

Instinctual Behavior, Emotions

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- drugs that modify human behavior (hallucinogenic agents, tranquilizers, antidepressants, etc) act by modifying transmission at synaptic junctions in brain.

EMOTIONS

1. MENTAL (subjective) component – **CORTEX**:
 - 1) **cognition** - awareness of sensation and usually its cause
 - 2) **affect** - feeling itself
 - 3) **conation** - urge to take action
 2. PHYSICAL component – **somatic** and **visceral** (e.g. hypertension, tachycardia, sweating) responses – integrative action of **HYPOTHALAMUS**.
- emocijos gali kilti tiek dėl **psychic activity** (association cortical areas project to gyrus cinguli), tiek dėl **hypothalamic activity**.
 - **LIMBIC system** connects **CORTEX** with **HYPOTHALAMUS**.

Anatomical basis for emotions – **Papez circuit**. see A138 (4) p.

Characteristics of limbic system:

- 1) **paucity of connections with neocortex** ("neocortex sits astride limbic system like rider on horse without reins"); actually, there are few connections - **neocortical activity does modify emotional behavior** and **vice versa**.

Characteristics of emotion is that it **cannot be turned on and off at will**.

- 2) **prolonged after-discharge** (of limbic circuits) following stimulation → prolonged emotional responses outlasting stimuli that initiate them.

SEXUAL BEHAVIOR

also see p. Psy1

- several areas of **limbic system** (incl. hippocampus, amygdala) are involved in sexual behavior.

- connections between cortical areas, hypothalamic areas, and limbic nuclei may be responsible for various complex emotional, behavioral, and physiologic aspects of sexual functioning.

MATING is **basic but complex phenomenon** - many parts of nervous system are involved.

- COPULATION itself - series of reflexes integrated in **spinal & lower brain stem** centers.
- BEHAVIORAL COMPONENTS (coordinated sequence of events that lead to pregnancy) - regulated to large degree in **limbic system** and **hypothalamus**.
 - basic responses are innate (present in *all mammals*);
 - in *nonprimates*, successful mating can occur with no previous sexual experience;
 - in *primates*, learning plays part in mating behavior;
 - in *humans*, sexual functions extensively encephalized and conditioned by social and psychic factors.

ENDOCRINE CONTROL

Nonprimate mammals

- **removal of gonads** →→→ absent sexual activity (loss is slow to develop in males of some species).
- **injections of gonadal hormones** (testosterone, estrogens) in castrate animals revive sexual activity.
 - N.B. large doses of testosterone in castrate females initiate female behavior;
 - large doses of estrogens in castrate males trigger male mating responses!

Human adults

- **ovariectomy** does not necessarily reduce libido or sexual ability (postmenopausal women continue to have sexual relations, often without much change in frequency) - due to **adrenal cortex secretion** (steroids converted to circulating estrogens) + **learned sexual functions**.
- **treatment with sex hormones** increases sexual interest;
 - testosterone or estrogen (e.g. used to treat carcinoma of prostate) increase libido in males.
 - behavioral pattern that was present before treatment is stimulated but not redirected (e.g. testosterone to homosexuals intensifies their homosexual drive but does not convert it to heterosexual drive).

NEURAL CONTROL IN MALE

Animals

- removal of **neocortex** inhibits sexual behavior;
 - partial cortical ablations also produce some inhibition (inhibition degree independent of coexisting motor deficit; most marked when lesions are in frontal lobes).
- bilateral limbic lesions localized to **piriform cortex overlying amygdala** → sexual activity↑↑↑ (mount immature females, other males, attempt to copulate with other species and with inanimate objects).
- **hypothalamus** is also involved:
 - appropriately placed anterior hypothalamic lesions abolish interest in sex.
 - stimulation along medial forebrain bundle and in neighboring hypothalamic areas → penile erection with considerable emotional display.
 - intrahypothalamic implants of testosterone restore sexual behavior in castrated rats.

Men - bilateral lesions in or near **amygdaloid nuclei** → hypersexuality.

PHEROMONES

- animal substances that **act at distance** to produce hormonal, behavioral, or other physiologic changes in another animal.

- in monkeys, sex drive of male is greater when he is exposed to female at time of ovulation than when he is exposed to female at another time of her cycle (due to olfactory fatty acids in vaginal secretions).
- armpit odor of women is capable to modify menstrual cycle; women who are good friends or roommates tend to synchronize their menstrual cycles.
- infants prefer breast or axillary pads from their own mothers (vs. unfamiliar mothers).

SEXUAL BEHAVIOR IN FEMALE

Mammals - sexual activity of **male** is more or less continuous, but sexual activity of **female** is cyclic:

- most of time, *female avoids male* and repulses his sexual advances.
- periodic abrupt change in behavior - *female seeks out male*, attempting to mate - **heat (estrus)**; sexual cycle in species that do not menstruate is named **estrous cycle**.

- estrus is brought on by rise in blood estrogen.
- *some animals* (rabbit, ferret) remain estrous until pregnancy results; ovulation is due to neuroendocrine reflex (stimulation of genitalia provoke release of LH).
- *in many other species*, spontaneous ovulation occurs at regular intervals (periods of heat coincide with its occurrence).
- removal of **neocortex** and **limbic cortex** abolishes active seeking out of male during estrus, but other aspects of heat are unaffected.
- **periamygdaloid** lesions do not produce hypersexuality (as in male).
- **anterior hypothalamus** (some part is sensitive to estrogen!):
 - discrete lesions abolish behavioral heat without affecting regular pituitary-ovarian cycle.
 - implantation of minute estrogen amounts causes heat in ovariectomized rats.

Women - sexual activity occurs *throughout menstrual cycle*, but there is more spontaneous female-initiated sexual activity at *about time of ovulation*.

EFFECTS OF SEX HORMONES IN INFANCY

Rats are particularly immature at birth

- *female rats* treated with single small dose of androgen before 5th day of life develop "masculinized" brains - do not have normal heat periods when they mature, no cyclic release of pituitary gonadotropins characteristic of adult female but, rather tonic, steady secretion characteristic of adult male + male sexual behavior.
- *male rats* castrated at birth develop "female hypothalamus" - female pattern of cyclic gonadotropin secretion, considerable female sexual behavior (when given doses of ovarian hormones that do not have this effect in intact males).

N.B. development of "female hypothalamus" depends simply on **absence of androgens in early life** (rather than on exposure to female hormones).

vs. humans - estrogen also has masculinizing effect upon brain and appears to be necessary for normal development of both male and female brains (it previously was believed that only androgens were important in sexual differentiation of brain, i.e. developing brain without androgen exposure would develop into "female" brain).

Animals of other species (more fully developed at birth) - do not show these changes when exposed to androgens during *postnatal period*.

- develop genital abnormalities when exposed to androgens *in utero*.

Human females - exposure to androgens in utero does not change cyclic pattern of gonadotropin secretion in adulthood; some masculinizing effects on behavior do occur.

MATERNAL BEHAVIOR

- depressed by lesions of **limbic cortex** (cingulate and retrosplenial portions).

- hormones are not necessary (PROLACTIN facilitates maternal behavior).
- in female mice, knockout of *fos-B* gene → failure to retrieve and care for pups.

FEAR & RAGE

FEAR (fleeing, avoidance reaction) and **RAGE** (fighting, attack reaction) are related instinctual protective responses to threats in environment:

- when animal is threatened, it usually attempts to **flee**.
- if animal is cornered, it **fight**s.
- both reactions can be produced by **hypothalamic** stimulation.

FEAR

- function of:

- 1) **hypothalamus**
- 2) **amygdaloid nuclei:**
 - amygdaloid nuclei *encode memories that evoke fear* (e.g. viewing faces that have fearful expressions activates left amygdala; happy faces fail to produce response).
 - afferent sensory inputs that trigger conditioned fear responses may pass directly to amygdalas (without going through neocortical sensory areas).
 - *destruction* of amygdalas → absent fear reaction (and its autonomic and endocrine manifestations).

RAGE & PLACIDITY

- most animals, including humans, maintain **balance** between *rage* and its opposite (*placidity*).
- *major irritations* make normal individuals "lose their temper," but *minor stimuli* are ignored.
- in certain brain lesions, this balance is altered:

Rage responses to minor stimuli are observed after:

- a) removal of **neocortex**
- b) destruction of **ventromedial hypothalamic nuclei** and **septal nuclei** with intact cerebral cortices.

In humans, rage attacks follow number of nervous diseases (esp. epidemic influenza and encephalitis, which destroy neurons in limbic system and hypothalamus).

Abnormal placidity - after bilateral destruction of **amygdaloid nuclei** (but converted into rage by subsequent destruction of ventromedial nuclei of hypothalamus).

In Japan, bilateral amygdaloid lesions have been made in aggressive mental patients → patients became placid and manageable.

- in male animals, aggression is decreased by **castration** and increased by **androgens**.
- rage is also conditioned by **social factors**; it is more prominent in males that live with females and increases when stranger is introduced into animal's territory.

"Sham Rage"

- it was originally thought that rage attacks induced by *minor stimuli* in animals with **diencephalic & forebrain** lesions are *undirected* and represented *only physical, motor manifestations* of anger (reaction was therefore called "sham rage").
- this appears to be incorrect (term "sham rage" should be dropped) - rage attacks are usually *directed with great accuracy* at source of irritation; rage attacks include as well as **mental manifestations** of rage.

SELF-STIMULATION & ADDICTION

Self-stimulation experiments

Points where electrical brain stimulation leads to repeated bar pressing (*self-stimulation*) are located in **medial band of tissue** extending from frontal cortex through hypothalamus to midbrain tegmentum (dopaminergic pathway from ventral tegmental area to nucleus accumbens) - **reward (approach) system**.

- *humans* report pleasurable sensations, using phrases like "relief of tension", "quiet, relaxed feeling" ("joy" or "ecstasy" are only rarely reported); some persons with highest self-stimulation rates cannot tell why they keep pushing bar.
- drugs that block postsynaptic D₃ receptors reduce self-stimulation; dopamine agonists increase it; main site of relevant receptors appears to be **nucleus accumbens**.

Points where stimulation is **avoided** are in **lateral portion** of posterior hypothalamus, dorsal midbrain, and entorhinal cortex - **punishment (avoidance) system**.

- *humans* report sensations ranging from vague fear to terror.
- areas where bar pressing is repeated are much more extensive than those where it is avoided; in rats: repeated pressing is obtained from 35% of brain, avoidance - from 5%, indifferent responses (neither repetition nor avoidance) - from 60%.

ADDICTION (PSYCHIC DEPENDENCE)

- repeated compulsive use of substance despite negative consequences.

- addiction is associated with **reward system** (particularly with **nucleus accumbens**, located at base of striatum, and *mesocortical dopaminergic neurons* that project from midbrain to this nucleus and frontal cortex).
- all known addictive substances affect brain in different ways, but all increase amount of dopamine available to act on D₃ receptors in nucleus accumbens.
- in long term, addiction involves development of **tolerance** - need for increasing amounts of drug to produce "high"; **withdrawal** produces psychological and physical symptoms.

Causes of tolerance and withdrawal are poorly understood.

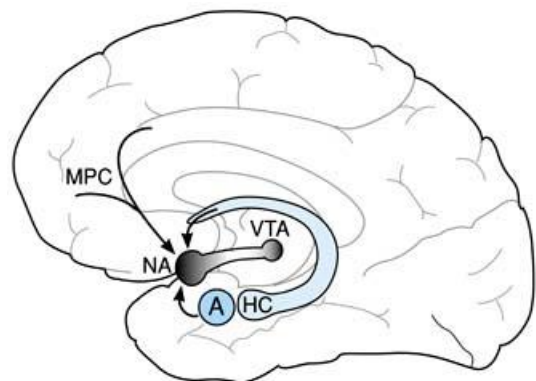
Presence of tolerance and / or withdrawal is called **PHYSICAL DEPENDENCE**.

- addicts characteristically **relapse after treatment** (esp. by exposure to environmental cues and memories associated with drug use).

Key brain areas involved in addiction:

ventral tegmental area (VTA) projects via mesocortical *dopaminergic* system to **nucleus accumbens** (NA);

medial prefrontal cortex (MPC), hippocampus (HC), and amygdala (A) send excitatory *glutamatergic* projections to **nucleus accumbens**.



BIBLIOGRAPHY for ch. "Cranial Neuropathies" → follow this [LINK >>](#)

Ganong "Review of Medical Physiology", 2002

Viktor's NotesSM for the Neurosurgery Resident
Please visit website at www.NeurosurgeryResident.net