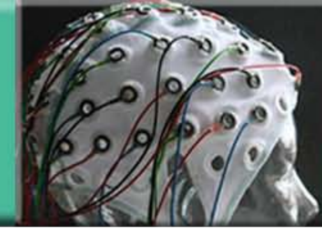
UNIVERSITÄT
DES
SAARLANDES

Überblick

14.04.	Konzepte der Biol. Psychologie
21.04.	--- entfällt ----
28.04.	Messmethodik
05.05.	Elektrophysiologische Verfahren
12.05.	Bildgebende Verfahren
19.05.	Gehirnerkrankungen
26.05.	Das visuelle System
02.06.	Mechanismen der Wahrnehmung
09.06.	Das sensomotorische System
16.06.	Lernen und Gedächtnis
23.06.	Hemisphärenasymmetrie
30.06.	Entwicklung und Plastizität
07.07.	Sprache
14.07.	**Emotion**
21.07.	Prüfungsvorbesprechung



Kolloquium Psychologie Sommersemester 2010

Mittwoch 18.00 – 20.00 h; Seminarraum IIa; Geb. A2 4

28.04.10	Dietrich Manzey (TU Berlin) *Complacency* and *automation bias* effects in interaction with automation: An attentional synthesis
19.05.10	Matthias Riemenschneider (UKS Homburg): Genetik der Alzheimer-Krankheit und assoziierte Endophänotypen.
26.05.10	Matthias Weigelt (SWI, Saarbrücken). Intelligente Hand- und Kopfarbeit: Ein Beitrag aus der experimentellen Bewegungsforschung zur Planung und Organisation motorischer (Alltags-)Aufgaben.
09.06.10	Thomas Elbert (Universität Konstanz): Maladaptive minds: Wie traumatische Erfahrungen Geist und Gehirn verändern.
16.06.10	Igor Schindler (University Hull): TMS as a tool in Neuropsychology.
23.06.10	Andreas Eder (FSU Jena): Mechanismen affektiver Handlungssteuerung.
07.07.10	David Donaldson (University of Stirling):
14.07.10	Christian Döller (University College London): Raum, Gedächtnis und neuronale Repräsentation.

Heute !!

Lernkontrolle

Welche Gehirnregionen sind dem Wernicke-Geschwind-Modell zufolge am lauten Lesen beteiligt?

Wernicke – Geschwind Modell

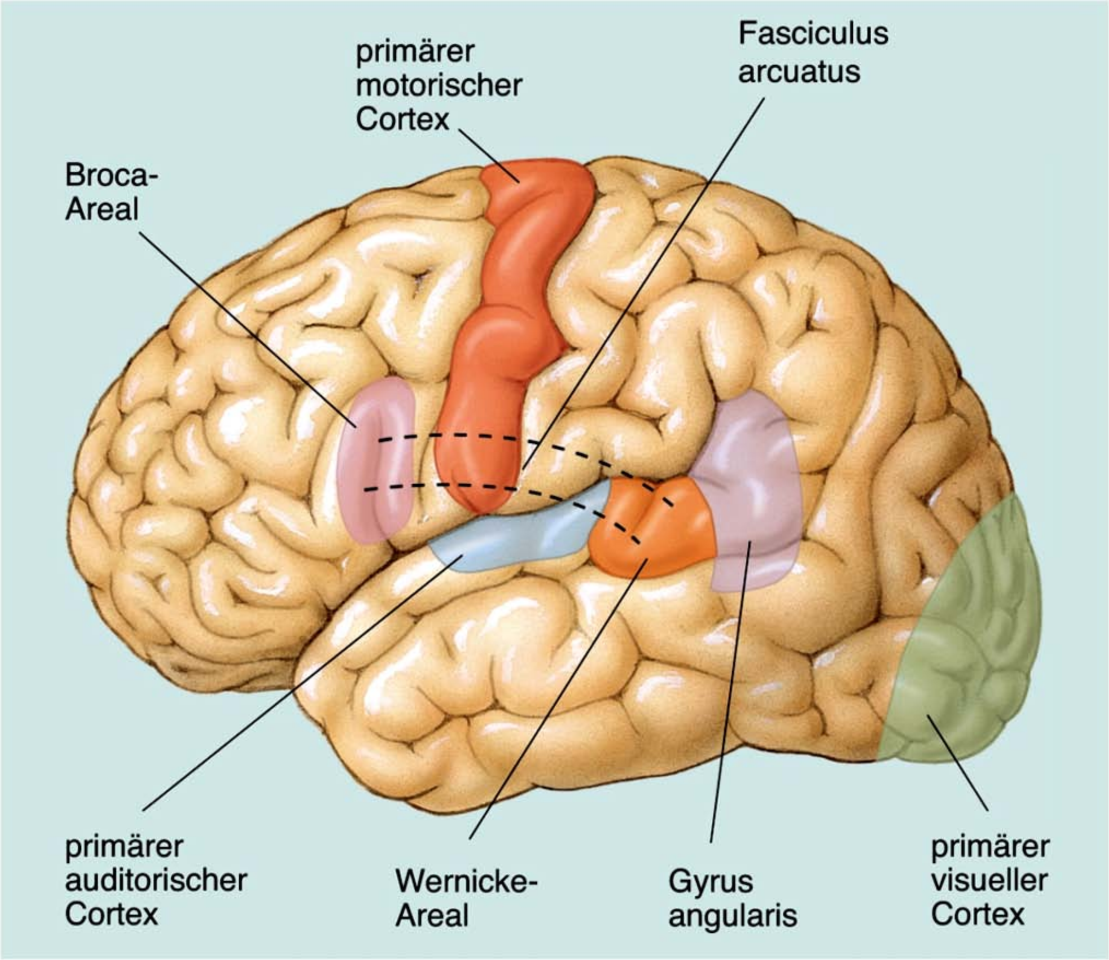
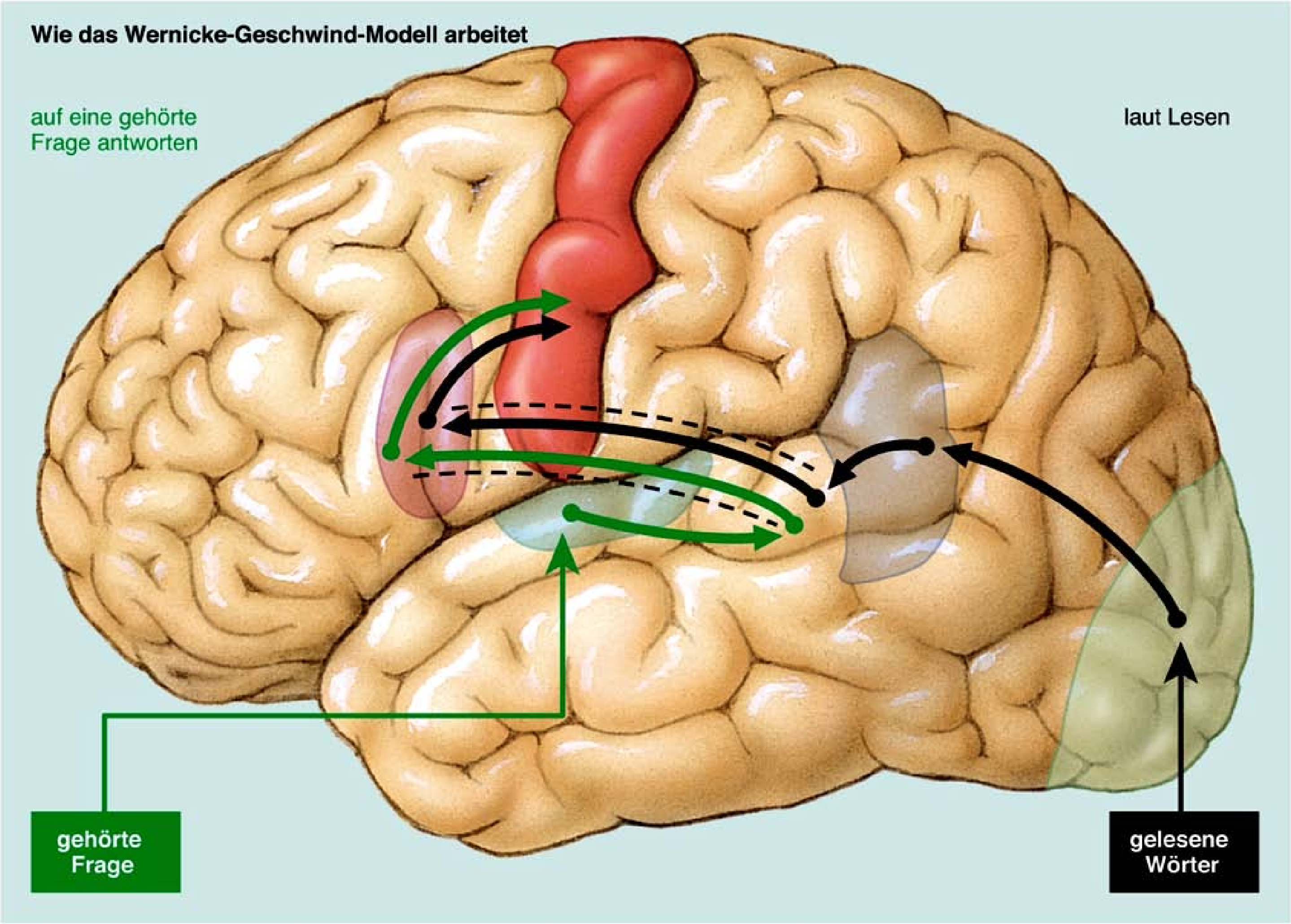


Abbildung 16.9: Die sieben Komponenten des Wernicke-Geschwind-Modells.

Wie funktioniert das Wernicke-Geschwind Modell

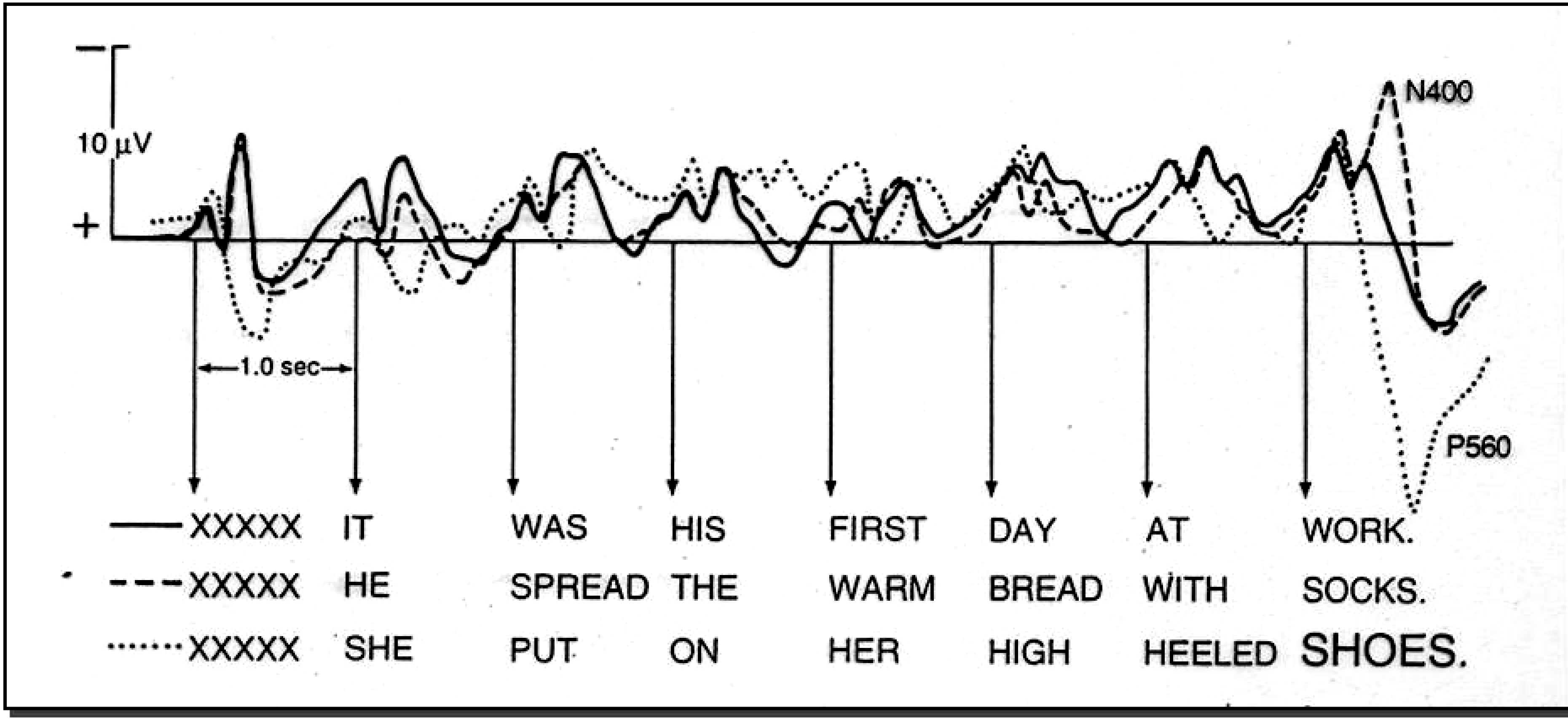


Ereigniskorrelierte Potentiale und Sprachverarbeitung

- ***Semantik:*** *N400*
  - lexikalisch-semantische Integration
- ***Syntax:*** *P350*
  - Disambiguierung
- ***Syntax:*** *P600*
  - Integrationsprozesse, Reanalyse, ‚Repair'

Ereigniskorrelierte Potentiale und Sprachverarbeitung: Die N400

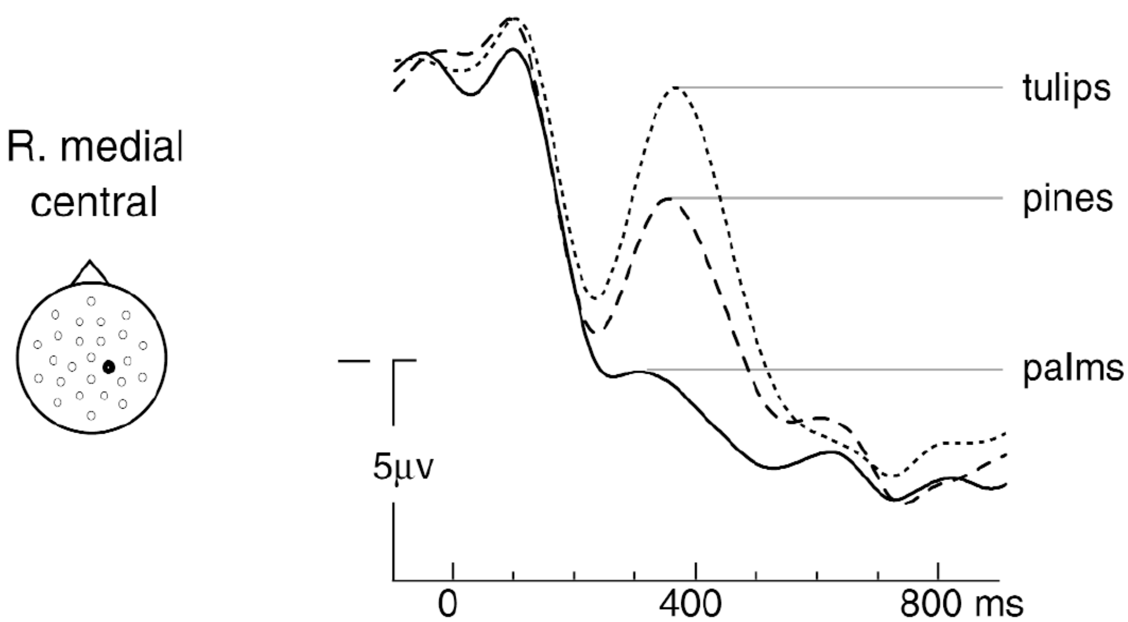
Negativierung auf semantische Abweichungen um etwa 400 ms



Ereigniskorrelierte Potentiale und Sprachverarbeitung: Die N400

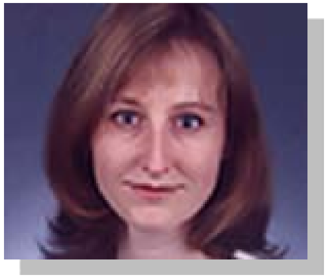
... reflektiert Einfluss semantischen Wissens

‘They wanted to make the hotel look more like a tropical resort.
So along the driveway they planted rows of ...’



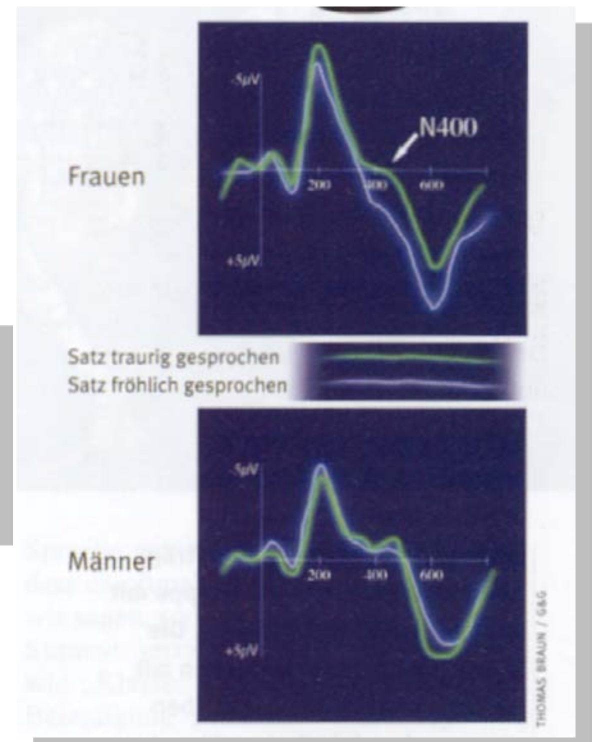
Kutas & Federmeier, 2000

Gender Differences in the Processing of Emotional Prosody



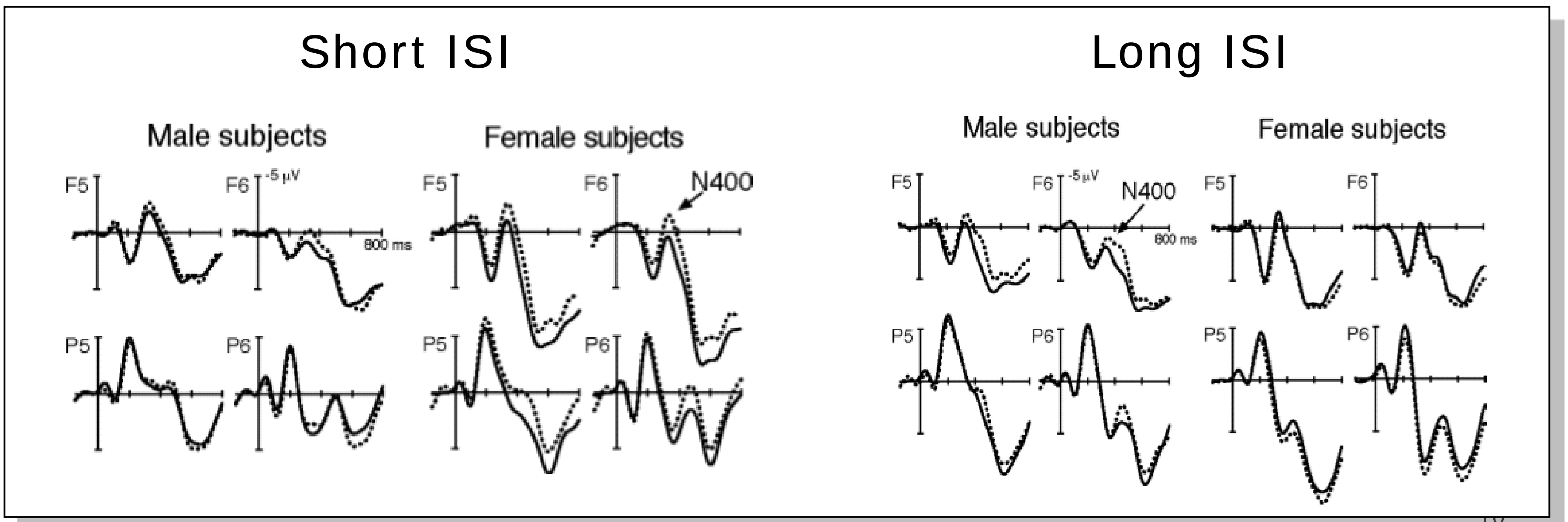
"Men don't really understand !

There are pronounced gender differences in the time course of processing emotional prosody"



Schirmer et al. (2002, 2003)

Gender Differences in the Processing of Emotional Prosody



Schirmer et al. (2002, 2003)

Disambiguierung

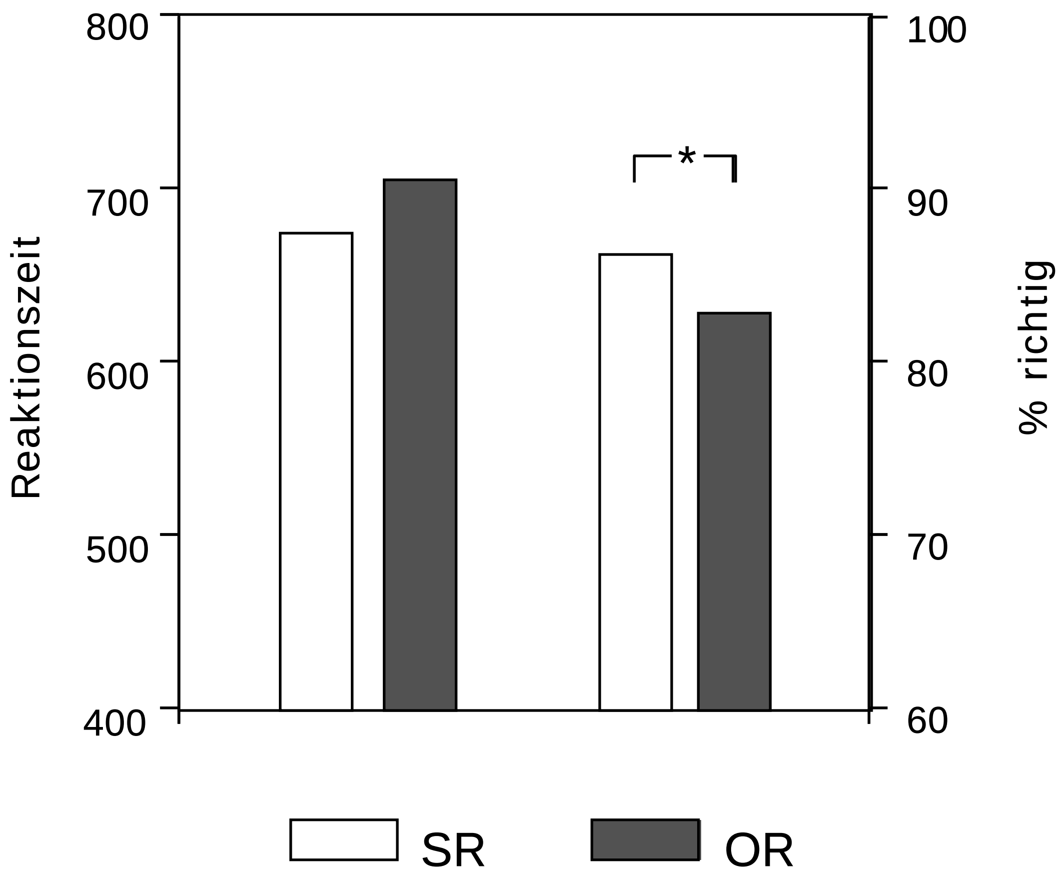
SubjektRelativSatz:

Das sind die Managerinnen, die die Arbeiterin gesehen haben.

ObjektRelativSatz:

Das sind die Managerinnen, die die Arbeiterin gesehen hat.

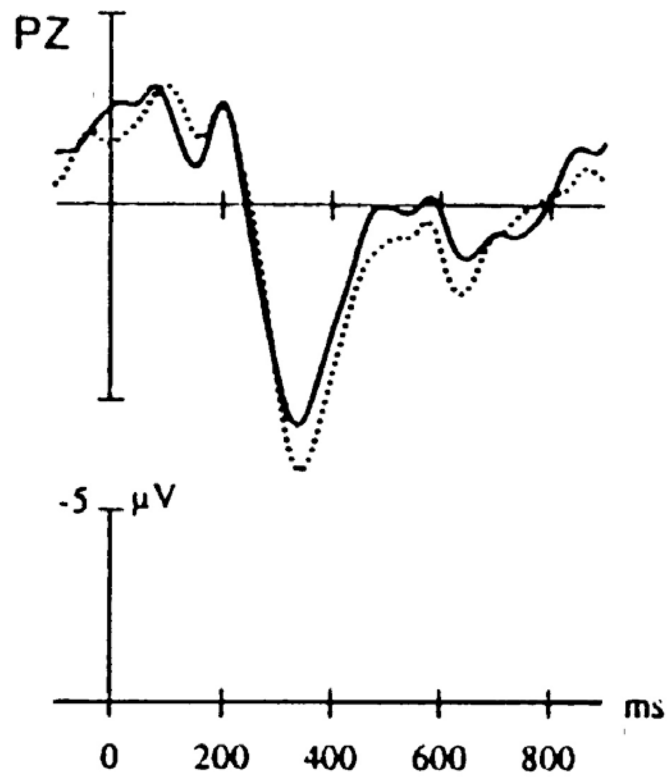
Satzverstehen



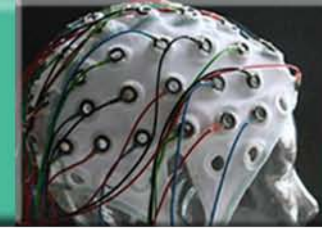
Ereigniskorrelierte Potentiale und Sprachverarbeitung: Die P350

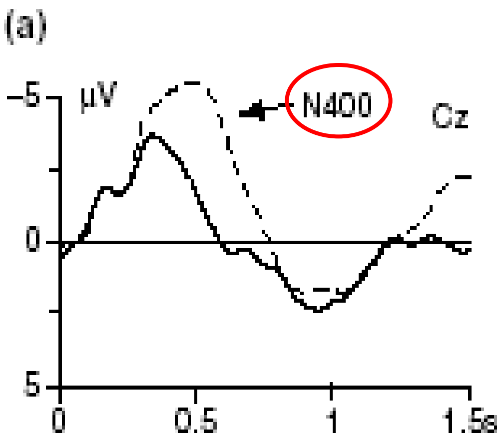
—— *SR: Das sind die Managerinnen, die die Arbeiterin gesehen HABEN.*

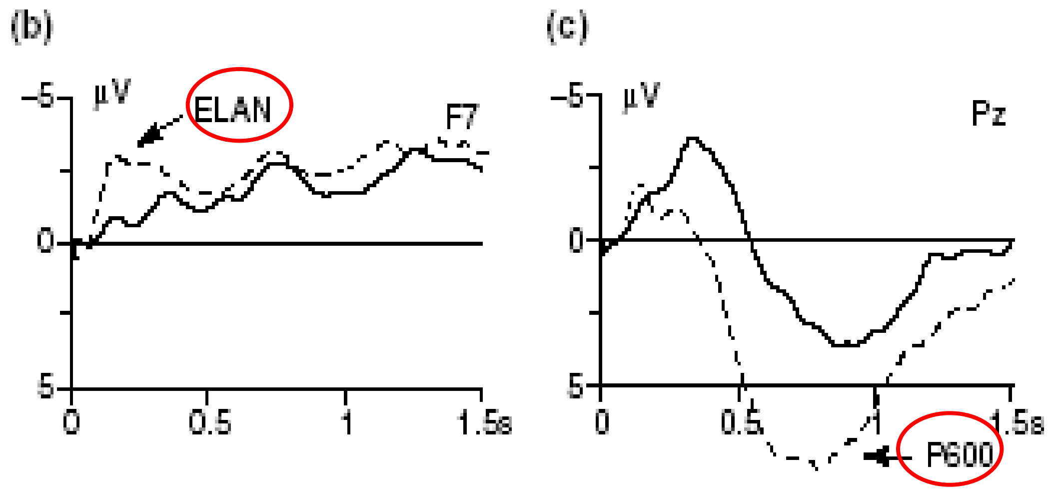
······· *OR: Das sind die Arbeiterinnen, die die Managerin gesehen HAT.*



Ereigniskorrelierte Potentiale und Sprachverarbeitung: Die P600

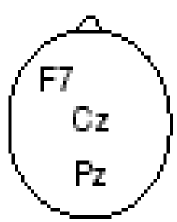






— Das Hemd wurde gebügelt

-- Das Gewitter wurde gebügelt



— Das Hemd wurde gebügelt

-- Die Bluse wurde am gebügelt

Friederici, 2002

Take Home

- Aphasien
  - Brocaaphasie
  - Wernickeaphasie
  - Leitungsaphasie
- Wernicke-Geschwind Modell und seine Kritik
  - Neuropsychologische Ansätze
  - kognitiv neurowissenschaftliche Ansätze
- Kognitiv-neurowissenschaftliche Befunde (EKP-Komponenten)

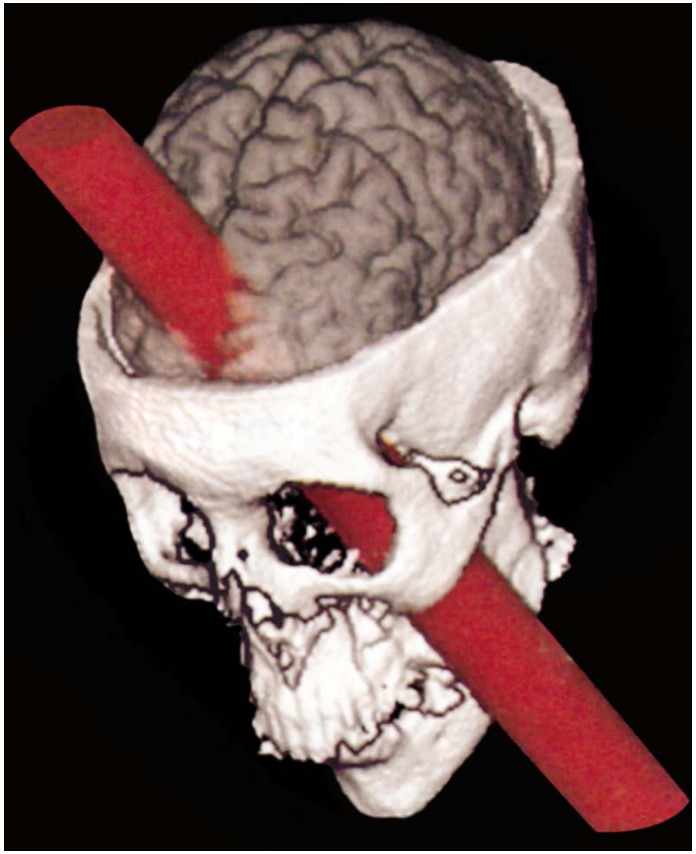
Emotionen

Pinel (6. ed) Kapitel 17

Inhalt

- Emotionstheorien
- Emotionsauslöser
- Ausdruck von Emotionen (emotionale Gesichtsausdrücke)
- Neuronale Grundlagen von Emotionen
- Emotionales Lernen
- Emotionen und Gedächtnis

Phineas Gage



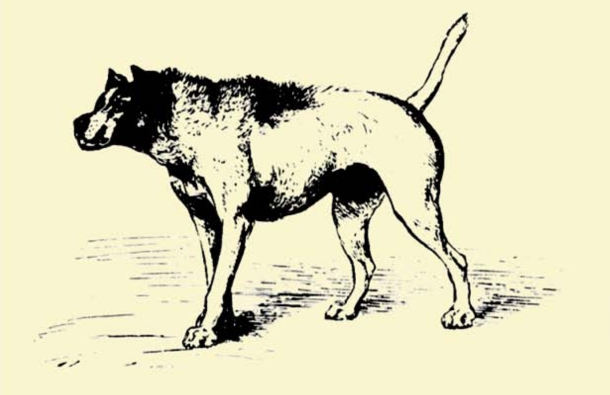
A.R. Damasio (1998). Descartes Irrtum

Darwin (1872)
Evolution von Emotionen

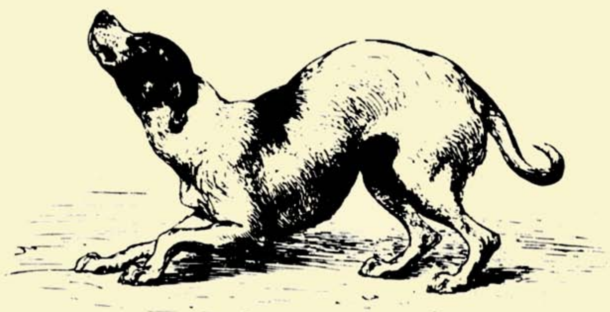
- Emotionen als Ergebnis der Evolution

1. Emotionale Ausdrücke entwickeln sich aus Verhaltensweisen, die zukünftiges Verhalten vorhersagen (z.B. Drohgebärden)
2. Bei entsprechendem Nutzen → Weiterentwicklung der kommunikativen Funktion (evtl. Verlust der ursprünglichen Funktion)
3. Gegensätzliche Verhaltensweisen signalisieren gegensätzliche emotionale Botschaften (Prinzip der Antithese)

Darwin (1872)
Das Prinzip der Antithese



Aggression



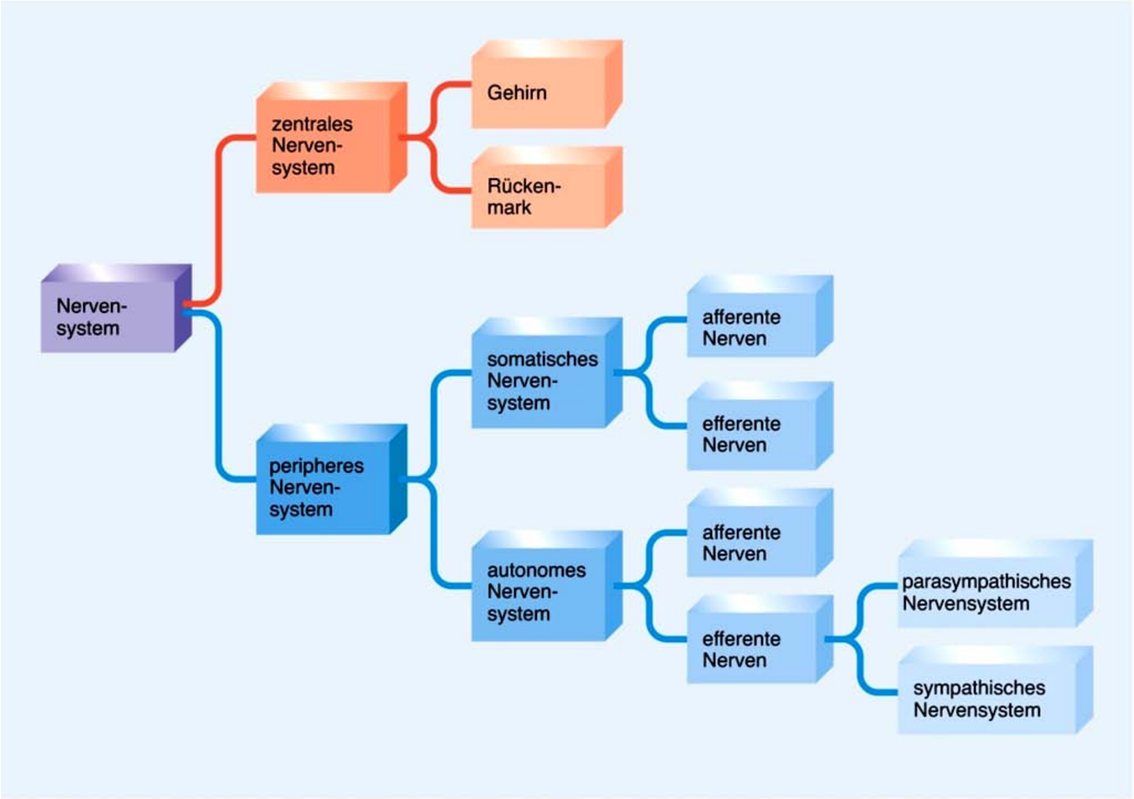
Submission

- **aggressive Haltung**
  vorwärtsgerichtete Ohren,
  aufgestellter Rücken,
  gesträubte Haare ,
  aufgerichteter Schwanz

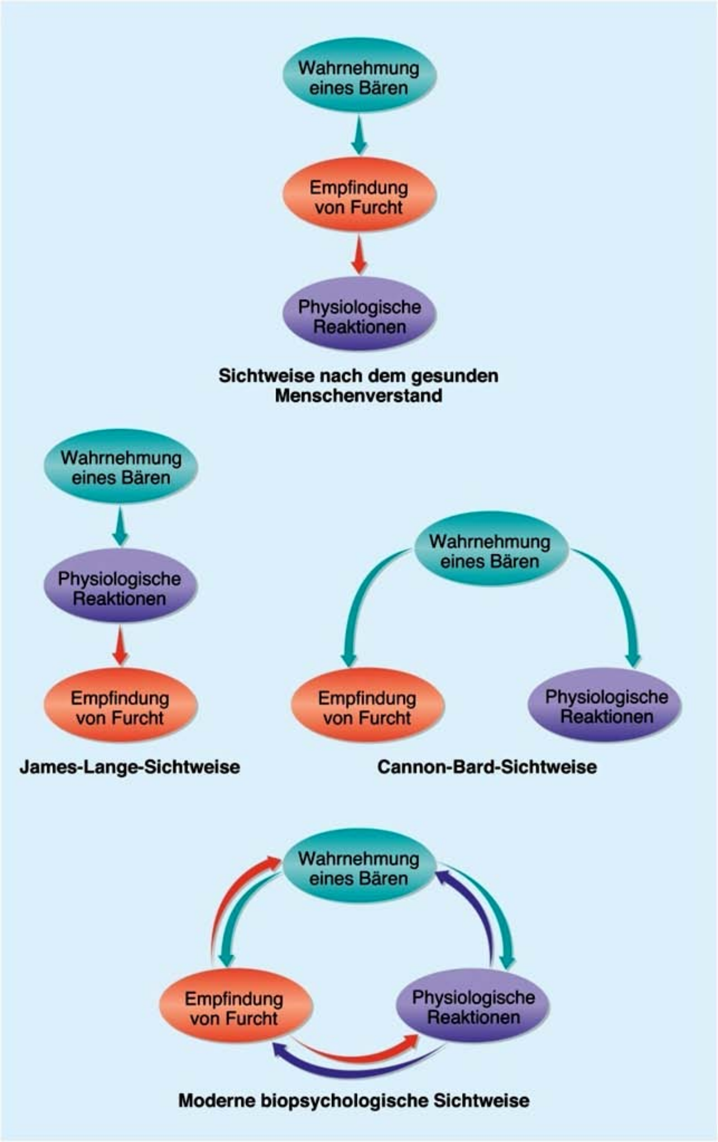
- **submissive Haltung**
  zurückgelegte Ohren,
  durchgedrückter Rücken, angelegte Haare
  nach unten gerichteter Schwanz

James-Lange (1884)
physiologische Emotionstheorie

Emotionen entstehen aus der Wahrnehmung von Reaktionen des somatischen und vegetativen Nervensystems



James-Lange Theorie und Cannon Bard Theorie



Beziehung zwischen Wahrnehmung emotionsauslösender Stimuli,

autonomen und somatischen Reaktionen auf die Stimuli

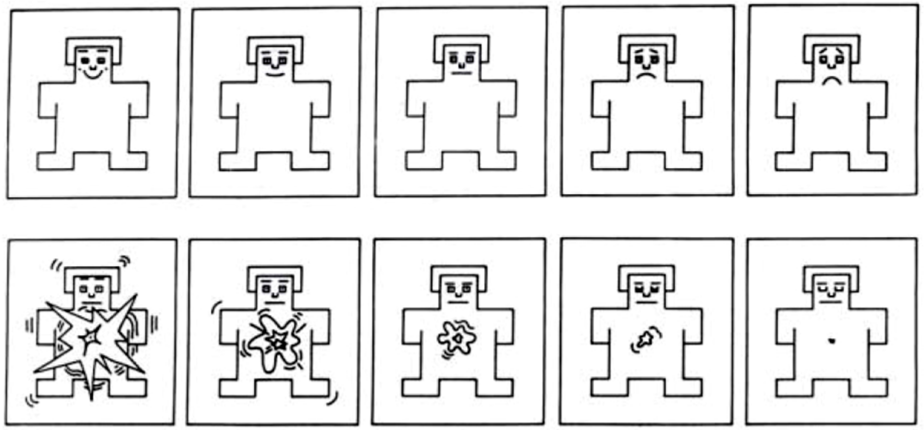
emotionalen Erleben

Techniken zum Auslösen von Emotion

International Affective Picture Scale (IAPS)

Emotionsdimensionen

- Valenz / Arousal (P. Lang & M. Bradeley)

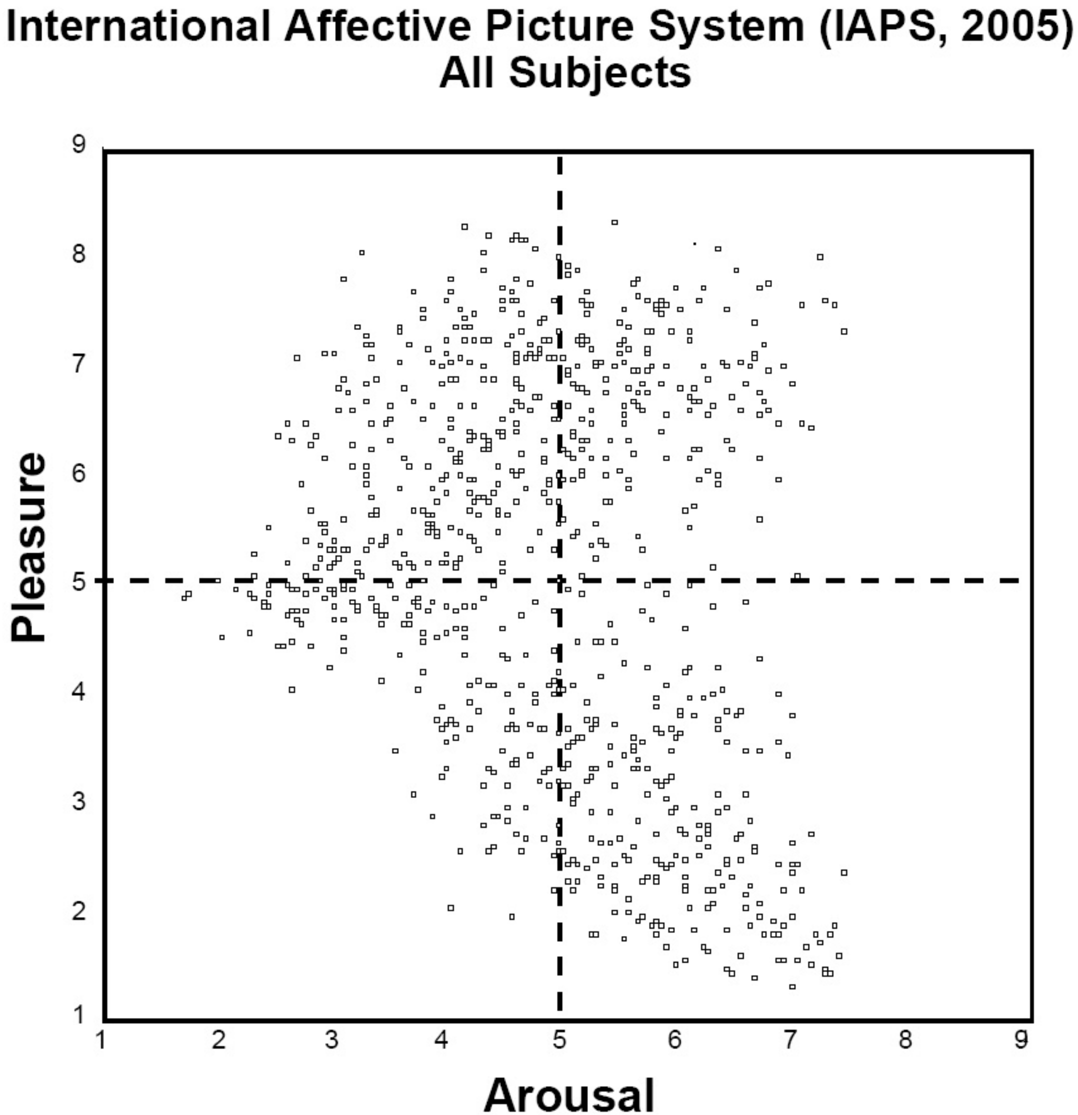


Gib an wie glücklich / erfreut Dich das Bild macht

Gib an wie angeregt / aufgeregt Du Dich durch das Bild fühlst

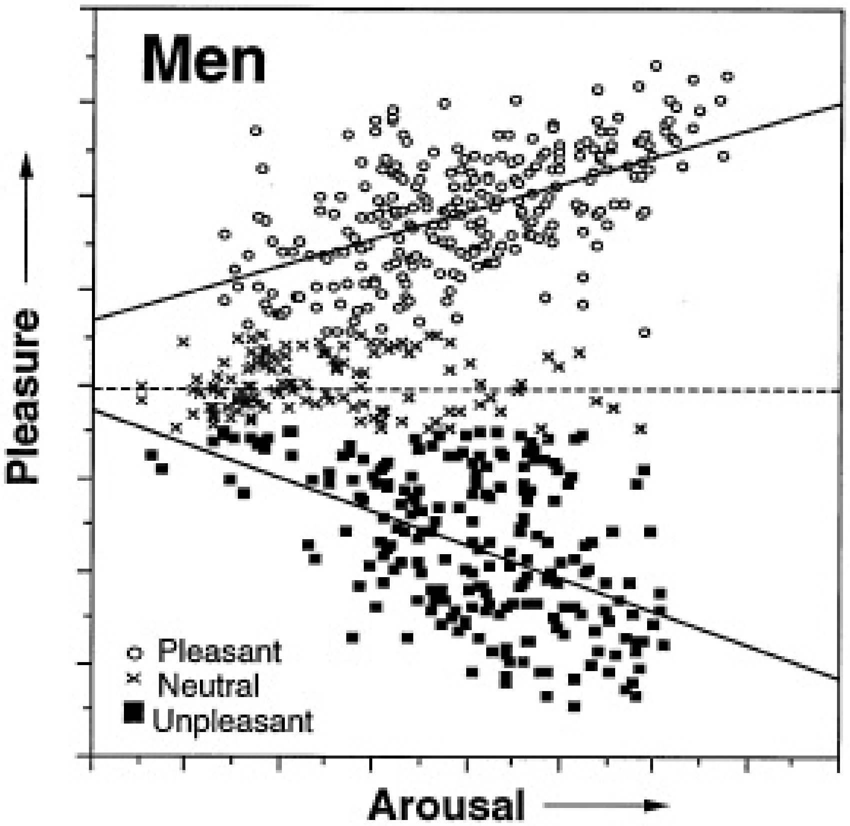
- Approach / Withdraw (R. Davidson)

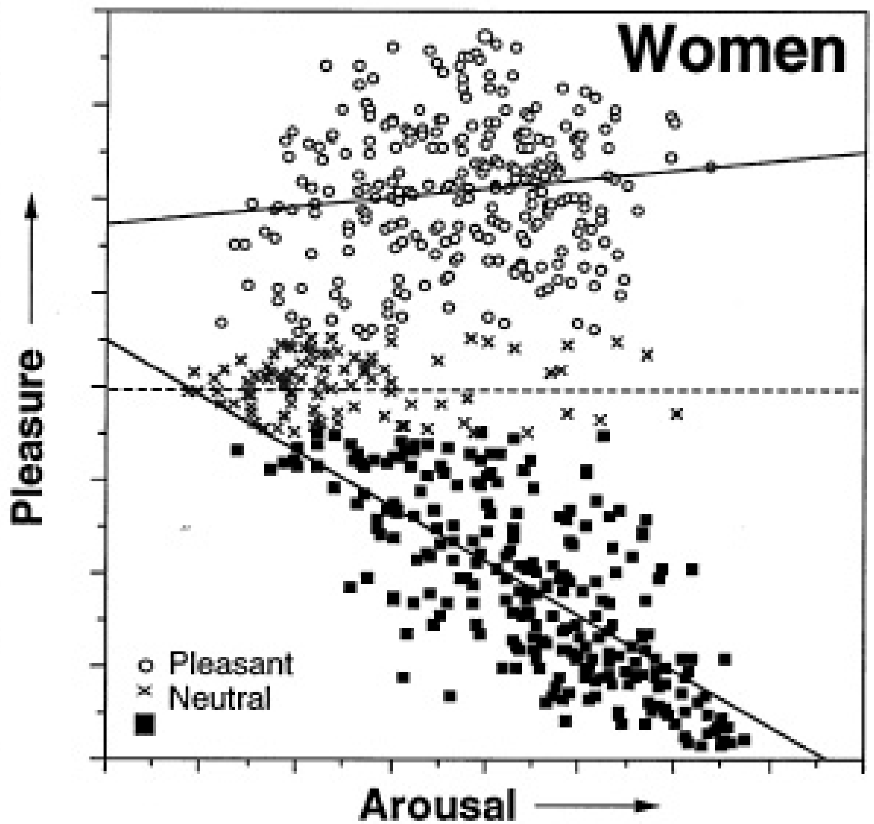
Arousal x Valenz Interaktion



Arousal x Valenz Interaktion

International Affective Picture System (IAPS, 1998; 600 pictures)





Emotionale Gesichtsausdrücke

sechs primäre Gesichtsausdrücke (Ekman und Friesen ,1975)

Überraschung, Wut, Trauer, Ekel, Furcht und Freude

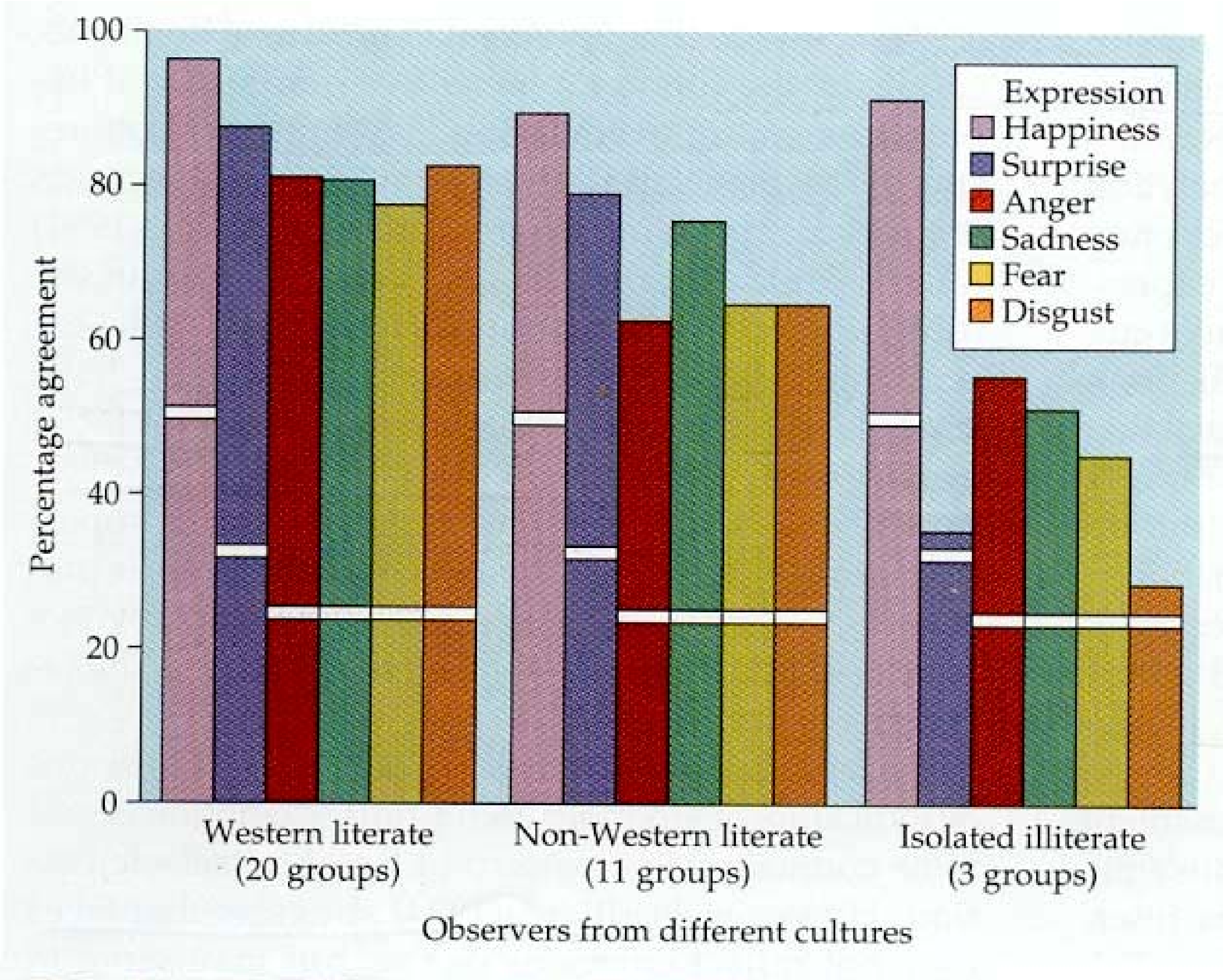
Alle anderen emotionalen Gesichts-ausdrücke wurden als Kombinationen dieser sechs bewertet.

Kombination aus Trauer und Freude

Emotionale Gesichtsausdrücke

Figure 13.2 The six emotional facial expressions Ekman and colleagues found to be universal across cultures. See how well you can pick out the faces showing anger, happiness, disgust, surprise, sadness, and fear. Adapted from Ekman (1973).

Transkulturelle Übereinstimmung bei der Beurteilung emo. Gesichtsausdrücke



Facial-Feedback Hypothese

Wird man besser gelaunt, wenn man ein glückliches Gesicht macht?

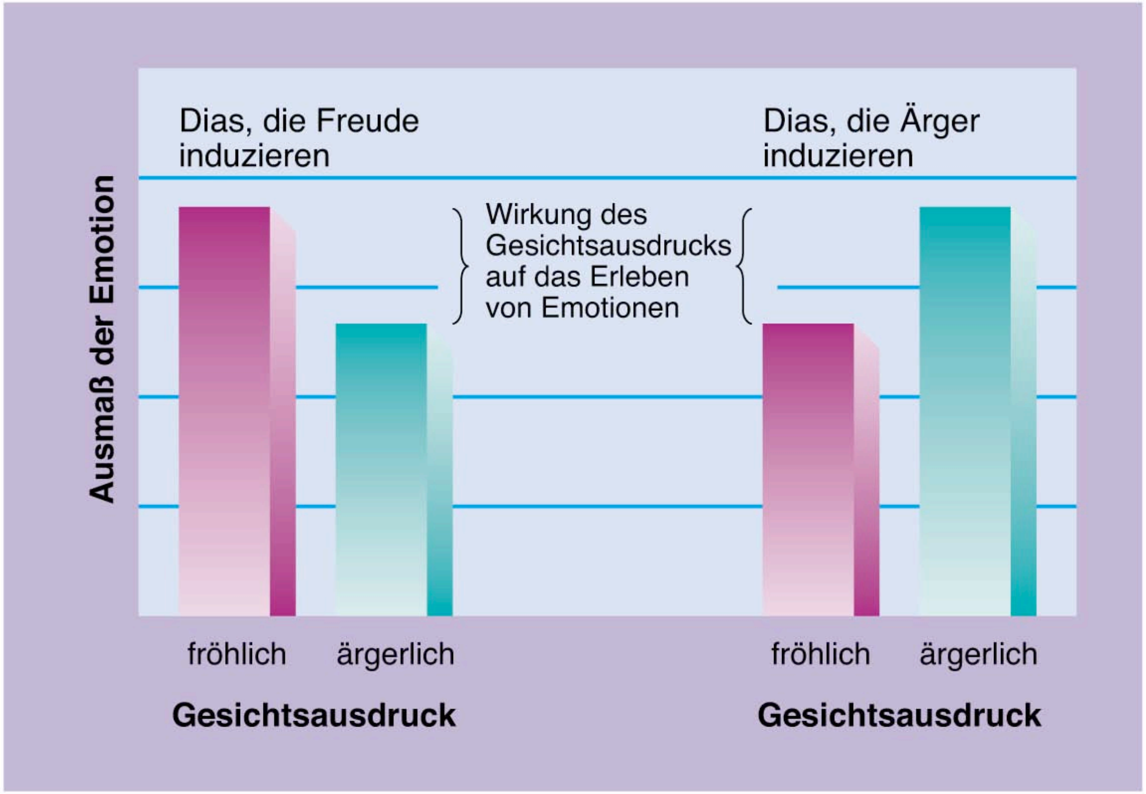
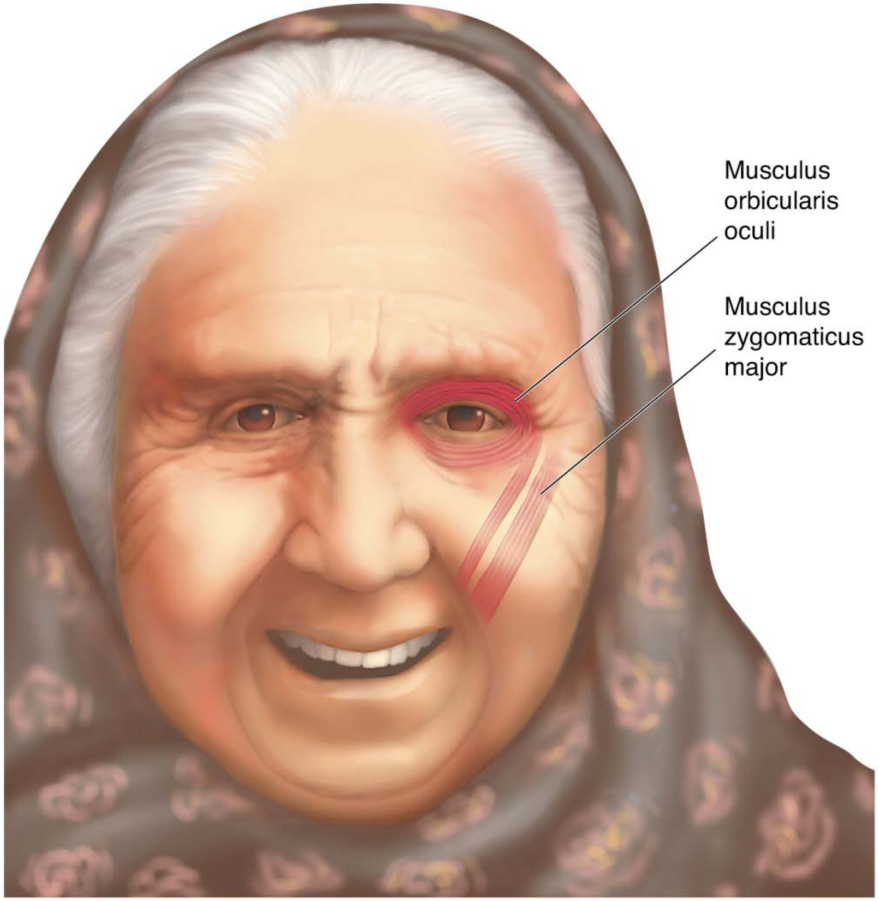


Abbildung 17.6: Die Auswirkungen des Gesichtsausdrucks auf das Erleben von Emotionen. Probanden berichteten, dass sie sich fröhlicher und weniger ärgerlich fühlten, wenn sie während der Betrachtung von Dias ein fröhliches Gesicht machten, und dass sie sich weniger fröhlich und ärgerlicher fühlten, wenn sie Dias betrachteten, während sie ein ärgerliches Gesicht machten (adaptiert nach Rutledge & Hupka, 1985).

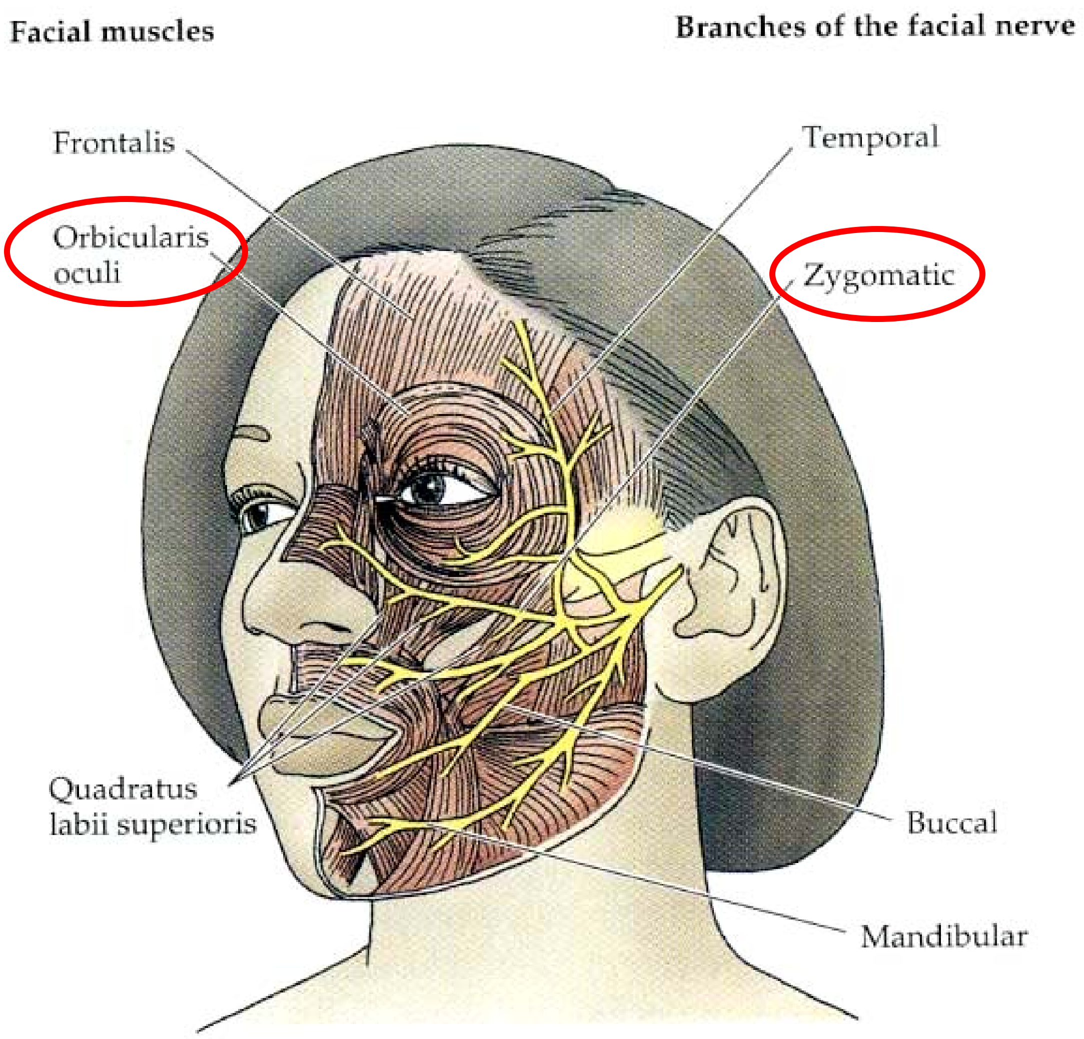
Duchenne Lächeln



Kann man sein wahres Gesicht verbergen?

Abbildung 17.7: Der M. orbicularis oculi und der M. zygomaticus major sind die beiden Muskeln, die sich während eines echten (Duchenne) Lächelns kontrahieren. Weil es für die meisten Menschen schwer ist, den lateralen Anteil des M. orbicularis oculi willkürlich zu kontrahieren, fehlt einem vorgetäuschten Lächeln normalerweise diese Reaktionskomponente.

Gesichtselektromyogramm



15.5 Facial Muscles and Their Neural Control

Neuronale Grundlagen von Emotionen

Das Limbische System / der Papez Kreis

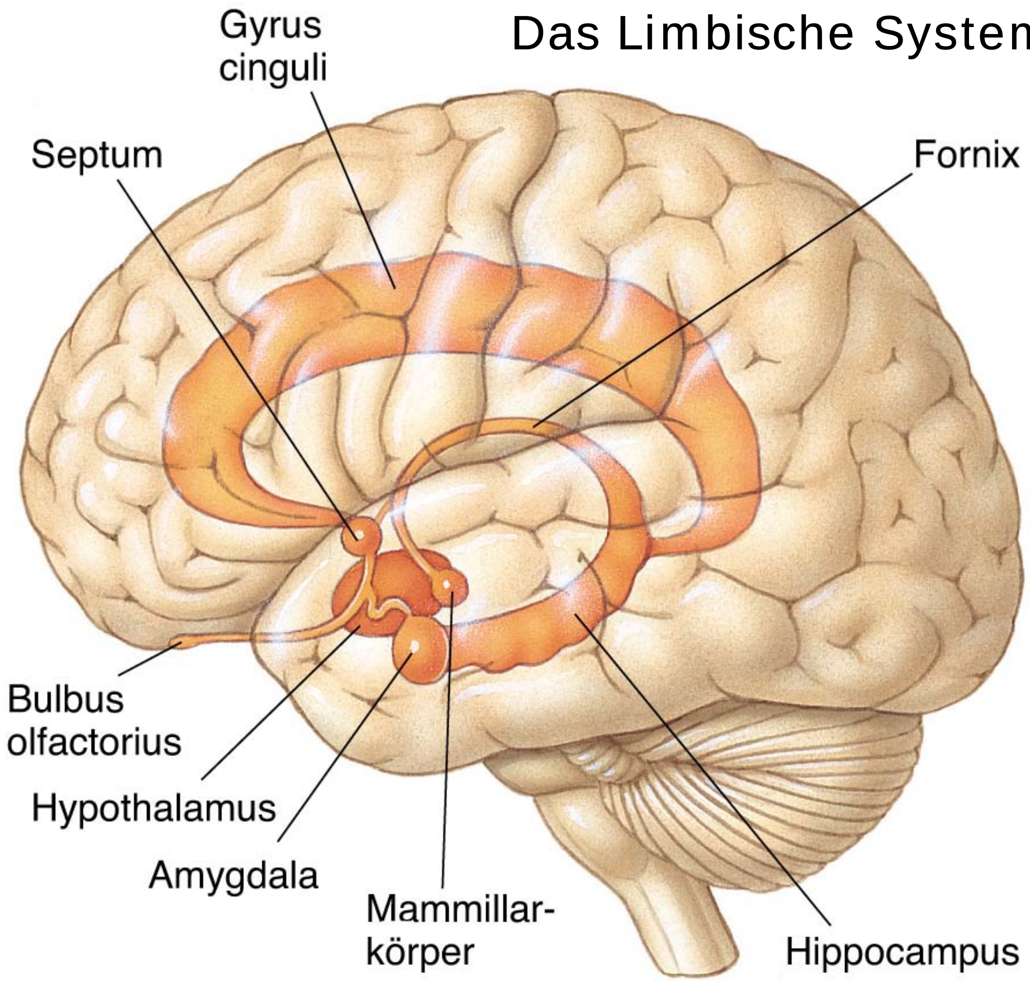
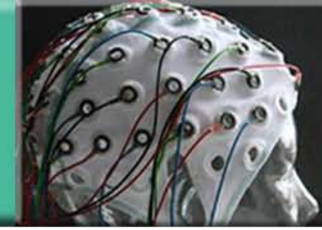


Abbildung 17.4: Die Lage der wichtigsten Strukturen des limbischen Systems. Im Allgemeinen sind sie seitlich der Mittellinie ringförmig um den Thalamus angeordnet (siehe auch Abbildung 3.28).



Zwei Gehirnstrukturen für Emotionsverarbeitung

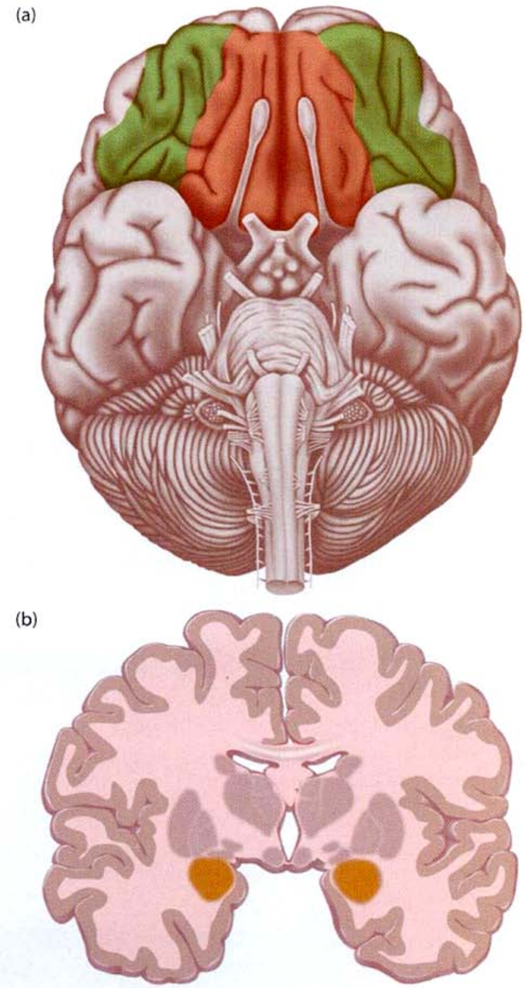


Figure 13.5 **(a)** The human orbitofrontal cortex, which is often divided into the ventromedial prefrontal cortex (red) and the lateral orbitofrontal cortex (green). **(b)** The human amygdala is highlighted in orange. From Davidson et al. (2000).

Der orbitofrontale Cortex und die Amygdala

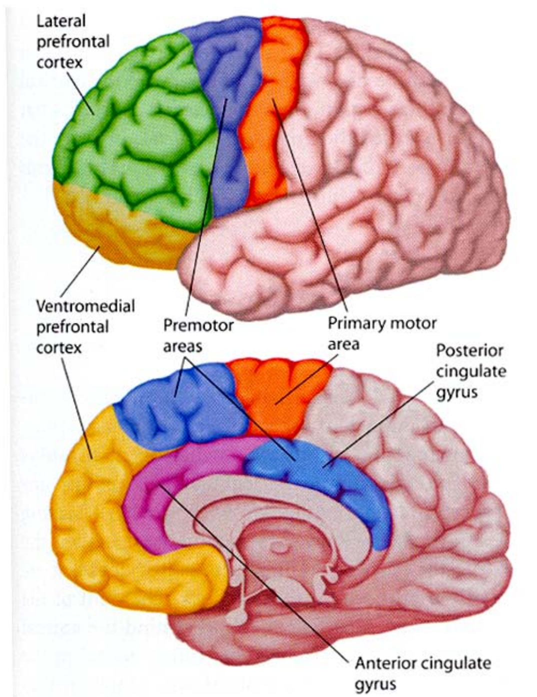
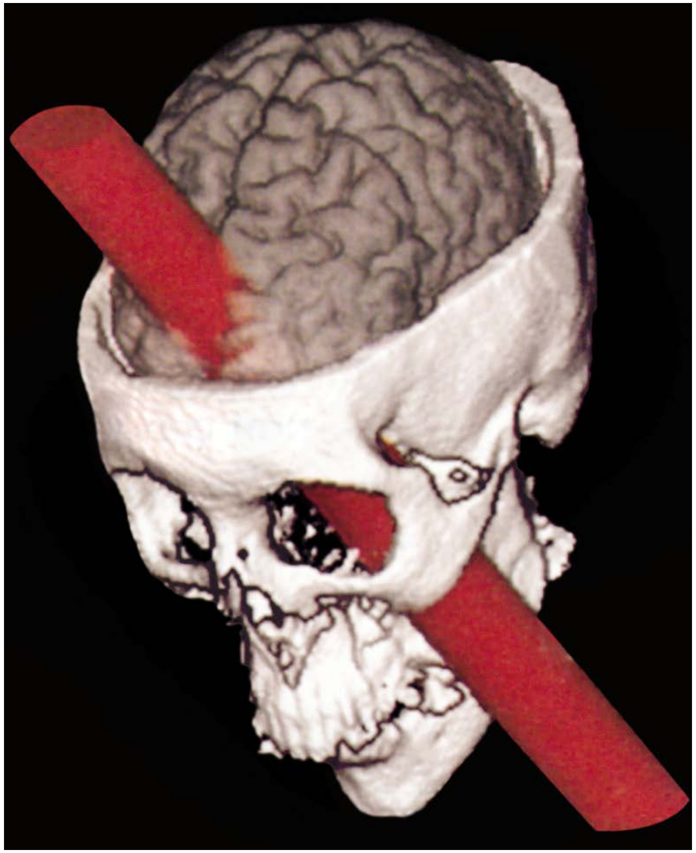


Figure 12.1 The areas of the frontal lobe. The prefrontal cortex includes all of the areas in front of the primary and secondary motor regions. The three major subdivisions of prefrontal cortex are the lateral prefrontal, ventromedial prefrontal, and the anterior cingulate cortex.

Phineas Gage



Die Rolle des orbitofrontalen Cortex

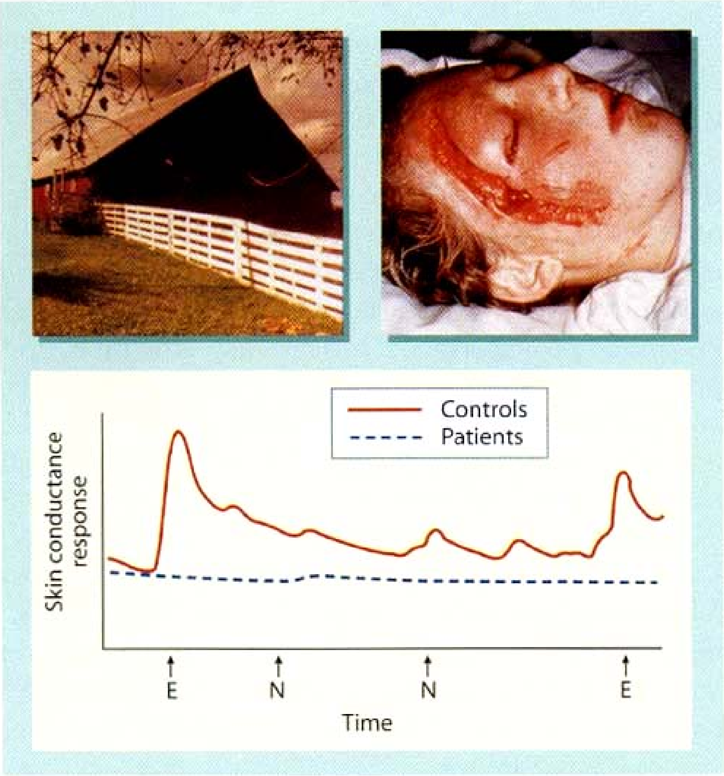
Imitative and utilization behaviors

Figure 13.7 Imitative and utilization behaviors are two signs of prefrontal damage, usually associated with lesions in the ventromedial region. **(a–d)** Imitative behaviors. The patient mimicks the physician making a threatening gesture **(a)**, putting on spectacles **(b)**, smelling a flower **(c)**, and praying **(d)**. **(e–g)** Utilization behaviors. When objects are placed in front of him, the patient puts on three pairs of glasses **(e)** or proceeds to use the makeshift urinal **(f, g)**.

Die Rolle des orbitofrontalen Cortex

emotionale Bewertung

Figure 13.8 Patients with ventromedial cortical damage fail to show autonomic, emotional responses to arousing stimuli. Subjects were shown a series of stimuli while measurements were made of their skin conductance response (SCR), a measure of emotional responsivity. Some of the stimuli were affectively neutral (N) such as photographs of the Iowa countryside. Others were expected to evoke strong emotional responses (E). The control subjects showed a large SCR to the emotional stimuli, whereas the prefrontal lesion patients had a "flat" SCR. Bottom panel is adapted from Damasio (1994).



Entscheidungsverhalten nach orbitalen PFC Läsionen

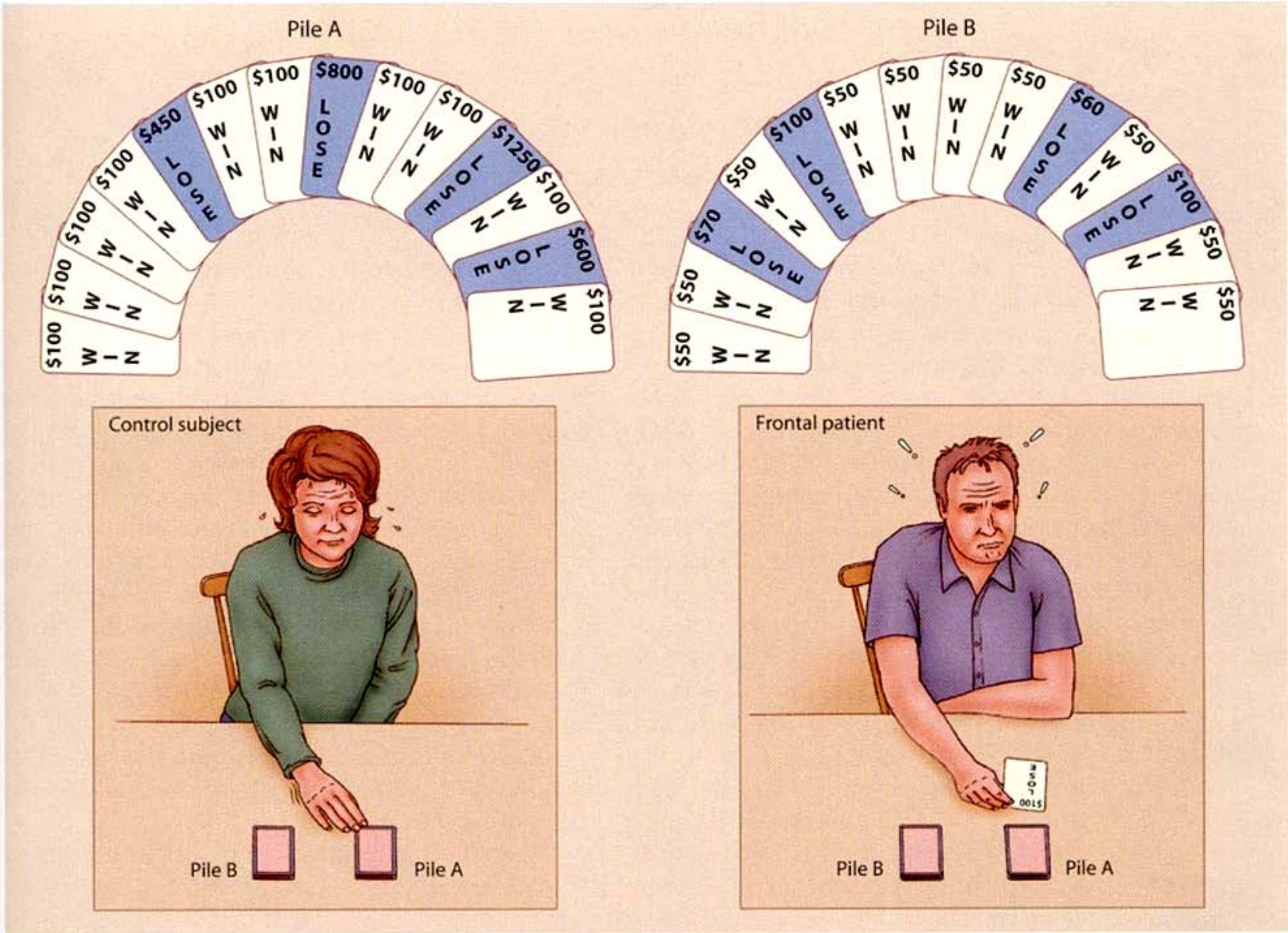


Figure 13.9 Emotional responses occur in reaction to stimuli but also are useful in guiding our decision processes. Subjects were required to choose cards from one pile or the other, with each card specifying an amount won or lost. Through trial and error, the subjects could learn that pile A was riskier than pile B. Control subjects not only tended to avoid the high-risk pile but also showed a large SCR when considering choosing a card from this pile. The patients with prefrontal lesions failed to show these anticipatory SCRs. Interestingly, they did show a large SCR upon turning over a card and discovering they had lost $1000 (of play money).

Orbitaler PFC

… reguliert die Fähigkeit zur Inhibition, Evaluation und zum Prozessieren sozialer und emotionaler Information.

Zwei Gehirnstrukturen für Emotionsverarbeitung

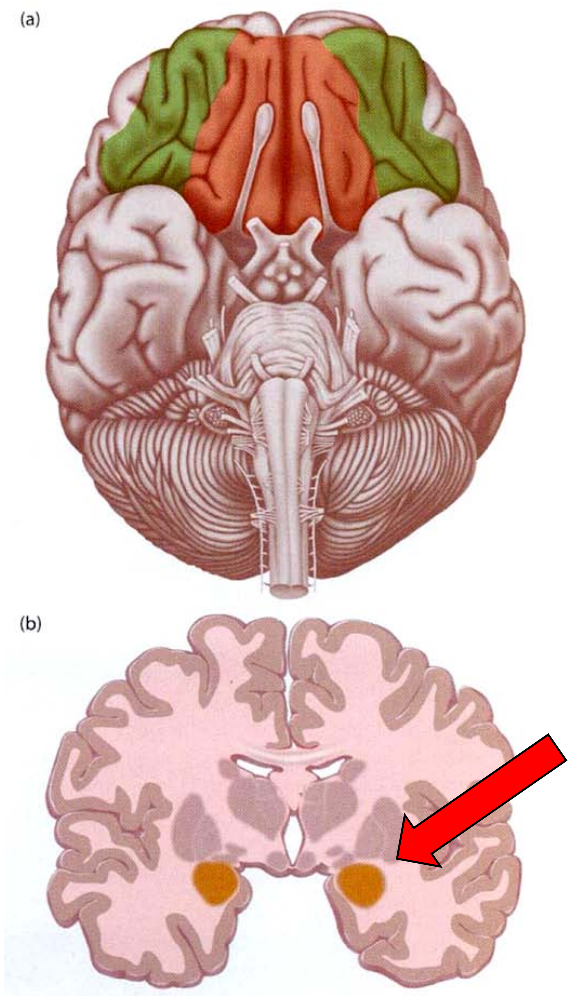


Figure 13.5 **(a)** The human orbitofrontal cortex, which is often divided into the ventromedial prefrontal cortex (red) and the lateral orbitofrontal cortex (green). **(b)** The human amygdala is highlighted in orange. From Davidson et al. (2000).

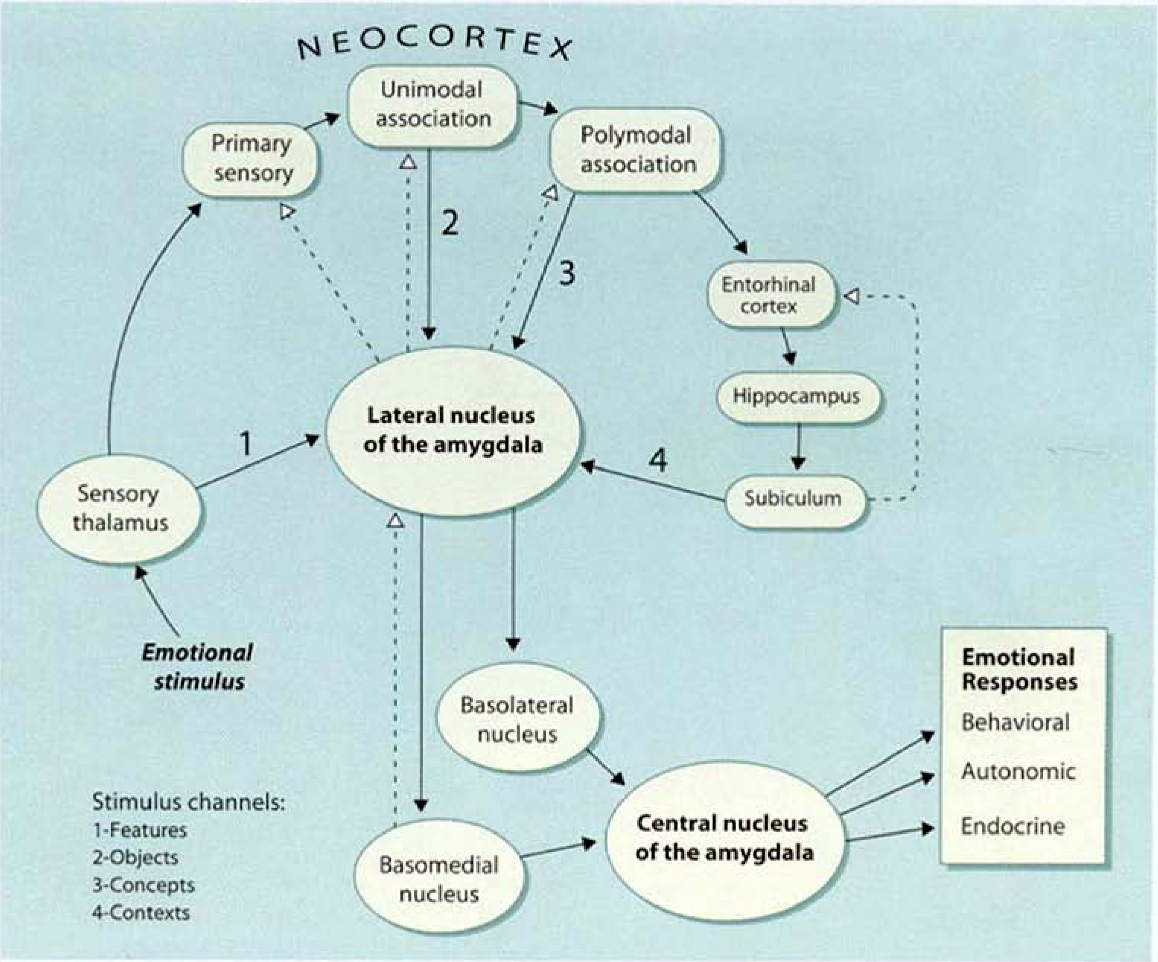
Der orbitofrontale Cortex und die Amygdala

Das Klüver-Bucy Syndrom (1939)

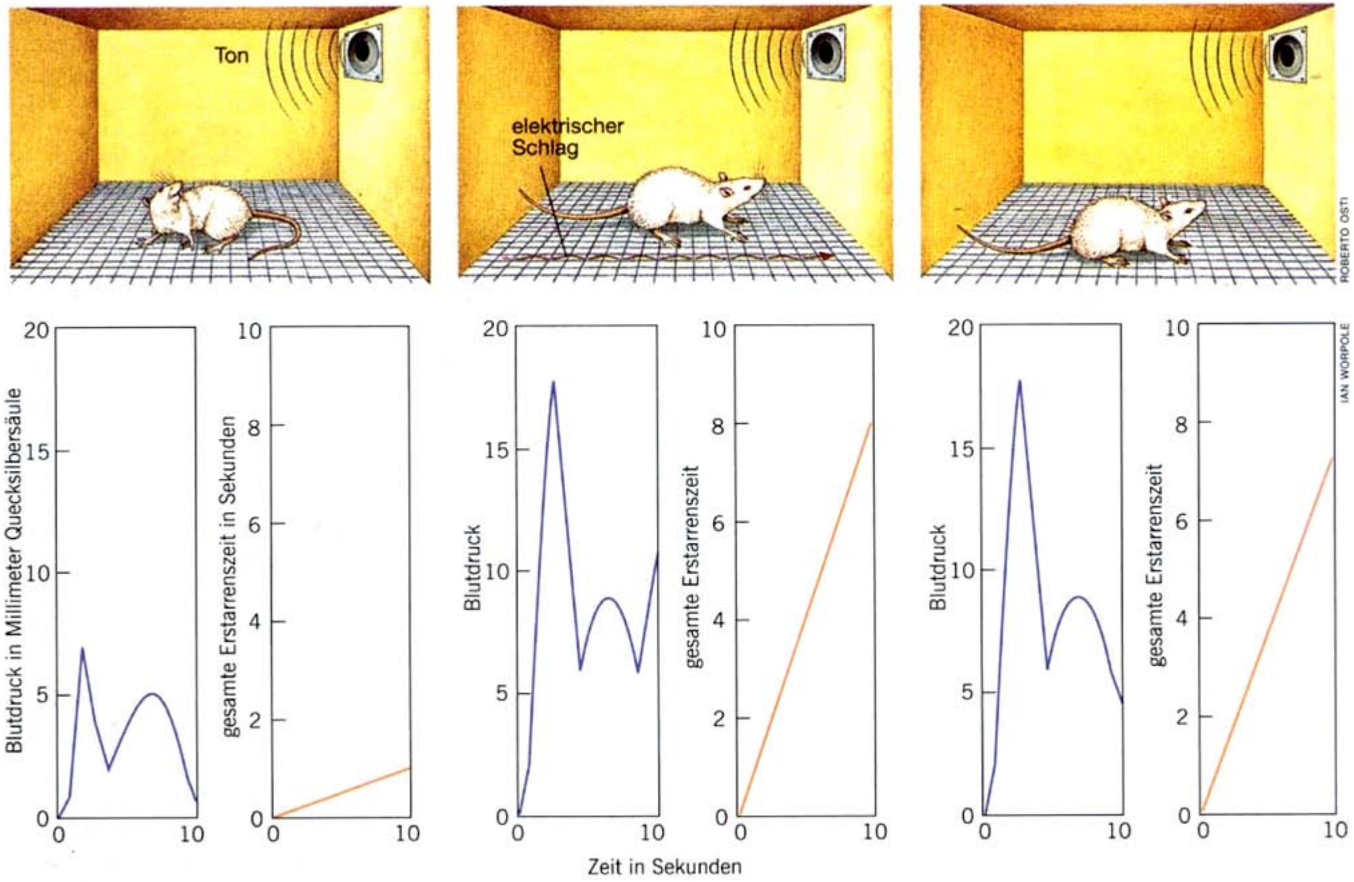
- Entfernung des anterioren Temporallappens bei Affen einschließlich der Amygdala
- Gesteigerte sexuelle Aktivität mit unpassenden Objekten
- Neigung bekannte Objekte wiederholt zu untersuchen
- Mangel an Furcht (z.B. vor Schlangen)
  → zahme Tiere

Die Amygdala

Figure 13.11 Amygdala pathways and fear conditioning. Adapted from Le Doux (1995).

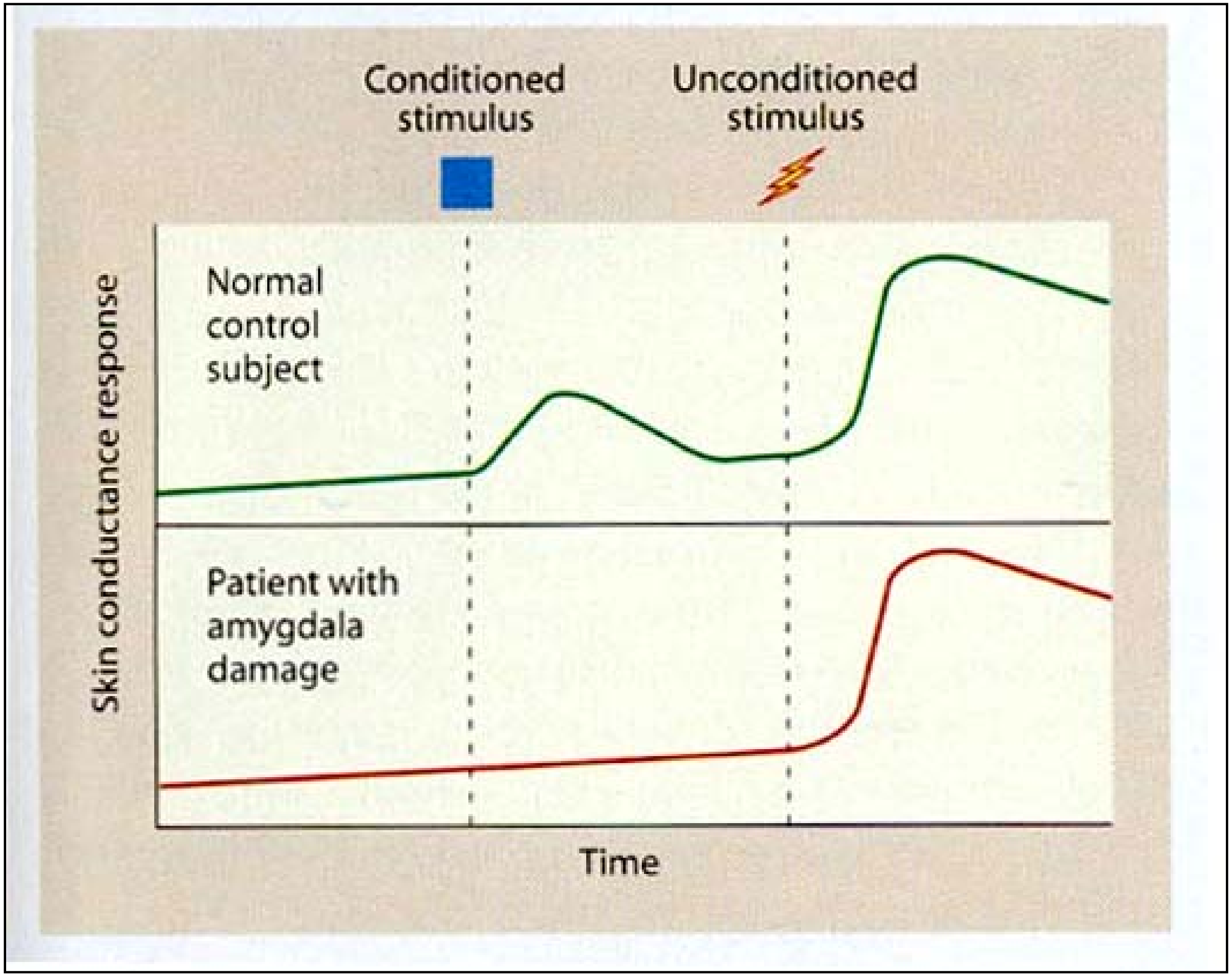


Emotionales Lernen: Angstkonditionierung

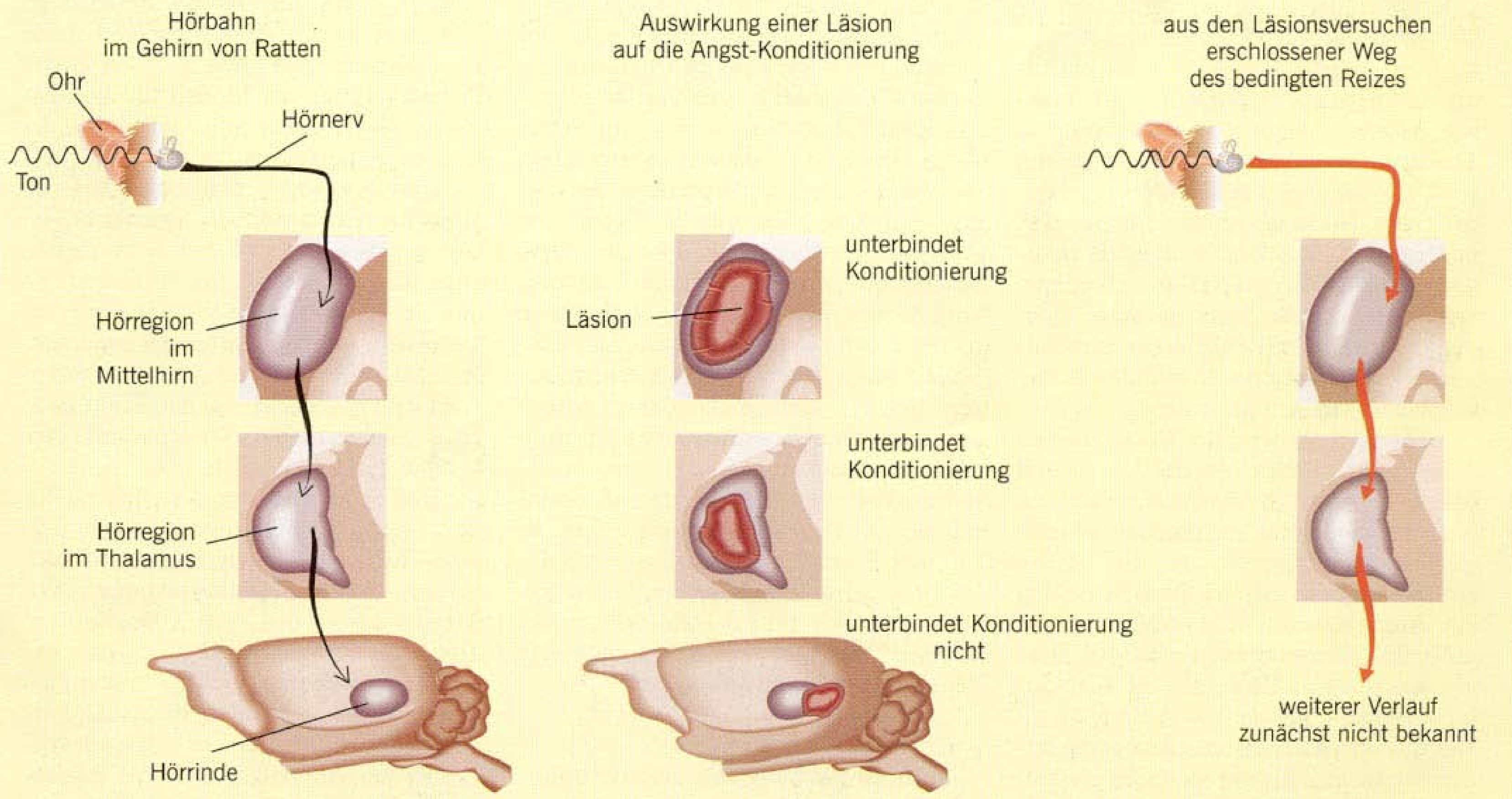


Bei der Angst-Konditionierung in den Experimenten des Autors erhielt eine Ratte über das Bodengitter des Käfigs einen schwachen Stromschlag, während sie einen Ton hörte. So wird der Ton zum bedingten oder konditionierten Reiz, bei dem allein sie später Angstreaktionen zeigt. Bevor das Tier den Zusammenhang gelernt hat, geschieht dies nicht – der Blutdruck steigt wenig, und das Tier verharrt kaum (links). Erst wenn es zugleich den Schmerzreiz spürt, zeigt es eine deutliche physiologische Reaktion und erstarrt (Mitte). Nachdem ihm dies mehrmals widerfahren ist, tritt die Reaktion schon beim Ton allein auf (rechts).

Auswirkung der Amygdalektomie auf Angstkonditionierung

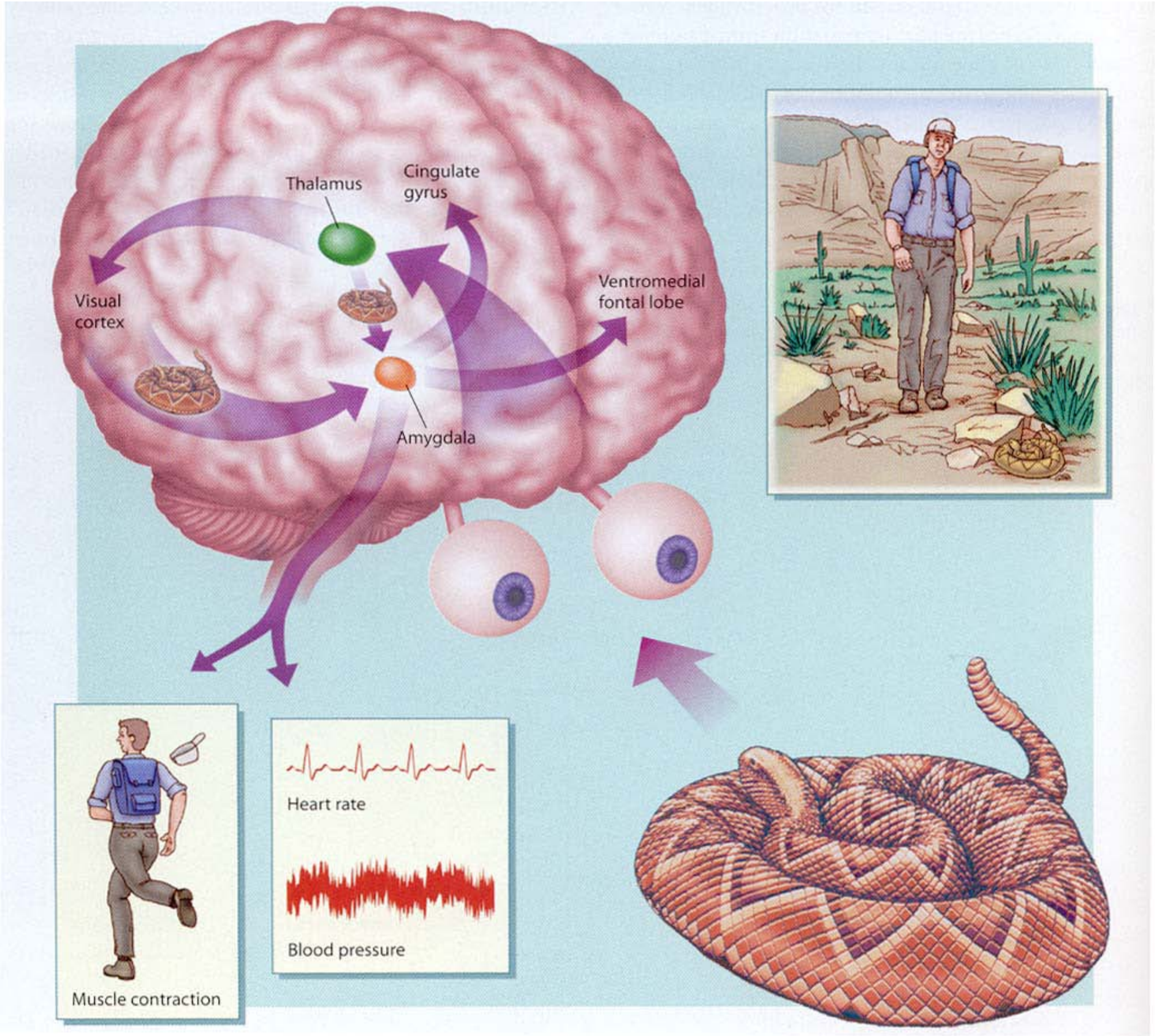


Angstkonditionierung



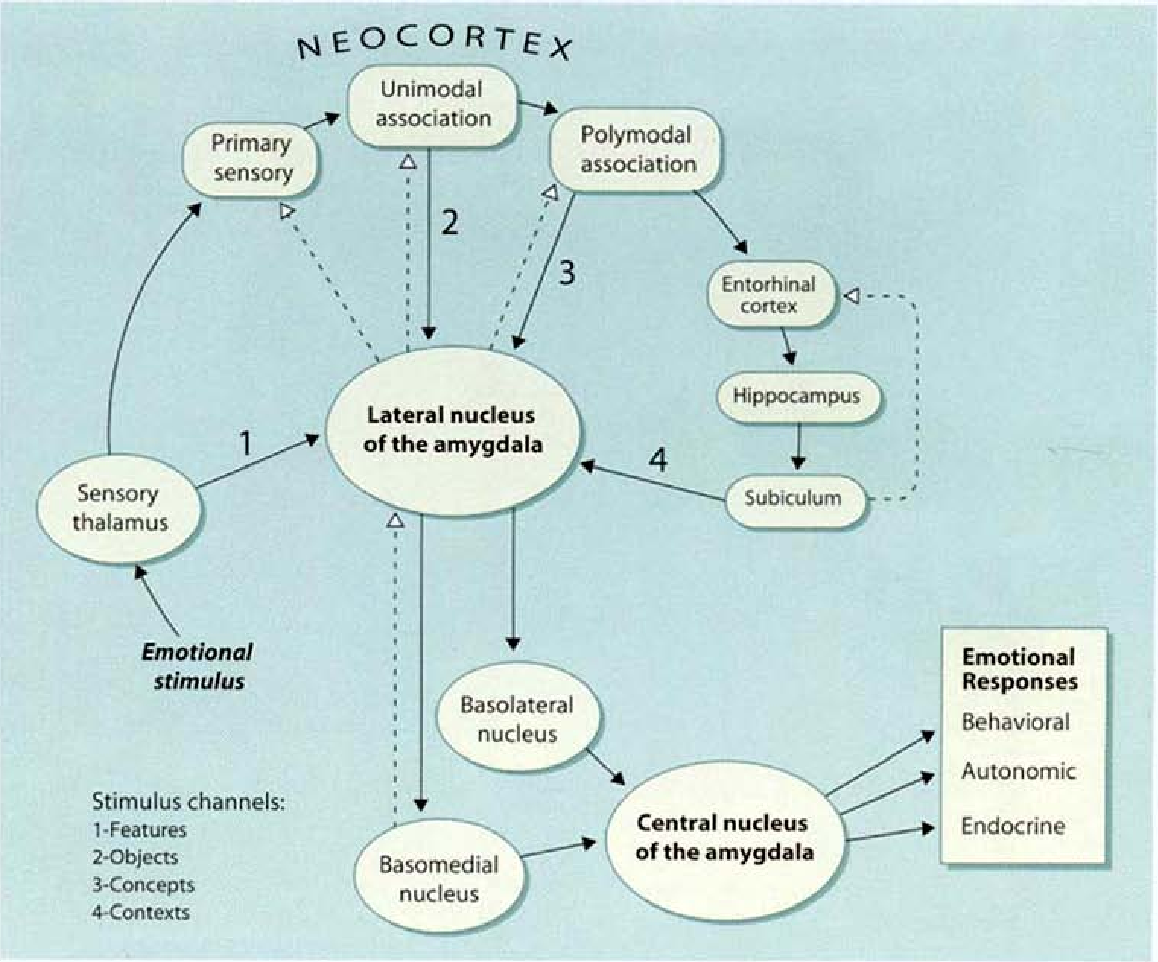
Durch gezielte Hirnläsionen ließ sich eingrenzen, welche Strukturen und Verbindungen bei der Angst-Konditionierung mitspielen. Die Hörbahn verläuft vom Ohr über Mittelhirn und Thalamus zur Hirnrinde (links). Sowohl die Zerstörung der Hörregion im Mittelhirn als auch die derjenigen im Thalamus unterbindet die bedingte Angstreaktion; eine Läsion der Hörrinde tut dies nicht (Mitte). Demnach müssen noch andere Gehirnstrukturen bei dieser Form der Gedächtnisbildung mitwirken (rechts).

Low road / high road



Die Amygdala

Figure 13.11 Amygdala pathways and fear conditioning. Adapted from Le Doux (1995).



Emotionsgedächtnis vs. deklaratives Gedächtnis

- Doppelte Dissoziation nach Amygdala und Hippocampusläsionen
- Amygdalaläsion: keine SCR (kein emotionales Lernen) aber intaktes deklaratives G
- Hippocampusläsion: intakte SCR (emotionales Lernen) / aber kein deklaratives G

Emotionsgedächtnis vs deklaratives Gedächtnis

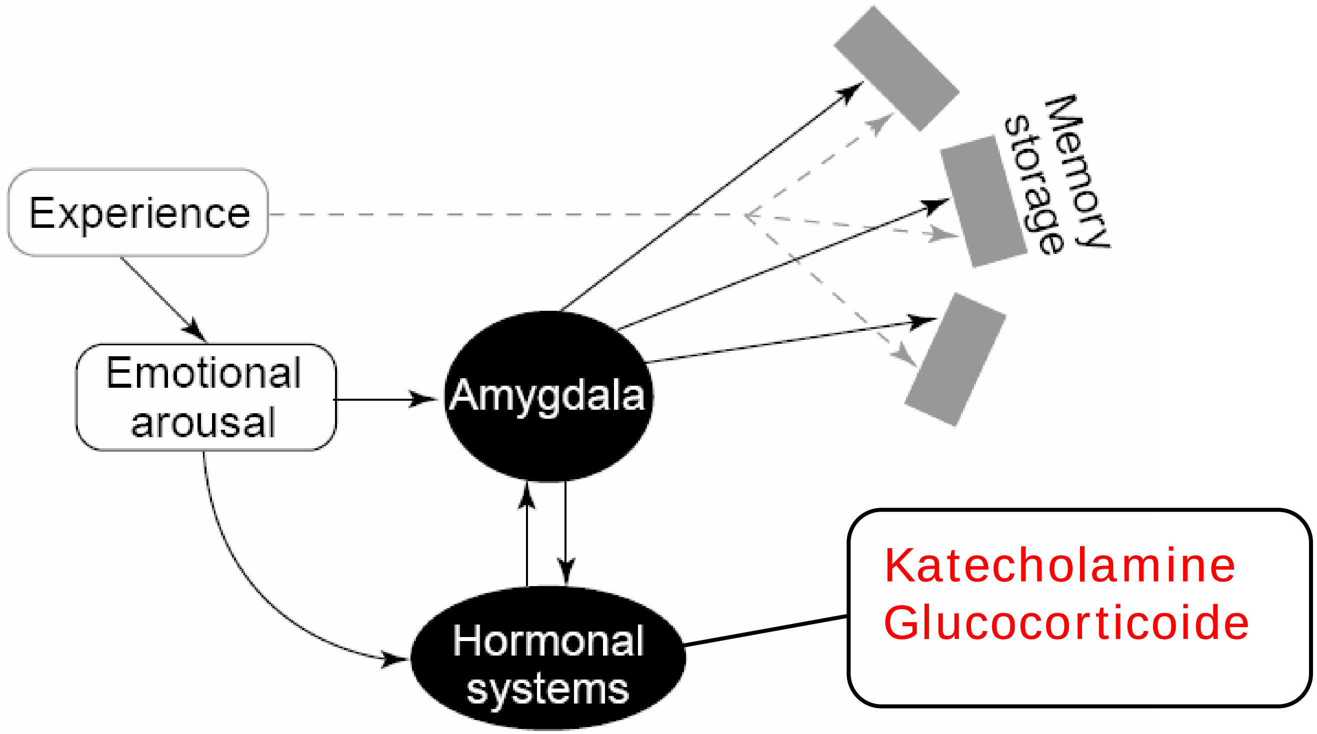


Fig. 5. ***Hypothetical memory-modulatory mechanism for emotionally arousing events.*** *Experiences can be stored in various brain regions with little or no involvement of either stress-hormone activation or the amygdaloid complex (AC). During periods of emotional arousal, stress-hormone systems interact with the AC to modulate memory-storage processes occurring in other brain regions.*

Die Amygdala und Gedächtnis-konsolidierung

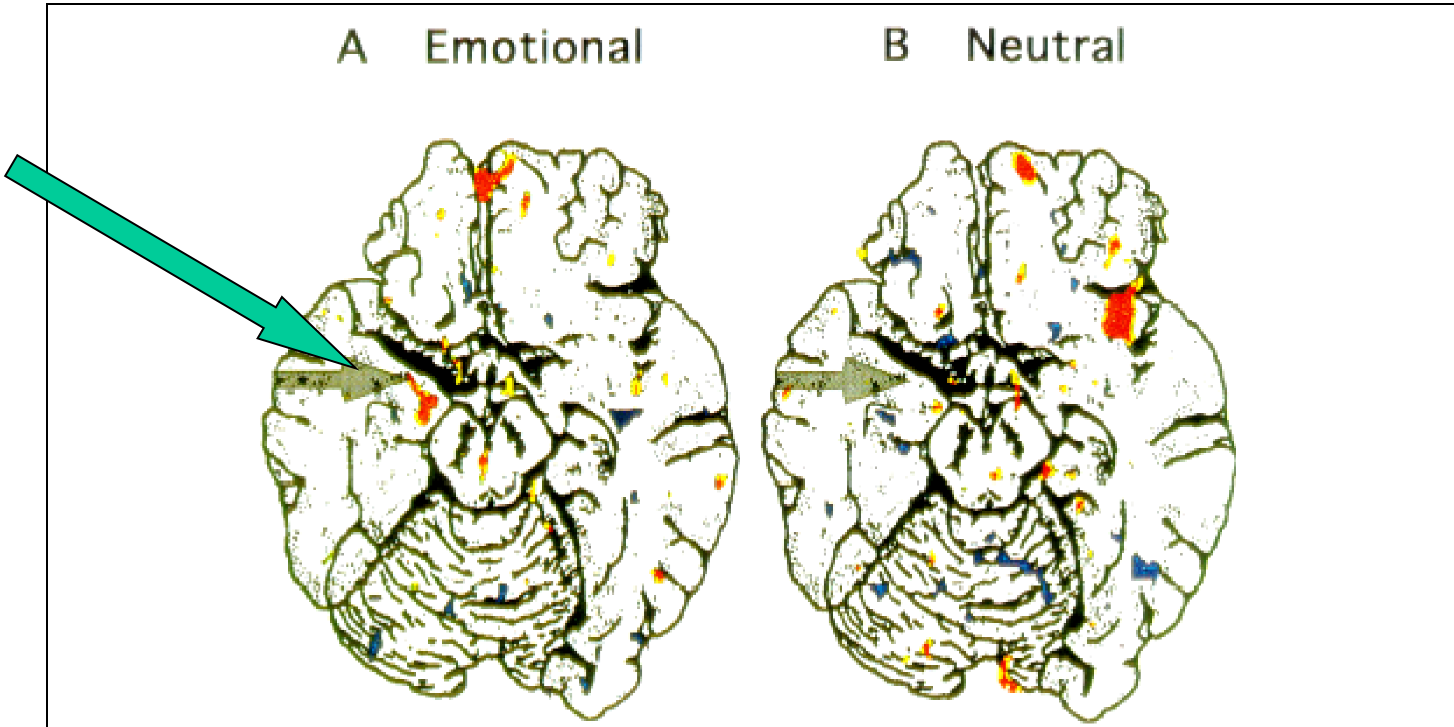


FIG. 2. (*A*) Axial brain section at the level of the AC [section 11 from Matsui and Hirano atlas (22), 21% of head height from CM line] showing regions whose relative GMR during the E session was significantly correlated positively (orange/yellow) or negatively (blue/green) with the number of films recalled from that session. (*B*) Brain regions in which relative GMR from the N session was significantly correlated with the number of films recalled from that session. The arrow points to the right AC in both cases. (Following radiologic convention, the right side of the brain is displayed on the left side of the image.) Note the area within the right AC whose activity correlated positively with recall of E, but not N, films.

Cahill et al. (1996)

Die Amygdala und Gedächtnis-konsolidierung

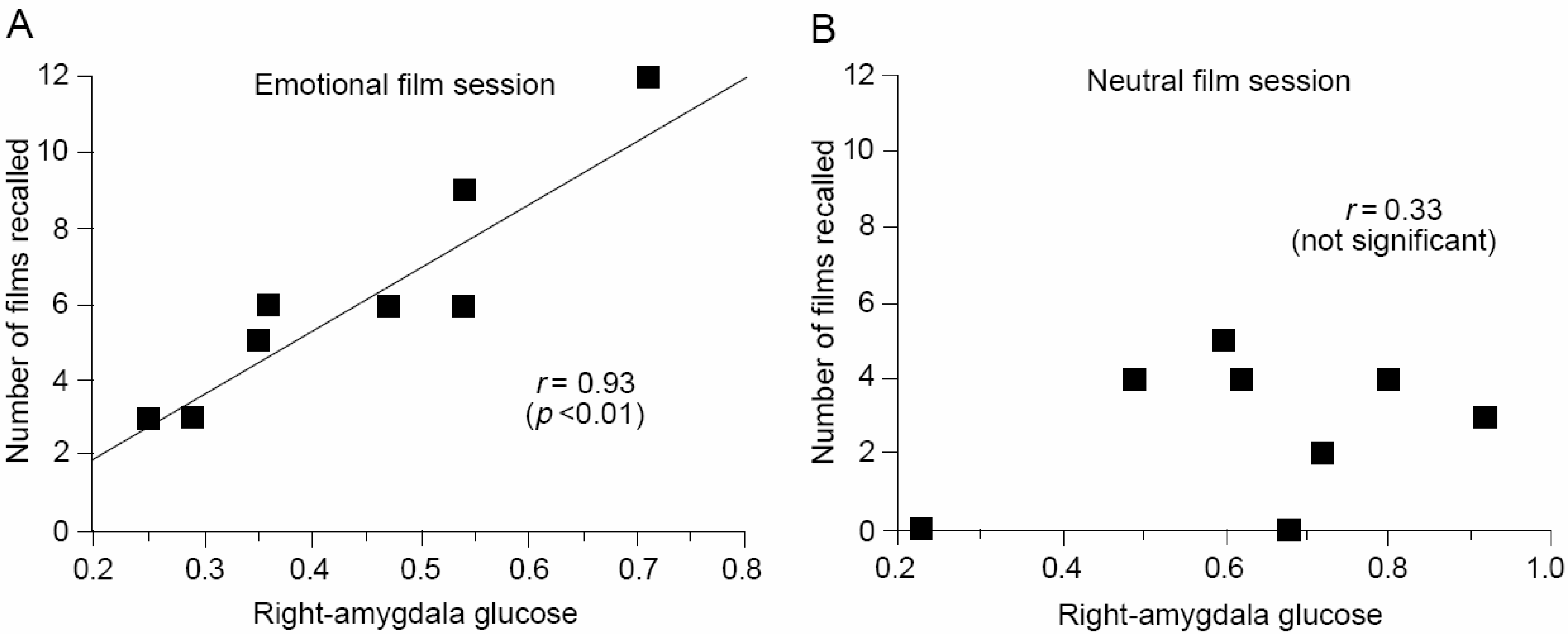
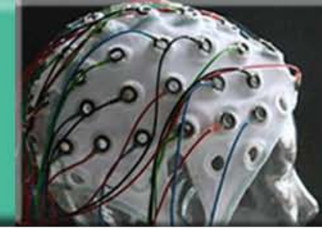


Fig. 4. ***Amygdala activity in healthy humans selectively correlated with the formation of declarative memory for emotionally arousing information.*** *Correlations between* **(A)** *glucose utilization in the right amygdaloid complex (AC) of healthy subjects while viewing a series of relatively emotionally arousing films and long-term recall of those films and* **(B)** *glucose utilization in the right AC of the same subjects while viewing a series of relatively emotionally neutral films and long-term recall of those films.* Modified from Ref. 67.

Die Amygdala und Gedächtnis-konsolidierung




Behavioral/Systems/Cognitive

Amygdala Activity Is Associated with the Successful Encoding of Item, But Not Source, Information for Positive and Negative Stimuli

Elizabeth A. Kensinger and Daniel L. Schacter

Table 1. Mean (SE) retrieval responses as a function of emotion

	Presented items			New items	
	Item-and-source	Item-not-source	Miss	Correct rejections	False alarms
Pictures					
Negative	0.61 (0.04)	0.30 (0.04)	0.09 (0.04)	0.93 (0.01)	0.04 (0.01)
Positive	0.61 (0.04)	0.27 (0.04)	0.13 (0.02)	0.92 (0.02)	0.04 (0.01)
Neutral	0.62 (0.04)	0.17 (0.02)	0.18 (0.02)	0.94 (0.02)	0.03 (0.01)
Words					
Negative	0.52 (0.03)	0.28 (0.03)	0.21 (0.02)	0.79 (0.04)	0.09 (0.02)
Positive	0.54 (0.04)	0.29 (0.02)	0.20 (0.03)	0.83 (0.04)	0.08 (0.02)
Neutral	0.52 (0.04)	0.18 (0.03)	0.25 (0.02)	0.88 (0.03)	0.06 (0.02)
All items					
Negative	0.56 (0.03)	0.30 (0.02)	0.15 (0.02)	0.87 (0.03)	0.06 (0.01)
Positive	0.57 (0.03)	0.28 (0.02)	0.16 (0.02)	0.88 (0.03)	0.06 (0.01)
Neutral	0.57 (0.04)	0.17 (0.02)	0.22 (0.02)	0.91 (0.02)	0.04 (0.01)

Die Amygdala und Gedächtnis-konsolidierung

Behavioral/Systems/Cognitive

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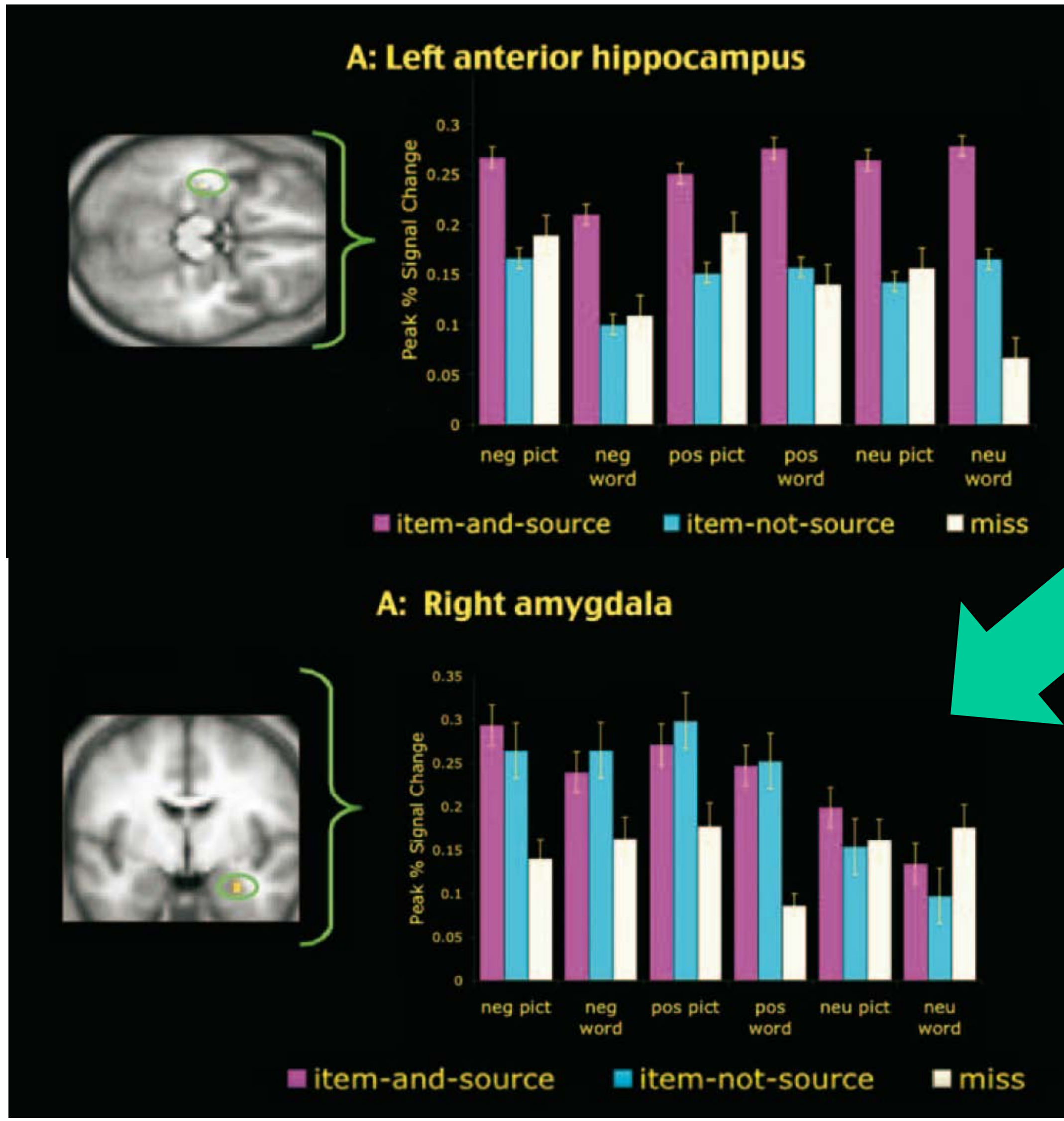

Posttraumatische Belastungsstörungen (PTBS)

- Flashback Memories

  Wiedererleben traumatischer Ereignisse (sensory reexperience)

- Wie ist die Erinnerung an ein Trauma im Gedächtnis repräsentiert?

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Cognitive abnormalities in post-traumatic stress disorder

Richard J. McNally

Department of Psychology, Harvard University, 1230 William James Hall, 33 Kirkland Street, Cambridge, MA 02138, USA

Characteristically arising in response to overwhelmingly terrifying events, post-traumatic stress disorder (PTSD) is a disorder of memory: sufferers seemingly relive their trauma in the form of involuntary recollection. Prominent cognitive abnormalities, especially in memory functioning, have motivated research designed to elucidate the mediating mechanisms that produce PTSD symptoms, especially those involving involuntary recollection. Recent developments suggest a pathophysiological model of PTSD which includes hyporesponsive prefrontal cortical regions and/or a hyper-responsive amygdala. Other work has also identified above-average cognitive ability as a protective factor and below-average hippocampal volume as a vulnerability factor for PTSD among the trauma-exposed. These attempts to elucidate the mediating mechanisms of PTSD have been both cognitive and, more recently, cognitive-neuroscientific in emphasis.

Introduction

Post-traumatic stress disorder (PTSD) is an anxiety disorder that develops usually in response to an overwhelmingly terrifying, often life-threatening event [1]. Symptoms include avoidance of reminders of the trauma, irritability, sleep disturbance, exaggerated startle, and emotional numbing. But its hallmark characteristic is the recurrent, involuntary recollection of the trauma in the form of intrusive thoughts, nightmares, and vivid sensory memories ('flashbacks'). Rather than merely remembering it as an event from their past, PTSD sufferers seemingly relive the trauma with all its original emotional intensity [2].

Striking disturbances in cognition, especially memory, have prompted research on the cognitive mechanisms of PTSD. Early studies are reviewed elsewhere [3]; recent breakthroughs are reviewed here. The most important developments include phenomenological studies that have yielded clues about how traumatic experiences are represented in memory; the incorporation of neuroimaging methods into studies on intrusive cognition that have led to a model of the pathophysiology of PTSD; and studies convincingly showing that above-average cognitive ability serves as a resilience factor and that small hippocampi serve as a risk factor for PTSD among the trauma-exposed. Finally, cognitive science methods have been brought to bear on the most explosive issue in the trauma field: the debate regarding allegedly repressed and recovered memories of childhood sexual abuse (CSA) (see Box 1).

Phenomenological and meta-cognitive findings

Among sufferers of PTSD, memory for trauma can be expressed in different ways, and phenomenological research has provided clues to how memory for trauma is represented in memory [4]. For example, a person might experience repetitive, unwanted thoughts *about* the trauma, such as '*Why did this thing happen to me?*', or experience intrusive memories *of* the trauma, such as vivid, sensory 'snapshots' of a horrific accident [5]. One study revealed that intrusive memories typically involved brief, visual flashbacks of stimuli that preceded the most terrifying aspect of the trauma rather than the most painful or distressing aspect of the experience per se [6]. Flashbacks, it seems, embody the antecedents – the 'warning signals' – that predicted the most worst part of the event. For example, one survivor of a head-on collision reported flashbacks of the headlights of the oncoming vehicle, not of the crash itself. Other work shows that flashbacks are more likely to involve certain sensory modalities than others. Visual flashbacks are most common, followed in frequency by bodily/kinesthetic (e.g. pains), auditory, olfactory, and gustatory ones [5].

Intrusive cognition *about* the trauma is expressible in language, and includes narrative descriptions of the trauma itself and meta-cognitive appraisal of the meaning of one's acute PTSD symptoms. Although the sensory reexperiencings are especially dramatic features of PTSD, they are less frequent than intrusive thoughts about the trauma. A study of assault victims indicated that trauma memories are more disorganized among those with PTSD symptoms, and that the magnitude of disorganization predicts subsequent PTSD pathology [7]. Victims suffering PTSD symptoms did not exhibit disorganization when recounting a non-traumatic, control event dating from the time of the trauma. Early work on disorganized narrative memory for trauma did not incorporate control events, and disorganization itself appeared to be an artifact of limited verbal ability [8].

Meta-cognitive appraisal of one's acute post-traumatic symptoms predicts whether one will develop chronic PTSD [9,10]. For example, if flashbacks are interpreted as harbingers of impending psychosis, or if exaggerated startle reactions and nightmares are interpreted as signs

Corresponding author: McNally, R.J. (rjm@wjh.harvard.edu).
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Hyperaktivität der Amygdala / Hypoaktivität
im orbitofrontalen PFC bei emotionalen Reizen

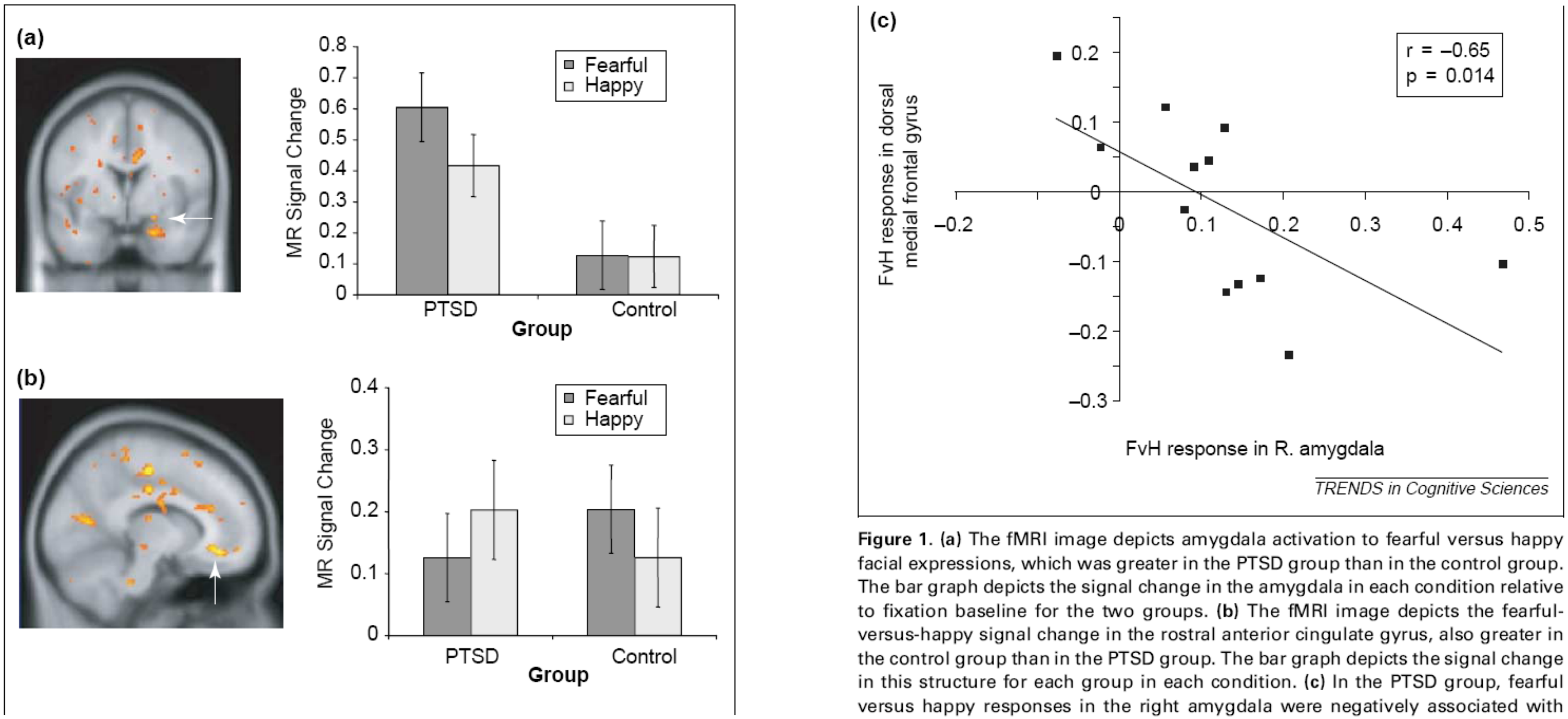


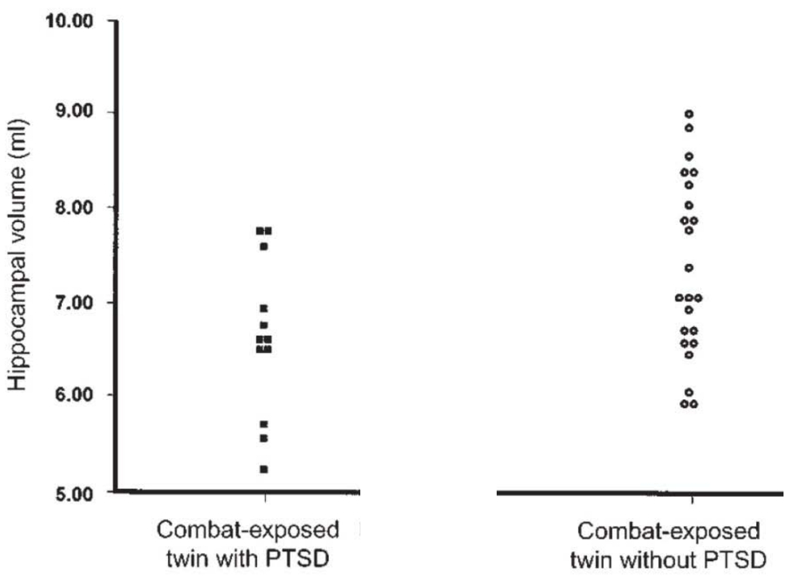
Figure 1. **(a)** The fMRI image depicts amygdala activation to fearful versus happy facial expressions, which was greater in the PTSD group than in the control group. The bar graph depicts the signal change in the amygdala in each condition relative to fixation baseline for the two groups. **(b)** The fMRI image depicts the fearful-versus-happy signal change in the rostral anterior cingulate gyrus, also greater in the control group than in the PTSD group. The bar graph depicts the signal change in this structure for each group in each condition. **(c)** In the PTSD group, fearful versus happy responses in the right amygdala were negatively associated with fearful versus happy responses in the dorsal medial frontal gyrus.

Gibt es prämorbide Risikofaktoren für PTBS?

Gilbertson et al (2002) Nat Neurosci, 5, 1242-1247

Hippocampusvolumen korreliert mit der Traumaschwere

→ kleiner Hippocampus: Vulnerabilitätsfaktor für PTBS ?



Gibt es prämorbide Risikofaktoren für PTSD?

Gilbertson et al (2002) Nat Neurosci, 5, 1242-1247

Hippocampusvolumen korreliert mit der Traumaschwere
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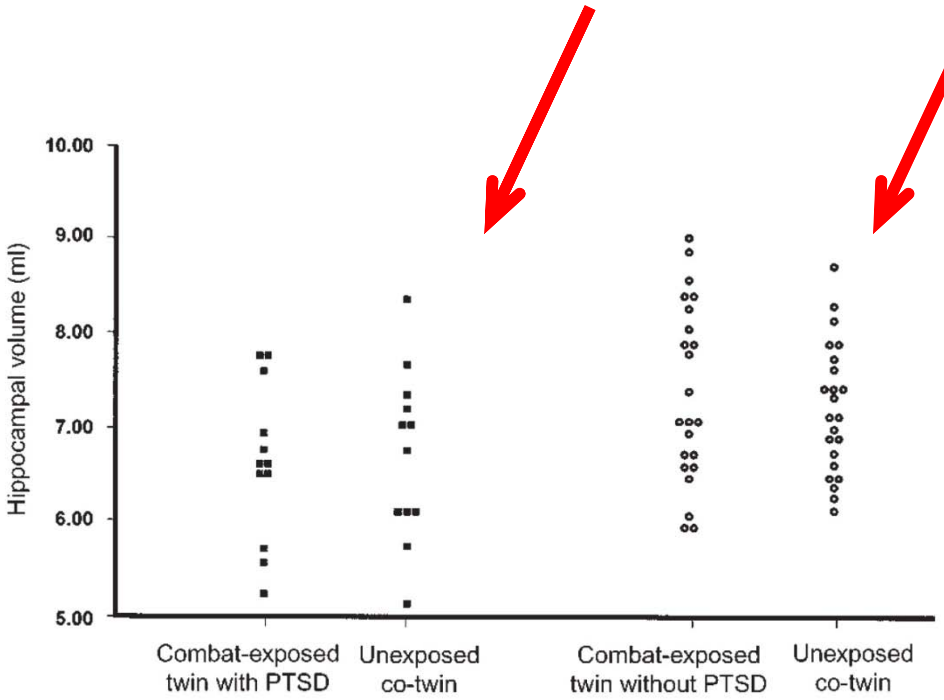
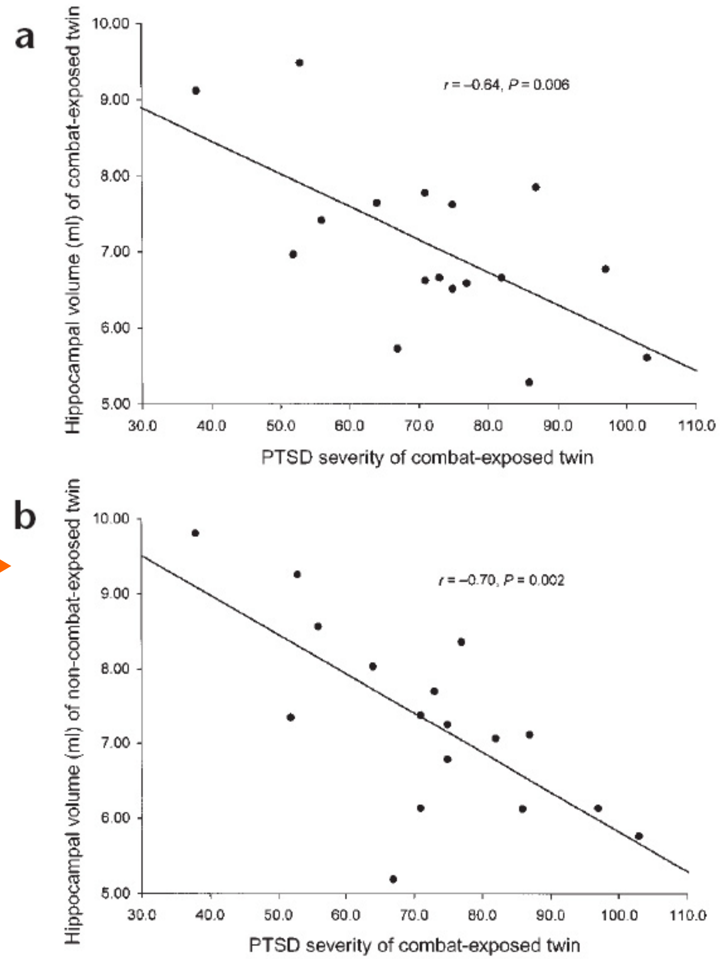


Gibt es prämorbide Risikofaktoren?



Research Article

Putting Feelings Into Words

Affect Labeling Disrupts Amygdala Activity in Response to Affective Stimuli

Matthew D. Lieberman, Naomi I. Eisenberger, Molly J. Crockett, Sabrina M. Tom, Jennifer H. Pfeifer, and Baldwin M. Way

University of California, Los Angeles

ABSTRACT—*Putting feelings into words (affect labeling) has long been thought to help manage negative emotional experiences; however, the mechanisms by which affect labeling produces this benefit remain largely unknown. Recent neuroimaging studies suggest a possible neurocognitive pathway for this process, but methodological limitations of previous studies have prevented strong inferences from being drawn. A functional magnetic resonance imaging study of affect labeling was conducted to remedy these limitations. The results indicated that affect labeling, relative to other forms of encoding, diminished the response of the amygdala and other limbic regions to negative emotional images. Additionally, affect labeling produced increased activity in a single brain region, right ventrolateral prefrontal cortex (RVLPFC). Finally, RVLPFC and amygdala activity during affect labeling were inversely correlated, a relationship that was mediated by activity in medial prefrontal cortex (MPFC). These results suggest that affect labeling may diminish emotional reactivity along a pathway from RVLPFC to MPFC to the amygdala.*

Putting feelings into words has long been thought to be one of the best ways to manage negative emotional experiences. Talk therapies have been formally practiced for more than a century and, although varying in structure and content, are commonly based on the assumption that talking about one's feelings and problems is an effective method for minimizing the impact of negative emotional events on current experience. More recently, psychologists have discovered that merely putting pen to paper to express one's emotional ailments has benefits for mental and physical health (Hemenover, 2003; Pennebaker, 1997). Although conventional wisdom and scientific evidence indicate that putting one's feelings into words can attenuate negative emotional experiences (Wilson & Schooler, 1991), the mechanisms by which these benefits arise remain largely unknown.

Recent neuroimaging research has begun to offer insight into a possible neurocognitive mechanism by which putting feelings into words may alleviate negative emotional responses. A number of studies of *affect labeling* have demonstrated that linguistic processing of the emotional aspects of an emotional image produces less amygdala activity than perceptual processing of the emotional aspects of the same image (Hariri, Bookheimer, & Mazziotta, 2000; Lieberman, Hariri, Jarcho, Eisenberger, & Bookheimer, 2005). Additionally, these studies have demonstrated greater activity during linguistic processing than during nonlinguistic processing of emotion in right ventrolateral prefrontal cortex (RVLPFC), a region associated with the symbolic processing of emotional information (Cunningham, Johnson, Gatenby, Gore, & Banaji, 2003; Nomura et al., 2003) and with top-down inhibitory processes (Aron, Robbins, & Poldrack, 2004). Finally, the magnitude of RVLPFC activity during affect labeling has been inversely correlated with the magnitude of amygdala activity during affect labeling in these studies. Together, these results suggest that putting feelings into words may activate RVLPFC, which in turn may dampen the response of the amygdala, thus helping to alleviate emotional distress.

In studies of affect labeling, an emotionally evocative image is usually shown along with two options for categorizing the image. The images in Figures 1a and 1b provide examples of typical *affect-label* and *affect-match* trials, respectively. During affect-label trials (i.e., linguistic processing of affect), a pair of affective labels is presented at the bottom of the screen, and the subject chooses the label that best characterizes the emotion displayed by the target face at the top of the screen. During affect-match trials (i.e., nonlinguistic processing of affect), a

Address correspondence to Matthew Lieberman, Department of Psychology, Franz Hall, University of California, Los Angeles, Los Angeles, CA 90095-1563, e-mail: lieber@ucla.edu.

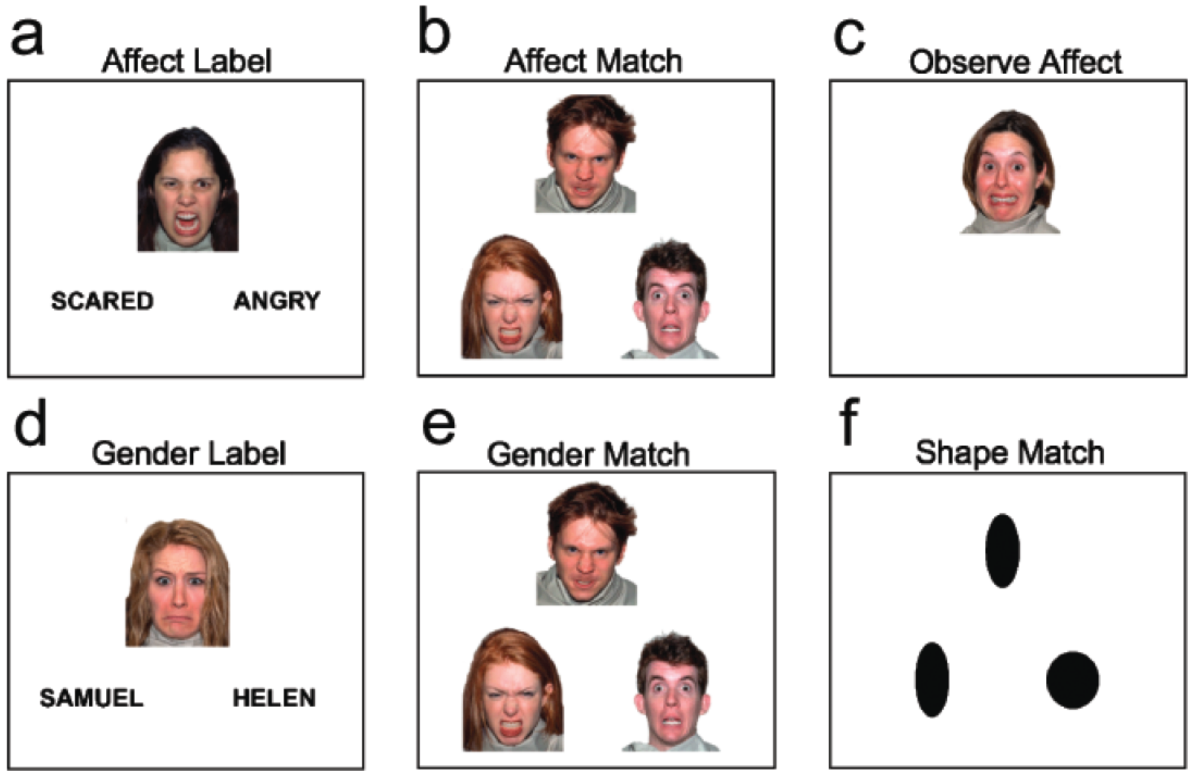


Fig. 1. A sample display from each of the six types of experimental trials.

Affektkennzeichnung reduziert Amygdala Aktivität

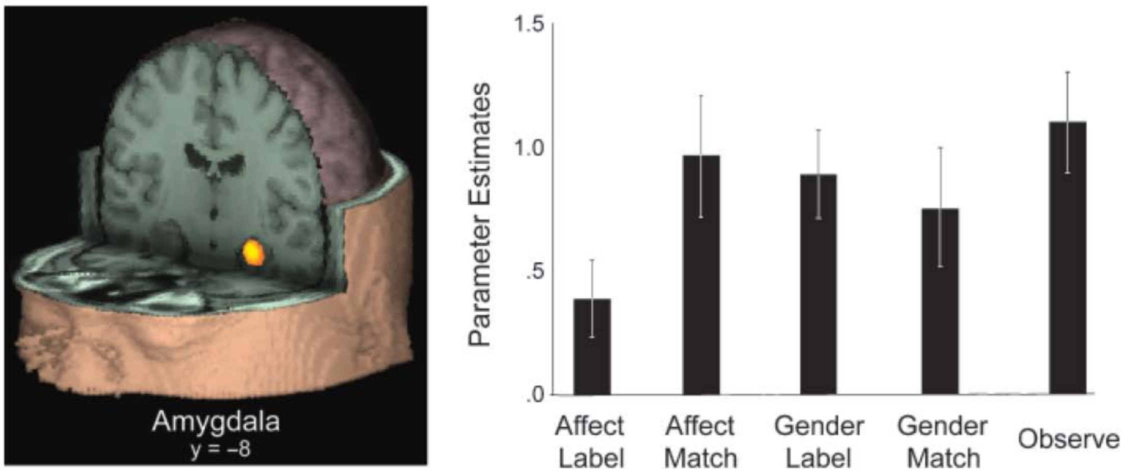


Fig. 2. Parameter estimates of activity during five conditions (relative to activity in the shape-match control condition) in an amygdala region of interest (ROI). The ROI was identified by comparing activity in the observe condition and activity in the shape-match condition. The illustration on the left shows an axial slice indicating the extent of the ROI.

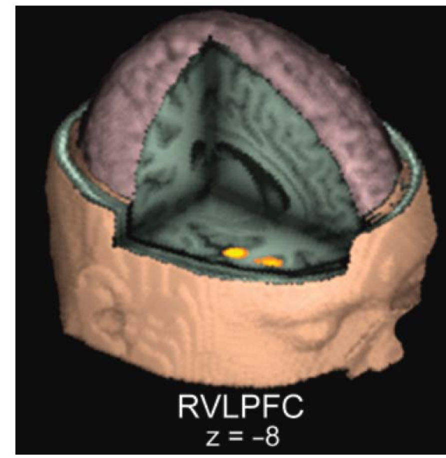


Fig. 3. Illustration of a canonical brain showing two clusters in right ventrolateral prefrontal cortex (RVLPFC) where activity was greater during affect labeling than during gender labeling.

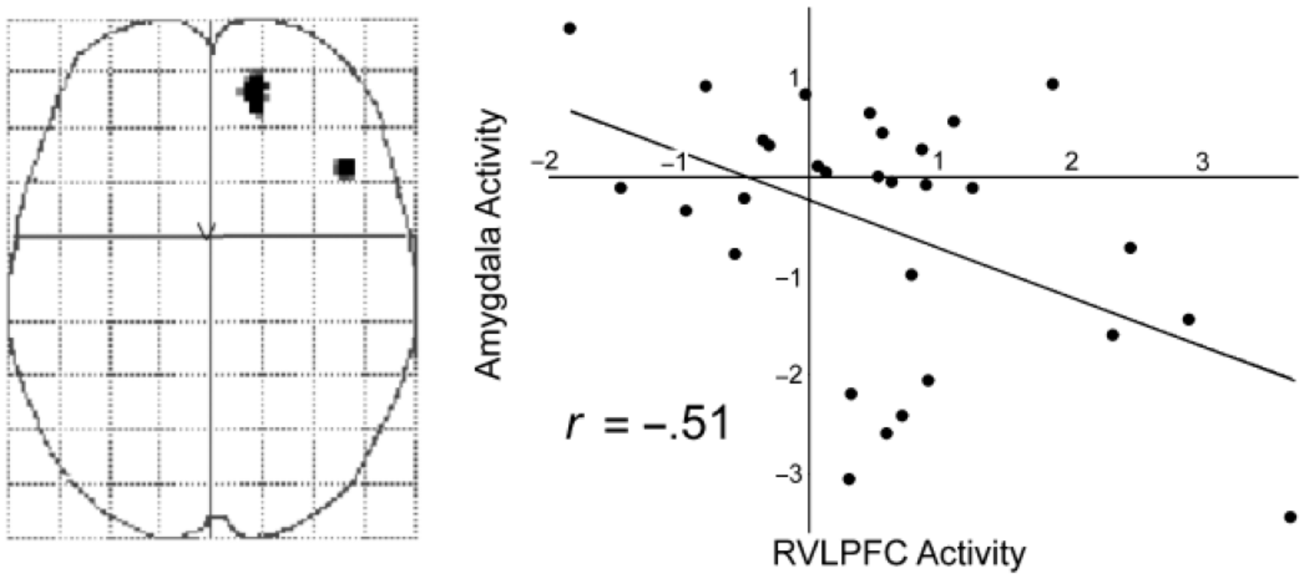


Fig. 4. Correlation between right ventrolateral prefrontal cortex (RVLPFC) and amygdala activity. Each plotted point represents the parameter estimates for a single subject's activity in RVLPFC and the amygdala during affect labeling, relative to gender labeling. The view of the glass brain on the left shows all brain regions (RVLPFC and medial prefrontal cortex) for which activity was inversely correlated with amygdala activity during affect labeling, relative to gender labeling.

Take Home

- 3 Emotionstheorien und deren Vergleich
- emotionale Gesichtsausdrücke
  echtes vs unechtes Lächeln
- Neuronale Grundlagen von Emotionen
  orbitofrontaler Cortex, Amygdala
- Emotionen und Gedächtnis
  modulatorischer Einfluss der Amygdala
- Neuronale Grundlagen der PTBS