SOMATOSENSORY FUNCTION, PAIN, & HEADACHE

CHAPTER 50
IT'S ALL IN YOUR MIND—HAVE YOU BEEN DOING ANY THINKING LATELY?
ACUTE PAIN

- Distinction between acute & chronic pain is important as treatment is very different.
- Arbitrarily defined as lasting less than 6 months; caused by an acute, self-limiting process expected to resolve.
- See table 50-1 Pg 1175 for comparison, autonomic and psychological components.
- Teleologically, acute pain is the body’s way of getting your attention re a problem; can serve to immobilize an area to prevent further trauma.
CHRONIC PAIN

- Arbitrarily defined as lasting more than 6 mos.
- A better definition is pain lasting longer than the normal expected time of healing of the causative pathology (Int’l Assoc for the Study of Pain).
- Clinical course is quite variable.
- A leading cause of disability in the U.S.
- Serves no “useful purpose,” unlike acute pain, which is a signal to the organism of an underlying condition needing medical attention.
CHARACTERISTICS OF CHRONIC PAIN

- Can be unrelenting and severe.
- Can have episodes of escalation of severity.
- Acute pain can coexist / be superimposed on chronic pain: sickle cell disease.
- More significant impact on psychological, physiological, familial, and economic stresses.
- Can be associated w/: loss of sleep, appetite, and depression.
- Autonomic responses absent, decreased.
CHARACTERISTICS OF CHRONIC PAIN

DEPRESSION AND DECREASED PAIN TOLERANCE

- Chronic pain depletes endorphins and serotonin.
- No endorphins $\rightarrow$ no natural pain relievers.
- No serotonin $\rightarrow$ depression.
- Depression $\rightarrow$ worsening of pain.
- Treating depression pharmacologically can be effective in the mgmt. of chronic pain; SSRI’s tricyclic antidepressants.
A WORD ABOUT “ADDICTION”

- DRUG ABUSE.
- TOLERANCE.
- DEPENDENCE- psychological, physical.
- ADDICTION.
- PSYCHOLOGICAL DEPENDENCE AND ADDICTION.
NEUROPATHIC PAIN

- Pain arising from “damage” to peripheral nerves.

**CAUSES**

- **SINGLE AREA:** local trauma / crush injuries; nerve entrapment / compression; neuralgias such as trigeminal neuralgia, post-herpetic neuralgia.

- **SYSTEMIC:** diabetic neuropathy, alcohol, hypothyroidism, renal failure, Rx w/ neurotoxic agents, complex regional pain syndrome (CRPS).
NEUROPATHIC PAIN

MANIFESTATIONS

- Highly variable.
- Pain can be accompanied by paresthesias, numbness.
- Persistent or intermittent.
- Burning, stabbing, jabbing, shooting.
- **Allodynia** - pain that results from a non-injurious stimulus to the skin.
TRIGEMINAL NEURALGIA

- aka Tic Douloureux.
- Involves pain in the distribution of the trigeminal (5th) cranial nerve; usually the mandibular or maxillary branches.
- Usually unilateral.
- Intermittent, but excruciating pain, that is stabbing. Allodynia.
- Cause is controversial → ? anomalous artery or vein impinging on the trigeminal nerve root.
TRIGEMINAL NEURALGIA

DIAGNOSIS

- Clinical, look for and rule out other neurologic disorders.
- R/O multiple sclerosis in young people.
POSTHERPETIC NEURALGIA

- Pain that persists for more than a month after the onset of Herpes zoster (shingles).
- 10-70% of patients w/ shingles will develop postherpetic neuralgia, higher risk in the elderly.
- Working hypothesis is “damage to the peripheral nerves, dorsal root ganglia, and alteration in CNS processing of pain stimuli.
- Affected nerves fir spontaneously and have lower thresholds for discharge → exaggerated response to stimuli
POSTHERPETIC NEURALGIA

Incidence /development of postherpetic neuralgia may be reduced by early treatment w/ antiviral drugs effective against Herpes: Zovirax (acyclovir), Valtrex (valcyclovir).
COMPLEX REGIONAL PAIN SYNDROME

“CRPS”

Formerly Reflex Sympathetic Dystrophy (RSD).

2 EXCITING FLAVORS: Type I and Type II.

Pretty much the same, except in Type II there is history of damage to a peripheral nerve.

“A regional, neurotrophic pain problem that affects one or more limbs.”
COMPLEX REGIONAL PAIN SYNDROME

MANIFESTATIONS

- Allodynia, hyperalgesia, or spontaneous pain.
- Severe, burning pain.
- Pain increases with touch, activity, emotions, anything that increases peripheral sympathetic stimulation.
COMPLEX REGIONAL PAIN SYNDROME

SYMPATHETIC COMPONENTS

- Vascular and trophic changes to the skin, soft tissues, bone.
- Rubor vs pallor.
- Sweating vs dryness.
- Edema, skin atrophy.
- Patchy osteoporosis.
- Gave rise to the former name Reflex Sympathetic Dystrophy.
COMPLEX REGIONAL PAIN SYNDROME

ETIOLOGY

- See your text.
- Obscure.
- Increased sensitivity to sympathetic stimulation.
- Heightened afferent activity.
- Neurogenic inflammation by Substance P, histamine, etc.
HEADACHES

- We’ll cover:
  - 1) Migraines.
  - 2) Cluster headaches.
  - 3) Tension headaches.

- These are “primary” headaches.
- Read text re causes and characteristics of secondary headaches: neoplasms, infection, etc. When to be concerned, refer, etc.
MIGRAINES

- From the Greek hemi = half and kranion = head.
- Hemi + kranion = hemikranion → migraine.
- 18% of women, 6% of men.
- Familial, inherited as an autosomal dominant w/ variable penetrance.
MIGRAINES

- **FLAVORS:**
  - 1) Migraine w/ aura - 15%.
  - 2) Migraine w/ out aura - 85%.
  - 3) Mixed - combination of migraine w/ tension headache, sinus headache, etc.

- **SUB-TYPES:** ophthalmoplegic migraine, hemiplegic migraine, aphasic migraine, retinal migraine.
- Also “classic” migraine and atypical migraine.
MIGRAINE W/OUT AURA

- Unilateral, pulsatile, throbbing.
- 1-2 days, worse w/ activity.
- Nausea, vomiting.
- Sensitivity to light (photophobia) and sound (phonophobia), Can be extreme.
- Visual symptoms- common; flashes of light, “stars,” “sparks.”
MIGRAINE W / AURA

- Similar symptoms as migraine w/out aura, but preceded by a prodrome, the “aura.”
- Aura develops over 5-20 minutes, followed by the headache.
- Visual symptoms- flashes of light, scotomata.
- Neurologic symptoms- tingling or numbness on one side of the face or in one hand / fingers; when severe can have motor difficulty.
- The trick is to abort the headache w/ the onset of the aura.
MIGRAINES

ETIOLOGY / PATHOPHYSIOLOGY

- See text. “Vascular headache.”
- Vasodilatation is a constant, preceded by vasospasm / vasoconstriction, accounting for some of the Sx’s of the aura.
- See text re vascular ion channels, trigeminal nerve involvement, neuropeptides, etc.
MIGRAINES

OTHER FACTORS / TRIGGERS

- Estrogen- “menstrual migraine.”
- Dietary triggers- MSG, smoked stuff (cheese, meats), chocolate, certain wines w/ nitrites and sulfites
- Helpful in establishing the triggers is a diary keeping track of when migraines occur in relation to menses, diet, etc. Careful reading of labels is crucial.
CLUSTER HEADACHES

- Uncommon. 1 / 1000. Mostly men (80%).
- Occur in clusters over weeks to months, followed by pain-free intervals.
- Rapid onset. Short duration- 15 min to 3 hrs.
- Often assoc w/ restlessness, agitation, also conjunctival redness, rhinorrhea, nasal congestion, forehead / facial pain. As such, often mistaken for a sinus headache.
CLUSTER HEADACHES

ETIOLOGY / PATHOPHYSIOLOGY

- “Not completely known.”
- See text.
- Familial / hereditary.
- “Interplay of vascular, neurogenic, metabolic, and humoral factors.”
TENSION HEADACHE

- The most common type.
- Not as severe, typically does not interfere with function.
- Dull, aching, diffuse, non-descript.
- “Hat band” distribution.
- No N / V.
- Infrequent, episodic, chronic.
TENSION HEADACHE

ETIOLOGY / PATHOPHYSIOLOGY

- ? Sustained contraction (tension) of the muscles of the head and neck.
- Lately, a vascular component has been proposed.
- Caffeine- is a vasoconstrictor. Withdrawal results in rebound vasodilatation. Symptoms of this type of headache more consistent w/ those of tension headache rather than migraine.
TENSION HEADACHE

**ETIOLOGY / PATHOPHYSIOLOGY**

- Some headache meds contain ingredients addressing both muscle contraction / tension AND vasodilatation.

- Midrin- contains tylenol, a vasoconstrictor, and a muscle relaxer.

- With pharmacologic treatment of all types of headache, need to be careful for “rebound” headaches when used chronically.
TMJ – RELATED HEADACHES

- Mentioned only for completeness.
- Consider in cases where treatment for the “usual” types of headaches is not effective.
- Deserves referral to practitioner w/ experience, skill, and the set-up to treat disorders of the TMJ.
HERNIATED INTERVERTEBRAL DISK

- See fig 51-9, Pg 1206.
- “H.N.P.”- herniated nucleus pulposus, “slipped disk.”
- Nucleus pulposus protrudes thru the annulus fibrosus.
- Herniation is usually posterior, into the intervertebral foramen containing the nerve root.
- 95% occur at L4-L5 or L5-S1.
- When cervical, C5-C6, C6-C7.
HERNIATED INTERVERTEBRAL DISK

**MANIFESTATIONS**

- Depends on the nerve root being compressed.
- See Fig 51-10, Pg 1207 for dermatomes.
- Pain: but it may be absent. Intensified by any Valsalva maneuver- coughing, lifting, etc
- Sensory: paresthesias, numbness.
HERNIATED INTERVERTEBRAL DISK

DIAGNOSIS

- Hx and PE.
- Tests of muscle strength, DTR’s.
- Straight leg raising test: positive if pain occurs upon raising the leg to 60° or less in the supine position.
- MRI / CT, myelogram.
MONONEUROPATHIES

- Involve one nerve root.
- Caused by: trauma, infection.
- Symptoms referable to the nerve root involved.
- Example: Carpal tunnel syndrome.
POLYNEUROPATHIES

- Involve multiple nerve roots, with demyelination or axonal degeneration.
- Signs / Sx’s- motor, sensory, both.
- Caused by: immune mechanisms (Guillian-Barre), toxins (lead, alcohol), metabolic disease (diabetes, uremia).
PARKINSON’S DISEASE

- Degenerative disorder of the basal ganglia resulting in destruction of the nigrostriatal pathway and decreased production of dopamine
- Caused by: idiopathic; encephalitis; antipsychotic drugs; chemicals / toxins; repeated head trauma; cerebrovascular disease; genetic.
- Onset usually after age 50, 1% of the population over 60.
PARKINSON’S DISEASE

MANIFESTATIONS

- Triad of:
  - 1) Tremor.
  - 2) Rigidity.
  - 3) Bradykiesia.
PARKINSON’S DISEASE

MANIFESTATIONS: THE TREMOR

- “Pill rolling” tremor or the hands / fingers.
- Also alternating flexion & contraction of the feet, head, neck, face, lips, tongue, jaw.
- Begins as a unilateral tremor, progresses to bilateral.
- Resting tremor, resolves w/ movement and sleep.
PARKINSON’S DISEASE

MANIFESTATIONS: RIGIDITY

- Resistance to movement of the flexors and extensors thru their full range of movement.
- “Cog-wheel rigidity-” evident with passive range of motion.
- May develop flexion contractures.
- Unilateral → bilateral.
PARKINSON’S DISEASE

MANIFESTATIONS: BRADYKINESIA

- Difficulty in both initiating and stopping voluntary movement.
- The most disabling feature of Parkinson’s.
- Slow, “shuffling” gait.
- Expressionless face- “Parkinson’s Facies.”
- Speech- slow, monotonous, w/ out modulation.
PARKINSON’S DISEASE

MANIFESTATIONS: OTHER

- Autonomic features: Due to the effects of the basal ganglia on the ANS: excess sweating, salivation, lacrimation, constipation, impotence, orthostatic hypotension, dysphagia, thermal regulation.

- Dementia. 20%. Develops late.
MULTIPLE SCLEROSIS

- A demyelinating disease.
- The most common cause of non-traumatic neurologic disability among young and middle-aged adults.
- Onset between age 20 and 40 in 2/3.
- More common in colder, northern latitudes.
- Women 2X more than men.
MULTIPLE SCLEROSIS

PATHOPHYSIOLOGY

- Demyelination of the nerve fibers in the white matter of the brain, spinal cord, and optic nerve.
- Myelin- formed by the oligodendrocytes; serves as an electrical insulator.
- Demyelination results in: decreased conduction velocity, conduction blocks.
- End result is a sclerotic patch of nerve tissue; the “plaque.”
MULTIPLE SCLEROSIS

PATHOPHYSIOLOGY

- Felt to be “an immune-mediated disorder in genetically-susceptible individuals.”
- Initiating factor(s) not known. ? Viral.
- Immune components involved: CD8 and CD4 T-cells, antibodies, macrophages.
- Not clear as to whether the process effects the myelin itself or the oligodendrocytes that produce it. Maybe both.
MULTIPLE SCLEROSIS

MANIFESTATIONS

- See text for broad range of Sx’s.
- Common are:
  - 1) Paresthesias.
  - 2) Optic neuritis- loss of vision, clouding, loss of parts of the visual field, pain w/ eye movement, diplopia, gaze paralysis.
- Also problems w/ : speech, muscle strength, gait and coordination, balance.
MULTIPLE SCLEROSIS

MANIFESTATIONS

- Paresthesias: numbness, tingling, burning on the face or extremity.
- Pain & spasticity- 80% have it at some point.
- Other: sexual and bladder dysfunction (incontinence), vertigo, nystagmus, fatigue (very common,).
- Psychological: mood swings, depression, apathy, inattentiveness, memory loss.
MULTIPLE SCLEROSIS

CLINICAL COURSE

- Diagnosis often not considered / delayed due to vague, variety of symptoms.

- 4 CATEGORIES:
  - 1) Relapsing-remitting.
  - 2) Secondary progressive.
  - 3) Primary progressive.
  - 4) Progressive relapsing.
MULTIPLE SCLEROSIS

CLINICAL COURSE

RELAPSING-REMITTING

- Worsening → recovery → remission → relapse

SECONDARY PROGRESSIVE

- Relapsing-remitting course transforms into gradual neurologic deterioration w/ or w/ out superimposed acute relapses.

PRIMARY PROGRESSIVE

- Continuous deterioration from the onset of Sx’s
MULTIPLE SCLEROSIS

CLINICAL COURSE

PROGRESSIVE RELAPSING

- Gradual deterioration from onset of Sx’s but with superimposed relapses.
MULTIPLE SCLEROSIS

DIAGNOSIS

- Clinical- Hx & PE.
- Lab- CSF analysis; elevated IgG. Not specific for MS.
- MRI. BUT → normal findings DO NOT preclude the Dx.
CHAPTER 52
STROKE

■ 4 EXCITING FLAVORS:
■ 1) ISCHEMIC.
■ 2) HEMORRHAGIC.
■ 3) THROMBOTIC. A TYPE OF ISCHEMIC STROKE.
■ 4) EMBOLIC. A TYPE OF ISCHEMIC STROKE.
ISCHEMIC STROKE

- Most common type- 70-80%.
- Ischemia resulting from embolism or thrombosis.
- See text for 5 sub-types.
- The TIA is a type of ischemic stroke, akin to angina; ie, temporary, and a precursor to a subsequent stroke. The “mini stroke.”
ISCHEMIC STROKE

THROMBOTIC TYPE

- Results from thrombosis in an arteriosclerotic vessel.
- Most commonly at arterial bifurcations.
- Internal carotids, vertebral arteries, junction of vertebral and basilar.
- Seen in patients with underlying atherosclerosis: ASCVD, peripheral vascular disease.
ISCHEMIC STROKE

EMBOLIC TYPE

- Results from a thrombus that embolizes, lodging in a cerebral vessel; most commonly the middle cerebral artery.

- Originate from:
  1) The heart- atrial fib, MI, rheumatic heart disease, ventricular aneurysm, etc.
  2) The carotids- on an atherosclerotic plaque.
HEMORRHAGIC STROKE

- The most frequently fatal stroke.
- RISK FACTORS:
  - Most common are age and hypertension.
- OTHER RISK FACTORS:
  - Aneurysm (congenital berry), coagulopathy, trauma, AVM, vasculitis.
  - Blood plus resulting edema progresses rapidly to coma and death. Those that survive have significant deficits / impairments.
STROKE

MANIFESTATIONS

- See text.
- Varying degrees of motor and sensory deficit depending on location and degree of cerebral ischemia / infarction.
- Motor deficit is contralateral, except on the face which is ipsilateral.