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 called nerves**

    c. Divisions

        (1) Functional

            (a) Somatic -- mainly controls skeletal muscles

            (b) Visceral (autonomic)

                -- controls involuntary muscles

                -- controls glands

                -- CNS component as well [details later]

(2) Structural

(a) Spinal

-- Spinal Cord

-- 31 pairs

(b) Cranial

-- Brain

-- 12 pairs

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. More 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

-- Hormone-like (overlaps endocrine)—very few nerve cells do this

-- For chemical communication—transmitters—all nerve cells

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 only slightly to non-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 primary 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^{+} µm 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 (sense organs)

-- dendrites contact receptor--axon away to another neuron

-- 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 and 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 proteins

-- passageway (hole) through molecule

-- various kinds, each handling specific ion(s)

-- Ungated (free or open) channels

-- always permits its ion(s) to pass through

-- however, only diffuses if concentration gradient exists

-- Gated (restricted) channels

-- its ion(s) 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 (Na^{+})

-- 10-20 times more concentrated in ECF than cytoplasm

-- Membrane has gated diffusion channels

-- Membrane has active transport pump

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

-- 10-12 times more concentrated in ECF than cytoplasm

-- Membrane has gated diffusion channels

-- Membrane has weak active transport pump

c. Potassium (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 (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 2×10^{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 equaled by 2 factors

-- greatly repulsed by cytoplasmic negativity

-- weak active transport outward

3. Amount

a. Quantification introduction

-- Voltmeter leads -- traditional hookup

-- negative to ECF

-- positive 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 an altered 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 (under experimental conditions)

b. Membrane effect

-- Sodium gates opened wider -- channel protein altered

-- Na^+ 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 negative)

-- Cannot be counteracted

-- Na^+ pump can work no faster

-- resting K^+ effects overwhelmed—but does **not** stop inward net gain

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

3. Amount

-- >#70 mV (e.g. # 65 mV)

-- Variable

-- Varies with amount of stimulus

-- Due to Na^+ gates opening proportionally

Repolarization

1. Importance

-- Depolarization effects only needed for short time

-- Necessary to restore resting potential for next depolarization

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 potential ?

-- 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

-- Supra-threshold depolarization

-- above-threshold

-- action potential no stronger

-- Sub-threshold depolarization

-- does represent effect on membrane

-- rapid additional depolarization could reach threshold

--**extremely important** [explained later]

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 sodium gates now being closed

-- This prevents any behavior analogous to wave summation for muscle

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

Hyper-polarization

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 (threshold)

-- #100 mV less #40 mV = +60 mV (sub-threshold)

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 later]

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 = pre-synaptic; right of dash = post-synaptic]

a. These will be considered for rest of this topic

--axo - dendritic

--axo - somatic

--axo - axonic

b. These are exceptions, not to be mentioned again (they **are** important)

--dendro - dendritic [exception

--dendro - axonic [exception

--two way (bi-directional or reciprocal)

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

-- give added functionality

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

-- from neurosecretory endings

-- 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 myo-neural 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 surface shapes exist (e.g. flat)

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 cannot operate

-- Transmitter stores replenished

6. Transmitter disappearance

a. Excretion

b. Hydrolyzed or otherwise degraded

-- This is the most used technique

-- e.g. acetylcholinesterase (AChE) hydrolyzes acetylcholine (Ach) to acetate (widely metabolized) and choline (reused by neurons)

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. Hyper-polarization

-- Either potassium or chloride channels opened

(any one synapse will use one <u>or</u> 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 **<u>always</u>** 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

There are many transmitters—only those used as examples will be considered

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 EPSP x 1 mV) # (10 IPSP x 1 mV) = +30 mV

-- e.g. to prevent threshold depolarization

-- either inadequate EPSP or counteracting IPSP

-- 25 EPSP x 1 mV = +25 mV (subthreshold)

-- (30 EPSP x 1 mV) # (5 IPSP x 1 mV) = +25 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 EPSP) x 1 mV = +30 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 EPSP) + 15 EPSP] x 1 mV = +30 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) Sub-threshold 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

--Use – e.g. determining how long an afterdischarge pool will be active

3. Other Types

a. Introduction

--There are more, but not all will be considered

--Note: the naming of some will be my own (in parenthesis) and not standardized

b. “On/Off” pool

--This would activate (on) or deactivate (off) another pool

--E.g. activating/deactivating a reverberation pool

c. **“On /Off Switching” pool** – would control an “On/Off” pool

d. Parallel Processing

-- This is a fundamental, very important mechanism for integration of numerous mental and physical mechanisms

--This involves the simultaneous transmission over varied sensory and/or motor pathways

-- Thus, there can be various responses that are logically correlated, occurring at the same time

-- E.g. When feeling a sudden sharp pain on one’s arm, the following reactions might occur:

-- looking at the arm

-- recognizing a bee

-- knocking the bee off

-- jumping up from the chair

-- probably shouting or saying something (bleep!)