Nervous System: Neural Integration
(Chapter 15 &16)
Lecture Materials
for
Amy Warendra Czura, Ph.D.
Suffolk County Community College
Eastern Campus

Primary Sources for figures and content:

Neural Integration

Central Nervous System
(higher order function)
↑↓
Peripheral Nervous System
↑ ↓
Afferent Division  Efferent Division
(sensory) (motor)

Afferent Division
sensory receptors → sensory pathways →
1. (somatic sensory info)
   -sensory cortex of cerebrum
   -cerebellum
2. (visceral sensory info)
   -reflex centers in brainstem
   -reflex centers in diencephalon
Sensory receptors

general senses:
- temp, pain, touch, pressure, vibration, proprioception
- simple receptors located anywhere on body

special senses:
- olfaction, vision, gustation, hearing, equilibrium
- complex receptors located in specialized sense organs

transduction = conversion of environmental stimulus into action potential by sensory receptor

sensation = the sense info; action potentials

perception = conscious awareness of sensation
-receptors specific for particular type of stimulus

-specificity is due to structure of receptor
-simplest receptors are dendrites (free nerve endings), least specific
receptive field = area monitored by single receptor (e.g. touch: arm vs. fingertip)

labeled line = link between receptor and processing site in CNS
-stimulation anywhere on labeled line will produce same perception (e.g. phantom limb)

stimulus → receptor → transduction → action potential → sensation → CNS perception
Tonic receptors:
- always active
- signal at different rate when stimulated
- monitor background levels

Phasic receptors:
- activated by stimulus
- monitor intensity and rate of change of stimulus

Adaptation = reduced sensitivity to a constant stimulus
1. Peripheral adaptation = reduction in receptor activity
   - phasic $\rightarrow$ fast adapting
   - tonic $\rightarrow$ slow or non adapting
2. Central adaptation = inhibition of nuclei along labeled line
   - not all pathways will adapt
Four types of general sensory receptors:
1. pain = nociceptor
2. temperature = thermoreceptor
3. physical = mechanoreceptor
4. chemicals = chemoreceptor
-al! can be found in both somatic (exteroceptors) and visceral (interoceptors) locations except:
-proprioceptors (a mechanoreceptor) are somatic only (receptors on handout)
Somatic Sensory Pathways
- consist of two or three neurons:

1. first order neuron:
   - sensory neuron
   - connects from receptor to CNS
   - cell body is in dorsal root ganglion/ cranial nerve ganglion

2. second order neuron:
   - interneuron (stimulated by first order)
   - located in spinal cord or brain stem
   - subconscious processing of info

3. third order neuron:
   - located in thalamus
   - relays info to primary somatosensory cortex of cerebrum for conscious awareness (perception)

sensory homunculus
-only ~1% of somatic sensory info reaches cerebrum (major changes only, “background” is filtered)
(-LSD interferes with sensory damping/filtering = sensory overload)
-all sensory info undergoes decussation in spine before reaching target in CNS

Visceral Sensory Pathways
-interoceptors transmit info to solitary nucleus of medulla oblongata for relay to visceral centers in brainstem and diencephalon (no perception)
-two neurons: 1st and 2nd order
Efferent Division
conscious and subconscious motor centers in brain → motor pathways →
1. Somatic Nervous System → skeletal muscles
2. Autonomic Nervous System → visceral effectors (smooth & cardiac muscle, glands, adipose)
Somatic Nervous System
-motor control of skeletal muscle
-consists of two neurons:

1. upper motor neuron
   -has soma in CNS processing center:
     a. primary motor cortex of cerebrum
        (voluntary control)
     b. cerebrum, diencephalon, and brain stem (subconscious control: reflex)
     c. basal nuclei of cerebrum and cerebellum (coordination, balance, fine tuning)

2. lower motor neuron
   -soma in brain stem or spinal cord
   -links to skeletal muscle motor unit
Motor Related Disorders

*Parkinson’s Disease*
-jittery movements: lack fine tuning of motor
-results from degeneration of dopamine neurons of substantia nigra (inhibits basal nuclei)
-overactive basal nuclei = “ticks”

*Amylotrophic Lateral Sclerosis*
-degeneration of motor neurons in CNS
-causes muscle atrophy and death

*Epilepsy*
-1/25 people
-wide range in condition: absence seizures (blank) to grand mal seizures (convulsions, unconscious)
-uncontrolled/chaotic neuron activity in brain: blocks normal messages
Autonomic Nervous System
-motor control of visceral effectors
-involves three neurons:

1. visceral motor nuclei in hypothalamus to autonomic nuclei in CNS
2. autonomic nuclei to autonomic ganglia in PNS
3. autonomic ganglia to visceral effector
two subdivisions

1. sympathetic = “fight or flight”
   (“s” for “stress”)
2. parasympathetic = “rest and digest”
   (“p” for “peace”)
-typically oppose each other on same effector
-some effectors innervated by only one:
   -blood vessels and sweat glands - sympathetic only
   -smooth muscle of eye - parasympathetic only
Sympathetic Division
-prepares body for heightened somatic activity
-ganglia:
  1. located near spinal cord
  2. adrenal medulla
    -center of adrenal gland (above kidney)
    -releases epinephrine and norepinephrine as hormones into blood to control effectors body wide at once (endocrine function)
-Sympathetic activation results:
  -increased alertness
  -insensitivity to pain
  -elevation in BP, heart, respiratory rate
  -elevation in muscle tone
  -mobilization of energy reserves
  -secretion from eccrine sudoriferous glands
-Neurotransmitters:
1. preganglionic neurons release acetylcholine (cholinergic synapse) → EPSP on ganglionic neuron
   - directly open ion channel
   - fast acting, short lived
2. Ganglionic neurons / postganglionic fibers release norepinephrine at effector (adrenergic synapse)
   (-NE and E from adrenal medulla: hormones)
   - result depends on type of receptor:
     1. $\alpha_1$ and $\beta_1$ receptors = excitatory/stimulatory to effector
     2. $\alpha_2$ and $\beta_2$ receptors = inhibitory
        (beta-blockers: block $\beta_1$ receptors)
   - G protein → second messengers
   - slow acting but long lasting
Parasympathetic Division
- stimulates visceral activity
- maintains homeostasis
- ganglia located in or near effector
- vagus nerve carries 75% of parasympathetic innervations
- Parasympathetic activation results:
  - constriction of pupils
  - secretion by digestive glands
  - secretion of hormones for nutrient uptake
  - sexual arousal
  - activation of digestive tract
  - defecation and urination
  - constriction of respiratory pathways
  - reduction in heart rate
Neurotransmitters:
- all release Ach: all cholinergic synapses
- effects quick, localized, short-lived
- type of effect depends on receptor:
  1. Nicotinic receptor = excitatory effect on target
  2. Muscarinic receptor = inhibitory or excitatory, depending on target cell

Higher Order Functions
1. involve cerebral cortex
2. involve both conscious and subconscious processing
3. are not part of genetic wiring (reflex): can be modified
   e.g. memory and consciousness

Memory
   memory = storage and retrieval of info
   fact memories = specific bits of info
   skill memories = learned motor behaviors
short term memory (STM)
- primary / working memory
- rapid recall but short retention
- store 7-8 bits of info at one time
- STM can be converted to long term memory for more permanent storage

memory consolidation = STM $\rightarrow$ LTM
- performed by hippocampus
- depends on:
  1. emotional state
  2. rehearsal
  3. association
  4. automatic memory

long term memory (LTM)
- infinite info
- can be stored for lifetime
- secondary memories: fade with time, can be difficult to recall much later
- tertiary memories: part of one’s consciousness (e.g. name)
-LTM are broken into component parts to store in appropriate cerebral cortex (e.g. visual, olfactory, etc.)

Mechanism of memory storage not clearly understood but involves:
1. new mRNA and protein synthesis in neurons involved
2. change of shape of dendritic spines
3. change in size and number of synaptic terminals
4. release of more neurotransmitter

Amnesia = loss of memory, due to disease or trauma of hippocampus and amygdala
1. retrograde amnesia = lose memories of past events, remember now → forward
2. anterograde amnesia = unable to store new memories, only remember past

Consciousness
conscious = aware of external stimuli
unconscious = range of unawareness:
   drowsy → →→→→→→→ brain dead
Sleep

Sleep = partial unconsciousness from which a person can be aroused with stimuli

1. Deep sleep
   - relaxed state
   - heart and respiratory rate decreased
   - minimal activity in cerebral cortex

2. REM (Rapid Eye Movement) sleep
   - active, dreaming state
   - cerebral cortex as active/ more active than in conscious state
   - but little reaction to outside stimuli
   - skeletal muscles inhibited
-alternate between deep and REM sleep throughout sleep period
-sleep required for life, but not clear why
-lack of sleep leads to serious disturbance in mental function
-during sleep protein synthesis in neurons increases: sleep may be used to repair and recharge neural tissue

*Narcolepsy* = condition where person lapses abruptly into sleep for ~15min
-usually follows pleasant event
-cause unknown
-sufferers show reduced levels of REM sleep at night
Sleep apnea = person stops breathing until hypoxia (lack of O₂) wakes them
-hypoxic wake response ability declines with age or respiratory illness

Arousal
-requires Reticular Activating System (RAS)
-RAS located in brainstem, provides consciousness

mechanism:
- stimulation of RAS → activation of cerebral cortex
- positive feedback (reverberation) on RAS maintains consciousness after initial stimulus
- over time RAS becomes less responsive = sleepy feeling

-internal clock in suprachiasmatic nucleus of hypothalamus sets normal sleep-wake cycle
Age Related Changes
- ↓ brain size and weight (cerebrum)
- ↓ number of neurons
- ↓ blood flow to brain (↑ chance of stroke)
- ↓ number of synapses
- ↓ neurotransmitter production
- accumulation of deposits:
  a. inside cells:
     - lipofuscin = granular pigment
     - neurofibrillary tangles = packed neurofibrils
  b. extracellular
     - plaques = collections of fibrillar proteins entangling abnormal cell processes
     (amyloid proteins = normal proteins misfolded become sticky)
- all forms of deposits affect processing and memory ability, motor speed, and sensory sensitivity
- increased diseases:
1. Alzheimer’s Disease
   - loss of higher order functions
   - occurs in 15% over 65 years
   - progressive, untreatable
   - due to reduction in Ach levels and accumulation of beta amyloid peptide (plaques and tangles)
   - current treatments block Ach breakdown

2. Huntington’s Disease
   - genetic, middle age onset
   - accumulation of huntington protein kills neurons of basal ganglia and cerebral cortex → ticks, cognitive dysfunction
   - progressive and fatal
   (onset → death in ~15 years)