Mind, Brain & Behaviour 1
Behavioural Neuroscience

The neural basis of emotion

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Learning objectives

- Outline the roles of the amygdala and orbitofrontal cortex in emotional behaviour
- Understand the three main theories of how feelings are associated with emotions
- Explain the somatic marker hypothesis of social decision making
- Describe the role of the amygdala in fear conditioning
- Describe the neural pathways underlying voluntary and involuntary facial expression

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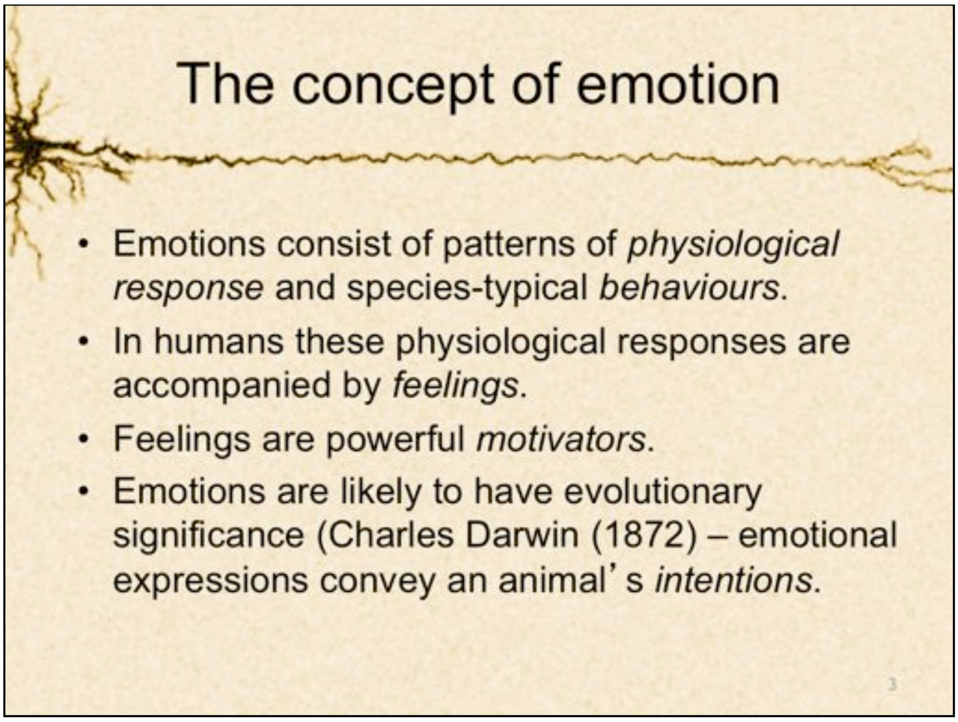


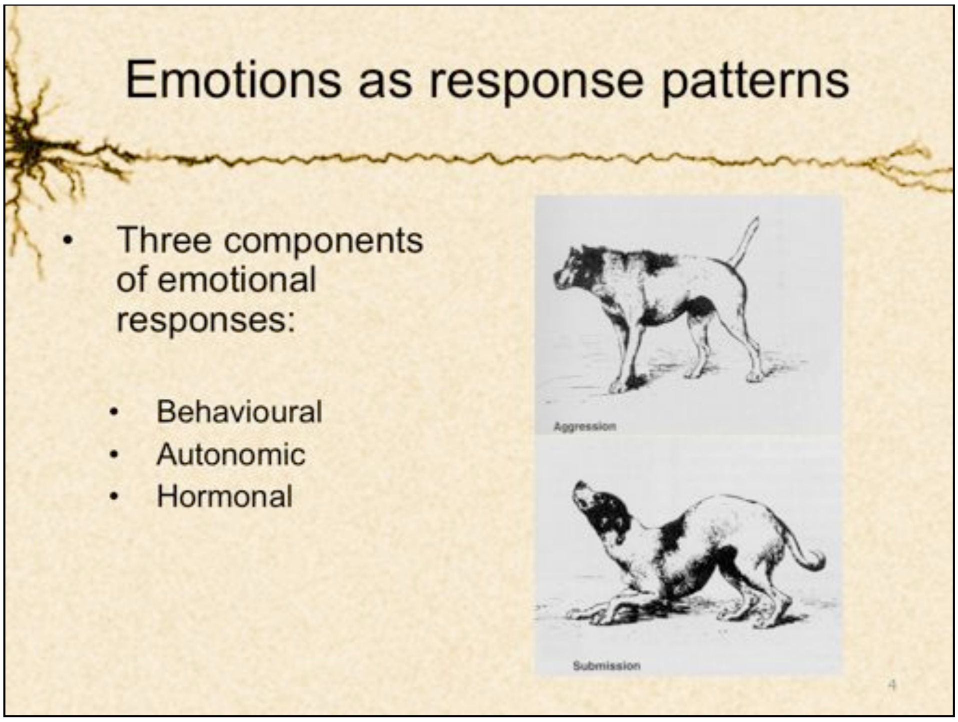
The concept of **emotion** has several different meanings. It is often used to refer to the positive or negative **feelings** we experience in particular situations, such as happiness when we see a close friend, or fearfulness when we are confronted by a wild dog. Emotion can also be used to refer to the **physiological** or **behavioural** changes that accompany certain situations, such as an increase in heart rate, rapid breathing, changes in posture or eye gaze, and so on.

Feelings are strong **motivators** (factors that form the basis for action), and thus they exert an influence on how we are likely to behave in particular situations. Emotional behaviours are likely to have played an important part in the evolution of our central nervous system.

Charles Darwin, in his 1872 publication *The Expression of Emotion in Man and Animals*, suggested that particular, stereotyped responses tend to accompany similar emotional states in all members of the same species. Thus, for example, in humans facial expressions tend to accompany the same feelings of emotion in all individuals, regardless of culture.

Darwin believed that expressions of emotion, like all other behaviours, evolve through the process of natural selection. He suggested that expressions of emotion evolve from behaviours that indicate what an animal is likely to do next (i.e., behaviours that have **predictive value**), and that if these expressions are of benefit to the animal they may evolve in ways that enhance their **communicative value** even though their original function may be lost.

The idea behind Darwin's proposition is that behaviours that originally served a function such as fighting would evolve to become a display of the **intention** to fight, without actually being enacted. In this way an elaborate threat display might evolve and obviate the need for real fighting, which in turn would lead to fewer deaths within the species due to conflict. To be effective, displays that convey intentions must be easily **recognisable** and readily **distinguishable** from other emotional displays within the species.

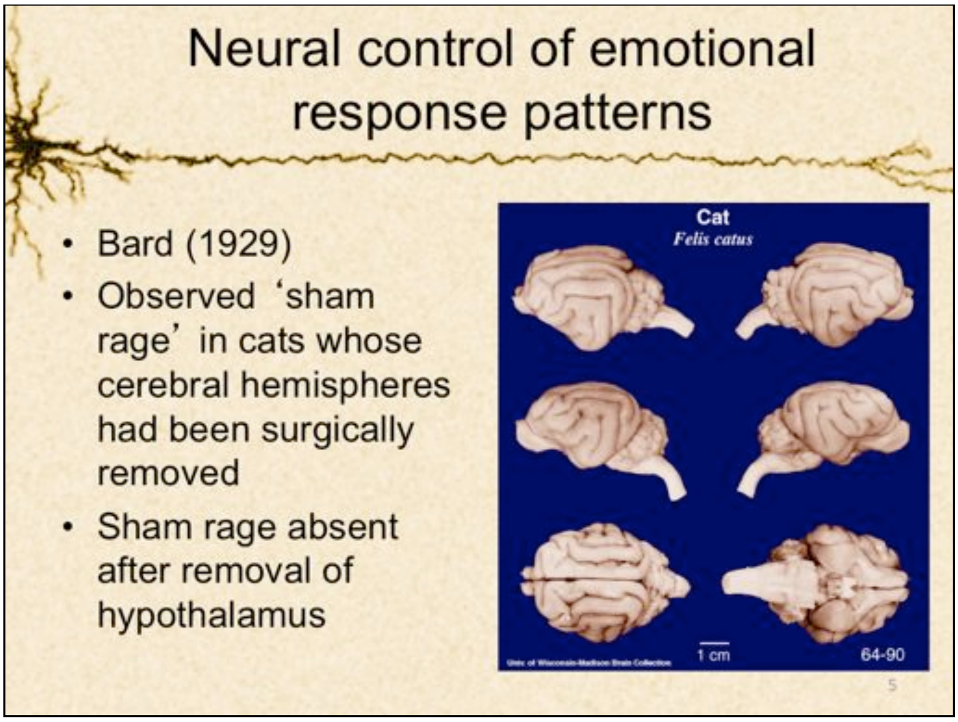


Emotional responses have three components:

1) **Behavioural** – muscular changes that are appropriate to the situation that elicits them. For example, a dog may seek to defend its territory in the face of an intruder by adopting an aggressive posture (growling, ears forward, back and tail up, teeth bared). If this behavioural display is sufficiently effective, the intruder may in turn adopt a submissive posture (ears back, back and tail down). If it is not effective, the defending dog may run toward the intruder and attack. Note that the displays of aggression and submission in this example are clearly distinguishable by the opposite movements and postures (what Darwin called the **principle of antithesis**).

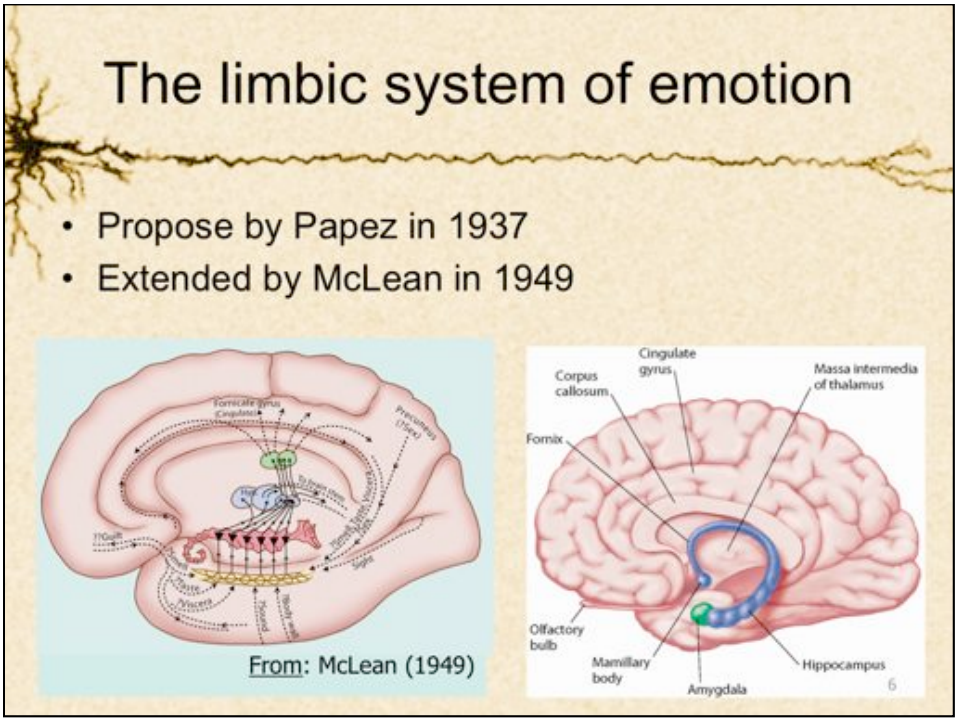
2) **Autonomic** – physiological changes induced by the autonomic nervous system facilitate the behavioural responses. In the example of the dog defending its territory, activity of the sympathetic branch of the autonomic nervous system increases whereas activity of the parasympathetic branch decreases; the dog's heart rate increases, and blood is diverted from the digestive system to the muscles.

3) **Hormonal** – these reinforce the autonomic changes. To continue the example, the dog's adrenal medulla secretes epinephrine and norepinephrine, which act to further increase blood flow to the muscles and cause nutrients stored in the muscles to be converted into glucose.



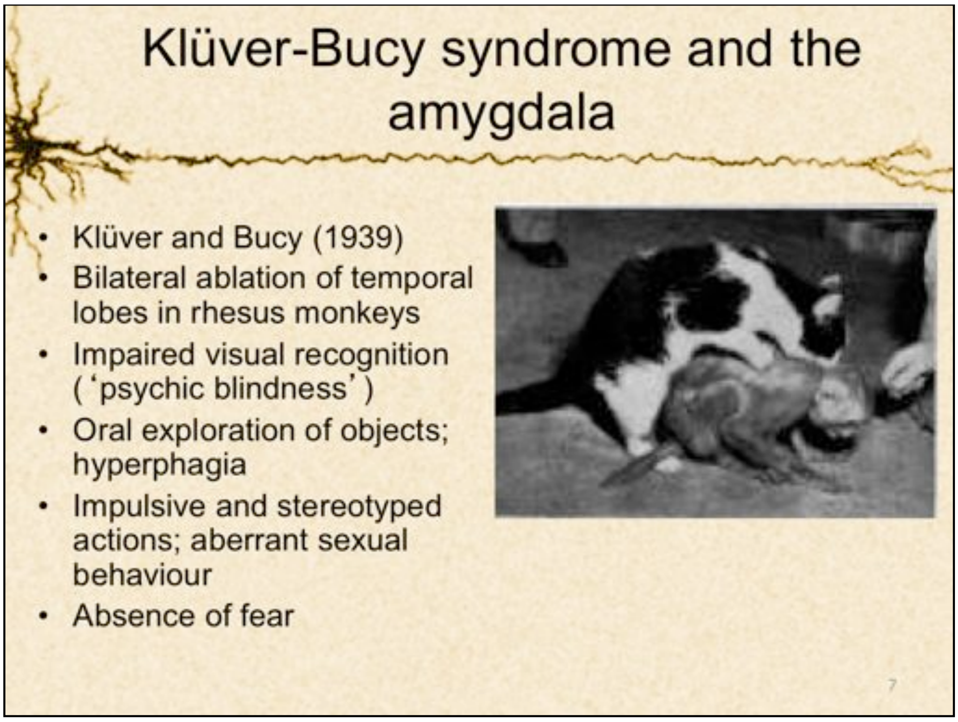
In a series of ablation studies conducted in the 1920s, Bard found that cats who had had their cerebral cortex surgically removed from both hemispheres (**decortication**) behaved in an excessively aggressive manner in response to the slightest stimulation. A simple touch would make the cats hiss, arch their backs, raise their fur and bare their teeth. Such behaviour is abnormal in two respects: first, it is particularly exaggerated, and second, it is not directed at an environmental stimulus that would normally elicit such an emotional reaction.

Bard called this behaviour following decortication **sham rage**. Crucially, when the surgical removal of brain tissue also included the **hypothalamus**, sham rage was no longer elicited, which led Bard to conclude that the hypothalamus is responsible for the expression of aggressive behaviour, and that the cortex normally inhibits and controls such behaviour.



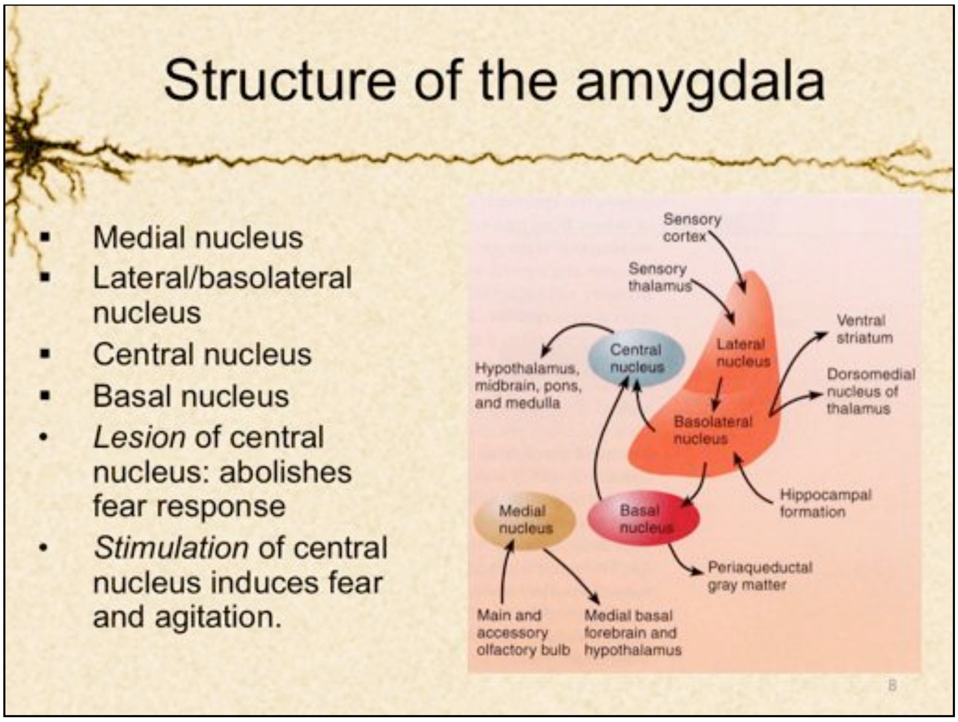
The neuroanatomist James Papez (pronounced ‘Payps’) in 1937 suggested that a distinct circuit of brain structures subserves emotional expression; this circuit included the **hypothalamus**, **anterior thalamus**, **cingulate gyrus**, **fornix** and **hippocampus**. Some years later, in 1949, Paul McLean coined the term the ‘Papez circuit’, and he added the **amygdala**, **orbitofrontal cortex**, and some nuclei of the **basal ganglia**. He called this extended neural circuit the **limbic system**. (The circuit borders the thalamus, and limbic means ‘border’.)

The hippocampus formed the central element of McLean’s limbic system (denoted here by a seahorse). It was believed to receive inputs from the various senses, as well as from the internal organs. Although the concept of the ‘limbic system’ is still used, it is now clear that it has several shortcomings, including the fact that the hippocampus plays a much more important role in learning and memory than in emotion.



In 1939 Klüver and Bucy described a syndrome in monkeys that followed surgical removal of the anterior temporal lobes. The monkeys ate virtually anything that was edible, showed increased sexual activity (often directed at inappropriate objects), a tendency to explore all items with the mouth, and an **absence of fear**. Monkeys that were previously difficult to handle became completely tame after the surgery, showing no fear whatsoever, even toward snakes, an animal that terrifies most monkeys. It has been suggested that the **Klüver-Bucy syndrome** arises from damage to the **amygdala**, which is located in the anterior temporal lobe (and hence was removed by the surgery).

Klüver-Bucy syndrome is occasionally described in humans. One such patient, described by Marlowe, Mancall & Thomas (1985), became ‘flat’ and indifferent to people and events after his brain damage (caused by an infection). He would gaze for hours at the television, even when it was turned off; he would imitate the actions of others; he engaged in oral exploration of all objects within his reach, lifting them to his mouth and sucking or chewing on them. Despite being heterosexual prior to his illness, he made frequent sexual advances toward other male patients.



It is now recognised that the amygdala plays a key role in the physiological and behavioural reactions to objects and situations that have particular biological significance (e.g., those that are likely to be painful, those that signify the presence of food or water, or the presence of a potential mate or rival).

The amygdala (or more accurately, the **amygdaloid complex**) is located in the anterior temporal lobe, and consists of several nuclei with different functions:

Medial nucleus – receives sensory input (including information from the olfactory system concerning odours and pheromones) and relays information to the basal forebrain and hypothalamus.

Lateral/basolateral nuclei – receive sensory information from the primary sensory cortex, association cortex, thalamus and hippocampus. They send axons to parts of the basal ganglia, thalamus, and the central nucleus of the amygdala.

Central nucleus – sends axons to regions of the hypothalamus, midbrain, pons and medulla that are responsible for the expression of various emotional responses.

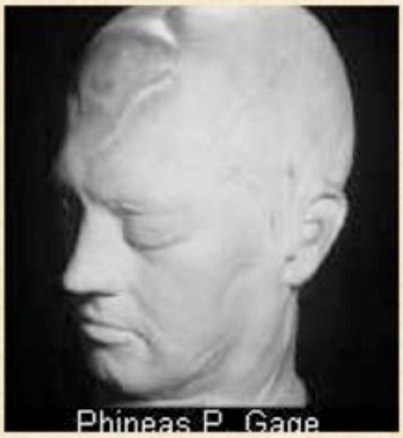
Basal nucleus – receives axons from the lateral and basolateral nuclei and sends axons to other amygdaloid nuclei and to the midbrain.

The central nucleus is the most important part of the brain for the expression of emotional responses to **aversive** or **threatening** stimuli. Damage to the central nucleus abolishes fear responses to stimuli that have been associated with aversive events. By contrast, electrical stimulation of the central nucleus induces a reaction of fear or agitation.

Some of the effects of **anxiolytic** (anxiety-reducing) drugs are produced through the central nucleus of the amygdala. The central nucleus contains a high concentration of opiate receptors. It seems that some anxiety disorders may be due to overactivity of the central nucleus.

Don't forget Phineas…

- Phineas Gage
  - railway foreman in Cavendish, Virginia
  - accident involving 'tamping iron' (1848)
  - survived profound damage to frontal lobe
  - behavioural changes (*'no longer Gage'*)



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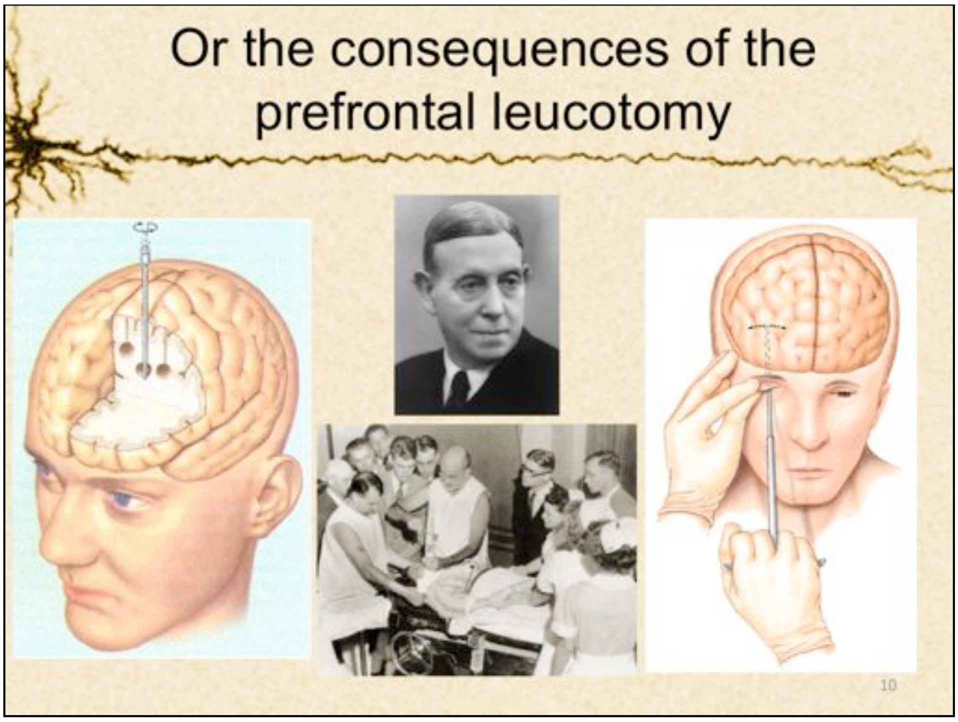
You will recall from Lecture 1 the case of Phineas Gage, and the personality changes that occurred after damage to his frontal lobes. Here are the key details of the Gage case, as outlined originally in Lecture 1:

Phineas Gage was using a tamping iron to lay charges in rock, which was being blasted in order to create a cutting for a railroad track. While tamping a charge into the rock the tamping iron created a spark that ignited the gunpowder and sent the tamping iron shooting up from the hole and into Phineas' head. Phineas sustained extensive damage to the frontal lobes, but survived the accident and lived for several years thereafter.

Some months after the accident, probably in about the middle of 1849, Phineas felt strong enough to resume work. But because his personality had changed so much, the contractors who had employed him would not give him his place again. Before the accident he had been their most capable and efficient foreman, one with a well-balanced mind, and who was looked on as a shrewd smart business man. He was now fitful, irreverent, and grossly profane, showing little deference for his fellows. He was also impatient and obstinate, yet capricious and vacillating, unable to settle on any of the plans he devised for future action. His friends said he was "No longer Gage."

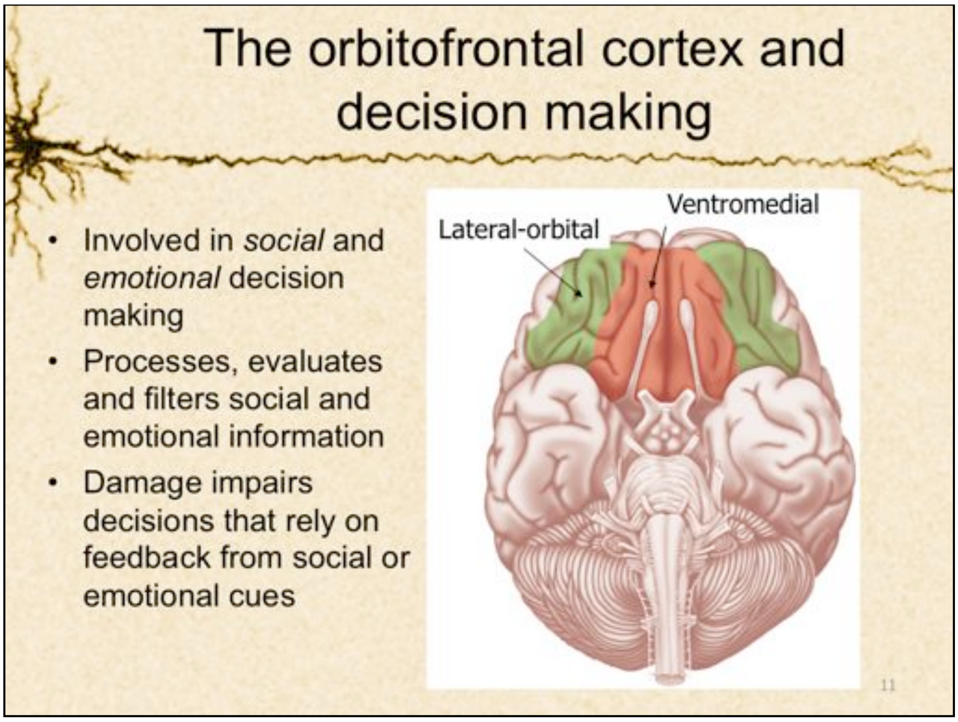
It might be argued that many aspects of Phineas' personality change are attributable to alterations in his emotional reactions to people and situations in everyday life.



You will also recall from Lecture 1 that frontal leucotomy was developed as a treatment for psychiatric disorders, particularly those in which anxiety is a prominent symptom.

As outlined in that lecture, the long-term effects of the frontal leucotomy were not properly assessed for many years, by which time tens of thousands of individuals had undergone the operation. Only later did it become clear that prefrontal leucotomy was of little therapeutic value, and that it had many serious cognitive side-effects such as a **apathy** (lack of motivation), **emotional unresponsiveness**, **disinhibition** (lack of self-control), **lack of foresight** and **inability to plan**, and so on.

The operation was eventually abandoned in most parts of the world, but not before more than 40,000 operations had been performed in the United States alone (and many hundreds in Australia).

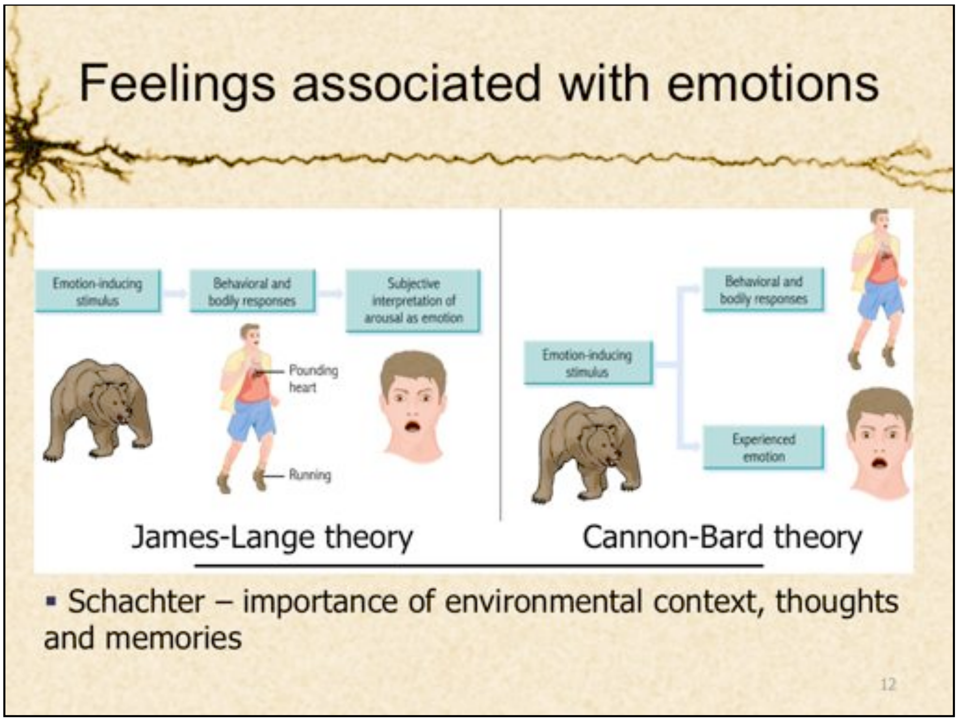


The human **orbitofrontal cortex** is located on the ventral surface (underside) of the frontal lobes, immediately above the orbits that contain the eyes. The more medial region is known as the **ventromedial prefrontal cortex**, whereas the more lateral region is called the **lateral-orbital prefrontal cortex**.

The precise role of the orbitofrontal cortex is unclear, partly because the behaviours it is responsible for controlling are themselves difficult to categorise. It seems likely that Phineas Gage had damage to this region, either in the left hemisphere alone or bilaterally, after the tamping iron was blasted through his head. After the accident, you will recall, Gage was considered a changed man, no longer reliable and shrewd, but impatient, rude, and indecisive.

In general terms, current opinion favours the view that the orbitofrontal cortex is crucially involved in regulating our ability to evaluate social and emotional information, inhibit inappropriate responses and develop plans for acting appropriately. Some workers have classified the role of the orbitofrontal cortex as one of **decision making**.

There is evidence that patients with lesions of the orbitofrontal cortex tend to engage in antisocial behaviours, and may in particular have difficulty in inhibiting aggressive impulses. One study found that individuals with a history of antisocial or violent behaviours show reduced metabolic activity in the orbitofrontal cortex, as measured by functional imaging techniques.



So far we have considered the behavioural and physiological aspects of emotion. But what accounts for the unique **feelings** associated with emotional experiences?

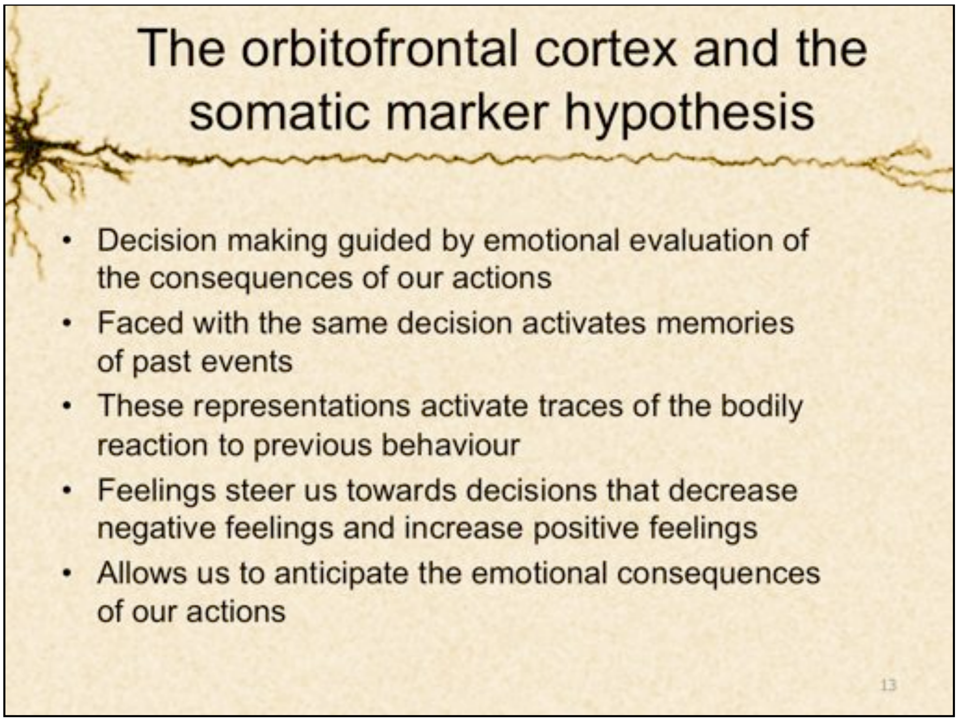
William James (1842-1910) and Carl Lange (1834-1900) independently suggested similar explanations for the feelings associated with emotions. According to the **James-Lange** theory, an emotion-inducing stimulus or event triggers an appropriate set of physiological responses (controlled by the autonomic nervous system), such as increased heart rate and breathing, sweating, etc., as well as particular behaviours, such as clenching of the fists and teeth. The brain receives feedback from sensory receptors in the skin, muscle and internal organs that produce these responses, and this feedback generates the subjective feeling of emotion. In other words, according to the James-Lange theory our emotional feelings are determined by the way in which we interpret the sensory feedback we receive from our physiological response to an emotion-inducing stimulus. This is opposite to the view many people have, which is that emotional feelings are experienced directly.

In 1927, the physiologist Walter Cannon criticised this theory, saying that the internal organs provide only very diffuse and slow sensory feedback which could not account for the salience and immediacy of emotional feelings. He proposed instead that emotion-inducing stimuli have two independent effects: they excite a feeling of emotion and also a range of physiological effects controlled by the somatic and autonomic nervous systems. The theory was expanded and promoted by Bard, and has come to be known as the **Cannon-Bard** theory.

In fact, both the James-Lange and Cannon-Bard theories appear to be incorrect in some respects. One prediction of the James-Lange theory is that patients with spinal cord damage, which deprives the brain of somatic input, should experience reduced feelings of emotion. The evidence on this is mixed: some researchers suggest that such patients do indeed experience less intense emotions, whereas others have shown that such patients are capable of a full range of emotional feelings. On the other hand, as we shall see there is also evidence that autonomic and somatic responses to emotional stimuli can influence emotional experience.

Failure to find unequivocal support for either the James-Lange or Cannon-Bard theories led to the development of a third theory, originally proposed by Stanley Schachter. He suggested that a general state of visceral arousal was necessary for the experience of emotion, and that an individual's **interpretation** of the experience is determined by the **current environmental context, thoughts and memories**. Thus, instead of specific physiological responses generating specific emotional experiences, as suggested by the James-Lange theory, Schachter suggested that different emotional experiences reflect the variety of ways in which a common physiological response can be interpreted.

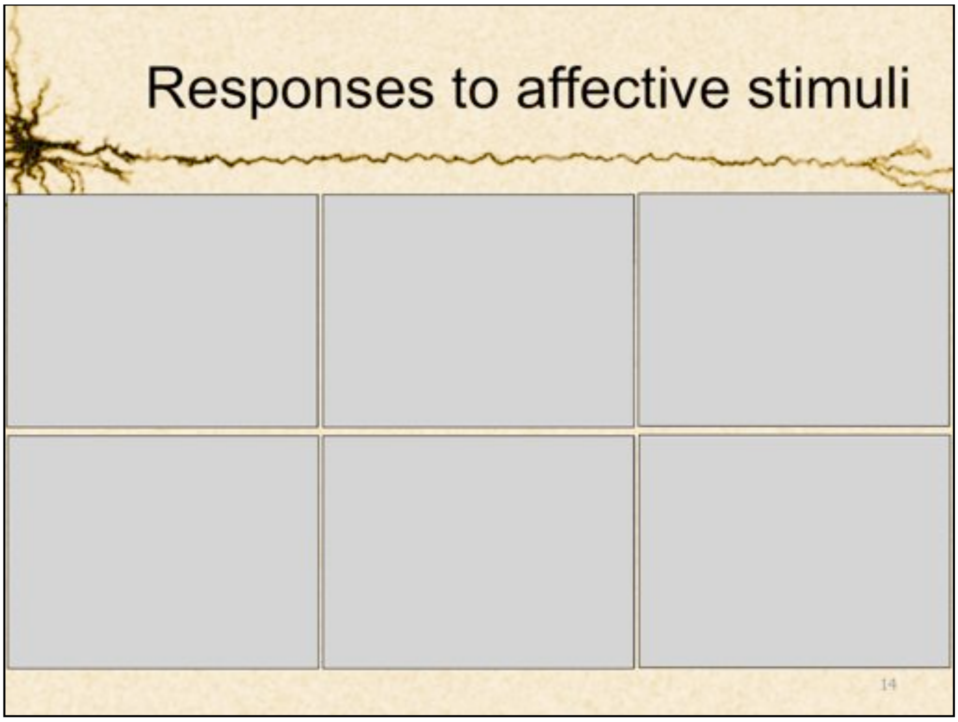
To give a concrete example, going on a rollercoaster ride produces a set of physiological responses. The experience will seem exciting or terrifying depending on what we expect about the ride, our previous encounters with such rides, who we are with, whether we feel in control of the situation, and so on. Some will love the experience and others will hate it. We may ourselves love the experience on one occasion and hate it the next,



Emotion plays an important role in everyday decision making. Damasio (1994) suggested that decision making is guided by the **emotional evaluation** of the consequences of our actions. Every time we make a decision we rely upon our **recollection of the outcomes of previous choices**, in terms of their costs and benefits, and on our anticipation of the consequences of our current actions.

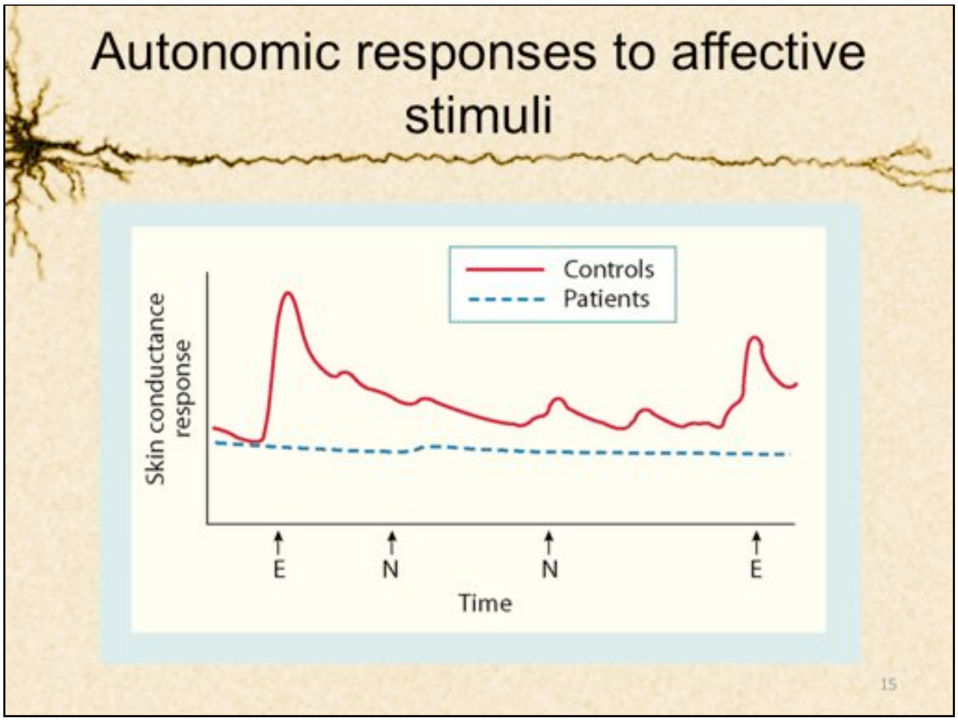
According to Damasio, when we learn by experience the consequences of our actions become associated with the various physical reactions that were triggered at the time. Recall that the autonomic nervous system controls various bodily responses to environmental events, such as increased or decreased heart rate, sweating, and so on. When faced with having to make a decision about a current set of circumstances, we activate representations of past events that were similar in nature, and these representations carry with them a trace of the bodily reactions to our previous behaviour. To take a concrete example, imagine that in the past you bought expensive tickets to a concert being given by your favourite band, but on the night the sound was bad and the group only played a few songs. You went away feeling angry at having paid so much money for a disappointing night's entertainment, and spent the next few weeks working overtime to cover the cost of the tickets. Some months later a friend tells you the same band is back in town, and suggests you go along to see them. You have to make a decision. On the one hand, you hope things will be better this time; on the other, you have a "gut feeling" that it might be wiser to decline given what happened last time.

Damasio argues that this gut feeling is a kind of **somatic marker** that helps you to make the best decision. According to his **somatic marker hypothesis**, such feelings steer us away from behaviours linked to negative feelings and toward behaviours connected with positive feelings. In a sense, somatic markers enable us to **anticipate** the emotional consequences of our actions. Crucially, Damasio has suggested that the somatic reactions that underlie our decision making are mediated via the orbitofrontal cortex.



One way to measure the somatic marker hypothesis is to measure the emotional reactions of patients with lesions of the orbitofrontal cortex to stimuli that normally elicit affective responses.

Damasio et al. (1984) measured the skin conductance response (SCR; a physiological function controlled by the autonomic nervous system) of patients with lesions of the orbitofrontal cortex. These patients had a normal SCR to innately negative stimuli, such as loud sounds, but showed no SCR to stimuli with learned affective value, such as pictures of mutilated bodies, disaster scenes and nudity. Normal control patients by contrast, showed an increase in SCR to the affective stimuli compared with pictures of neutral stimuli, such as photographs of the countryside.



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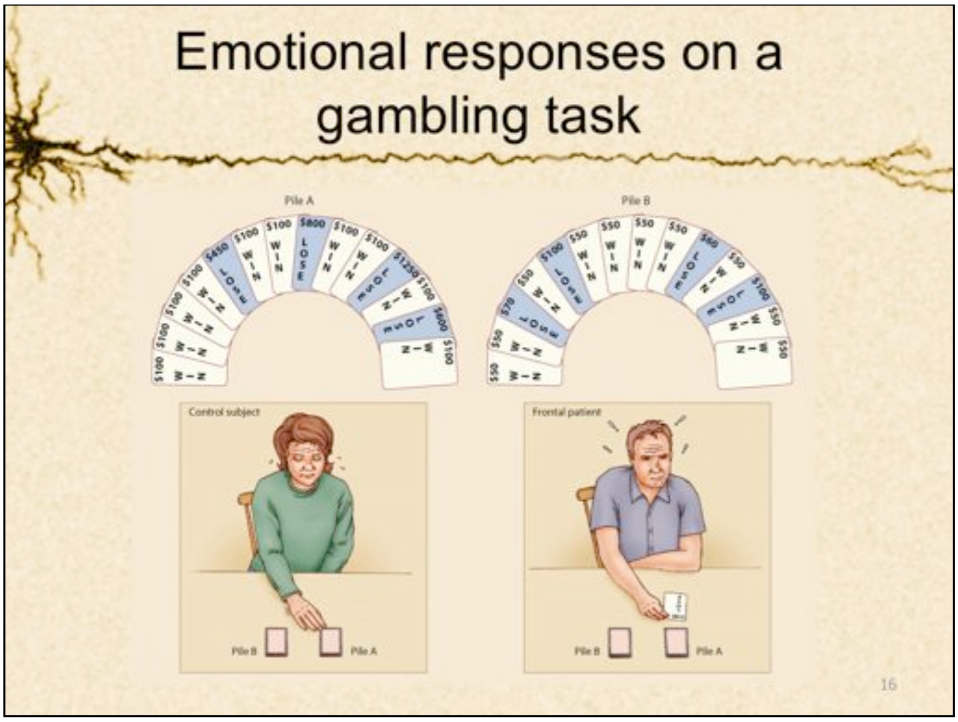
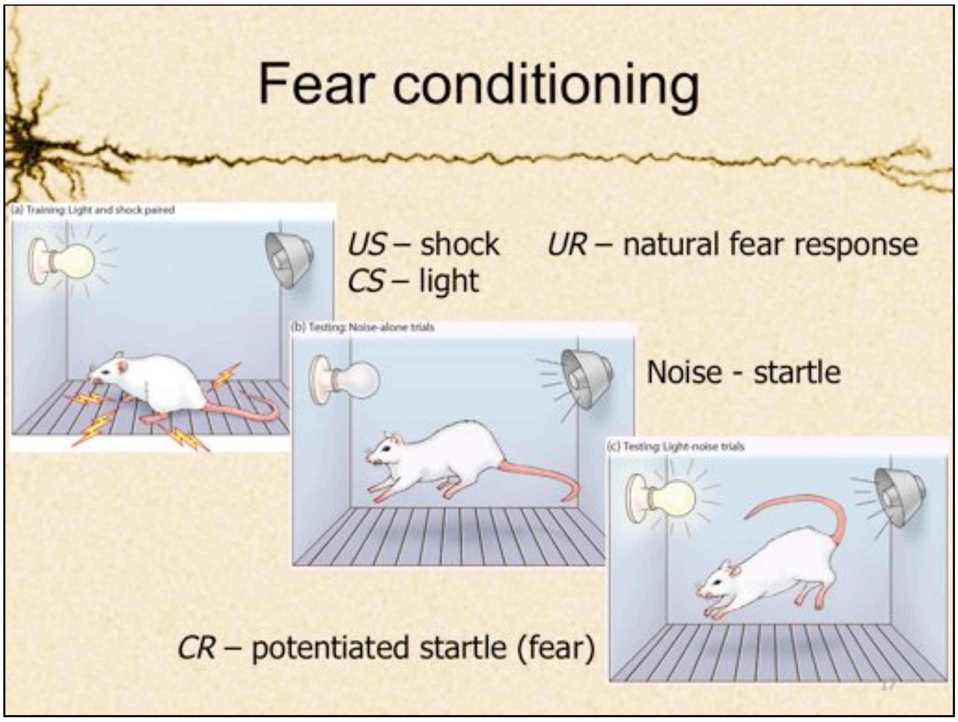


The absence of a significant autonomic response to learned emotional stimuli in patients with orbitofrontal lesions provides some support for Damasio’s somatic marker hypothesis. But to what extent does their lack of an emotional reaction have an impact on decision-making in such individuals?

To address this question, Damasio et al. had their patients and controls perform a **simulated gambling task**. Participants were confronted with two packs of cards, each of which contained the word WIN or LOSE and an associated amount of money. The participants were free to choose cards from either deck with the aim of maximising their winnings. Unbeknownst to the participants, one deck contained cards with modest payouts and and modest losses, whereas the other contained cards with large payouts and hefty losses. In the long run, choosing from the latter deck resulted in the loss of money, whereas choosing from the former led to a small overall winning.

Whereas controls quickly learned to choose from the deck with small wins and losses, patients with orbitofrontal lesions continued to choose from the other deck, lured by the occasional big win, and apparently immune to the effects of hefty losses. By measuring the SCR in both groups, Damasio et al. were able to assess the autonomic consequences of the individuals’ choices. Both groups showed an increase in SCR after turning over each card, demonstrating that they registered the rewards and penalties. Over time, though, the SCR for controls began to rise when they were *contemplating* choosing a card from the risky deck. By contrast, the SCR of the orbitofrontal patients failed to show any such anticipatory effect, suggesting that their choices were not accompanied by the physiological effects of emotion.

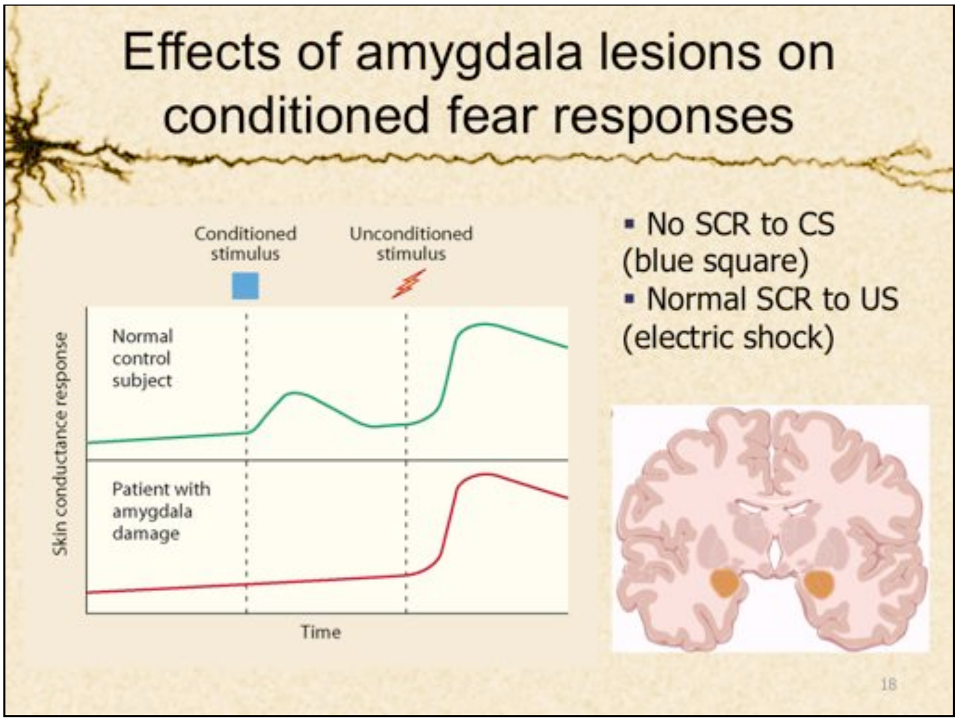


Psychologists have known for a long time that emotional responses can be **conditioned**. One particular example is **fear conditioning**, in which a previously neutral stimulus acquires aversive properties because it has been paired repeatedly with an aversive event, such as a painful stimulus.

A common example of fear conditioning is provided by this example of a rat inside a cage whose floor can deliver an electric shock. Initially a light is switched on for a few seconds and turned off again. The light is the **conditioned stimulus (CS)**: it might surprise the rat the first few times it is turned on, but it is a neutral event and the rat quickly habituates to it. Once the rat has habituated to the light, it is paired with an electric shock (the **unconditioned stimulus, US**), which is delivered immediately before the light goes off. The rat has a natural fear response to the shock, which is called the **unconditioned response (UR)**. After a few pairings of the light (CS) and the shock (UR), the rat learns that the light predicts a shock and develops a fear response in association with the light alone; this is called the **conditioned response (CR)**.

In the example shown above, the CR is a **potentiated startle reaction**. Most animals show a natural startle reaction to sudden, unexpected stimuli such as a loud noise. Such natural startle reactions can be exaggerated when the animal is already in a fearful or anxious state. In this example, the rat shows a potentiated (i.e., much greater) startle reaction when the noise is preceded by the light, which elicits a fearful state.

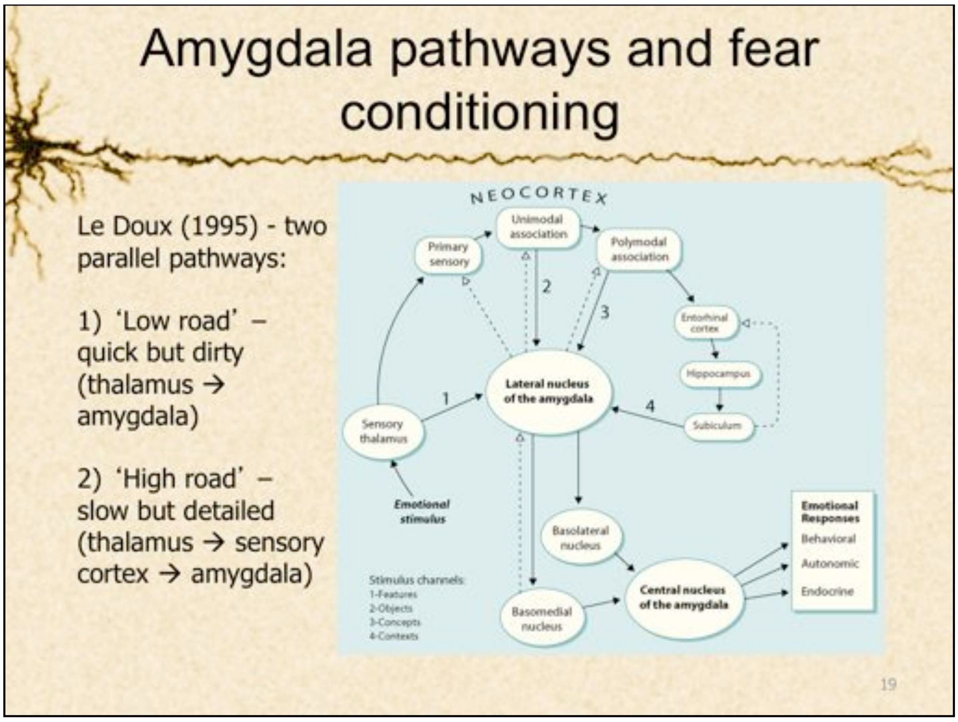
Movie directors often capitalise on the potentiated startle reaction in their films. A scenario is established which elicits an anxious state in the viewer: a woman walking slowly through a graveyard at night, with fog hovering all around and strange noises emanating from unseen places. Suddenly there is a shriek and an object flashes into view; the audience jumps and screams, only to realise it is a harmless cat that has jumped down from an overhanging branch!



A consistent finding to emerge from the human neuropsychological literature is that damage to the **amygdala** impairs conditioned fear responses. Interestingly, amygdala damage does NOT disrupt the normal **unconditioned** response to an aversive event, suggesting that the amygdala itself is not required to show a fear response. Instead, amygdala damage **blocks** the ability of the individual to **learn a conditioned response** to a neutral stimulus that is paired with an aversive unconditioned stimulus.

In this example from Phelps et al. (1998), a woman with bilateral lesions of the amygdala participated in a fear conditioning experiment in which a blue square was presented on a computer display for a few seconds, and was immediately followed by a mild electric shock. The patient showed a normal fear response to the shock, as reflected by the increase in her **skin conductance response** (SCR; a brief change in skin conductance due to autonomic activity associated with a stimulus or event). But she failed to show any change in SCR when the blue square was presented, despite many learning trials, indicating a **failure to acquire a conditioned fear response**. This is in contrast with a normal control participant, who shows an increased SCR to both the conditioned and unconditioned stimuli.

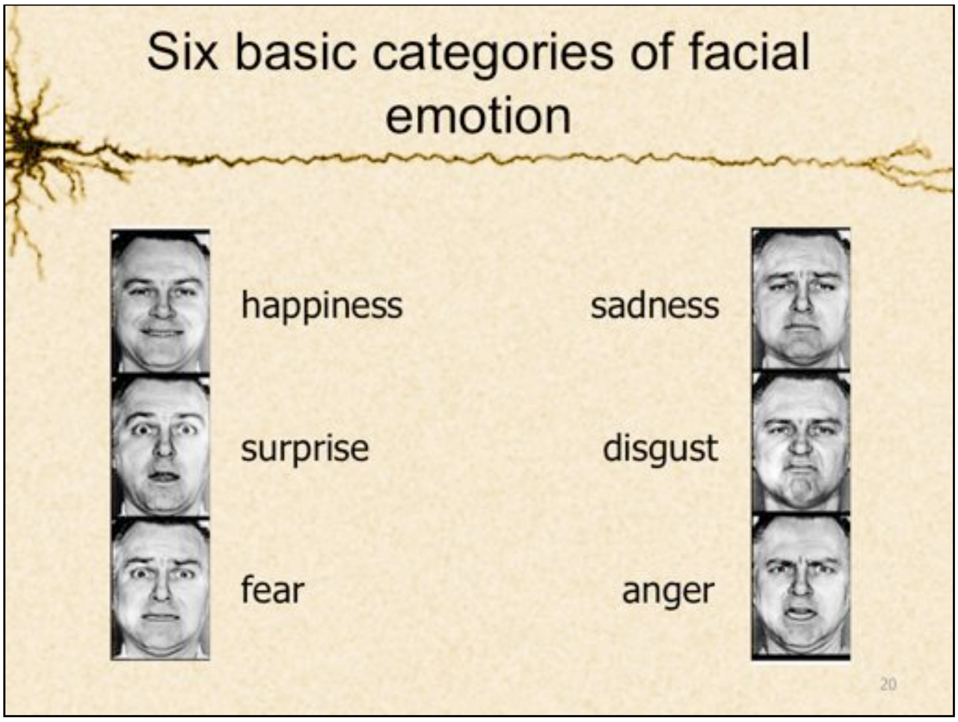
Interestingly, after the experiment the patient stated she was well aware that the appearance of the blue square signalled an impending electric shock. Thus, even though she had **explicit knowledge** of the relationship between the conditioned stimulus (CS) and the unconditioned stimulus (UCS), this evidently had no impact on her autonomic responses (as reflected in her SCR). When patients with bilateral damage to the hippocampus, but an intact amygdala on each side, are tested on the same task, they show the opposite pattern to the patient just described: they exhibit a normal SCR to the conditioned stimulus, but remain unaware of the relationship between the CS and the UCS. (You will recall from Lecture 5 that hippocampal damage can cause severe memory impairments, as illustrated by the famous amnesic patient HM, whose hippocampi had been removed surgically to relieve his epilepsy.)



Joseph Le Doux and his colleagues have suggested that there are two pathways subserving fear conditioning in the human brain. The lateral nucleus of the amygdala is reciprocally connected with the sensory and association cortices, and so is well suited to gather information that will underlie new associations. The lateral nucleus of the amygdala projects to the central nucleus, which in turn sends projections to other parts of the brain to initiate an emotional response, if the stimulus is determined to represent a potential threat.

One crucial feature of Le Doux's model of the neuroanatomical basis for fear conditioning is that information concerning a stimulus can reach the amygdala via **two separate, parallel pathways**. One pathway, sometimes called the **'low road'**, involves information transmitted from the sensory thalamus directly to the lateral nucleus of the amygdala. This pathway conveys information very quickly, but in a fairly crude form, and simply indicates whether a given stimulus is likely to represent the conditioned stimulus. A second pathway, called the **'high road'**, conveys information from the sensory thalamus to the primary sensory cortex and association cortex, which together analyse the stimulus in much more detail, before sending this information on to the lateral nucleus of the amygdala.

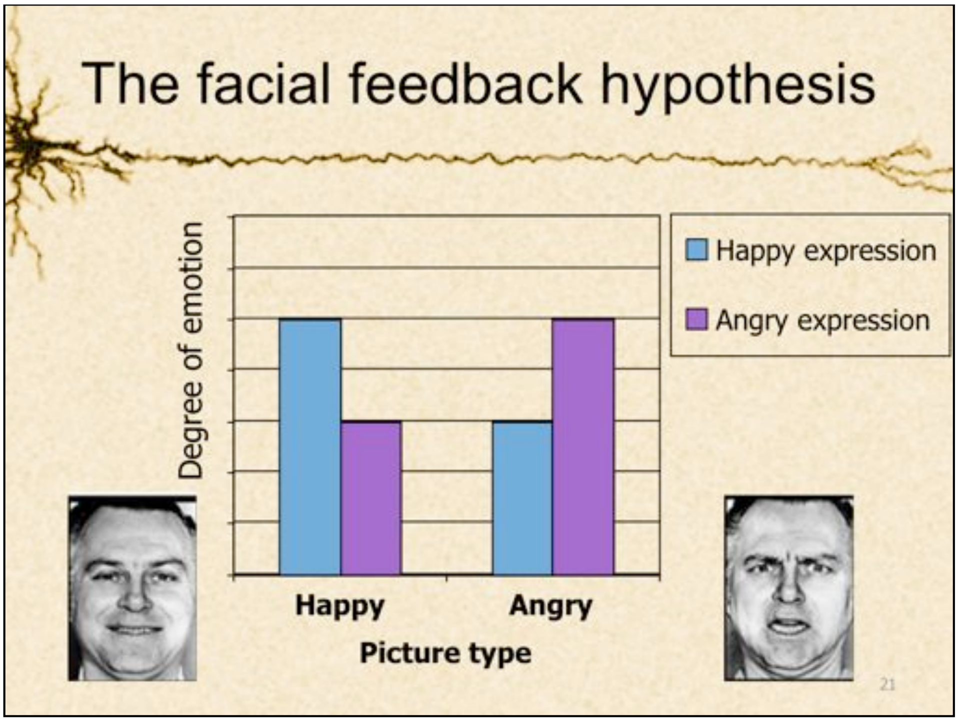
The low road is thought to ready or prime the amygdala quickly, so that it is ready to respond to the more detailed information from the high road if this corresponds to the conditioned stimulus. This two-pathway mechanism allows for both fast and accurate responses to potentially threatening events.



Charles Darwin (1872) suggested that human expressions of facial emotion evolved from similar expressions in other animals (Darwin, 1872).

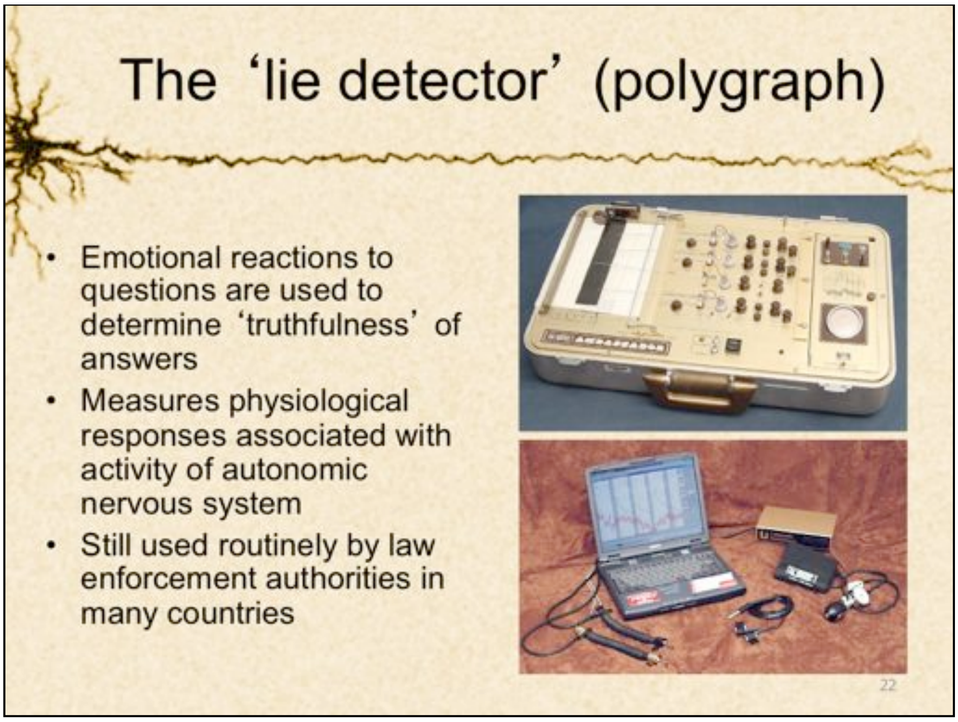
Facial expressions of emotion do not need to be learned; they appear to be **innate.** For example, congenitally blind children express the same facial emotions as normally sighted children (Izard, 1971), even though blind children have never seen facial emotions in others. Moreover, people of different cultures use the same patterns of movement of face muscles to convey particular emotions. Ekman & Friesen (1971) studied members of an isolated tribe in New Guinea. They found that tribe members readily recognised the emotions conveyed by Westerners.

Facial emotion conveys important information about the feelings and intentions of others (e.g., fear or anger may signal potential threat). Efficient and accurate recognition of facial expressions of emotion in others thus has **adaptive value**. The human **limbic system**, particularly the **amygdala**, plays a crucial role in the expression and recognition of emotion. Damage to the amygdala impairs the ability to recognise facial expressions of emotion, particularly expressions of **fear** (Adolphs et al., 1994).



Is there any truth to the idea that 'putting on a happy face' can actually make us feel better. It has been suggested that our facial expressions can influence our emotional experience, an idea known as the **facial feedback hypothesis**. The basic idea behind this hypothesis is that afferent information from the muscles of the face is conveyed back to the brain and interpreted as reflecting an emotional state.

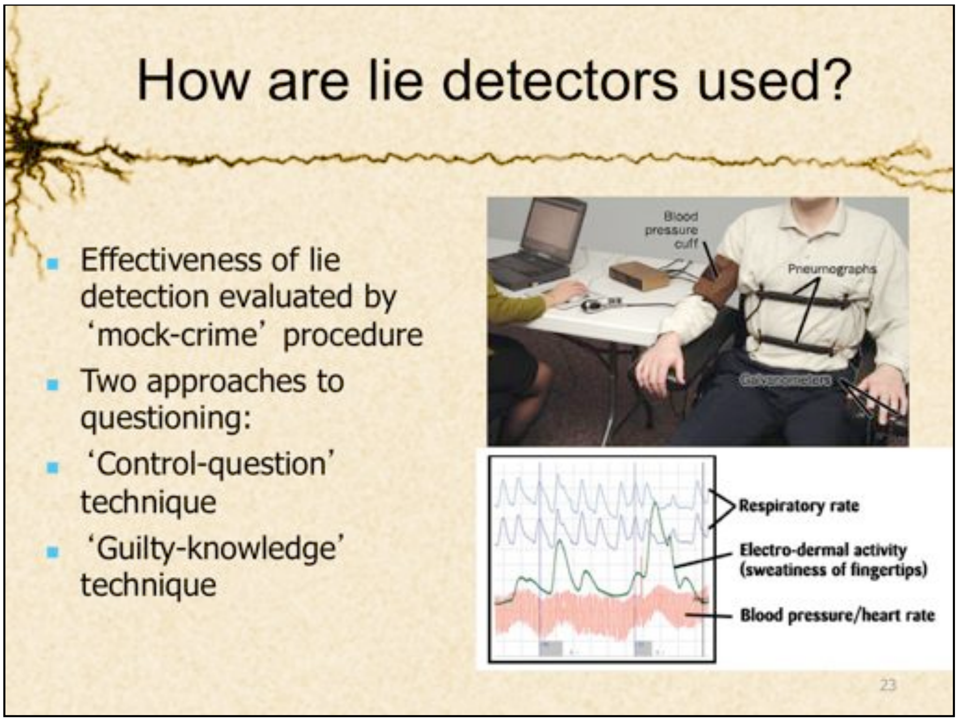
Rutledge and Hupka (1985) tested the facial feedback hypothesis by having participants adopt one of two patterns of facial-muscle contractions while viewing a series of pictures. The facial patterns corresponded to the muscles that would be contracted for a happy or angry expression, though the participants were not told this. Participants reported that the pictures made them feel more happy and less angry when they were making happy faces and more angry and less happy when they were making angry faces.



The autonomic response that accompanies emotional reactions to stimuli and events forms the basis for the so-called **lie-detector (polygraph).** Lie detectors rely on autonomic responses associated with emotion to infer whether a person is telling the truth in response to specific questions.

Most modern lie detectors measure such physiological functions as heart rate, blood pressure, breathing and skin conductance (hence the name **polygraph**, meaning ‘many graphs’).

Lie detectors are routinely administered during police interrogation, particularly in the United States, and can provide useful information; but they are not infallible.



The main problem with evaluating the effectiveness of lie detectors is that it is rarely possible to determine with certainty whether a particular suspect is guilty or innocent.

To overcome this problem, psychologists have used simulated crimes, known as **mock-crime procedures**, in which volunteers participate in a pretend crime and are then tested by an examiner who does not know the 'guilt' or 'innocence' of the individual. In this way any determination made by the examiner can be checked objectively.

Most interrogation techniques involving lie detectors adopt a method called the **control-question technique**, in which the physiological response to the key question (e.g., 'Did you steal that car?') is compared with the physiological response to a control question ('Have you ever been in jail before?'). The technique assumes that lying will be associated with greater activity by the sympathetic branch of the autonomic nervous system. Research using the control-question technique in mock crimes has found that success rates are around 80%.

Of course, lie detectors don't actually detect lies at all; rather, they detect the physiological changes that accompany an individual's **emotional response** to particular questions. For this reason it is more difficult to detect lies in real-life situations than in experimental simulations. In real life, many people are likely to have some emotional reaction to the question 'Did you steal that car?', regardless of their guilt or innocence.

In an effort to overcome this problem, questioners use the **guilty-knowledge technique**. This requires that the questioner has some specific pieces of information concerning a crime that only the guilty person could know. Instead of trying to detect a lie, the questioner simply uses the polygraph to assess a suspect's emotional reaction to a series of actual and contrived details of the crime. People who are innocent will react in the same way to all details because they have no knowledge of the crime; by contrast, the guilty person should show an emotional reaction to the actual details.

In a mock-crime study by Lykken (1959), participants waited for an occupant of an office to leave the room, then stole the purse from her desk, removed the money and left the purse in a locker. At the time of interrogation, the questions were as follows: 'After the theft, where do you think we found the purse…In the toilet? In the filing cabinet? In the locker?'. Using skin conductance as the only physiological measure, 88% of the mock criminals were identified; even more importantly, none of the innocent parties was judged to be guilty.

Summary

- Emotional behaviour – role of hypothalamus, amygdala and orbitofrontal cortex
- Feelings of emotions – James-Lange, Cannon-Bard, Schachter
- Somatic marker hypothesis – mechanisms underlying social and emotional decision making
- Fear conditioning – role of the amygdala, dual pathways (Le Doux)
- Facial expression of emotion – voluntary and involuntary control, facial feedback hypothesis

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