (Lower Extremity Left)

MOTOR *(SCORING ON REVERSE SIDE)*

0 = total paralysis
1 = palpable or visible contraction
2 = active movement, gravity eliminated
3 = active movement, against gravity
4 = active movement, against some resistance
5 = active movement, against full resistance
5* = normal corrected for pain/disuse
NT = not testable

SENSORY *(SCORING ON REVERSE SIDE)*

0 = absent 2 = normal
1= altered NT = not testable

(DAP) Deep Anal Pressure (Yes/No) ☐

MOTOR SUBSCORES

UER ☐ + UEL ☐ = *UEMS TOTAL* ☐ LER ☐ + LEL ☐ = *LEMS TOTAL* ☐

MAX (25) (25) (50) MAX (25) (25) (50)

SENSORY SUBSCORES

LTR ☐ + LTL ☐ = *LT TOTAL* ☐ PPR ☐ + PPL ☐ = *PP TOTAL* ☐

MAX (56) (56) (112) MAX (56) (56) (112)

NEUROLOGICAL LEVELS
Steps 1-5 for classification as on reverse

	R	L
1. SENSORY	☐	☐
2. MOTOR	☐	☐

3. NEUROLOGICAL LEVEL OF INJURY (NLI) ☐

4. COMPLETE OR INCOMPLETE? ☐
Incomplete = Any sensory or motor function in S4-5

5. ASIA IMPAIRMENT SCALE (AIS) ☐

(In complete injuries only)
ZONE OF PARTIAL PRESERVATION
Most caudal level with any innervation

	R	L
SENSORY	☐	☐
MOTOR	☐	☐

REV 11/15

Muscle Function Grading

0 = total paralysis

1 = palpable or visible contraction

2 = active movement, full range of motion (ROM) with gravity eliminated

3 = active movement, full ROM against gravity

4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position

5 = (normal) active movement, full ROM against gravity and full resistance in a functional muscle position expected from an otherwise unimpaired person

5* = (normal) active movement, full ROM against gravity and sufficient resistance to be considered normal if identified inhibiting factors (i.e. pain, disuse) were not present

NT = not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of > 50% of the normal ROM)

Sensory Grading

0 = Absent

1 = Altered, either decreased/impaired sensation or hypersensitivity

2 = Normal

NT = Not testable

When to Test Non-Key Muscles:

In a patient with an apparent AIS B classification, non-key muscle functions more than 3 levels below the motor level on each side should be tested to most accurately classify the injury (differentiate between AIS B and C).

Movement	Root level
Shoulder: Flexion, extension, abduction, adduction, internal and external rotation **Elbow:** Supination	**C5**
Elbow: Pronation **Wrist:** Flexion	**C6**
Finger: Flexion at proximal joint, extension. **Thumb:** Flexion, extension and abduction in plane of thumb	**C7**
Finger: Flexion at MCP joint **Thumb:** Opposition, adduction and abduction perpendicular to palm	**C8**
Finger: Abduction of the index finger	**T1**
Hip: Adduction	**L2**
Hip: External rotation	**L3**
Hip: Extension, abduction, internal rotation **Knee:** Flexion **Ankle:** Inversion and eversion **Toe:** MP and IP extension	**L4**
Hallux and Toe: DIP and PIP flexion and abduction	**L5**
Hallux: Adduction	**S1**

ASIA Impairment Scale (AIS)

A = Complete. No sensory or motor function is preserved in the sacral segments S4-5.

B = Sensory Incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5 (light touch or pin prick at S4-5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.

C = Motor Incomplete. Motor function is preserved at the most caudal sacral segments for voluntary anal contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments (S4-S5) by LT, PP or DAP), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body.
(This includes key or non-key muscle functions to determine motor incomplete status.) For AIS C – less than half of key muscle functions below the single NLI have a muscle grade ≥ 3.

D = Motor Incomplete. Motor incomplete status as defined above, with at least half (half or more) of key muscle functions below the single NLI having a muscle grade ≥ 3.

E = Normal. If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

Using ND: To document the sensory, motor and NLI levels, the ASIA Impairment Scale grade, and/or the zone of partial preservation (ZPP) when they are unable to be determined based on the examination results.

ASIA
AMERICAN SPINAL INJURY ASSOCIATION

INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

ISCOS
INTERNATIONAL SPINAL CORD SOCIETY

Steps in Classification

The following order is recommended for determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation.

2. Determine motor levels for right and left sides.
Defined by the lowest key muscle function that has a grade of at least 3 (on supine testing), providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5).
Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.

3. Determine the neurological level of injury (NLI)
This refers to the most caudal segment of the cord with intact sensation and antigravity (3 or more) muscle function strength, provided that there is normal (intact) sensory and motor function rostrally respectively.
The NLI is the most cephalad of the sensory and motor levels determined in steps 1 and 2.

4. Determine whether the injury is Complete or Incomplete.
(i.e. absence or presence of sacral sparing)
*If voluntary anal contraction = **No** AND all S4-5 sensory scores = **0** AND deep anal pressure = **No,** then injury is **Complete.** Otherwise, injury is **Incomplete.***

5. Determine ASIA Impairment Scale (AIS) Grade:

Is injury Complete? **If YES, AIS=A** and can record ZPP (lowest dermatome or myotome on each side with some preservation)

NO ↓

Is injury Motor Complete? **If YES, AIS=B**

NO ↓ (No=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)

Are at least half (half or more) of the key muscles below the neurological level of injury graded 3 or better?

NO ↓ **AIS=C** **YES** ↓ **AIS=D**

If sensation and motor function is normal in all segments, AIS=E
Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact; the ASIA Impairment Scale does not apply.

Amputation

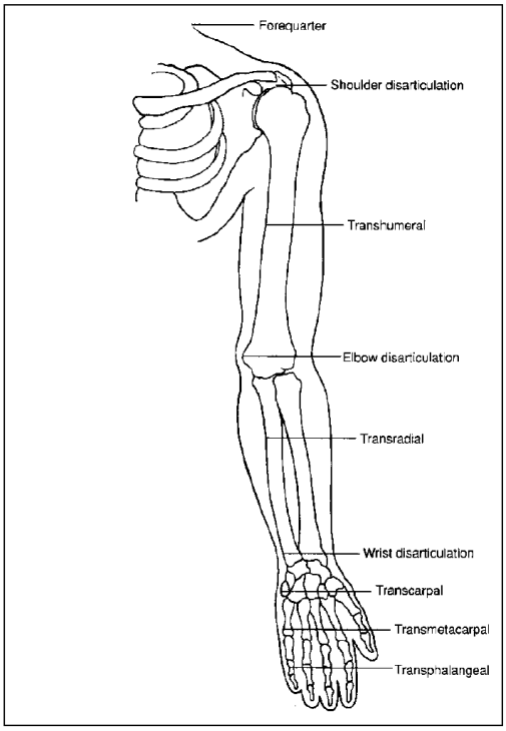
Epidemiology:

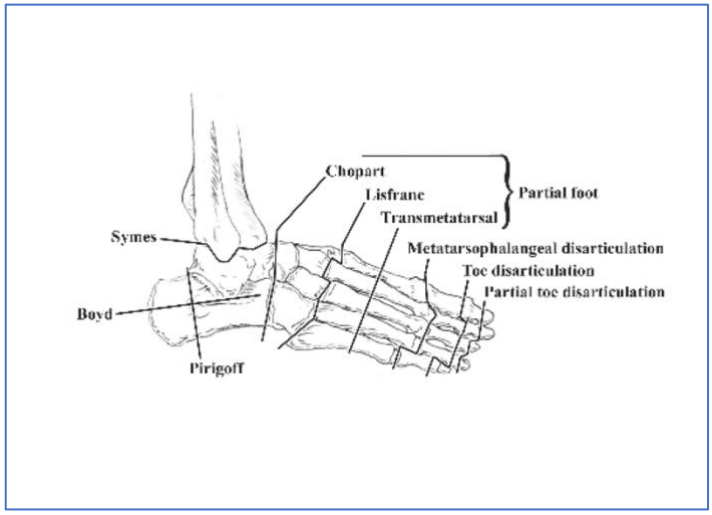
- Causes: DM, PAD/PVD, trauma, cancer, congenital
- DM is a contributing fator to 2/3rd of all LE amputations
- Trauma is leading cause of UE amputation

Road to Prosthesis:

- Start with shrinker for edema control right after amputation once wound is closed, then move to rigid removal dressing
- A few weeks to months later will be fitted for a temporary prosthesis and then another few months later will have permanent prosthesis

Upper and Lower Extremity Amputations :





Complications of LE Amputations

- Skin issues:
    - Folliculitis: hair root infection from poor hygiene. Clean w/ antispectic cleanser, may need PO abx
    - Boils/abscesses
    - Epidermoid cysts: sebaceous glands plugged by keratin, seen months after prosthesis is worn, need I&D
    - Choke syndrome: the socket is too tight leading to impaired venous return. Tx (relieving constriction, restoring total contact be/en socket and residual limb by reducing the number of socks)

- Bone problems:
    - Bone spurs - modify socket

    - Hypermobile fibula that is left longer than tibia
    - Bone overgrowth, HO - seen more in kids w/ acquired amputation and young adults w/ traumatic amputation

- Pain:
    - Incisional pain: can be d/t unprotected neuroma
    - Phantom sensation (not painful): sometimes diminishes w/ time, sometimes doesn't
    - Phantom pain: if it persists longer than 6 months --> prognosis for spontaneous recovery is poor. Doesn't occur with congenital limb deficiency
        - Tx: Physical modalities (acupuncture, TENS, vibration, us), meds (TCAs, GABA inhibitors, SNRI, SSRI, capsaicin, propanol, mexiletine), psych (hypnosis, biofeedback, cognitive therapy, behavioral)

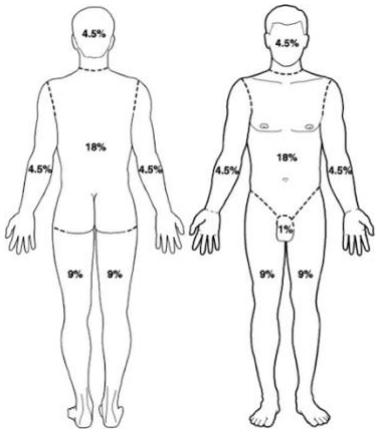
Functional K Levels Medicare Guidelines

K0: no ability or potential to ambulate or transfer, a prosthesis will not enhance QOL
K1: Household ambulator
potential or ability to transfer or ambulate on level surfaces at fixed cadence
K2: Limited Community Ambulator
Potential/ability to transfer or ambulate on low level barriers (curbs, stairs, uneven surfaces)
K3: Community Ambulator
Potential/ability to transfer or ambulate with variable cadence. Can transverse most environmental barriers.
K4: Active Adult/Athlete/Child
Potential or ability for ambulation that exceeds basic skills, includes high impact, stress, or energy levels

Burn Rehab

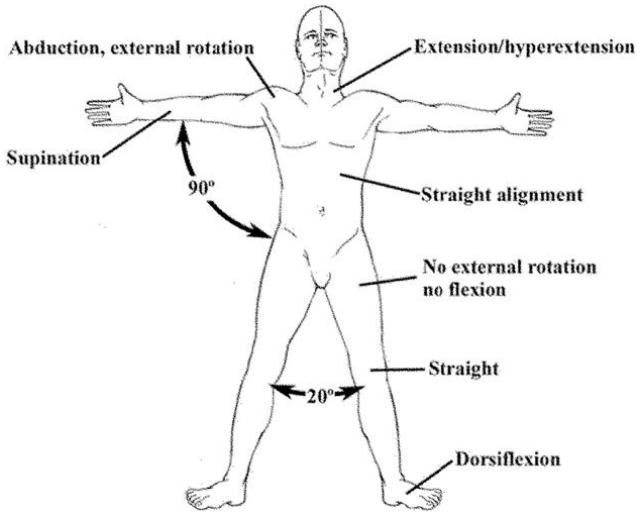
Assessment of Burns:

- Types of Burns: Thermal, electrical, chemical, radiation
- Size: Rule of 9's
- Depth:
    - 1st degree: outer layer of epidermis only
    - 2nd degree/superficial partial thickness: most of epidermis
    - 3rd degree/deep partial thickness: all the epidermis, upper 1/3rd of dermis
    - 4th degree/full thickness: all of epidermis and dermis



Complications after Burns:

- Heterotopic ossification – seen in elbows more commonly
- Contractures: scars after burns grow and contract causing decreased range of motion, pain, decrease functionality
    - To prevent contractures, patients should be placed in anti-contracture position
    - May need splinting to keep the patient in this position
- Nutrition: after a burn, there is a loss of body fat, may need more than 2000 calories a day in addition, may need PEG tube placement for feeds



Pediatric Rehab

Cerebral Palsy:

- Nonprogressive lesion to an immature brain causing disorder of movement control, posture, can also affect cognition and sensation
- Can occur in utero, near time of delivery, or within the first 3 years of life
- Risk factors: in utero intracranial hemorrhage, TORCH infections, prematurity, hyperbilirubinemia, trauma, infections, cancers, seizures
- Types:
    - Spastic – 75% of all CP cases
    - Dyskinetic (abnormal movements like chorea, athetosis, dystonia, ataxia)
    - Mixed (spastic and dyskinetic)
- Complications: mental retardation, seizures, hearing impairements, language disorders, behavorial disorders, bladder and bowel dysfunction, spasticity
- Therapeutic management: various exercise techniques with PT/OT
    - Bobath – neurodevelopmental treatment to normalize tone, inhibit abnormal primitive reflex patterns,
- Spasticity management with medications, bracing (AFOs, KAFOs, HKAFOs), nerve blocks with phenol or alcohol, Botox injections, surgery (selective dorsal rhizotomy)

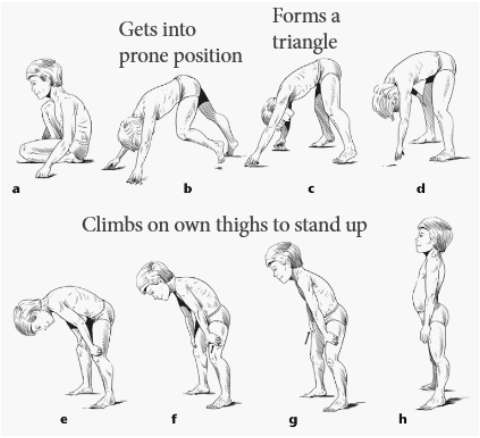
Spina Bifida:

- Neural tube defects (NTD)caused by congenital malformations of the spinal cord
- Risk factors: low socioeconomic class, midspring conception, maternal obesity, in utero exposure to anticonvulsant meds (valproic acid, carbamazepine), maternal febrile illness
    - *take folic acid during pregnancy to reduce the risk of NTDs
- Types:
    - Spina bifida occulta: failure of fusion of posterior elements of vertebrae resulting in tuft of hair on back or pigmented nevus, dimple,
    - Spina bifida cystica: protruding cystic sac bulging out on the back
- Complications: causing change in function, mentation,
    - Arnold Chiari Malformation: downward displacement of medulla and brainstem
    - Hydrocephalus: increased cerebrospinal fluid in the head
    - Tethered cord: abnormal attachment of spinal cord at the distal end
    - Syringomyelia: fluid filled central cavity in the spinal cord
    - Neurogenic bladder and bowel
    - Skin breakdown

Myopathic Neuromuscular Diseases:

<u>Duchenne Muscular Dystrophy (DMD):</u>

- X-linked, progressive disease leading to absence of dystrophin (supports muscle fiber strength)
- Symptoms: delay in walking, abnormal gait, frequent falling, difficulty climbing stairs
- Signs: waddling gait, Gower's sign, proximal muscle weakness (shoulders and hips more than hands and feet)
- Lose ability to walk by 8 to 12 years



- Complications:
    - Contractures
    - Scoliosis
    - Decreased pulmonary reserve
    - EKG abnormalities – the heart is a muscle therefore the lack of dystrophin affects the myocardium
- Management: promote activity, prevent contractures and deformities by passive stretching, may need bracing, avoid immobilization, minimize progression to scoliosis by preventing postural defects

Becker's Muscular Dystrophy (BMD):

- X-linked, later onset, slower progression than DMD, dystrophin is present but less than normal levels
- Symptoms: difficulty with running and climbing stairs, cramps with exercise
- Signs: mild functional disability, proximal muscle weakness, prominent calves, waddling gait
- Continue to ambulate well into their late teenage years
- Management: promote activity, prevent contractures and deformities by passive stretching, may need bracing,