

# INTRODUCTION

## System Concept

### 1. Central nervous system (CNS)

#### a. Significance

- \_ Not central in location
- \_ Central in importance -- controls entire system

#### b. Organs

- \_ Brain
  - highest level of control
  - coordinates entire system
- \_ Spinal cord
  - can operate independently
  - all actions influenced by brain

### 2. Peripheral nervous system (PNS)

#### a. Significance

- \_ Peripheral in location -- extends from central
- \_ Peripheral in importance -- cannot work alone

#### b. Organs -- all nerves

#### c. Divisions

- \_ Somatic -- mainly controls skeletal muscles
- \_ Visceral (autonomic)
  - controls involuntary muscles
  - controls glands
  - CNS component as well [ details later ]

### 3. Functional divisions

#### a. General

- \_ Different approach than structural
- \_ Includes both CNS & PNS

#### b. Sensory

- \_ Concerned with receptors (sense organs)
- \_ Incoming phenomena -- receiving information

#### c. Motor

- \_ Concerned with muscles & glands -- called effectors
- \_ Outgoing phenomena -- effecting an action

## Functions

### 1. General

#### a. System deals with information

- \_ Transmitting -- most basic
- \_ Receiving -- sensory
- \_ Storage -- memory
- \_ Processing -- associating varied info
- \_ Responding -- motor

#### b. Purposes

- \_ Integration -- interrelating all body parts
- \_ Coordination -- purposeful interrelation

#### c. Other integrating/coordinating systems

- \_ Endocrine -- close associations with nervous
- \_ Cardiovascular -- physical, not functional

## 2. Most basic function

- \_ Conduction of nervous impulses
- \_ Only means for all other specific functions

## 3. Specific functions

- a. Stimulation (excitation) -- to start or increase
- b. Inhibition -- to stop or decrease
- c. Reception
  - \_ Via receptors (sense organs)
  - \_ Gives awareness of environment
- d. Secretion
  - \_ Involves transmitters or hormones (overlaps endocrine)
  - \_ For chemical communication

# NERVOUS TISSUE

## Basic Characteristics

### 1. Irritability

- \_ Best response to environmental stimuli
  - \_ Results in cellular membrane alterations
  - \_ Utilized in performing functions
- \_ Muscle tissue responds somewhat less
- \_ Neuroepithelial tissue is less responsive
- \_ All other tissues slightly to not responsive

### 2. Conductivity

- \_ Logical outcome of extreme irritability

\_ Movement (conduction) of membrane alterations

\_ Constitutes actual nervous impulse

## Neurons

### 1. Importance

#### a. Basic unit

All system functions performed by these cells

#### b. Arrangement

\_ All neurons interconnected

\_ Directly or indirectly

\_ Necessary to integrate & coordinate whole body

\_ Permits information transmission throughout body

### 2. Structure [ mostly covered in lab ]

#### a. Cell body (soma)

\_ Can be 150<sup>+</sup>  $\mu\text{m}$  diameter

\_ Contains nucleus, most cytoplasm & organelles

#### b. Dendrites

\_ Smaller projections from body

\_ Most often more than one

\_ Tree-like branching -- max. thousands small tips

#### c. Axon (fiber)

\_ Largest projection from body -- up to 1 M. long

\_ Collateral branches -- few, right angle

\_ Endings (telodendria) -- variable number

\_ Usually special coverings [ details later ]

### 3. Functional parts

#### a. Dendrites

- \_ Receive incoming information -- some exceptions
- \_ Via axon endings of other neurons
- \_ Can receive up to several hundred thousand endings

b. Axon

- \_ Endings transmit outgoing info -- some exceptions
- \_ Endings contact other neurons
- \_ Above endings up to soma -- can receive other endings

c. Soma

- \_ Metabolic control of rest of cell
- \_ Membrane receives endings from other neurons

#### 4. Coverings -- axons only

a. Schwann sheathing (neurolemma)

- \_ PNS only
- \_ Highly flattened Schwann cell
- \_ Wraps around axon & itself several times
- \_ One Schwann cell covers only part of axon length
- \_ Nodes (of Ranvier) -- gaps between adjacent cells
- \_ Exception -- some wrap around several axons

b. Oligodendroglial sheathing

- \_ CNS only
- \_ Identical wrapping effect on axon
- \_ One cell has multiple flattened projections
- \_ Each projection wraps around different spot on axon

c. Myelin sheathing (medullation)

- \_ Myelin = lipids + glycolipids + glycoproteins

- \_ Secreted by Schwann or oligodendroglial cells
- \_ Part of axon's covering as well
- \_ Can be absent -- not produced by all sheathing cells

## 5. Types

### a. Structural classification

#### 1) Unipolar

- single projection from soma
- splits into two branches
- functional axon & dendrite
- e.g. dorsal root ganglion

#### 2) Bipolar

- single dendrite from soma -- usually unbranched
- one axon from soma
- e.g. rod from retina -- neuroepithelial

#### 3) Multipolar

- one axon from soma
- more than one dendrite from soma
- broad category, due to varying dendrites
- most neurons in this category

### b. Functional classification

#### 1) Sensory (afferent)

- form pathways from receptors
- dendrites contact receptor/axon away
- dendrites towards receptor/axon away
- dendrites in PNS/axon into CNS

-- dendrites lower in CNS/axon higher in CNS

## 2) Motor (efferent)

-- form pathways to effectors (muscles/glands)

-- dendrites in higher CNS/axon goes lower in CNS

-- dendrites in CNS/axon into PNS

-- dendrites towards CNS/axon towards effector

-- dendrites towards CNS/axon contacts effector

## 3) Association (intermediate; interneuron)

-- completely within CNS

-- most numerous

-- basic resident neurons

-- join sensory with motor

## 6. Arrangements

### a. White matter

\_ Mostly medullated (myelinated) axons

\_ PNS -- forms nerves

\_ CNS -- forms tracts (nerve equivalent)

\_ Brain -- forms general widespread white matter

### b. Gray matter

\_ Mostly somas & dendrites

\_ PNS -- forms ganglia

\_ CNS -- forms nuclei & diffuse gray networks

\_ Brain -- forms cerebral & cerebellar cortex

## Non-nervous Auxiliary Cells

### 1. PNS

a. Schwann cells [ already covered ]

b. Satellite (capsule) cells

- \_ Only present in ganglia
- \_ Form coverings around neuron bodies

## 2. CNS

a. Ependymal cells

- \_ Line brain ventricles & spinal cord's central canal
- \_ Covers choroid plexuses which project into ventricles
- \_ Secrete cerebrospinal fluid

b. Oligodendroglial cells

- \_ [ already covered ]
- \_ Reason that CNS neurons cannot regenerate -- ?

c. Astroglial cells (astrocytes)

- \_ Largest & most numerous glial cells
- \_ Numerous projections from body of cell
- \_ Attach to neurons -- for physical support
- \_ Attach to blood vessels -- blood-brain barrier
- \_ Attach to other structures -- e.g. oligodendroglia

d. Microglial cells

- \_ Smallest glial cells
- \_ Phagocytic -- protective
- \_ Connective tissue origin -- most likely

# NERVOUS IMPULSE

## Importance

- \_ All functions accomplished by impulses
  
- \_ This is the only way in which neurons operate

## Basic Functional Concepts

### 1. Membrane ionic behavior

#### a. Relevance

- \_ Recall -- irritability leads to membrane changes
- \_ Changes conducted -- this is impulse
- \_ Caused by ionic changes called potentials

#### b. Review of ionic concepts

- \_ Ion = electrically charged particle (+ or -)
- \_ Many different ions in cells & ECF
- \_ Constantly moving & changing locations
- \_ Move both into & out of cells

#### c. Passive membrane transport concepts

- \_ These mechanisms control some ionic entry & exit
  
- \_ General
  - ions follow concentration gradient
  - more net movement from greater to lesser
  - must pass through specific diffusion channels
  
- \_ Diffusion channels
  - specialized membrane protein

- passageway (hole) through molecule
- various kinds, each handling specific ion

\_ Ungated (free or open) channels

- always permits its ion to pass through
- only diffuses if concentration gradient, though

\_ Gated (restricted) channels

- its ion may or may not be permitted to pass
- can close down passageway completely
- can open passageway variable amounts
- controlling stimulus causes opening/closing

d. Active membrane transport concepts

\_ This mechanism can control same ions as passive

\_ General

- ions forced to move against gradient
- more net movement from lesser to greater
- must pass through transport pump

\_ Active transport pumps

- specialized membrane proteins
- different from diffusion channels
- expend energy from ATP to force ions through

\_ Ions handled

- some only transport one specific ion (e.g. Cl)
- others handle more than one (e.g. Na/K)

## 2. Electrical potential

### a. Basic concept

- \_ Potential for electrical current (energy flow)
- \_ Due to imbalance of charges between two areas
- \_ Relative -- one area being compared with another
- \_ Only realized when areas joined by conductor
- \_ Can be detected with voltage measuring device
- \_ e.g. electrical energy stored in battery

### b. Equal ionic distribution (balance)

- \_ Ionic movement into & out of cell balanced
  - particular ions pass through (in or out)
  - equal number of same kind pass other way
  
- \_ Mechanism
  - initial movement usually involves diffusion
  - active transport used for counter-balancing
  
- \_ Importance
  - movements into & out of cell unavoidable
  - massive diffusion destabilizes membrane
  - maintains dynamic ionic distribution
  
- \_ Not the same as concentration equilibrium
  - few ions in equilibrium between outside/inside
  - this maintains needed concentration differences
  
- \_ This is the usual, desirable situation in most cells

### b. Unequal distribution

\_ Imbalanced ionic movements into & out of cell

- particular ions pass through (in or out)
- fewer numbers of same kind pass other way

\_ Mechanism

- massive diffusion through ungated channels
- pump inadequate to counter-balance
- termed leaky membrane

\_ Importance

- would destabilize & harm most cells
- nerve, muscle & neuroepithelial cells survive
- fundamental principle which permits irritability

c. Membrane application of electrical potentials

\_ Equal distribution

- ionic balance creates no potential
- charged particles relatively balanced
- note this is a dynamic concept
- voltmeter would read "0"

\_ Unequal distribution

- establishes potential electrical energy
- due to net ionic gain in or out of cell
- not electric current, only a potential
- voltmeter would register "< or > 0"

\_ Polarity

- potential creates + & - polarized areas

- orientation depends on ion & direction
- quantification depends on voltmeter hookup

### 3. Ions involved

#### a. Sodium ( $\text{Na}^+$ )

- \_ 10-20 times more concentrated in ECF than cytoplasm
- \_ Membrane has gated diffusion channels
- \_ Membrane has active transport pump

#### b. Chloride ( $\text{Cl}^-$ )

- \_ 10-12 times more concentrated in ECF than cytoplasm
- \_ Membrane has gated diffusion channels
- \_ Membrane has weak active transport pump

#### c. Potassium ( $\text{K}^+$ )

- \_ 40-50 times more concentrated in cytoplasm than ECF
- \_ Membrane has ungated diffusion channels
- \_ Membrane has gated diffusion channels, also
- \_ Membrane has active transport pump -- shared with Na

#### d. Others

- \_ Not insignificant, but secondary to above 3
- \_ Intracellular anions
  - variable impermeable organics
  - e.g. proteins
  - create important internal negativity
- \_ Calcium ( $\text{Ca}^{++}$ )
  - more concentrated in ECF
  - stabilizes membrane for Na
  - Na permeability inverse to Ca concentration

# Resting Potential

## 1. Importance

- \_ Establishes basic membrane conditions
  - \_ No nervous impulse in existence
  - \_ No related conditions which could lead to impulse
  
- \_ Like a reference point
  - \_ Alteration establishes conditions for impulse
  - \_ Restoration causes impulse to disappear
  - \_ Variant change can make impulse extremely unlikely

## 2. Cause

### a. Potassium imbalance

- \_ Leaky membrane -- ungated (always open) channels
- \_ Extremely steep gradient causes much diffusion out
- \_ Pump not efficient enough to counteract all leakage
- \_ Actual number of ions
  - net loss rate 7 million
  - insignificant (total in cell  $2 \times 10^{11}$ )
- \_ Polarity
  - inside more negative (has lost positive K)
  - ECF more positive (has gained positive K)

### b. Role of sodium

- \_ Equally distributed
- \_ Equal inward diffusion & outward active transport

- \_ Important so as not to interfere with K
- \_ Steep concentration gradient has another role [later]

c. Role of chloride

- \_ Equally distributed
- \_ Inward diffusion equalled by 2 factors
  - greatly repulsed by cytoplasmic negativity
  - weak active transport outward

### 3. Amount

a. Quantification introduction

- \_ Voltmeter leads -- traditional hookup
  - - to ECF
  - + to cytoplasm
- \_ Thus, cytoplasm measured relative to ECF

b. Resting potential reading

- \_ -70 mV
- \_ Caused only by K imbalance
- \_ "-" reflects voltmeter hookup
  - inner reference point has lost positive K
  - makes it now less positive (i.e. more negative)
  - reversal of hookup would produce "+" reading
- \_ "70" reflects amount of net K loss to ECF
- \_ "mV" reflects small electric current generated
- \_ Different neurons may have higher or lower reading

# Depolarization

## 1. Importance

- \_ Necessary to reverse resting polarity
- \_ Only under new polarity can impulse be generated
- \_ Amount of depolarization variable but critical

## 2. Cause

### a. Stimulus

- \_ Neuron membrane must be altered
- \_ Affected by some form of energy
  - chemical (most often)
  - mechanical
  - thermal
  - radiant
  - electrical

### b. Membrane effect

- \_ Sodium gates opened wider -- channel protein altered
- \_ Na<sup>+</sup> diffusion increased
  - wider passages through membrane
  - follows steep concentration gradient

### c. Results

- \_ Reversal of resting polarity
  - inside suddenly gains great number + ions
  - outside suddenly loses + charges (more -)
- \_ Cannot be counteracted
  - Na pump can work no faster

-- resting K effects overwhelmed

d. Time -- lasts about 200 nsec (0.2 msec)

### 3. Amount

- \_  $> -70$  mV -- e.g. - 65 mV
- \_ Variable
  - \_ Proportional with amount of stimulus
  - \_ Due to Na gates opening more or less

## Repolarization

### 1. Importance

- \_ Depolarization effects only needed for short time
- \_ Necessary to restore resting potential

### 2. Causes

#### a. Sodium

- \_ Diffusion channel gates close
- \_ Occurs automatically at right time
- \_ Now back to resting position
- \_ Necessary to prevent further depolarization

#### b. Potassium

- \_ Gated diffusion channels open
- \_ Occurs automatically as sodium gates close
- \_ Even more  $K^+$  diffuses out than during resting
- \_ Reverses depolarization from inward  $Na^+$  diffusion

- \_ Gates close when resting reached
- \_ Ungated channel leakage again maintains resting

c. Why is K, & not Na, used to restore resting -- ?

- \_ Active transport would be required
- \_ K diffuses very freely

c. Time -- lasts about 500 nsec (0.5 msec)

### 3. Amount

- \_ From  $>-70$  mV back to  $-70$  mV -- e.g.  $-65$  to  $-70$  mV
- \_ Variable -- depends on exact amount of depolarization

## Action Potential

### 1. Importance

Absolutely necessary for nervous impulse generation

### 2. Cause

a. Concept

- \_ It is an extra strong depolarization
- \_ It only follows a threshold depolarization

b. Threshold depolarization

- \_ Critical, minimum amount to produce action potential
- \_ Varies -- about  $-40$  mV
- \_ Stimulus must be strong enough to reach this level

c. All-or-none law

- \_ When threshold reached
  - action potential occurs
  - nervous impulse always follows
- \_ If threshold not reached

- no action potential at all
- no nervous impulse possible

\_ Suprathreshold depolarization

- above-threshold
- action potential no stronger

\_ Subthreshold depolarization

- does represent effect on membrane
- rapid additional could reach threshold

d. Sodium gates

\_ Depolarization [ previously described ]

\_ Upon reaching threshold amount

- gates open more, to their maximum
- no additional stimulus occurs
- caused by positive feedback

e. Spike

\_ Describes sharp peak on oscilloscope

\_ Produced by action potential

- upward part is depolarization
- downward part is repolarization

\_ Lasts for 1 msec, maximum

### 3. Amount

- \_ +20 mV
- \_ Quite variable in different neurons

## Actual Impulse

### 1. Concept

- \_ Series of action potentials
- \_ Occur in succession -- one after the other
- \_ Each one generates the next
- \_ Each occurs farther away on membrane
- \_ Eventually conducted over entire membrane of neuron
- \_ Begins at original site of threshold stimulus/depolarization

## 2. Refractory period

- \_ 500 nsec (0.5 msec) period -- during spike
- \_ Affected membrane unresponsive to further stimulation
- \_ Reason
  - \_ Sodium gates opened maximally
  - \_ No more depolarization possible
- \_ Absolute refractory period
  - \_ Early in spike
  - \_ As just described
- \_ Relative refractory period
  - \_ Later in spike
  - \_ Much stronger stimulation could depolarize
  - \_ Due to Na gates now being closed
- \_ This prevents any behavior analogous to wave summation

## 3. Variations

### a. Strength

- \_ New action potentials generated as impulse spreads
- \_ No loss in magnitude
- \_ Same strength at any point over membrane
- \_ Variations in mV of different impulses irrelevant

### b. Velocity

- \_ Varies greatly in different neurons
- \_ Range 1 - 120 M/sec
- \_ Thicker neurons conduct faster
- \_ Medullated neurons conduct faster

c. Medullation

- \_ Each spike only occurs at nodes
- \_ Termed saltatory -- jumping
- \_ Fastest neurons have farthest spaced nodes
- \_ Exact mechanism debated

d. Impulse frequency

- \_ Maximum number possible -- about 1000/sec
- \_ Limiting factor -- each spike requires 1 msec

## Fatigue

### 1. Concept

- \_ Neuron which is unable to be depolarized
- \_ No action potential or impulse possible
- \_ Not a frequent occurrence

### 2. Reason

- \_ Sodium gradient inside/outside neuron too low
- \_ No excess sodium in ECF to cause depolarization
- \_ Caused by too many impulses in rapid succession
  - \_ 100,000 impulses at maximum frequency
  - \_ At lower frequencies, sodium gradient maintained

### 3. Recovery

- \_ Sodium pump increases activity

\_ Recovers as soon as gradient at minimal level

# Hyperpolarization

## 1. Importance

- \_ Prevents or inhibits impulses
- \_ Equally important
  - \_ Provides a moderating balance in system
  - \_ [ explained later ]

## 2. Causes

### a. Concept

- \_ Increase in resting potential
- \_ Opposite of depolarization

### b. Initiation

- \_ Inhibitory influences on neuron membrane
  - [ explained later ]
- \_ Very low level, persistent stimulus as well

### c. Two possible responses (same resulting potential)

#### 1) Potassium

- gated channels open
- enhances resting potential
- different beginning point than repolarization

#### 2) Chloride

- channel gates open
- inward rush of negative ions
- same effect as outward  $K^+$

- \_ Note: any one neuron has either  $K^+$  or  $Cl^-$  response

### 3. Amount

- \_ <-70 mV -- e.g. -100 mV
  - \_ Tremendous variation
  - \_ Depends on degree of inhibition required
- \_ Amount of depolarization to reach threshold now greater
  - \_ -70 mV less -40 mV = +30 mV
  - \_ -100 mV less -40 mV = +60 mV

## SYNAPTIC TRANSMISSION

### Importances

#### 1. Direction of conduction

- \_ Determines impulse direction over connected neurons
- \_ Only allows axon endings to affect other neurons
- \_ Act as one-way valves [ exceptions -- below ]

#### 2. Timing

- \_ Causes strategic delays in impulses over grouped neurons
- \_ Produces effects when they are needed
- \_ Slower action between neurons -- impulse over each faster

### Concepts

#### 1. Synapse

- a. When used as a noun
  - \_ Denotes a structure
  - \_ This connects one neuron with another
  
- b. When used as a verb

- \_ Denotes a function
- \_ The action which occurs between 2 neurons

## 2. Relative roles of connected neurons

### a. Pre-synaptic

- \_ Axon endings
- \_ Where each cell influences other neurons

### b. Post-synaptic

- \_ Any neuron region except axon endings
- \_ Where each cell is influenced by other neurons

## 3. Types

[ left of dash = presynaptic; right of dash = postsynaptic ]

### a. Axo - dendritic

### b. Axo - somatic

### c. Axo - axonic

### d. Dendro - dendritic [ exception -- not mentioned again ]

### e. Dendro - axonic [ exception -- not mentioned again ]

\_ Two way (bi-directional or reciprocal)

\_ e.g. axo-dendritic/dendro-axonic combination

\_ Not just exceptions -- give added functionality

[ not mentioned again ]

## Structure

### 1. Pre-synaptic parts

#### a. End bulb (synaptic knob or bouton)

Swelling at tip of axon ending

b. Vesicles

- \_ Within end bulb
- \_ Similar to Golgi secretion vesicles
- \_ Clustered near membrane adjacent to post-synaptic

c. Transmitter

- \_ Chemical substance
- \_ Contained within vesicles
- \_ Exception
  - neurosecretory endings
  - this is a hormone, not a transmitter

d. Pre-synaptic membrane

- \_ Bottom of end bulb
- \_ Conforms to contour of post-synaptic
- \_ Modified to permit transmitter release from vesicles

2. Synaptic gap (cleft)

- \_ No physical contact between pre- & post-synaptic
- \_ Same as myoneural junction
- \_ Gap of about 25 nm
- \_ Transmitter released here -- diffuses across

3. Post-synaptic parts

a. Spine

- \_ Knob-like projection from dendrite or soma
- \_ Contact structure for end bulb
- \_ Other, flat surfaces can be contacted as well

b. Post-synaptic (sub-synaptic) membrane

- \_ Covers spine or flat surface
- \_ Corresponds in area to pre-synaptic membrane
- \_ Modified to respond to transmitter

c. Membrane receptors

- \_ Membrane proteins
- \_ Primarily sensitive to the transmitter

## Mechanism of Action

### 1. Pre-synaptic

a. Nervous impulse

Conducted down axon & all endings

b. End bulb

- \_ Vesicles fuse with pre-synaptic membrane
- \_ Exocytosis causes transmitter release into cleft

### 2. Cleft

- \_ Transmitter diffuses across
- \_ Was more concentrated in vesicles

### 3. Post-synaptic

a. Receptors

- \_ Bind with transmitter
- \_ Undergo change in shape & activity

b. Diffusion channels

- \_ Receptors are gate controllers for channels

- \_ Control gates directly or indirectly
- \_ Gates open due to shape change
- \_ Diffusion of particular ion now increased
- \_ Variable effects
  - depends on synapse type
  - [ details later ]

#### 4. Delay time

- \_ Above mechanism takes about 500 - 600 nsec
- \_ Significant delay compared with spike & faster impulses

#### 5. Fatigue

- \_ Frequent impulses can deplete transmitter
- \_ About 10,000 impulses would be required
- \_ Fatigued synapse can not operate
- \_ Transmitter stores replenished

## Effects (Responses)

### 1. Excitation (stimulation)

#### a. General

- \_ Only one of two possible responses
- \_ Any one synapse has only this response or the other

#### b. Depolarization

- \_ Sodium channels opened by receptors
- \_ Potential goes  $> -70$  mV
- \_ Exact amount & impact [ later ]

c. Excitatory post-synaptic potential

- \_ Called EPSP
- \_ Used to identify this synaptic type

## 2. Inhibition

a. General -- this is the only other possible response

b. Hyperpolarization

- \_ Either potassium or chloride channels opened  
(any one synapse will use one or the other)
- \_ Potential goes  $< -70$  mV

c. Inhibitory post-synaptic potential -- called IPSP

## 3. Mechanisms for two synaptic types

a. Different transmitters

- \_ Many specific transmitter chemicals
- \_ Some always in EPSP synapses
- \_ Some always in IPSP synapses

b. Different post-synaptic membrane receptors

- \_ Examples of one transmitter in both EPSP & IPSP
- \_ Different receptor types
- \_ Actually, receptor type is always responsible

## 4. Importance

- \_ Why is IPSP necessary ?
- \_ What are the functions & benefits of IPSP ?
- \_ Isn't EPSP adequate -- it can lead to action potentials ?

- a. Sharpens (refines) responses
  - \_ Permits discrimination of information
  - \_ Turning process off is a response
- b. Limits responses
  - \_ Blocks unnecessary pathways & actions
  - \_ Allows discrete, direct responses
- c. Maintains order
  - \_ EPSP only can stimulate
  - \_ Prevents over-stimulation -- braking mechanism
- d. Feedback circuits
  - \_ Negative feedback permitted
  - \_ EPSP & IPSP can negate each other

## Transmitter Chemistry

\_ SKIP THIS \_

## Potentials

### 1. Post-synaptic net potential

- \_ Change per synapse
  - \_ Effect of release of transmitter from one end bulb
  - \_ Potential (EPSP or IPSP) only changes 1 mV
- \_ Implication
  - \_ Individual synapse has little effect
  - \_ e.g. EPSP would only depolarize from -70 to -69 mV
- \_ How synapses effectively utilized

- \_ More than one synapse required
- \_ Must be coordinated effects
  
- \_ Think of this as groups of end bulbs
  - \_ Groups affect one neuron (in post-synaptic role)
  - \_ Effect of group's end bulbs collective -- summation
  - \_ Any one group could produce threshold depolarization
  
- \_ Reason for different groups
  - \_ Each represents unique combination
  - \_ Permits variable effects of different endings
  - \_ Any impulse generated from many other impulses

## 2. Summation

### a. General group principles

- \_ Endings come from more than one neuron's axons
- \_ Possible to have more than one ending from one axon
- \_ Each ending only has 1 mV potential effect
- \_ Endings of both EPSP & IPSP type
- \_ Summed effect = EPSP less IPSP

### b. Spatial summation

- \_ One of 2 ways group's summed effects accomplished
- \_ Simply the number of end bulbs simultaneously active
  
- \_ e.g. to produce threshold depolarization
  - 30 mV of depolarization required
  - 30 EPSP end bulbs must "fire" together
  - 30 EPSP x 1 mV = +30 mV
  - threshold reached (AP & impulse follow)

- \_ IPSP end bulbs usually active as well
  - for threshold, additional EPSP required
  - e.g.  $(40 \text{ EPSP} \times 1 \text{ mV}) - (10 \text{ IPSP} \times 1 \text{ mV}) = +30 \text{ mV}$
  
- \_ e.g. to prevent threshold depolarization
  - either inadequate EPSP or counteracting IPSP
  - $25 \text{ EPSP} \times 1 \text{ mV} = +25 \text{ mV}$  (subthreshold)
  - $(30 \text{ EPSP} \times 1 \text{ mV}) - (5 \text{ IPSP} \times 1 \text{ mV}) = +25 \text{ mV}$

#### c. Temporal summation

- \_ The other way group's summed effects accomplished
- \_ This represents the frequency of end bulb "firings"
  - end bulbs do not just have to "fire" once
  - can "fire" several times in rapid succession
  - if fast enough, effects are accumulated
  
- \_ e.g. threshold depolarization with only 10 EPSP's
  - $3(10 \text{ EPSP}) \times 1 \text{ mV} = +30 \text{ mV}$
  - each of 10 active 3 times in rapid succession
  - same effect as 30 EPSP's working spatially

#### d. Combination

- \_ In reality, both spatial & temporal occur together
- \_ e.g. threshold, using only EPSP end bulbs
  - $[ 3(5 \text{ EPSP}) + 15 \text{ EPSP} ] \times 1 \text{ mV} = +30 \text{ mV}$
  
- \_ Various degrees of counteraction between EPSP & IPSP

### 3. Facilitation

a. Concept

- \_ When EPSP's are more prevalent
- \_ But, depolarization held just below threshold

b. Significance

- \_ State of readiness of post-synaptic group sites
- \_ Only slightly increased depolarization causes threshold

c. Mechanism

- \_ Usually, IPSP's used to diminish depolarization
- \_ For threshold, enough IPSP's simply stop "firing"

d. Lack of resting potential

- \_ Most neurons kept perpetually facilitated
- \_ So, resting is mainly of theoretical interest
- \_ Usually only seen in experimental situations
- \_ It is important
  - its possibility required as reference point
  - all knowledge of other potentials from this

e. Two most important situations

1) Threshold depolarization

- permits AP & impulse
- impulses required to cause end bulb "firings"

2) Subthreshold depolarization

- this is facilitation
- required to prevent threshold when not needed

# NEURONAL POOLS

## Concept

### 1. Definition

Logical groupings of whole neurons

### 2. Basis

- \_ Every neuron has some connection with all others
- \_ Each neuron directly or indirectly synapses with all others
- \_ There are no isolated neurons

## Purposes

### 1. Permits specific tasks

- \_ Logic for choice of neurons in each pool
- \_ Different neuron combinations carry varied information

### 2. Basis for a group of EPSP end bulbs

- \_ These would affect one post-synaptic site
- \_ This is essence of how an impulse can be produced

### 3. Basis for a group of IPSP end bulbs

- \_ These would affect one post-synaptic site
- \_ Work in concert with EPSP end bulbs
- \_ This is essence of how impulses are prevented

# Basic Principles

## 1. Convergence

- \_ One of 2 basic facts about extent of interconnections
- \_ Many pre-synaptic endings converge on any one neuron
- \_ Represents the effects of the many upon one other
- \_ Extent
  - \_ Hundreds up to hundreds of thousands of endings
  - \_ From a few up to thousands of pre-synaptic axons

## 2. Divergence

- \_ The other basic fact about interconnections
- \_ Involves one neuron in its pre-synaptic role
  - \_ Its axon endings diverge
  - \_ Will contact other neurons in post-synaptic role
- \_ Represents the effects of one upon many others
- \_ Extent -- a few up to hundreds of other neurons

# Types

## 1. Afterdischarge pool

### a. Concept

- \_ Pool which produces certain frequency of impulses
- \_ Series of rapid impulses, one after another

### b. Uses

- \_ Skeletal muscle wave summation for tetanus
- \_ Complex mental functions

-- those that require repetitive impulses

-- e.g. mathematical calculations

### c. Mechanism

\_ Most simply -- chain of neurons

-- endings of #1 synapse with each of others

-- endings of #2 synapse with each remaining

-- each remaining neuron does the same

\_ Assumptions for this example

-- "synapse" actually means a group of endings

-- EPSP will predominate

-- impulse will cause threshold at post-synaptic

\_ Impulse on #1 reaches all others at same time

-- generates impulses on #2, #3 & so on

-- impulses on others are simultaneous

\_ Impulse on #2 reaches all others at same time

-- occurs shortly after #1's impulse

-- generates 2nd impulse on #3 & so on

\_ Series of impulses accomplished

-- 2 impulses in succession on #3

-- 3 impulses on #4

-- 4 impulses on #5 & so on

\_ Good example of delays caused by synapses

## 2. Reverberation pool

### a. Concept

\_ Cyclical (circular) stimulation of neuronal chain

\_ Impulses (or inhibition) continuous

b. Uses

\_ Timing of breathing movements from medulla

\_ Short-term memory

-- neurons continuously stimulating each other

-- outside interruption (distraction) halts

c. Mechanism

[ same assumptions as above for afterdischarge ]

\_ Most simply demonstrated with 2 neurons

-- #1 synapses with #2

-- #2 synapses back to #1

\_ Impulse from #1 to #2

-- impulse generated on #2

-- goes back to #1

\_ Second impulse over circuit

-- generated by impulse from #1 to #2

-- causes #1 to stimulate #2 again

-- #2 then stimulates #1 again as well

\_ Reverberation accomplished

-- 3rd, & subsequent, impulses generated

-- so, #1 & #2 reverberating (back & forth)

\_ How is this stopped ?

-- other neurons required (#3 minimum)

-- could use IPSP to stop #1 or #2