Theories of Emotion

Emotion is hard to study and even to define...

**James-Lange Theory** (1880s) ...is the label we give after-the-fact to autonomic arousal and associated behavior
- i.e. Our subjective “feelings” are an interpretation we make of our body’s reaction to stimuli

**Cannon-Bard Theory** (1930s) Once threat perceived, visceral & subjective experience of emotion is simultaneous
- Via perceptual input via Thalamus to Cortex & activation of the ANS (for somatic responses)

**Schacter-Singer Theory** (1980s) ...is the interaction between cognitive appraisal and autonomic/limbic activity
- Physiology determines how strong emotion is, but ID’ing emotion depends on a cognitive appraisal of situ
- Feedback between variety of brain regions involved (e.g. Prefrontal Cortex, Amygdala, Hypothalamus, etc!)
  - That is, influence moves both bottom up (visceral to cognitive) AND top-down (cognitive to visceral)

Behavioral and neurological data support aspects of all of these:
- e.g. Subjects given arousing drug (e.g. amphetamine) - or placebo - and shown scary, funny, or sad images
  - Aroused subjects all reported stronger emotion, but type reported varied with stimuli
- e.g. Subjects hold pen-in-teeth vs. pen-in-lips while judging comics; Rate them funnier if teeth exposed
- e.g. Direct subjects to make specific changes in facial muscles (e.g. Raise brows, pull them together, raise upper eyelids, tighten lower eyelids, stretch lips horizontally) w/o mentioning an emotion
  - When asked, subjects reported “feeling” appropriate emotion (e.g. Above expression => fear)
  - Expressions also produced changes in heart-rate, skin temp, etc. (e.g. Fear: heart up, temp down)

COMPLEX! Emotion plays role in all evaluative (discrimination, motivation, learning) & communicative processes
- May depend in part on learned associations, but basic emotional expressions mimicked by newborn humans
  - So, from its inception, emotional expression is shaped by social interaction
- PLUS: Facial expressions of basic emotions are similar across cultures, although “display rules” vary

Many brain areas are implicated in the generation and control of emotional behavior…
- Including array of structures of Limbic System, and the Thalamus, the Hypothalamus, and the Frontal Cortex
- Here, we will focus on role of **Amygdala** (part of Limbic System) and **Frontal Cortex**

**Amygdala**
- Stimulation of particular regions (most of which interact with each other) leads to typical reactions
  - Including array of structures of Limbic System, and the Thalamus, the Hypothalamus, and the Frontal Cortex

- **Corticomedial Area** => Primed to **Attack**, prolonged inclination toward aggression
  - e.g. Rabies, from a virus that attacks, especially, temporal region of brain => extreme aggression

- **Lateral Nuclei** => **Startle Reflex** Sudden loud noise => Freeze, Tense neck to protect spine, Inc heart rate
  - Startle Reflex is influenced by Amygdala connections…
  - => from Pain fibers, and Visual and Auditory input, to detect and learn negative associations
  - => to **Central Gray Area** of Midbrain = Part of Tegmentum for motor control, esp of neck muscles
  - => to **Hypothalamus** => Influences Autonomic NS response (e.g. inc blood pressure, heart rate, etc)
    - Recall that 2 of the Hypothalamic “4 Fs” = Fighting & Fleeing

- **Central & Basolateral Nuclei** => **Conditioned fear**, via integration of sensory info (e.g. vision + pain)
  - Unlearned Startle Reflex (e.g. loud noise > !) can be further conditioned (modified via learning)
    - If visual stim (e.g. light) is paired w/noxious stimulus (e.g. shock) light alone comes to elicit fear
    - Once association is learned, paired signal enhances the reflex (i.e. jump higher to noise if light on)
  - In fact, any unpleasant stimulus (e.g. disturbing photo, sound or odor) presented simultaneously, whether or not previously paired with unconditioned (noxious) stimulus, enhances Startle Reflex
  - Alternatively, a paired signal previously associated w/pleasant stimulus will decrease Startle Reflex
    - e.g. Light paired w/soothing warmth => Not jump as high to noise if light is on
  - All of above aid memory of emotionally-laden stimuli, via connections to/from **Hippocampus**
    - i.e. Emotion-provoking (esp aversive) images or words, if not too intense, remembered better than neutral

- Contemporary research on Amygdala focused on **shared emotion**
  - Amygdala active not just when feel/express but also when observe emotion in others
    - e.g. In many primates, infants learn what to fear by watching/imitating mother’s reaction
  - Urbach-Wiethe Disease = degenerative calcification in Amygdala, (or other damage) can lead to…
    - Impaired ability to recognize Facial Expression, especially fear and untrustworthiness
    - Patients recognize individuals, gender, but trouble naming emotion, judging if face is “approachable”
      - May be related to disinterest in eyes; forced to consider eyes only, judgments are a bit better
    - Patients generally placid, do not experience strong likes/dislikes, act less timid in strange environment
Anterior Insular Cortex (medial to anterior Temporal Lobes; also includes primary Gustatory Cortex)
- Damage => impairs recognition & production of Disgust: Co-opted in humans as a social reaction
- This area also connected to hindbrain cranial nerves for control of facial muscles
  - Damage=>Patients can show teeth voluntarily, but cannot spontaneously smile
    = Emotional Facial Paralysis
- Contrast to Primary Motor Cortex for facial area and/or its connections to facial nerves
  - Damage=> Patients can spontaneously smile, but cannot show teeth voluntarily
    = Volitional Facial Paralysis

Frontal Cortex
- Has many reciprocal connections with Amygdala, moreso than any other cortical area
- Important in expressing, inhibiting and reading emotion…
  - Prefrontal Cortex Damage > loss of inhibition, socially-inappropriate behavior, sudden aggression
    - Famous example: Phineas Gage, survived steel rod through cheek, in/out of Prefrontal Cortex
      - Cognition ok but “personality” radically changed, irresponsible, volatile, indifferent to consequences
      - Contemporary work implicates Prefrontal in Theory of Mind = attribute knowledge, emotion to others
      - Inappropriate Prefrontal-Amygdala interaction may be critical in Autism (involves ToM deficits)
        - ? May result from premature develop of Prefrontal, before sufficient connections with Amygdala?
  - Interpretation of own responses can influence impact of stress
    - e.g. Helplessness under duress can lead to overactive Parasympathetic Rebound > ulcers
      - 2 rats yoked together, both receive same shocks to tail if one does not run in wheel at signal
      - Rat which runs does NOT develop ulcers, but rat w/no active response DOES (the same shock)
      - But if non-active rat has Prefrontal lesion, does not react emotionally & does not develop ulcers
  - Gambling Task: Consider diff performance of Normals, Amygdala-Lesioned &Prefrontal-Lesioned subjects
    - Task: Pick cards from 4 piles to learn about each pile’s payoff/penalty profile
    - 2 have good payoffs but very bad occasional penalties. Other 2 have smaller payoffs & smaller penalties
    - After some experience with this task…
      - Normals feel anxiety before taking from bad piles & shift to better, even before can explain decision
      - Prefrontal-Lesioned – Do not develop anticipatory anxiety or shift, thos show neg emotion at penalty
      - Amygdala-Lesioned - No anticipatory anxiety, no shift, no feeling negative emotion at penalty

- Von Economo (“Spindle”) Cells
  - Long fibers, but branch little – for communication between distant brain areas w/out intervening influence
  - Found only in large-brained animals (humans, elephants, whales)
  - e.g. Connect Anterior Insula with Anterior Cingulate (social risk, cost/benefit analyses)

Neurotransmitters implicated in the generation and control of emotional behavior:
Serotonin (5HT) Turnover = reuptake & resynthesis, as determined by levels of metabolite 5-HIAA in blood
- Low Serotonin Turnover associated with increased levels of impulsive behavior including…
  - Sudden aggression, rapid cocaine addiction, no pause at choice pts, poor delayed gratification, etc.
  - Also associated with (esp seasonal disorders that can lead to) depression & suicide
  - Recall that drop in Serotonin associated w/crankiness that arises when overdue to begin sleep cycle
  - Anti-depression drug Prozac gets around Turnover issue by blocking 5HT re-uptake, prolonging synapse

GABA = Inhibitory NT, hyper-polarizes cell by admitting Cl- ions, affects emotion in Amygdala & Hypothalamus
- Most anxiety-reducing (axiolytic) drugs are Benzodiazepines (e.g. Valium, Xanax), act as GABA-agonists
  - Most GABA receptor sites also bind Benzo’s, enables GABA to bind more easily and longer
  - Note: Decreases both own experience of anxiety and how perceive anxiety in others!
- In contrast, blocking of GABA sites in Amygdala can lead to Panic Attacks

CCK - Stimulation of (excitatory) CCK sites in Amygdala => enhanced Startle Reflex
- CCK an NT/Hormone also involved in appetite suppression, released after eating when blood sugar rises
  - Some diet pills are CCK-agonists w/side effect of anxiety: Antagonists calming, but promote overeating