ELSEVIER

Progress in Neurobiology 75 (2005) 29–52

progress in Neurobiology

www.elsevier.com/locate/pneurobio

Emotion and cortical-subcortical function: conceptual developments

M.R. Bennett[a,*], P.M.S. Hacker[b]

[a]*The Neurobiology Laboratory, Department of Physiology, Institute for Biomedical Research, University of Sydney, Sydney, NSW 2006, Australia*
[b]*St John's College, Oxford, UK*

Received 4 June 2004; accepted 23 November 2004

Abstract

Biologists have studied the expression of emotions in man and other animals since at least 1806, when Charles Bell published his *Anatomy and Physiology of Expression*. We trace the main experimental developments since that time, including Darwin's investigations into the evolution of innate forms of expression of emotions, as well as those into cognitive versus precognitive forms of expression of emotions. In particular, contemporary studies by neuroscientists into the origins of emotional experiences are detailed, especially emotional responses to faces showing different expressions, on which much research has been carried out. We examine the various claims made by these researchers as to what their experiments show. Our conceptual analysis indicates that there is considerable confusion as to what experimental work to this time indicates about the role of cortical and subcortical structures in the expression of emotions. We attempt to clarify what can and cannot be justified as established concerning the workings of the brain and emotions.

Contents

* Corresponding author. Tel.: +61 2 9351 2034; fax: +61 2 9351 3910.
E-mail address: maxb@physiol.usyd.edu.au (M.R. Bennett).

0301-0082/$ – see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.pneurobio.2004.11.002

1. Introduction

Fruitful research on the emotions requires clarity with regard to the category of the emotions and the differentiation of emotions from feelings that are not emotions at all, although commonly confused with them. So before recounting some of the salient advances in research over the past century or so, we shall endeavour to provide a schematic map of the conceptual terrain.

Feelings must be differentiated into sensations, tactile perceptions, appetites, and affections (see Fig. 1 as well as Bennett and Hacker (2003, Chapter 7). Pains, tickles and tingles are bodily sensations with a more or less determinate bodily location. They are felt *in* a part of the body, but not *with* a part of the body. Unlike perceptions, sensations are not correct or incorrect, and are not susceptible to cognitive error. There is no such thing as thinking one is in pain but being mistaken. Phantom pains or reflected pains may lead one to judge that one has an injury where there is none, but what is erroneous is neither the sensation nor its felt location – it is the judgement concerning the location of the injury. Localised bodily sensations must be distinguished from sensations of overall bodily condition, such as feelings of weariness or lassitude. Feeling the heat, solidity, elasticity or dampness of an object are forms of tactile perception – exercises of a cognitive faculty that inform one how things are in one's environment in respect of perceptible qualities such as warmth, cold, hardness, softness, wetness, dryness, and so forth. Like all perceptions, they may be correct or incorrect, and we distinguish between things feeling thus and so to one, and things being thus and so. Natural appetites are such things as feelings of hunger, thirst or animal lust. Non-natural (acquired) appetites are addictions.

An appetite is a blend of desire and sensation. Sensations characteristic of appetites have a bodily location (sensations of hunger are located in one's midriff) and are forms of unease that dispose one to action to satisfy the desire. The desire that is blended with sensation is characterised by its formal object: hunger is a desire for food, thirst for drink, lust for sexual intercourse. The intensity of the desire is typically proportional to the intensity of the sensation. Fulfilling an appetite leads to its temporary satiation and so to the disappearance of the sensation. Appetites are not constant, but recurrent, typically caused by bodily needs or hormonally determined drives.

Affections too are felt. The feelings that are affections can be distinguished into agitations (e.g. astonishment, excitement), moods (e.g. cheerfulness, depression) and emotions (e.g. fear, love) – see Fig. 2. Unlike sensations, affections do not inform one about the state of one's body, though they are sometimes linked with sensations. One does not feel pride in one's chest, even though one's chest may swell with pride, or fear in one's mouth, even though one's mouth may be dry with fear. One's blush of embarrassment does not inform one of the state of one's facial arteries, although it may inform one that one is more embarrassed than one thought, and one's tears of grief do not inform one of the state of one's lachrymal glands, although they may inform one that one is grieving more than one anticipated. Unlike feelings that are perceptions, the affections do not inform one of the state of the world around one. Paradigmatic emotions are love, hate, hope, fear, anger, gratitude, resentment, indignation, envy, jealousy, pity, compassion, grief, as well as emotions of self-assessment such as pride, shame, humiliation, regret, remorse and guilt.

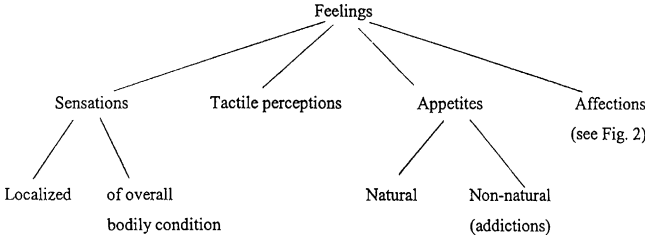

Fig. 1. Types of feelings distinguished.

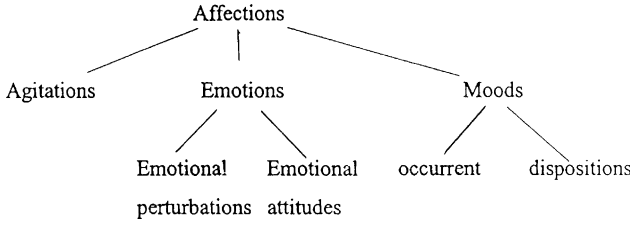

Fig. 2. Types of affection.

Agitations are short-term affective disturbances, commonly (but not only) caused by something unexpected, e.g. being and feeling excited, thrilled, shocked, convulsed, amazed, surprised, horrified, disgusted, delighted. They are caused by what we perceive, learn or realise. Because they are disturbances, caused by unanticipated disruptions, they are not motives for action as emotions may be, but temporarily *inhibit* motivated action. One's agitations are manifested in expressive behaviour *because* one is, for example, excited, surprised, or shocked, but one does not act *out of* excitement, surprise or shock as one may act *out of* love, pity or gratitude. Agitations are modes of *reaction*: one cries out *in* horror, recoils *with* revulsion, is convulsed *by* laughter, or is paralysed *with* shock. Occurrently felt emotions, as opposed to longer standing emotional attitudes, often bear a kinship to agitations, for example in the perturbations of rage, fear and grief.

Moods are such things as feeling cheerful, euphoric, contented, irritable, melancholic or depressed. They are states or frames of mind. They may be occurrent, or longer-term dispositional states (one may feel depressed for an afternoon, or one may be suffering from a depression that lasts for months – being then prone to feel depressed during one's waking hours). Moods are less closely tied to specific objects than emotions – one can feel cheerful without feeling cheerful about anything in particular, but one cannot love without loving someone or something in particular. They are not linked to specific patterns of intentional action, since, unlike emotions, they do not afford motives for action. Moods colour one's thoughts and pervade one's reflections. So they are linked to manners of behaviour, demeanour and tone of voice.

It is important, especially in the study of human emotions, to distinguish between episodic emotional perturbations and emotional attitudes. Emotional perturbations resemble agitations in certain respects. Some, e.g. fear or anger, have characteristic somatic accompaniments, both sensations that are felt and physiological reactions that are measurable. Others do not, e.g. feelings of pride, humility, compassion, and gratitude. They are manifest in *expressive behaviour* that may take various forms. It may be behaviour that is not action, such as blushes, perspiration, pallor. It may be voluntary (utterances of love and affection, hope or pride), partly voluntary (raised voice of anger, that can be suppressed) or involuntary action (cry of terror). It may be exhibited only in the manner of acting (e.g. tone of voice, impatient gestures).

Emotional attitudes, such as love, hate, pride, shame, remorse may last for long periods of time and motivate action done for reasons. One may love or hate a person, activity, a cause or place for years. One may be proud of the achievements of one's youth for the rest of one's life, and one may respect or detest, be jealous or envious of someone for years. One may be ashamed or guilty of one's misconduct for decades, and one's regrets for one's follies may never cease. One's judgement may be clouded not only by emotional perturbations but also by one's long-standing resentments, envies or jealousies. The emotions of love, hate or envy, for example, consist above all in the manner in which the object of one's emotion matters to one and the reasons one has for holding it to be important, and hence too in the motives it affords one for acting (for one acts *out of* love, hate or envy). One's emotions are then evident in the reasons that weigh with one in one's deliberations, in the desires one harbours in respect of the object of the emotion, and in associated thoughts and fantasies.

Emotions have objects as well as causes. What makes one afraid (a noise downstairs at night) need not be what one is afraid of (a burglar, who may not even exist). What one is frightened *by* is the cause of one's fear, what one is frightened *of* is its object. What makes one feel ashamed, e.g. someone's indignant tirade, is not the same as what one is ashamed of, namely one's own misdemeanour. A person need not know the cause of his emotion, but, save in pathological cases, he cannot be ignorant of the object of his emotion, i.e. whom he is angry with, what he is ashamed of, what he is afraid of.

It is obvious that the intensity of one's emotions is not proportional to the intensity of whatever sensations may accompany their occurrent manifestations. One's pride in one's children's achievements cannot be measured somatically, but may be manifest in one's behaviour, in the way one praises them and how one talks about them. One's fear of heights may be manifest above all in the lengths one goes to avoid them, rather than in any perturbations – given that one avoids heights at all costs. Unlike appetites, emotions do not display the same pattern of occurrence, satiation, and recurrence. They have a cognitive dimension absent from appetites. For the frightened animal is afraid of something it knows or believes to be dangerous or harmful, a mother is proud of her offspring, believing them to be meritorious, and the repentant offender is remorseful, knowing or believing himself to have done wrong and wishing to make amends. Human emotions, rooted though they are in our animal nature, are nevertheless run through, as mere animal emotions are not, with thought and belief, wish and want, fantasy and imagination – as should be expected of language-using, concept-exercising creatures.

2. Darwin

In 1872, Charles Darwin published his book *The Expression of Emotions in Man and Animals* (Darwin, 1965). The scope of the book was wider than suggested by the title, since Darwin investigated expressive behaviour in general, studying not only the expressions of fear, anger, grief, etc., but also behaviour expressing pain, assent, dissent, puzzlement, and helplessness, which are not emotions. Expressive behaviour can be differentiated into facial expression, movements of the body and limbs, posture and voice. Darwin concentrated upon facial expression, but also discussed gesture and mien.

The argument of the book was to show that the mode of expression of the emotions, at least in the range of the more

primitive emotions, is innate. It is common to the whole of mankind, and so supports the hypothesis of the descent of all races from a common progenitor. Furthermore, it shares common features with the modes of manifestation of emotions by other animals, in particular the apes, thus supporting the evolutionary hypothesis that man is continuous with the rest of animate nature. So Darwin's enterprise gave oblique support to evolutionary theory. It is very surprising that Darwin gave hardly any attention in his theory to the communicative role and evolutionary benefit of regular, determinate, and readily recognisable manifestations of basic emotions among animals in general and man in particular.

Of course, his researches were pursued also for their intrinsic interest. The question of *what* facial expression gives expression to *which* emotion had been raised by others. So too had the question of how these specific expressions are physiologically produced. Darwin relied upon Sir Charles Bell's physiological researches (see *Anatomy and Philosophy of Expression*, 1806) into facial musculature and its employment in the expression of the emotions, upon the work of Pierre Gratiolet (see *De la Physiognomie et des Mouvements d'Expression*, 1865) and G.B. Duchenne de Bologne (1990) (see *Méchanisme de la Physiognomie Humaine*, 1862) – in particular making use of the latter's methods of using photographs of facial expressions to illustrate the manifestations of emotion (see Fig. 3A, showing terror, and Fig. 3B, showing horror and agony, both being wood engravings made for Darwin's book from Duchenne's photographs). What was novel was Darwin's investigation into the question of *why* these innate forms of expression of emotion should have evolved, why just these specific muscles should be used to exhibit fear, anger, grief, etc., respectively.

The strategy he employed was to appeal to three distinct principles. The first was his *Principle of Serviceable Associated Habits*. According to this principle, expressive behaviour that is directly or indirectly useful in relieving or gratifying desires or feelings associated with an emotion will tend to be repeated whenever that emotion recurs (no matter how feebly) even if the behaviour is pointless. The second was *The Principle of Antithesis*, according to which contrary emotions tend to induce diametrically opposed expressive behaviour. The third was the *Principle of the Direct Action of the Nervous System*, as exhibited in blushing, perspiring, raised breathing and pulse rates, differential glandular secretions, etc. associated with specific emotions. Darwin hoped to be able to explain all of the forms of expressive behaviour by reference to these three principles and their interplay. Moreover, if they could also be invoked successfully to explain the expressions of emotion among primates and other animals, this would reinforce the argument for common evolutionary origins.

Darwin's first principle was supported by a great deal of illustrative data. It has won considerable backing in recent years, although Darwin's surprisingly Lamarkian explanation of its transmission is rejected. The second principle is generally thought to be more questionable. The third can hardly be challenged, but it is the one for which Darwin, as he admitted, had least explanation to offer.

Darwin's book, although an immediate best-seller, became forgotten in the course of the first half and more of the twentieth century. Its central doctrines of the *universality* and *innateness* of the fundamental forms of emotional expression were rejected. This was due partly to the rise of highly relativist forms of anthropology (the leading protagonists of which were Margaret Mead, Gregory

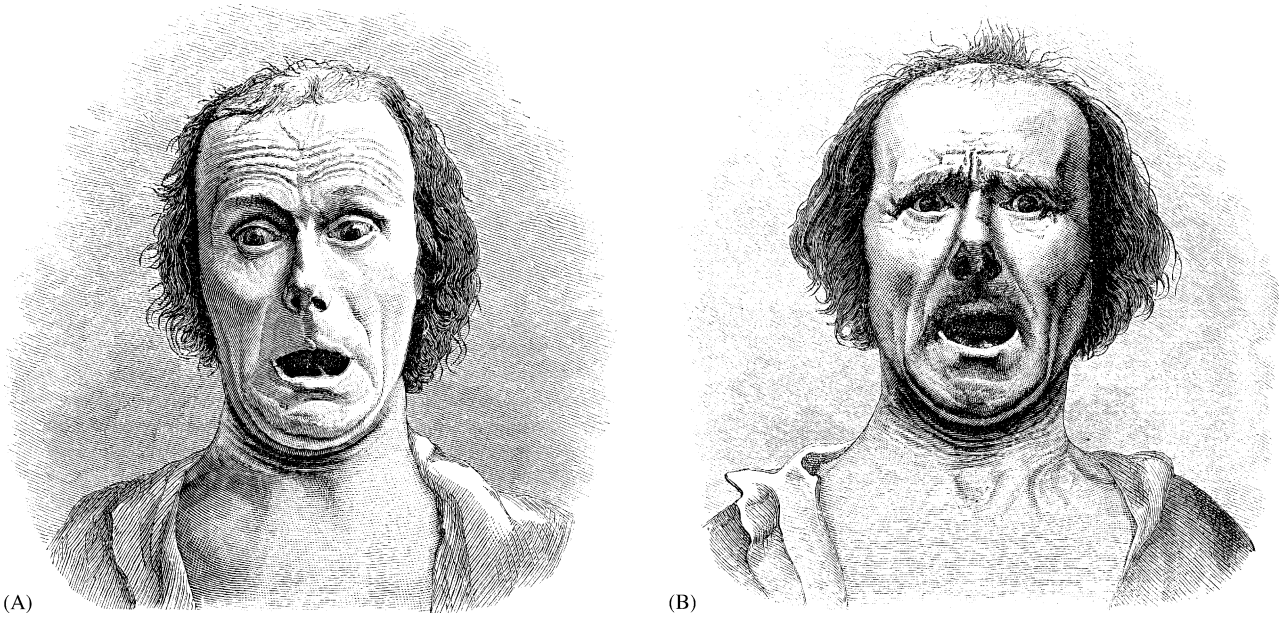

Fig. 3. Facial expressions illustrating the manifestations of emotion: (A) terror; (B) horror and agony (from figs. 20 and 21 in *The Expression of the Emotions in Man and Animals* C. Darwin (1872)).

Bateson and Ray Birdwhistell), and partly to the dominance of dogmatic behaviourism among experimental psychologists until the early 1960s. It was only with the repudiation of behaviourism and the questioning of Mead's anthropology that Darwin's theories received fresh reconsideration.

Darwin's evidential methods were, by later standards, excessively anecdotal, and his questionnaires often tended to suggest a bias in the answers. But his ideas were put to systematic tests meeting current standards of evidence by Paul Ekman in the 1960s and 1970s, and received powerful confirmation (Ekman, 1971, 1972). Ekman and his colleagues showed that there are distinct patterns of facial expression of the emotions corresponding to such primary emotions as fear, anger, happiness (presumably satisfaction, or being pleased, or contented) and disgust. These patterns appear to be behavioural universals, characteristic of literate and preliterate cultures alike, irrespective of the availability of specific emotion names in the language or of exposure to imagery in mass media. So, for example, in this research, data were gathered in New Guinea by telling the subjects a story (without mentioning any emotion terms) and asking them to select a face from a set of three photographs which was appropriate to the events recounted. The table in Fig. 4 shows the results appropriate for the stories listed in the first column. Within each row, the percentage of subjects who gave the correct response differentiating between three emotions was calculated across all subjects (regardless of whether the photographs used to represent them differed). The table shows that the appropriate face was chosen at a significant level for all discriminations (rows) except the differentiation of fear from surprise (last three rows). This demonstrates universality in emotion *recognition*. Other experiments demonstrated comparable universality in primary emotion *expression*. Of course, this does not imply that the expression of emotion is not subject to differential forms of social constraint and socially conditioned forms of behaviour.

ADULT RESULTS

Emotion described in the story	Emotions shown in the two incorrect photographs	No. *Ss*	% choosing correct face
Happiness	Surprise, disgust	62	90**
	Surprise, sadness	57	93**
	Fear, anger	65	86**
	Disgust, anger	36	100**
Anger	Sadness, surprise	66	82**
	Disgust, surprise	31	87**
	Fear, sadness	31	87**
Sadness	Anger, fear	64	81**
	Anger, surprise	26	81**
	Anger, happiness	31	87**
	Anger, disgust	35	69*
	Disgust, surprise	35	77**
Disgust (smell story)	Sadness, surprise	65	77**
Disgust (dislike story)	Sadness, surprise	36	89**
Surprise	Fear, disgust	31	71*
	Happiness, anger	31	65*
Fear	Anger, disgust	92	64**
	Sadness, disgust	31	87**
	Anger, happiness	35	86**
	Disgust, happiness	26	85**
	Surprise, happiness	65	48
	Surprise, disgust	31	52
	Surprise, sadness	57	28[a]

* $p < .05$.
** $p < .01$.
[a] Subjects selected the surprise face (67%) at a significant level ($p < .01$, two-tailed test).

Fig. 4. Table purporting to show that there are distinct patterns of facial expressions corresponding to different primary emotions (for description see text; taken from table 1 in Ekman and Friesen, 1971).

3. Cognitive versus precognitive theories for the expression of emotions

Scientists have differed over the relation between emotion and cognition. Zajonc (1984) has been a main proponent of the view that 'affect and cognition are separate and partially independent systems and although they ordinarily function conjointly, affect could be generated without a prior cognitive process.' Thus, according to Zajonc, affective judgements may be made on first meeting a person even though very little of what he refers to as 'cognitive processing' has taken place. Evidence which is taken to support this view has been provided by Murphy and Zajonc (1993) by means of the following experiments. Comparisons were made between the effects of what are called affective and cognitive priming under extremely brief 4 ms (suboptimal) and longer 1 s (optimal) exposure conditions. Subjects were told they would be presented with an assortment of Chinese characters that they were to rate on a 5-point scale where 1 indicated they did not like the ideograph at all and 5 indicated they liked the ideograph quite a lot. Subjects were then shown slides of 45 target Chinese ideographs. Four priming conditions, two control and two experimental, were investigated. The series of 45 trials began with five control trials having no prime at all (subsequently referred to as the no-prime control). The remaining 40 trials consisted of 20 control trials that had random polygons as primes (subsequently referred to as the irrelevant prime control) interspersed with 20 experimental trials that had facial primes. Of the 20 experimental trials, 10 of the target ideographs were shown twice, once primed with positive affect (i.e. preceded by an image of an individual smiling) and once primed with negative affect (i.e. preceded by an image of the same individual scowling). The results of these experiments are shown in Fig. 5. Clearly the liking ratings were significantly influenced by the affective primes when these were presented for 4 ms but not when they were presented for 1 s. The explanation suggested is that participants in the 1 s condition had sufficient time to reflect that their affective reaction, taken as a reflex response, was produced by the priming stimulus and so they were able to discount this when reacting to the second stimulus. A further study by Murphy and Zajonc (1993) forced subjects to make a cognitive judgement by asking them to rate Chinese ideographs for femininity after being presented with male or female priming faces. In this case the ratings were influenced by the priming faces if these were presented 1 s before the ideographs but not 4 ms before. According to Murphy and

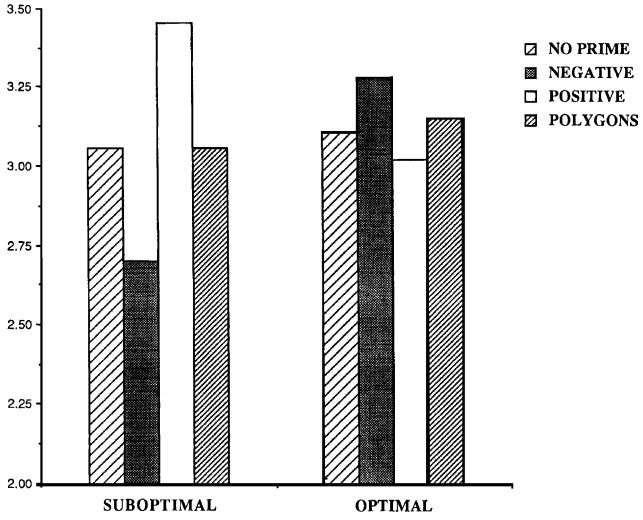

Fig. 5. Results of experiments taken as showing that affect and cognition are separate and partially independent systems (for description, see text; from fig. 1 in Murphy and Zajonc (1993)).

Zajonc (1993) these results indicate that the neural activity underpinning affective processing can sometimes occur faster than the neural activity required for cognitive processing and so lend themselves to supporting the hypothesis that the neural networks involved in affect and those required for cognition are separate and partially independent systems.

It is difficult to know what to make of such experiments. In the first place, they concern attitudes, in particular attitudes of liking and not liking, rather than emotions. Secondly, the object of the attitude, a hitherto unencountered shape of a Chinese ideograph, is so anomalous as to cast into doubt the nature of the judgements the subjects were being asked to make or what liking or not liking them showed. It is, after all, not unlike being asked whether Monday is fat or thin, or whether Wednesday is green or blue. Thirdly, it is interesting that the priming affected the arbitrary judgements of liking when exposure was suboptimal, but there is no reason whatsoever to explain the difference between these cases and the cases of judgements preceded by optimal exposure by reference to the subjects' having time for so called 'cognitive processing', given the complete unclarity about what 'cognitive processing' is supposed to mean. To suggest that subjects had time to discount the positive priming presupposes that the subjects were aware of the influence of the positive priming upon their (arbitrary) judgements. But is that evident? The supposition that the neural networks responsible for affective response differ from those responsible for knowledge may or may not be true, but in the case of many cognitively laden emotions, the component of knowledge or belief cannot, logically, be prized off the emotion, since without the element of knowledge or belief, the identity of the emotion evaporates, leaving no more than somatic agitation of one form or another.

Lazarus (1982) has argued the opposite point of view, namely that what he calls 'cognitive appraisal' is an important ingredient in emotional experience. Experiments alleged to support his case have been carried out using anxiety-evoking films presented to subjects with different accompanying sound tracks (Speisman et al., 1964). Such a film might involve a workshop accident in which a board is caught in a circular saw which rams with considerable force into a worker, who then dies in terrible pain on the floor. Another film showed a stone-age ritual involving adolescent boys having their penises deeply cut. Skin conductance of subjects were taken during the film, as indicative of their stress response, when the film was not accompanied by any sound track ('silent' condition) or when it was accompanied by the following sound tracks: one in which it is indicated that that the workshop film only involved actors or that the incision film did not involve any operation (constituting a 'denial' condition); another sound track appealed to the viewer to consider the workshop accident in an objective way and to view the incision film from the point of view of an anthropologist viewing native customs (constituting an 'intellectualisation' condition); finally, a sound track was presented which heightened the trauma produced by the visual presentation by emphasising the horror, cruelty and pain associated with the film (the 'trauma' condition). Fig. 6 shows skin conductance of subjects taken during the film, which was used as indicative of their stress response. The different conductance values are given for the three different sound tracks that accompanied the film. These are the trauma track, which pointed up the threatening aspects of the film, and gives the greatest increase in skin conductance; the intellectualisation sound track, which was accompanied by the smallest increase in skin conductance whilst watching the film; the denial sound track, which gave a skin conductance increase marginally greater that that of the intellectualisation process; and finally, no sound track at all, which was accompanied by skin conductance changes intermediate between those for the threatening sound track and the denial sound track. Lazarus and his colleagues interpreted these results as pointing to the fact that denial and intellectualisation both produced substantial reductions in stress. Thus by changing what they took to be the cognitive appraisal of the subjects witnessing the traumatic events the physiological indicator of their state of stress could be significantly altered. Lazarus and his colleagues have taken this to imply that cognitive appraisals play an important role in determining emotions (Folkman and Lazarus, 1990; Lazarus, 1991).

Unlike the previous experiments, the bearing of the experiment on the conclusion derived from it is clear. What is unclear is why it should be necessary to conduct experiments to show that thought and reflection can influence one's emotional responses. That is arguably a priori evident, in as much as reasons are involved in many emotional reactions, and reflection, by definition, can affect the weight and balance of reasons.

3.1. On physiological measurements of emotional responses

It is important to realise that neither the subjective nor the objective somatic accompaniments of an emotion, such as

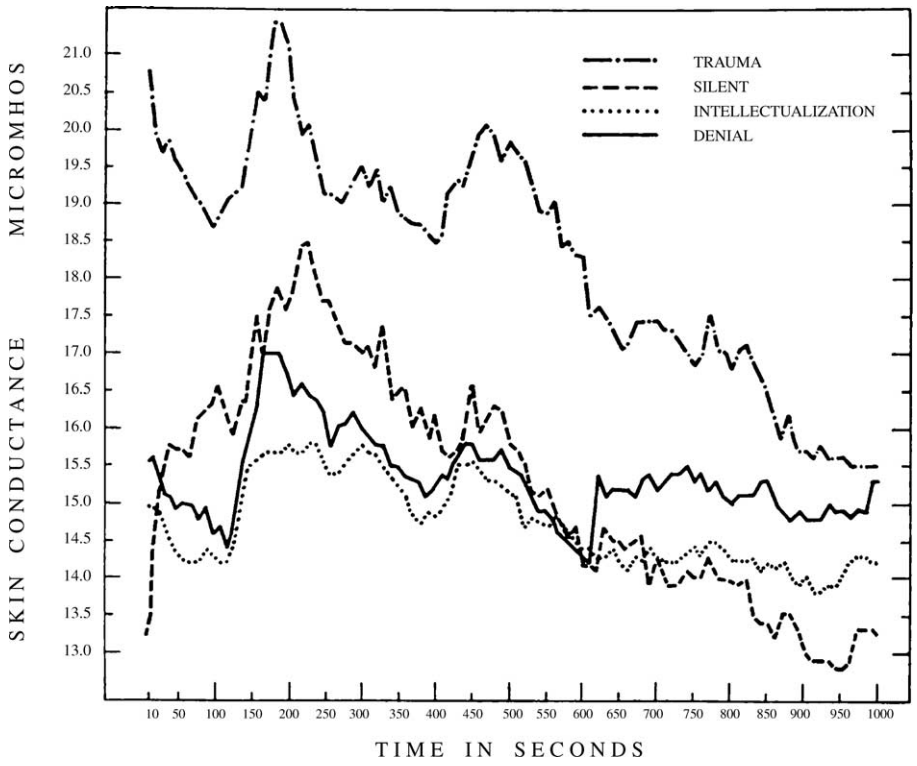

Fig. 6. Results of experiments taken as showing that cognitive appraisal is an important ingredient in emotional experience (for description, see text; from fig. 1 in Speisman et al. (1964)).

changes in skin conductance, are by themselves sufficient conditions for the identification and ascription of a given emotion such as that associated with stress. For one's bodily state subjectively experienced in terms of sensations or objectively determined in physiological terms, is not an emotion. It is only part of the syndrome of an episode of an emotional perturbation in appropriate circumstances, given the appropriate knowledge, beliefs and concerns of the agent. The measurements taken of skin conductance during the film provide only part of the somatic reaction of the individual and the appropriate knowledge, beliefs and concerns of the viewer must be carefully controlled for – no easy task! Whether these reactions are manifestations of one emotion or another, or have nothing to do with an emotion, depends upon the circumstances and on what the agent knows or believes of the circumstances in which he finds himself and upon what he cares about. We shall elaborate this point further below.

3.2. Involvement of the amygdala and the orbitofrontal cortex in the emotional responses to faces

Both animal and human studies indicate that the amygdala of the brain is involved in emotional reactions to faces (Rolls, 1995; LeDoux et al., 1990; LeDoux, 1993a, 1993b). Monkeys that have had their amygdala ablated are tame (Kluver and Bucy, 1939; Weiskrantz, 1956), and no longer make appropriate responses to signals of danger or threat. Radiotelemetry recordings of amygdala activity in monkeys during social interactions show the highest responses to ambiguous or threatening situations (for example, threatening face displays), and the lowest to tension-lowering behaviours (such as grooming and huddling; Kling and Steklis, 1976; Kling et al., 1979). Furthermore, lesions of the human amygdala are accompanied by disturbances in the recognition of fearful facial expression (Calder et al., 1996) as well as in fear conditioning (Bechara et al., 1996). All these observations point to the necessity of intact neural networks in the amygdala if animals and humans are to be able to respond fearfully in appropriate circumstances.

The orbitofrontal cortex of the brain is also involved in emotional reactions to faces and objects, for if it is damaged in primates there is considerable reduction in the normal aggression associated with fear at the sight of a human or a snake (Butter and Snyder, 1972). The orbitofrontal cortex is located in the ventral prefrontal cortex whereas the amygdala is a subcortical region in the anterior part of the temporal lobe. These areas are best identified with respect to the numbers on the cytoarchitectural map of the human cortex due to Brodmann (1909) shown in Fig. 7. Fig. 7A gives the lateral surface of the brain and Fig. 7B gives the medial surface. As the amygdala has a subcortical location it is not provided with Brodmann numbers for its location whereas the orbitofrontal cortex does and is located in Brodmann areas 11, 12, 13, and 14 which are delineated in a lateral view in Fig. 7.

There is much experimental research on the role of the amygdala and the orbitofrontal cortex in emotional responses to faces. We will therefore examine this in some

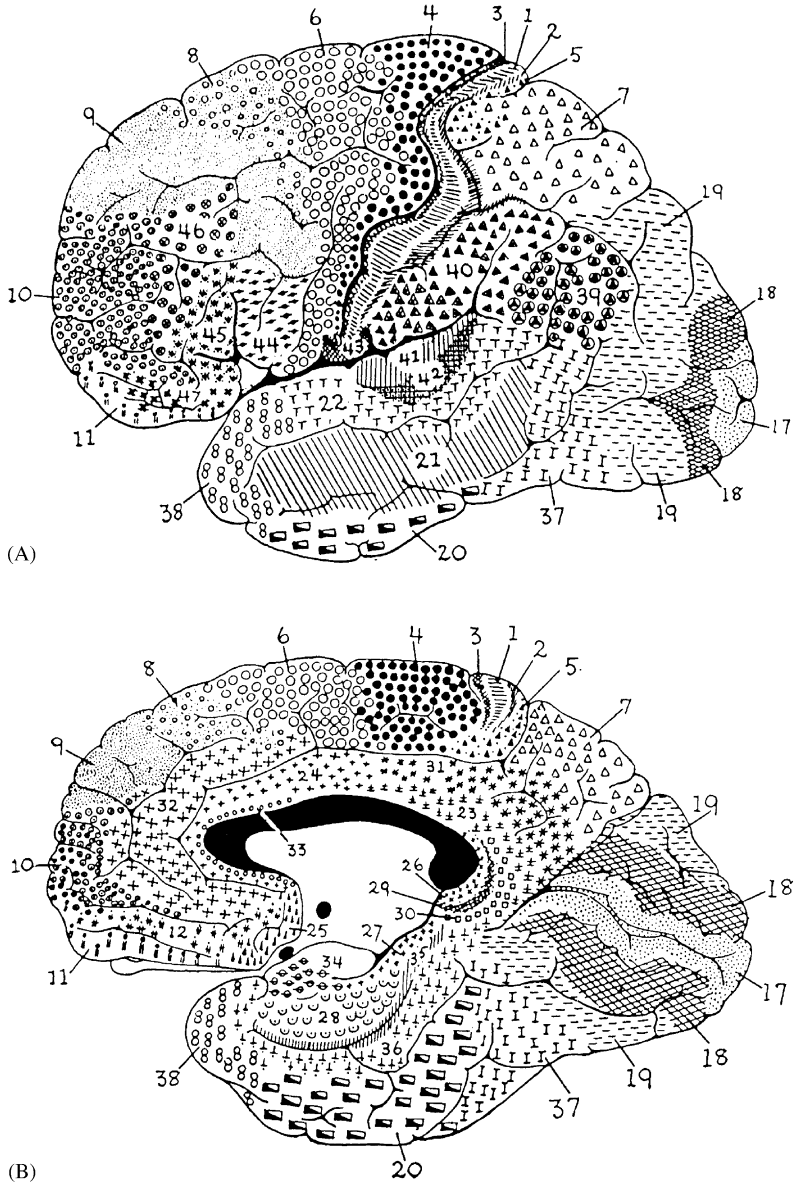

Fig. 7. Cytoarchitectural map of the human cortex (from Brodmann (1909) in Carpenter (1976)).

detail below in order to best elucidate recent conceptual developments in the study of emotion and neural activity. Activity in the amygdala and/or the orbitofrontal cortex often occurs in conjunction with enhanced activity in the anterior cingulate cortex as will be noted below. The next sections then focus on the properties of the amygdala and orbitofrontal cortex.

4. The amygdala

4.1. Faces expressing different emotions and the amygdala: PET and fMRI

There is direct in vivo evidence for a differential neural response in the human amygdala that accompanies facial expressions of fear and happiness (Morris et al., 1996). Positron-emission tomography (PET) measures of neural activity were acquired while subjects viewed photographs of fearful or happy faces, varying systematically in emotional intensity. This work showed that the neuronal response in the left amygdala is significantly greater when viewing fearful photographs as opposed to those showing happy expressions, of pleasure, or satisfaction and contentment. Furthermore, this response showed significant changes that were correlated with the intensity of emotion indicated by the expression displayed in the photographs (increasing with increasing fearfulness, decreasing with increasing contentment). The faces used in these experiments are shown in Fig. 8A. Faces a and e are prototypical neutral and fearful expressions. Faces b–d are interpolated between these prototypes. Computer morphing techniques were used to

shift the shape and pigmentation of the neutral prototype towards the fear prototype. Face b involves 25% fear (and 75% neutral), face c 50% fear (and 50% neutral) and face d 75% fear (and 25% neutral). Face f is an enhanced 125% fear expression, created by shifting the shape of the fear prototype 25% away from neutral (increasing by 25% any difference from neutral). Fig. 8B shows the results of these experiments. In 8Ba are shown the extent of regional cerebral blood flow (rCBF) values indicating the extent of activation of the left amygdala in the interaction of emotional category and intensity. The *x*-axis of the graph represents the proportion of the prototypical expression in the face stimuli, with fearful being positive (100% = 1.5) and happy negative (100% = −1.5). A regression line has been fitted to the data (with broken lines representing 95% confidence intervals for the gradient of the slope). The views of the brain are shown for orthogonal slices at the pixel of maximal activation within the left amygdala in Fig. 8Bb. This work has been taken to provide direct evidence that the human amygdala contains neural networks that function during the emotional salience of faces, with a specificity of response to fearful facial expressions. Functional magnetic resonance imaging (fMRI) studies support these PET studies in showing that there is a differential neuronal response in the amygdala to the presentation of fearful faces compared with that of happy faces (Breiter et al., 1996). It is unclear, however, what this differential response to facial expressions out of any relevant context means. Perhaps it signifies a greater sensitivity to expressions of fear of one's kin than to their expressions of contentment.

Increased neuronal activity occurs in the amygdala as well as the anterior cingulate when subjects view photos of facial expressions indicating various degrees of sadness, as shown in Fig. 9aA–aF (Blair et al., 1999). In this figure face A is taken as the 0% face expressing the neutral prototype whilst face F is the 100% prototype for sad expressions. Increasing intensity of sad facial expressions (Fig. 9aA–aF) was associated with enhanced activity in the left amygdala and right temporal pole as determined by PET (Fig. 9b). This figure shows views of the brain for orthogonal slices at the pixel of maximal activation within (A), the left amygdala and (B) the right temporal pole with significant areas of activation displayed on mean MRIs produced from the co-registered structural MRIs from all subjects.

In a separate study using fMRI, Killgore and Yurgelun-Todd (2004) asked subjects to view sad and happy faces, with each stimulus trial consisting of two rapidly presented stimuli (Fig. 10A): a 'target' face depicting either a sad or a happy emotional expression from one of eight posers and a 'mask' face consisting of a photograph of the same poser expressing neutral emotion; during each trial, the target face was presented visually for 20 ms and then immediately replaced by the neutral mask photograph for 100 ms. Each trial was separated by a 3 s interstimulus interval. Due to the brief duration of the target and its temporal proximity to the lengthier mask presentation, subjects lacked explicit

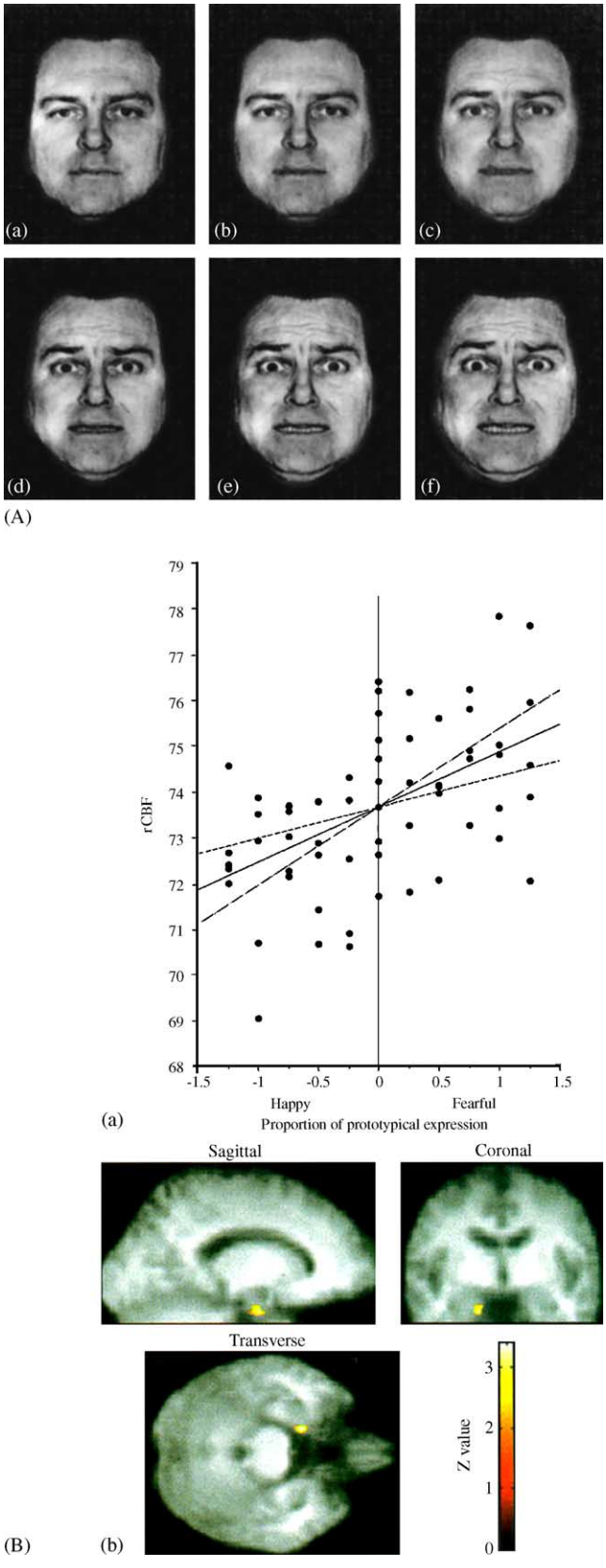

Fig. 8. Results of experiments taken as showing that neuronal responses in the left amygdala, as measured with PET, are significantly greater (B) when viewing fearful photographs as apposed to those showing happy expressions (A) (see text for further description; from figs. 1 and 3 in Morris et al. (1996)).

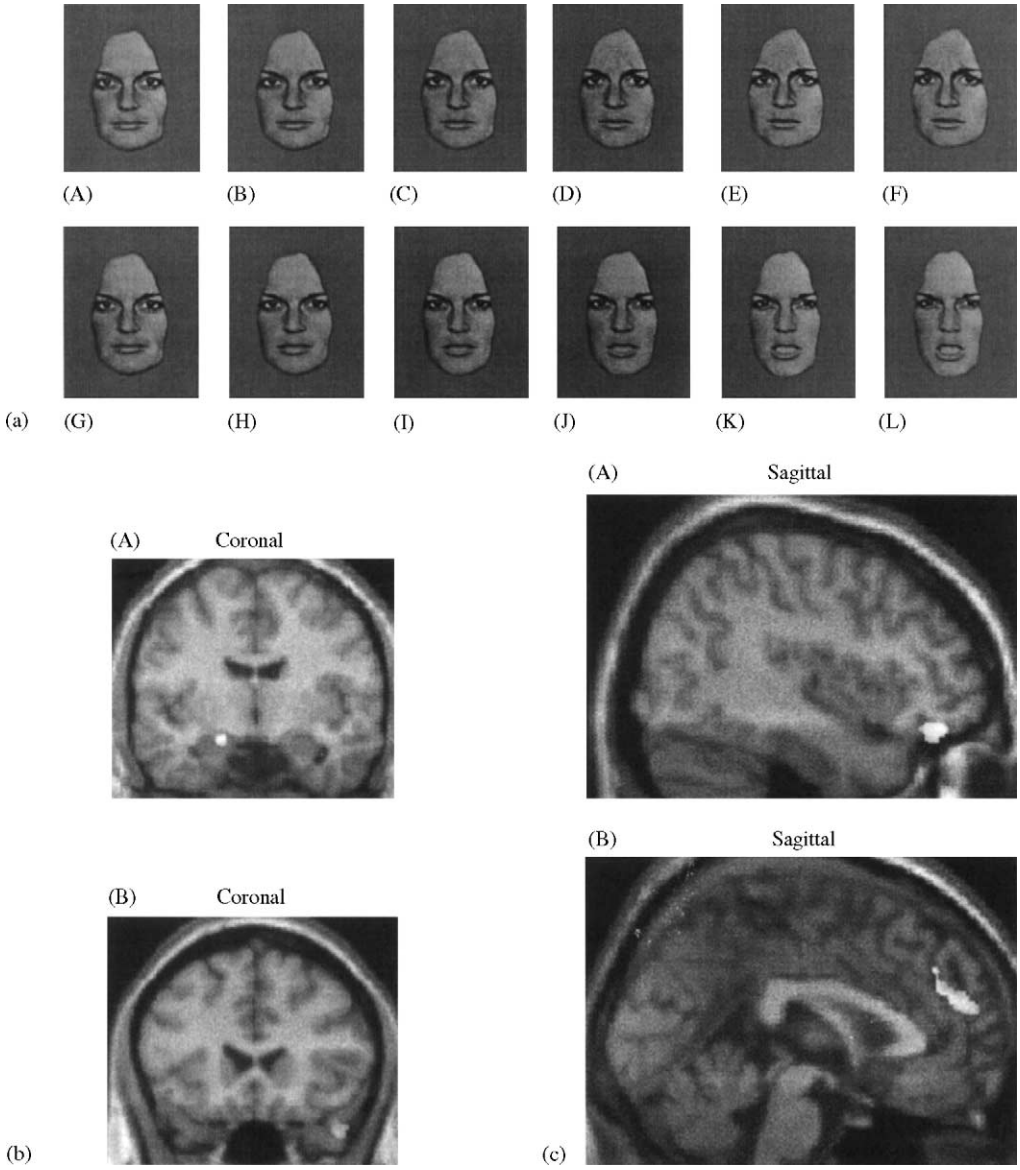

Fig. 9. Results of experiments taken as showing enhanced neuronal responses in the left amygdala (b), as measured with PET, are significantly greater when viewing increasingly sad expressions (aA–aF) whereas neuronal responses are enhanced in orbitofrontal cortex and anterior cingulate (c) when viewing increasing intensity of anger expression (aG–aL) (see text for further description; from figs. 1–3 in Blair et al. (1999)).

awareness of the mask. The results are shown in Fig. 10B in which the top row presents coronal views at the location of the anterior cingulate gyrus (areas 24 and 32 in Fig. 7B) and the bottom row a coronal view of the amygdala. The fMRI showed that masked happy faces were associated with significant bilateral activation within the amygdala and anterior cingulate gyrus whereas masked sadness yielded only limited activation. On the other hand masked sad faces are associated with significant activation within the left anterior cingulated gyrus (area 32 in Fig. 7B), but no significant activation within either amygdala (Fig. 10B). Killgore and Yurgelun-Todd (2004) conclude that the amygdala and the anterior cingulate are 'important components of a network involved in detecting and discriminating affective information presented below the normal threshold of conscious visual perception' (*that is neural networks in the amygdala and the anterior cingulate are active when subjects view happy faces for such short periods of time that they are unable to recall viewing them at all*).

4.2. Behavioural studies involving face recognition following damage to the amygdala

Contextual fear conditioning in rats involves the hippocampus, as well as the subiculum which projects to the B and AB nuclei of the amygdala (see Fig. 11), for if these are destroyed such fear conditioning is lost (see Frankland et al., 1998). This contextual fear conditioning is evident when rats not only exhibit fear in response to a particular conditioning stimulus (such as a tone), but also on

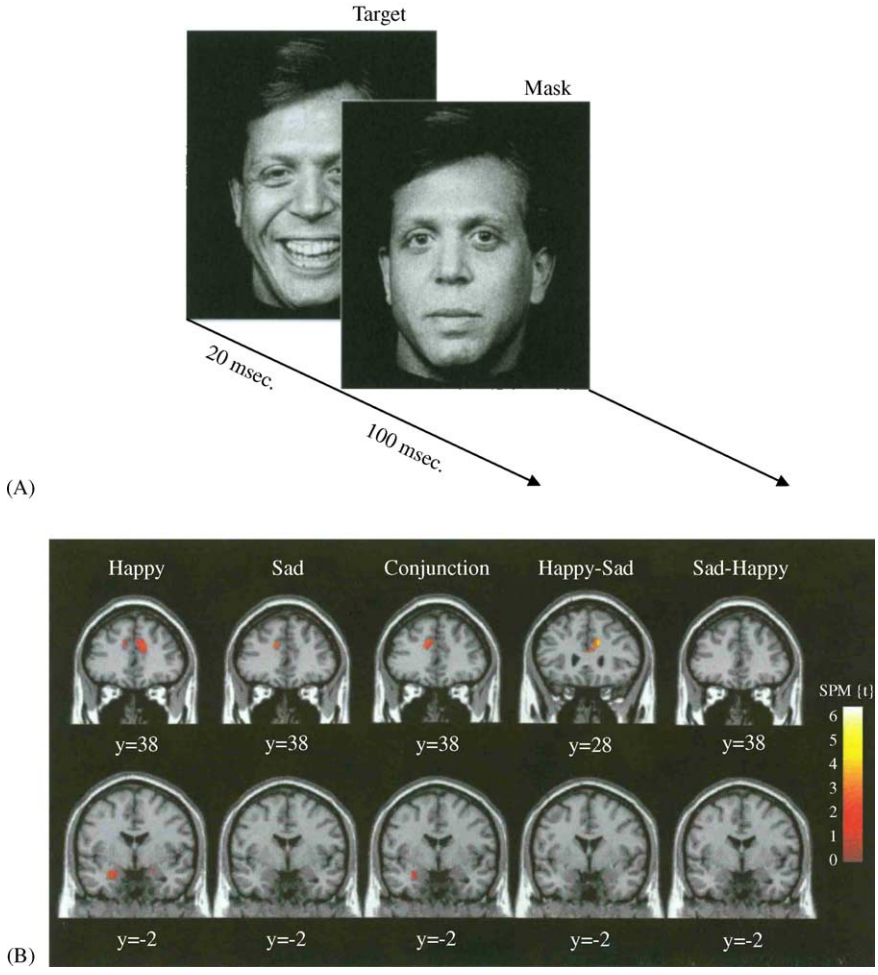

Fig. 10. Results of experiments taken as showing enhanced bilateral activation within the amygdala and anterior cingulate gyrus (B), as measured with fMRI, when viewing happy face or sad face expressions for a short period (20 ms) before these were masked with neutral facial expressions for longer periods (100 ms) (A). The top row presents coronal views at the location of the anterior cingulate gyrus and the bottom row a coronal view of the amygdala. Results labelled 'Conjunction', 'Happy-Sad' and 'Sad-Happy' have not been considered in the text but are described in Killgore and Yurgelun-Todd, 2004 (for further description, see text; from figs. 1 and 3 in Killgore and Yurgelun-Todd, 2004).

being returned to the box in which the tone and the unconditioned stimulus (such as a shock) are paired.

Bilateral removal of the amygdala in primates leads to dramatic changes in behaviour, including a lack of emotional responsiveness, tameness and eating inappropriate food (Weiskrantz, 1956). In general, such damage leads to impairments in the ability to learn associations between say a visual stimulus and a reinforcer such as pain. 'Emotional memories', sometimes referred to as 'implicit memories', are said to involve remembering what one felt in a similar situation without recollecting the previous occasion on which one experienced the emotion. 'Declarative memories', sometimes referred to as 'explicit memories', are those in which the details and circumstances of a previous occasion are recollected. Given these definitions, then (our explanatory interpretations are given in italics):

'In humans, damage to the amygdala interferes with the implicit emotional memories but not explicit memories about emotions, whereas damage to the medial temporal lobe memory system (*including the hippocampus*) interferes with explicit memories about emotions but not with implicit emotional memories (Bechara et al., 1995; LaBar et al., 1995). Although explicit memories with and without emotional content are formed by way of the medial temporal lobe system (*that is, are dependent on the integrity of the system*), those with emotional content differ from those without such content. The former tend to be longer lasting and more vivid (see Christianson, 1989; Cahill and McGaugh, 1988). At the same time, the medial temporal lobe memory system projects to the amygdala (Amaral et al., 1992). Retrieval of long-term memories of traumatic events may trigger fear reactions by way of these projections to the amygdala (*that is remembering traumatic events in the distant past may be accompanied by fear and this is dependent on the connections between the medial temporal lobe and the amygdala*)' (LeDoux, 2000, p. 175).

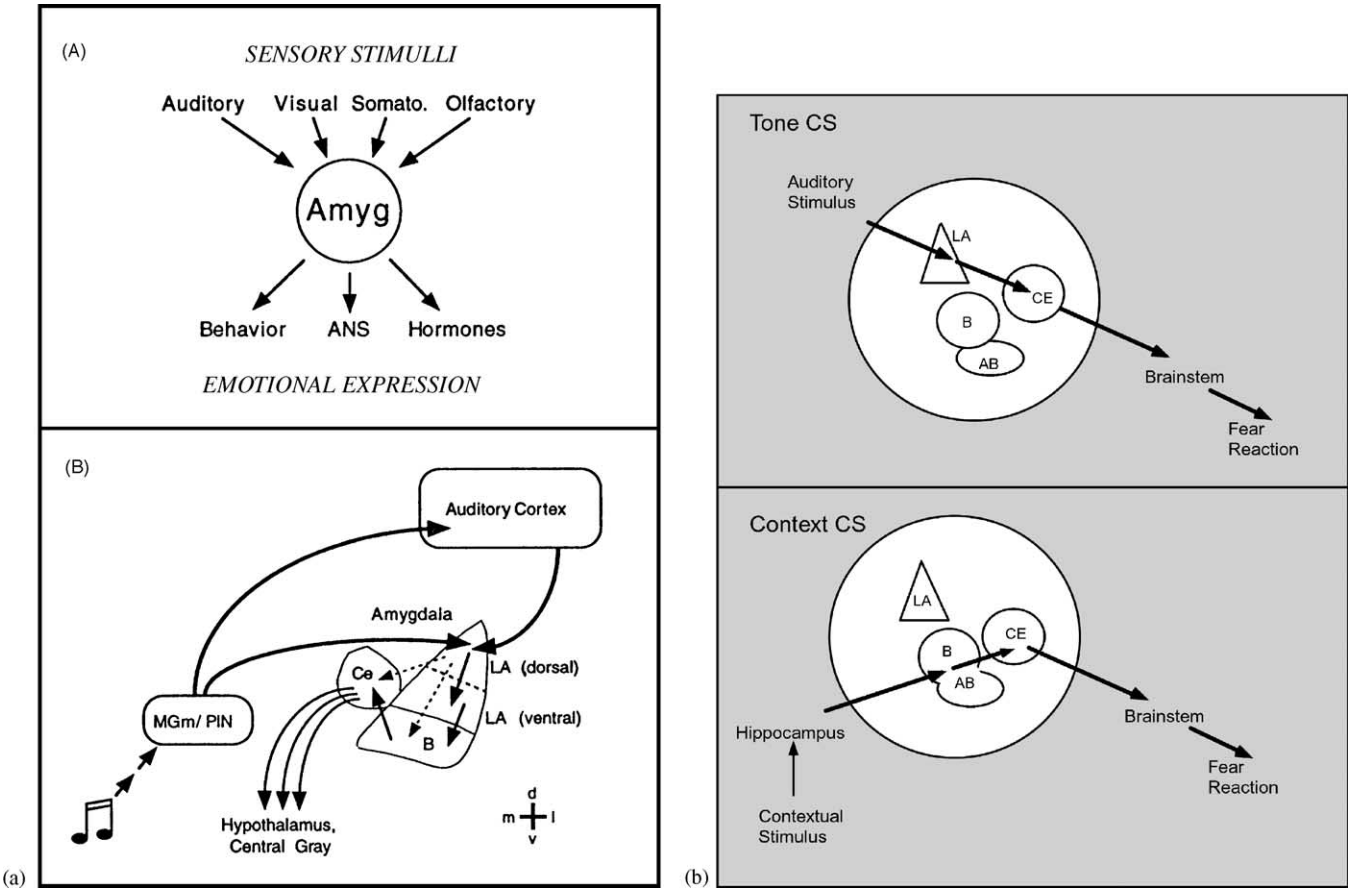

Fig. 11. Arrangement of nuclei in the amygdala and their relationship with other brain structures (for further description see text; a is from fig. 1 in Quirk et al. (1996); b is from fig. 4 in LeDoux (2000)).

4.3. Fear conditioning and the amygdala

The amygdala is important in some kinds of classical fear conditioning. LeDoux has determined in detail many of the pathways involved. These require some knowledge of the arrangement of nuclei in the amygdala (see Fig. 11aB and b). They include an output nucleus (CE, the central nucleus), together with the lateral nucleus (LA) which uniquely receives auditory input from the auditory cortex and the medial geniculate nucleus (MGn/PIN) of the thalamus as well as other sensory inputs and the basal (B) and accessory basal (AB) nuclei which receive input from the hippocampus (Fig. 11b; LeDoux et al., 1990). Thus the lateral nucleus is thought to be the means by which 'sensory information' reaches the amygdala, after which this is sent to some of the other nuclei such as the basal and the accessory basal and to the central nucleus. The amygdala then projects from the central nucleus to areas of the nervous system which are involved in different responses associated with emotion such as those involving the autonomic nervous system (e.g. hypothalamus and blood pressure) and the central (peraqueductal) grey (motor freezing response) (see Fig. 11a).

LeDoux has worked out the subcortical inputs that project to the amygdala through the medial geniculate nucleus which are necessary if conditioned fear stimuli are to affect an animal (see Fig. 11aB). The paradigm for his research follows that of Pavlov (1927). In this a conditioned stimulus (CS) which is initially neutral is temporally paired repeatedly with a stimulus which is not neutral as it produces a physiological response, the unconditioned stimulus (US). This pairing leads to a correlation between the conditioned stimulus and the unconditioned stimulus such that the physiological response is eventually evoked by the conditioned stimulus alone. In LeDoux's work the physiological measures are changes in heart rate, blood pressure and motor freezing which he interprets as indicating fear. This research, carried out mostly on rats, shows that if the conditioned stimulus is a tone and the unconditioned stimulus an electric shock to the legs, then the amygdala is implicated in the development of the conditioned stimulus fear response. Similar evidence for the amygdala being involved in fear responses is now available for primates (e.g. see Rolls, 1992).

4.4. Is cognitive appraisal an important ingredient in emotional experience? LeDoux's interpretations of his experiments on the amygdala

LeDoux has proposed that his work on the functioning of the amygdala in the conditioned fear response can help

resolve the question as to whether cognitive appraisal is an important ingredient in emotional experience, as held by Lazarus (1982) or whether affect and cognition are separate and at least partially independent systems, as proposed by Zajonc (1984). LeDoux regards the neural networks of the amygdala as essential in any animal's responding emotionally to a particular stimulus. He uses the unfortunate phrase 'emotional computer' to characterise the workings of the amygdala (LeDoux, 1992a, 1992b, 1996). The neural networks of the amygdala are necessary for an animal to feel and exhibit an emotion and the neocortex together with the hippocampus are responsible for 'cognitive processes', which is to say their networks must be functional for the appraisal of the situation which elicits an emotional response. LeDoux proposes that 'sensory information' about a stimulus that arouses an emotion is relayed simultaneously from the thalamus directly to the amygdala as well as to the cortex which then completes a circuit back to the amygdala (Fig. 11aB). The direct pathway is relatively fast-acting and is based on simple stimulus features (such as intensity). It allows rapid response to threatening situations and therefore provides for the animal's survival. The indirect pathway through the cortex is slow-acting and allows the animal to assess the situation. LeDoux suggests that the direct pathway from thalamus to amygdala provides for preconscious and precognitive 'emotional processing', that is the integrity of this pathway is required for us to experience an emotion without remembering the circumstances of the occasion in which the emotion was previously experienced. On the other hand, the indirect pathway through the cortex to the amygdala supports a postcognitive 'emotional processing', that is one which does involve recollecting such experiences. Accordingly both Lazarus (1982) and Zajonc (1984) are correct.

To what extent is the direct thalamic pathway involved in the normal fear behaviour of an animal? Roll argues that:

'It is unlikely that the subcortical route for conditioned stimuli to reach the amygdala, suggested by LeDoux (1992a, 1992b) (see also LeDoux, 1995, 1996), is generally relevant to the learning of emotional responses to stimuli. Animals do not generally want to learn that a particular pure tone is associated with reward or punishment. Instead, it might be a particular complex pattern of sounds such as vocalisation (or, for example, in vision, a face expression) that carries a reinforcement signal, and this may be independent of the exact pitch at which it is uttered. This LeDoux system (with the medial geniculate) may not reflect the way in which auditory-to-reinforcement pattern associations are normally learned' (Rolls, 1999, p. 104).

4.5. Fear is unrepresentative of the emotions

Although fear is a ubiquitous aspect of the animal, and hence too human, condition, it is a poor representative. For there are many emotions which typically involve little, if any, emotional perturbation or disturbance; for example humility, respect, admiration, contempt and gratitude. Indeed, not all instances of fear need involve fearful agitation, not because the fear is slight, but because of the character of the object of fear. What one is afraid of may preclude any particular, or at least any intense, fearful perturbation. Fear of imminent physical danger obviously involves perturbation. But fear of global warming need not – even though the ensuing motivation may be powerful, and the effect on the agent's mood may be substantial. Similarly, the depth of a person's remorse may be exhibited not in a syndrome of sensations and perturbations that he feels, but rather in his strenuous endeavours to make amends for his past actions and in his obsessive thoughts about his offence. Furthermore, it would be mistaken to suppose that the sole, or even privileged, measure of intensity of fear (or other emotions) is the intensity of emotional perturbation, expression, or neural stimulation caused. For this would wholly obscure the motivational force of the emotions. The intensity of an acrophobe's fear of heights, is exhibited above all in the lengths he will go to avoid heights. The intensity of a person's hatred is much more likely to be shown in the actions he plans to harm the object of his hatred that in the emotional perturbations he feels or the neural concomitants in his brain when in the presence of the object of his hatred.

Emotions evolved as animal responses to features of the environment apprehended as affecting in one way or another the good of the animal. Neither brain states (which are essential for feeling an emotion) nor somatic responses (which may characterise an emotional perturbation) are emotions. They lack the intentionality, or 'directness towards an object', which is constitutive of most emotions. One cannot individuate an emotion by reference to either brain states or somatic reactions independently of the circumstances of their occurrence and the knowledge or beliefs, as well as the desires or wishes, of the creature.

5. The orbitofrontal cortex

5.1. Behavioural studies involving face recognition following damage to the orbitofrontal cortex

As noted above, damage to the orbitofrontal cortex in primates leads to reduced aggression to sighted objects that would normally produce fear, such as a human being or a snake (Butter and Snyder, 1972). Furthermore, neurons that fire maximally when the animal sees a face are found in the orbitofrontal cortex, as shown in Fig. 12. This figure shows the firing rate of impulses for the neuron recorded from the orbitofrontal cortex in the left hand column when the macaque monkey was presented with one of the four images in the right hand column at time zero. It is clear that the neuron responds best when the macaque looks at a face (a), responds to a lesser degree to the other face presented (b), and does not respond to non-face stimuli, such as those in (c)

and (d) (results from Rolls et al., 1998). Following injury to this area of the brain in humans, social conventions are ignored and patients are unable to make appropriate plans for future action. These changes occur without impairment of intellectual functions or of memory and learning (Bechara et al., 2000).

5.2. *The orbitofrontal cortex and face recognition: PET and fMRI*

To investigate the possible significance of the effect of observing faces on the firing of orbitofrontal visual neurons described above, humans with damage to the ventral part of the frontal lobe have had their responses to faces tested. Impairments in the identification of facial emotional expression were demonstrated in a group of patients, with ventral frontal lobe damage, who had socially inappropriate behaviour (Hornak et al., 1996). Fig. 13A shows an fMRI scan of the lesion in such a patient, with the dark patch on the left of the figure centred at just 43 mm anterior in the right orbitofrontal region. The impairments in expression identification could occur independently of perceptual impairments, as the following experiments show. These consisted of presenting photographs of the following expressions: sad, angry, frightened, disgusted, surprised, happy and neutral. Patients were asked to choose from a list the adjective best describing the facial expression in each photograph. In order to then obtain evidence on whether there was a relation between problems in identifying facial expression and subjective emotional changes since their illness or injury, patients were asked whether there was any general change in their ability to experience emotion, in

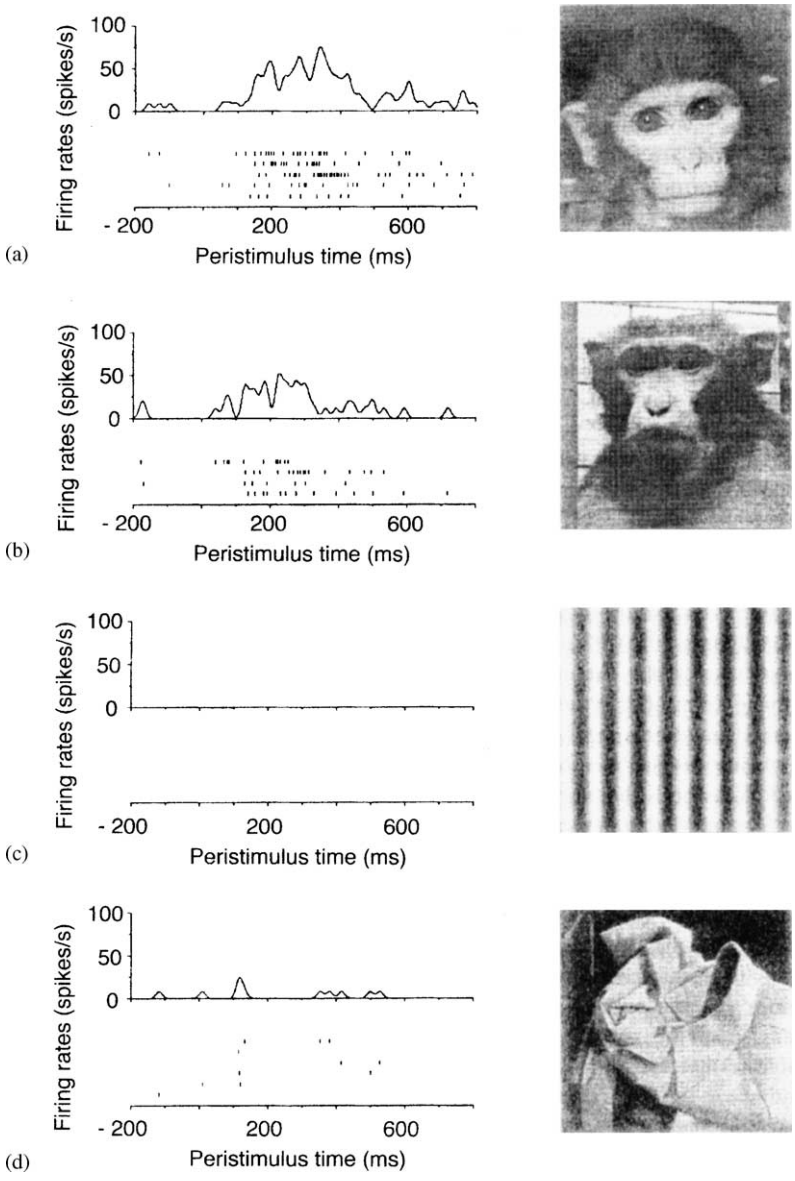

Fig. 12. Neurons exist in orbitofrontal cortex of macaque monkeys that fire maximally when the monkey sees a face (for descripton see text; from fig. 4.21 in *The Brain and Emotion* by Rolls (1999)).

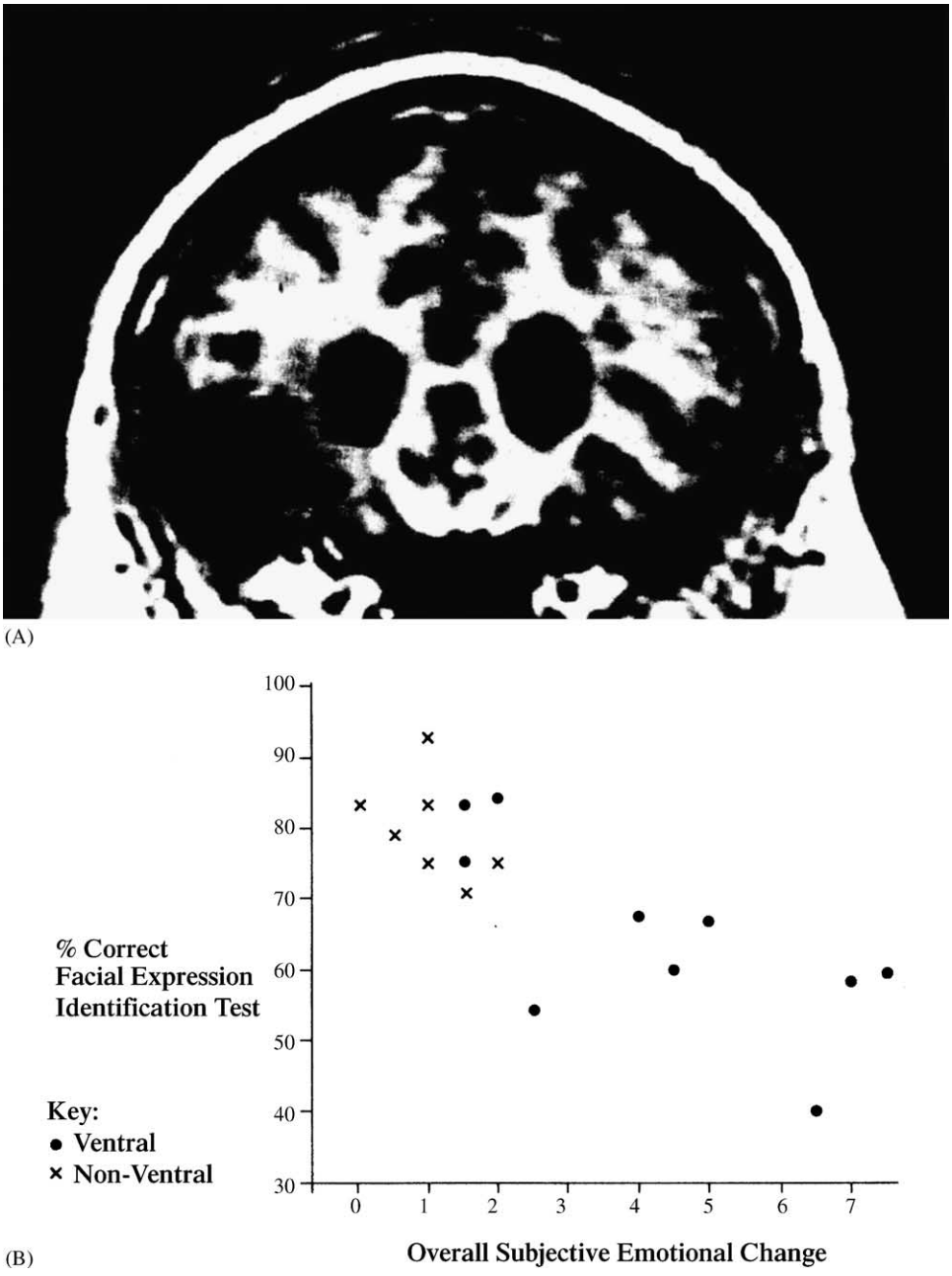

Fig. 13. Evidence that the orbitofrontal cortex of humans possesses neurons whose integrity must be intact for a human to correctly identify a facial expression: (A) fMRI scan of a patient with a right orbitofrontal lesion; (B) graphs showing changes in patients ability to correctly identify facial expressions (for further descriptions, see text; from fig. 1 in Hornak et al., 1996 and fig. 4.23 in *The Brain and Emotion* by Rolls (1999)).

particular the intensity or frequency of the following emotions: sadness (or regret), anger (or frustration), disgust (physical revulsion), excitement or enjoyment. Positive scores were given for changes in either direction. Fig. 13B shows that in such patients there is gradual decrease in their capacity correctly to identify facial expressions with an increase in their assessment of their own capacity to experience emotion. The demonstration that the identification of facial expression may be impaired independently of any difficulty in recognising the identity of faces is also consistent with what has been found in other studies.

5.3. Orbitofrontal cortex and the satisfying of appetites: Rolls's interpretation of his experiments on the orbitofrontal cortex

Rolls suggests that the neural networks of orbitofrontal cortex are implicated in the ability of animals to determine what he calls the 'reward value of food'. He and his colleagues have shown that if monkeys are gradually fed to satiety then the firing of taste neurons in their orbitofrontal cortex is gradually reduced to zero (Rolls et al., 1989). This is interpreted as showing that 'primates work to obtain firing of

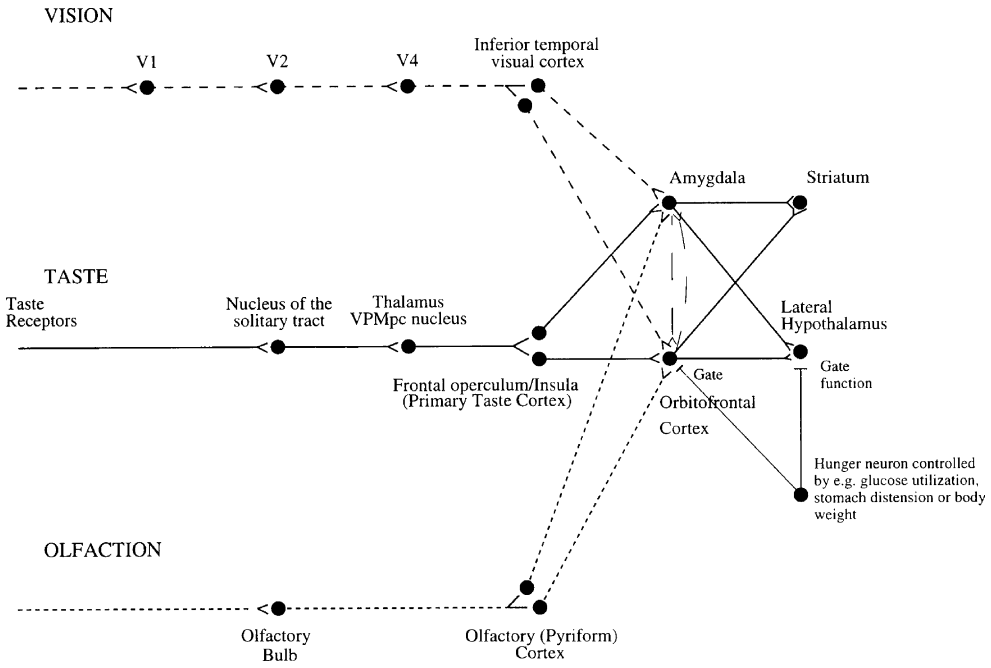

Fig. 14. Diagram providing a representation of some of the connections to and from the orbito-frontal cortex (from fig. 7.2 in *Neural Networks and Brain Function* by Rolls and Treves (1998)).

these neurons, by eating food when they are hungry' (Rolls, 1999, p. 32), but of course the primates can know nothing about these neurons. Rather as the animals feed less the firing of such neurons declines showing that interest in eating the food, taken as indicating the 'reward value' of the food, is correlated with activity of neurons in the orbitofrontal cortex. Rolls suggests that this idea is supported by experiments in which electrodes are placed in the orbitofrontal cortex of a primate which is then allowed to stimulate these at will. If the animal is gradually fed to satiety the extent of self-stimulation gradually falls away, indicating to Rolls and his colleagues that there must be neurons in this part of the cortex that fire in relation to the extent of satiety (Mora et al., 1979; Rolls et al., 1980). Experiments like these also indicate the existence of neurons which, according to Critchley and Rolls (1996), are involved in the 'reward value' of visual stimuli, such as the sight of food, that is they fire maximally under suitable conditions involving the sight of foods. The projection of the ventral visual pathway to the orbitofrontal cortex (Fig. 14) together with that from the primary taste cortex in the insular (Fig. 14) is required for this linkage to be possible.

5.4. Misconceptions about emotions and appetites

The research of Rolls described above does not distinguish between emotions and appetites. Rolls considers, as indicated above, that paradigmatic examples of emotions for experimental research are to be found in thirst, hunger and lust. These are appetites and not emotions. In what sense are appetites unlike emotions? First, emotions are not linked to localised sensations in the same way. Some emotions are associated with sensations (fear, rage), others are not (pride, remorse, envy). One does not have a feeling of pride in one's stomach or in one's chest, and although there are sensations characteristic of occurrent anger, such as throbbing temples and tension, one does not feel anger in one's temples or stomach muscles as one feels hunger in one's belly. Second, emotions have not only formal objects, in the sense that what one fears is what is thought to be frightening or harmful, they have specific objects, as when one fears tomorrow's examination or feels remorseful about lying to Daisy. Third, the intensity of emotions is not proportional to the intensity of whatever sensations may accompany their occurrent manifestation. How much one fears heights may be manifest in the lengths one goes to avoid them. Fourth, emotions do not display the pattern of occurrence, satiation and recurrence characteristic of the appetites described by Rolls, for the obvious reason that they do not have the same kind of physiological and hormonal basis as the appetites. Fifth, the emotions have a cognitive dimension absent from the appetites. The hungry animal wants food, the thirsty animal wants drink, but no particular knowledge or beliefs are essentially associated with these appetites. By contrast, the frightened animal is afraid of something it knows or believes to be dangerous. Finally, many human emotions are exhibited by characteristic facial expressions and manifested in typical tones of voice – as in the case of fear, anger, love and affection. Appetites are not.

6. Neural networks: amygdala and orbitofrontal cortex in vision

In this section we shall consider the networks of connections of the amygdala and orbitofrontal cortex. In

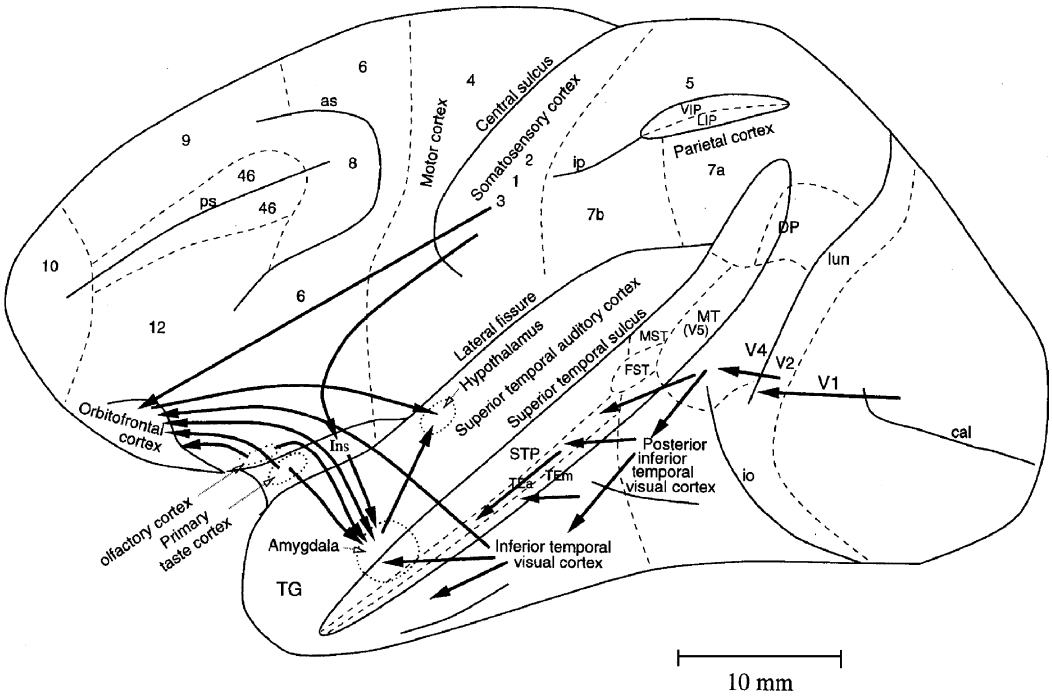

Fig. 15. Connections between the primate amygdala and oribito-frontal cortex and the origins of projections to these centres (for further description, see text; from fig. 4.1 in *The Brain and Emotions* by E.T. Rolls (1999)). The abbreviations on the figure are as follows: as, arcuate sulcus; cal, calcarine sulcus; cs, central sulcus; lf, lateral (or Sylvian) fissure; lun, lunate sulcus; ps, principal sulcus; io, inferior occipital sulcus; ip, intraparietal sulcus (which has been opened to reveal some of the areas it contains); sts, superior temporal sulcus (which has been opened to reveal some of the areas it contains); AIT, anterior inferior temporal cortex; FST, visual motion processing area; LIP, lateral intraparietal area; MST, visual motion processing area; MT, visual motion processing area (also called V5); PTI, posterior inferior temporal cortex; STP, superior temporal plane; TA, architectonic area including auditory association cortex; TE, architectonic area including high order visual association cortex, and some of it's subareas TEa and TEm; TG, architectonic area in the temporal pole; V1–V4, visual areas 1–4; VIP, ventral intraparietal area; TEO, architectonic area including posterior visual association cortex. The numerals refer to architectonic areas, and have the following approximate functional equivalence: 1–3, somatosensory cortex (posterior to the central sulcus); 4, motor cortex; 5, superior parietal lobule; 7a, inferior parietal lobule, visual part; 7b, inferior parietal lobule, somatosensory part; 6, lateral premotor cortex; 8, frontal eye field; 12, part of orbitofrontal cortex; 46, dorsolateral prefrontal cortex.

the overview of these networks provided in Fig. 15, connections are shown to the amygdala and the orbitofrontal cortex from the ventral visual stream including V1 to V2, V4 and the inferior temporal visual cortex as well as connections from the primary taste and olfactory cortices. In addition, connections are shown from the somatosensory cortical areas 1, 2 and 3 that reach the orbitofrontal cortex directly as well as via the insular cortex.

6.1. Amygdala

The principal reciprocal projections from the cortex to the amygdala, a subcortical region in the anterior part of the temporal lobe are shown in Fig. 16. Note in particular the reciprocal connections from the anterior cingulate (area 24) and the orbitofrontal cortex (areas 11, 12, 13 and 14). Principal outputs of the amygdala include those to the periaqueductal grey in the brain stem which controls defensive and aggressive behaviour, the hypothalamus which is involved in the control of blood pressure and other autonomic activities, the basal nucleus of Myenert concerned with startle responses and the ventral striatum (Fig. 11aB). There are outputs from the central nucleus of the amygdala, which if damaged leads to a loss of virtually all manifestations of conditioned fear, such as changes in heart rate and blood pressure, in hormone release and freezing behaviour.

There are neurons in the primate amygdala which respond to visual, auditory, olfactory, somatosensory and gustatory stimuli (Fig. 9aA; see Sanghera et al., 1979). It is not surprising then that direct inputs to the amygdala are from the most sophisticated areas of sensory integration, such as the inferior temporal visual cortex, the cortex in the superior temporal sulcus (see the arrows in Fig. 15), as well as from the superior temporal auditory cortex (not shown in Fig. 15). The amygdala also receives important inputs from cortical areas concerned with touch in the somatosensory cortex via the insula as well as concerned with taste, such as the primary taste cortex and the secondary taste cortex in the orbitofrontal cortex (Fig. 15). In addition, there are a number of subcortical inputs to the amygdala, such as the hippocampus, the subiculum which carries the output from the hippocampus, the hypothalamus, thalamic nuclei as well as the nucleus of the solitary tract which is an important centre for control of the autonomic nervous system.

Whilst there are neurons in the amygdala that respond to a range of sensory stimuli, as noted above, there are no

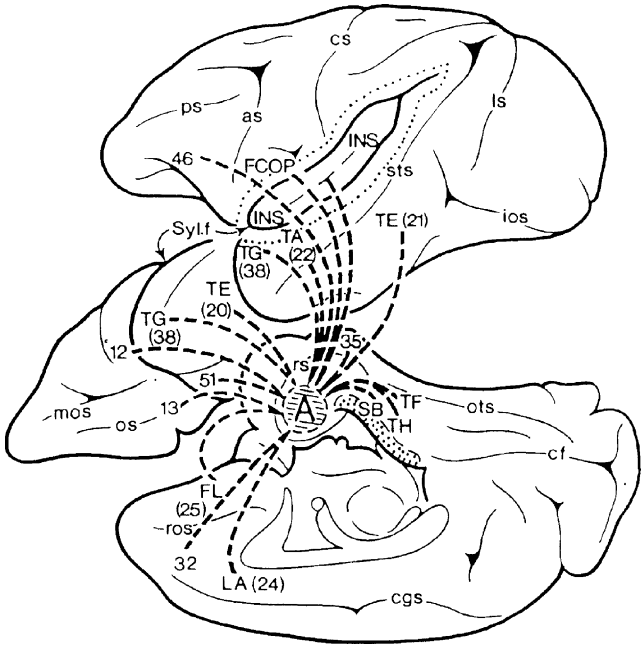

Fig. 16. Principal reciprocal projections from the primate cortex to the amygdala. Abbreviations are: as, arcuate sulcus; cc, corpus callosum; cf, calcarine fissure; cgs, cingulate sulcus; cs, central sulcus; ls, lunate sulcus; ios, inferior occipital sulcus; mos, medial orbital sulcus; os, orbital sulcus; ots, occipito-temporal sulcus; ps, principal sulcus; rhs, rhinal sulcus; sts, superior temporal sulcus; lf, lateral or Sylvian fissure (which has been opened to reveal the insula); A, amygdala; INS, insula; T, thalamus; TE (21), inferior temporal visual cortex; TA (22), superior temporal auditory association cortex; TF and TH, parahippocampal cortex; TG, temporal pole cortex; 12, 13, 11, orbitofrontal cortex; 35, perihinal cortex; 51, olfactory (prepyriform and periamygdaloid) cortex; for further description, see text; from fig. 10 in *The Amygdaloid Complex* in INSERM Symposium No. 20 (1981)).

neurons in this part of the brain that are excited solely by what Rolls calls 'reward stimuli' (for elucidation of this concept see below), for neurons that do respond to such stimuli also respond to sensory stimuli. However, as described below, the orbitofrontal cortex does possess neurons that are exclusively excited by reward stimuli.

6.2. Orbitofrontal cortex

The orbitofrontal cortex receives input from the ventral or object-identifying stream in the inferior temporal cortex, the primary taste and olfactory cortex in the insula and the somatosensory cortex (Brodmann areas 1, 2, and 3). It receives connections from a wide range of cortical and subcortical regions of the brain, including the olfactory, auditory, somatosensory, taste and visual cortices as well as the amygdala. Fig. 17 shows the cortical connections to the orbitofrontal cortex of the monkey brain, with lateral, ventral and medial views presented (the abbreviations in this figure are the same as those for Figs. 15 and 16). Fig. 16 shows the reciprocal connections between orbitofrontal areas and the anterior cingulated (area 25). These various inputs to the orbitofrontal cortex reflect, in part, the kinds of neurons which single unit studies reveal in the orbitofrontal cortex. Thus taste and olfactory neurons are found in medial and anterior parts of the primate orbitofrontal cortex, with some neurons activated by both olfactory and gustatory stimuli (see Fig. 12), indicating that they might be excited by the flavour of food (Rolls and Baylis, 1994). Furthermore, many of the neurons here are excited by visual stimuli as well as olfactory or taste stimuli (Rolls and Baylis, 1994). This might reflect the fact that (our italics): 'the orbitofrontal cortex has developed greatly in primates in learning about which visual stimuli have the taste and smell of food (*that is the neural networks of the relatively large orbitofrontal cortex of primates may be necessary in order for such animals to be able to learn which visual stimuli have the taste and smell of food*)' (Rolls, 1999).

7. The origins of emotional experience

7.1. The claims of LeDoux

The concept of 'working memory' plays an important part in LeDoux's ideas on the relation between activity in the brain and emotional experience. Goldman-Rakic and her colleagues argue that the neural networks involved in working memory support the capacity to hold an item of information for several seconds so that it may be utilised for the temporal integration of present sensory stimuli with those involving a memory of recent past occasions. Such networks in prefrontal cortex possess neurons which exhibit sustained tonic impulse activity when triggered by the brief presentation of a stimulus in contrast to sensory neurons that fire only during presentation of the stimulus. This integration involves interaction between the dorsolateral prefrontal cortex (taken as areas 12, 45 and 46; see Fig. 7; also Figs. 16–18) and the anterior cingulate (area 24) as well as areas (such as those in the inferior temporal cortex) required to identify an object visually. Other areas involved include the inferior parietal cortex (in order to enable the animal to discern the spatial arrangement of objects) together with the hippocampus of the temporal lobe (required in order for the animal to retain long-term memories) (Fuster, 1998; Goldman-Rakic, 1996; Levy and Goldman-Rakic, 2000; Braver et al., 1997; Carter et al., 2003).

LeDoux suggests that the activity in the neural networks involved in working memory is modulated by an animal's experiencing affectively charged events, such as those involving fear and hunger. This requires the integrity of projections from the amygdala and the orbitofrontal cortex to, for example, the anterior cingulate. In this way (our italics): 'working memory will become aware of the fact that the fear system of the brain has been activated *(that is, the neural networks involved in 'working memory' receive inputs that are activated by those parts of the brain whose integrity is required for an animal to feel fear)*'(LeDoux, 2000, p. 176). LeDoux further claims that (our italics): 'you

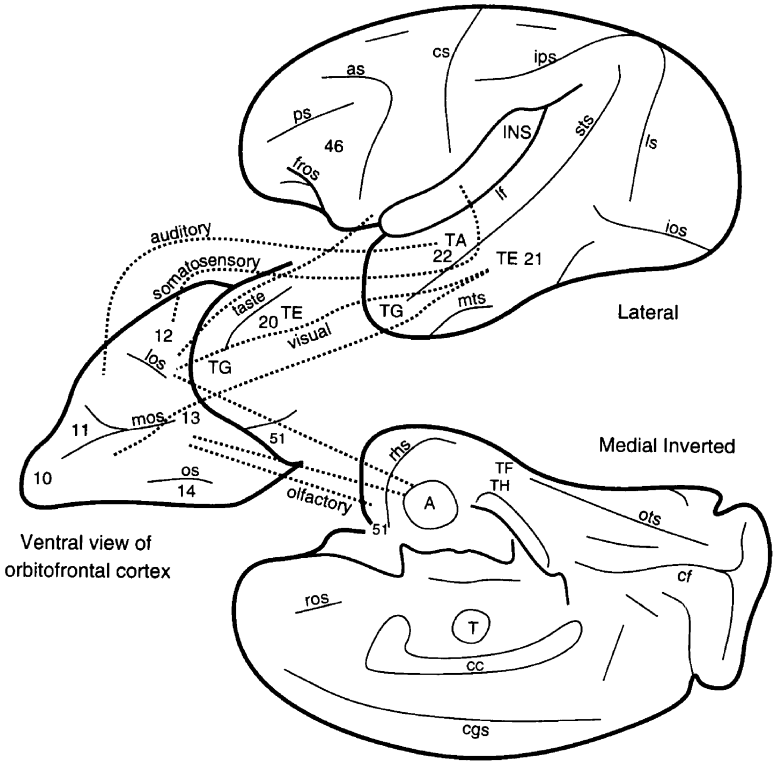

Fig. 17. Principal reciprocal projections from the primate cortex to the orbito-frontal cortex are shown (the abbreviations are the same as those in Figs. 15 and 16; from fig. 7.8 in *Neural Networks and Brain Function* by Rolls and Treves, 1998).

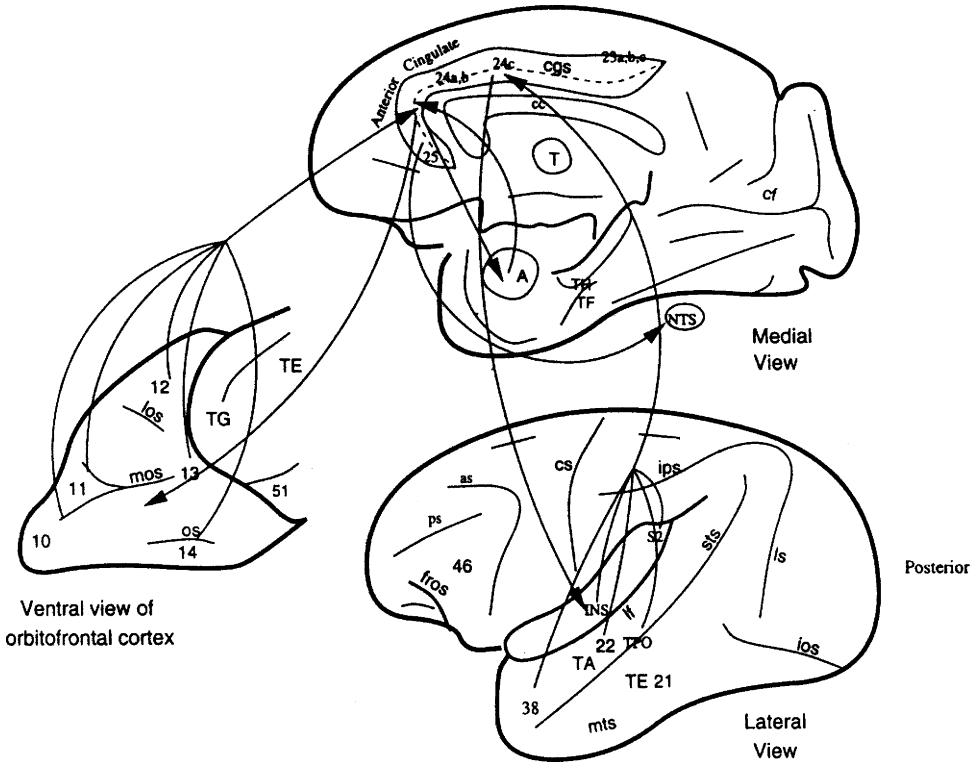

Fig. 18. Principal connections of the anterior and middle cingulate cortical areas (the abbreviations are the same as those in Figs. 15 and 16; From fig. 4.24 in *The Brain and Emotions* by Rolls (1999)).

can't have a conscious emotional feeling of being afraid without aspects of the emotional experience being represented in working memory. Working memory is the gateway to subjective experiences, emotional and non-emotional ones, and is indispensable in the creation of conscious emotional feeling *(that is, the neural networks which underpin 'working memory' must be active for one to have an emotional experience such as being afraid)*' (LeDoux, 1998, p. 276).

7.2. The claims of Rolls

Rolls suggests that 'the likely places where neuronal activity is directly related to the felt emotion' (Rolls, 1999, p. 73) are the orbitofrontal cortex in addition to the amygdala, together with those parts of the brain that receive connections from them. For example, he suggests that after identification of an object, which is dependent on the inferior temporal cortex, the amygdala and orbitofrontal cortex are required in order for the animal to associate the object with a punishment or a reward. Once this is done there is alteration in the activity of other parts of the brain by the output from these structures to the anterior cingulate (causally responsible for working memory) and to regions involved in determining autonomic responses in the hypothalamus and brainstem (Rolls, 1975; Rolls, 1993). This idea is similar to LeDoux's, except for the emphasis on reward and the functioning of the orbitofrontal cortex, rather than on fear and the functioning of the amygdala.

7.3. The claims of Damasio following James

William James, both in his *The Principles of Psychology* (James, 1890) and in his subsequent book *The Emotions* (James, 1922), written together with C.G. Lange, propounded a highly influential theory of emotions. James asserted that one could not attribute fear, for example, to a person if that person did not have a heightened pulse-rate, were not breathing shallowly, trembling, exhibiting gooseflesh and visceral stirring. Focusing exclusively upon emotional perturbations accompanying occurrent episodic emotion, James held that what excites an occurrent emotion is the *direct* cause of bodily changes, and the emotion is our own *perception* of those changes while they occur. Contrary to our common conceptions, it is not that we lose our fortune, are sorry and therefore cry, or meet a bear, feel afraid and therefore tremble, but rather we lose our fortune and cry and *therefore* feel sorry, meet a bear, tremble and *therefore* feel afraid. An emotion, according to James, is not the bodily and physiological perturbation consequent on apprehension of 'an exciting fact', but rather the person's *perception* of these somatic perturbations. We feel *emotions* because we perceive our bodily reactions. He therefore suggested that there are three essential steps in the production of an emotion. The first of these involves the initiation of particular visceral, vascular or somatic activities, for example, changes in the movement of the intestines, of blood pressure and heart rate and of skeletal muscles involved in locomotion and the defence reaction. In the second step, these changes are detected by peripheral sense receptors associated with each of these organs and muscles, and signals from these receptors transmitted to the brain. Here, in the third step, the brain generates activity which is necessary for feeling an emotion. For the emotion *is* the subjective apprehension of the relevant somatic changes.

Damasio (1994) has built on the Jamesian theory in his 'somatic marker hypothesis'. He conceives of an emotion as the collection of bodily changes in response to 'thoughts'. His conception of thoughts is rooted in the empiricist tradition. In his view, thoughts are visual or auditory mental images. The images constituting thoughts are comparable to the images that, according to him, constitute perceptions, differing from them primarily in being fainter. An emotion, he claims, is simply 'a collection of changes in a body state connected to particular mental images [thoughts] that have activated a specific brain system'. But he sharply distinguishes between *an emotion* and *feeling an emotion*. Feeling an emotion is a cognitive response to the cause of the emotion (namely the image or thought that excited the bodily response) coupled with the realisation of the causal nexus between the 'image' and the resultant somatic state (or the 'image' of such a state). So, feelings of emotion 'are first and foremost about the body, they offer us *the cognition of our visceral and musculoskeletal state* as it becomes affected by preorganised mechanisms and by the cognitive structures that have developed under their influence' (Damasio, 1994, p. 159*)*. Such feelings give us a glimpse of what is going on in our body in association with specific mental causes of those feelings, namely thoughts or images of other objects or situations. Accordingly, Damasio propounds his somatic marker hypothesis. The hypothesis is that somatic responses to 'images' serve to increase the accuracy and efficiency of decision processes, screening out certain options and allowing the agent to choose from among fewer. Further, 'when a negative somatic marker is juxtaposed to a particular future outcome the combination functions as an alarm bell. When a positive somatic marker is juxtaposed instead, it becomes a beacon of incentive' (Damasio, 1994, p. 174). The disposition to respond to 'images' (thoughts, perceptions, etc.) somatically in these ways was, Damasio suggests, 'probably created in our brains during the process of education and socialisation, by connecting specific classes of stimuli with specific classes of somatic state' (Damasio, 1994, p. 177). Roughly speaking, culturally inculcated 'gut-reactions' provide the basis for rational decision-making. In summary:

'When subjects face a situation for which some factual aspects have been previously categorised, the pertinent dispositions are activated in higher-order association cortices. This leads to the recall of pertinently associated facts which are experienced in imagetic form. At the same time, the related ventromedial prefrontal linkages are also

activated, and the emotional disposition apparatus is competently activated as well. The result of those combined actions is the reconstruction of a previously learned factual-emotional set' (Bechara et al., 2000, p. 297).

The somatic marker hypothesis leads Damasio to conjecture that the decision-making and executive deficiencies in patients suffering from lesions in the prefrontal cortices is explicable by reference to lack of somatic markers to guide them. More generally, emotions are essential for rational practical reasoning.

7.4. Misconceptions concerning the somatic marker hypothesis of James/Damasio

Damasio's theory of the emotions is a modification of James's. But James's theory is sorely defective from an analytic point of view, and cannot serve as the conceptual framework for the experimental and neurological investigation of the emotions.

James screens out all consideration of the emotions as long-standing attitudes, focusing exclusively upon occurrent emotions – passing episodes of emotional perturbation. This narrowing of focus led him to overlook crucial conceptual links that would perhaps have been more evident had he also examined emotional attitudes with their patent connection with motivation. He did not distinguish between the object of an emotion and its cause, failing to see that what makes us frightened (e.g. a noise in the night) need not be what we are frightened of (e.g. a burglar), hence too failed to see that while we may often not know the cause of our emotion (e.g. what made us angry), we must, at least in non-pathological cases, know the object of our emotion (who we are angry with or what we are angry about). Failing, as he did, to discriminate the object from the cause of an emotion, James also failed to see that there are not only causes of an emotion, but also reasons for an emotion – these being bound up with the character of the object of the emotion (if what we are frightened of (a snake) is indeed dangerous, then our fear is well founded, i.e. is supported by good reasons – if what we are frightened of is harmless (a mouse), then our fear is unwarranted). Human emotions can be reasonable, justifiable or unjustifiable – for they characteristically involve an element of judgement and appraisal. But apprehension of involuntary somatic perturbations can be neither reasonable nor unreasonable.

James made much of the alleged fact that one cannot feel an emotion without apprehending somatic perturbation. This is doubtful – feeling proud of an achievement involves no distinctive apprehended perturbations, feeling respect for another person entails no bodily agitations, and feeling gratitude to another for a favour done does not imply racing pulses or melting bowels. But even if we grant James the fact that typically episodic emotions involve somatic perturbations it nevertheless remains true that he completely overlooked that fact that one cannot (logically) feel an emotion the object of which is something about which one is altogether indifferent. Our emotions, in particular our emotional attitudes, show what we care about. That is why they are also logically bound up with our character traits (a loving disposition, a compassionate nature, an irascible temperament).

It should be evident that the mere apprehension of somatic changes consequent upon the perception of 'an exciting fact' is neither necessary nor sufficient for feeling an occurrent emotion. It is not sufficient, since to feel seasick in response to the perceived motion of the ship in which one is travelling is not to feel any emotion. It is not necessary, since to feel proud of one's children need involve no somatic perturbations. Having overlooked the essential difference between the cause and the object of an emotion, James cannot differentiate between emotions that may differ *only* with respect to their objects, e.g. shame and embarrassment, resentment and indignation, or remorse and regret. The somatic perturbations that accompany resentment may be identical with those accompanying indignation, but one feels resentful at the infringement of one's own rights, indignant at the infringement of the rights of others.

James screened out emotions understood as long-standing attitudes, focusing exclusively on emotional episodes and obscuring the connection between emotion and motivation. This is anything but a trivial and readily remediable oversight. For emotional attitudes are not mere dispositions to feel occurrent (episodic) emotional perturbations. To love another is not to have a disposition to feel one's heart melt in their presence, but to have an abiding protective concern for their welfare. To fear heights is not merely to have a disposition to feel the agitation of fear of heights, but to have a powerful motive for avoiding heights. Our long-standing emotional attitudes are primary springs of action. Our emotions are bound up with what we care about and what we care about is the source of the reasons that move us to action. Finally, our somatic perturbations are not normally objects of much thought and reflection (save perhaps in the case of hypochondriacs). But our emotions colour our thoughts and stimulate our imagination, inform our fantasy life, our wishes and our longings.

Damasio's theory inherits the defects of its Jamesian ancestor. There are extensive conceptual confusions involved in his somatic marker hypothesis. A very general misconception that runs through Damasio's theory is the idea that to think is a matter of having certain visual or auditory images. That is mistaken. Mental imagery is neither necessary nor sufficient for thinking. One can have mental images without thinking – as when one counts sheep in one's imagination or recites a mantra in one's imagination to *prevent* oneself from thinking. One can think without having any mental images, as when one talks thoughtfully to another, or when one engages in an activity with thought and concentration. (When a surgeon operates, he is thinking about what he is doing – but that does not mean that mental images are going through his mind.) To have or feel an emotion typically involves a *variety* of cognitive and

cogitative components – but having mental images, we shall argue, is not a necessary accompaniment of having an emotion, nor is it uniformly a cause of any somatic perturbation that may be involved in the emotional episode. Damasio's theory is open to the following objections.

1. An emotion is not an ensemble of somatic changes caused by a mental image of (i.e. a thought about) an object or an event. If a mental image of the swaying deck of a ship in a storm makes one's stomach turn, that does not mean that one is feeling an emotion – it means that one can induce seasickness by association.
2. There is no difference between having an emotion and feeling an emotion, just as there is no difference between having a pain and feeling a pain. There is a difference between having (feeling) an emotion and realising what emotion one is feeling – one can feel jealous without realising it. But realising what emotion one is feeling is not a matter of apprehending somatic changes as caused by images or thoughts. This conceptual error is a consequence of a further conceptual oversight.
3. Damasio, like James before him, fails to distinguish between the causes of an emotion and its object. (What made Othello jealous was Iago's tale; what he was jealous of was Desdemona's apparent love for Cassio.) Suppose that a certain emotional perturbation involves a given array of somatic changes. What makes those sensations of jealousy as opposed to envy, of fear as opposed to anger, of remorse as opposed to indignation, is not the mental image (or thought), if any, that *caused* them, but the *object* of the emotion, the agent's beliefs and desires concerning that object, and the circumstances in which the agent finds himself. What may make one afraid may be a noise in the night, but what makes one's racing pulses into sensations *of fear* is that one *believes* a burglar is in the house, and that one *apprehends* the (supposed) presence of a burglar as a *danger* (and, of course, it may just have been the cat – but that is not what one was afraid *of*).
4. If emotions were essentially ensembles of somatic changes caused by mental images, then learning the meaning of emotion words, and hence learning how to use them, would be a matter of learning the names of complexes of bodily changes with specific causes – akin to learning the meaning of an expression like 'giddiness' or 'seasickness'. But we do not learn the use of emotion words by learning sensation-names or names of overall bodily conditions. Rather, we learn what are appropriate *objects* of the relevant emotions, e.g. of fear (what is dangerous or threatening), of anger (what is annoying, offensive or in some way wrong), or pride (worthy achievement or possessions). Hence we learn how to use these terms ('afraid', 'angry', etc.) in the expression of our feelings towards the appropriate objects and in the description of the feelings (emotions, but not sensations) of others.
5. Damasio, like James, screens out the reasonableness or unreasonableness of many emotions, the justifiability or unjustifiability of many emotional responses (both occurrent emotional perturbations and long-standing emotional attitudes). This is a consequence of his failure to distinguish the cause of an emotion from its object, since the rationality or irrationality of an emotion depends (among other things) upon the agent's beliefs concerning the object of the emotion, the rationality of those beliefs, and the proportionality of the emotional response to its object. His Jamesian account is quite incapable of budgeting coherently for this aspect of emotion. For if emotions were simply somatic changes caused by mental images (or indeed the apprehension of such changes as so caused), then one could not have good reasons for feeling a certain emotion, and would not be answerable for one's emotions in the manner in which mature human beings are. For although there may be a reason (i.e. an explanation) why one has a headache, or why one's breathing rate or heartbeat (of which one is aware) rises, one cannot *have* a reason (i.e. a ground or warrant) for such things. By contrast, given appropriate circumstances, we can say that someone ought to, and has good reason to, feel proud or ashamed of himself. But we cannot say (save in a merely predictive sense) that his pulse rate ought to rise, or that his psycho-galvanic reflex reactions ought to change.
6. It should be noted that there are many emotions, even episodic emotions, that involve or that need involve, no somatic disturbance. One may feel gratitude towards a person for a favour done, and continue to feel grateful to that person for the rest of one's days, without perspiring or blushing or feeling one's pulse rate increase, etc. One may be proud of one's children or of one's work without any somatic perturbation. And one may hope that tomorrow's party will be a success without feeling one's blood pressure rise or fall.
7. One's feelings of emotion are not 'first and foremost about the body', nor do they essentially 'offer us the cognition of our visceral and muscoskeletal state' (Damasio, 1994, p. 159). It is mistaken to suppose that 'the essence of feeling an emotion is the experience of changes [in body state] in juxtaposition to the mental images that initiated the cycle' (Damasio, 1994, p. 145). One's emotions do not inform one either of the state of one's body or of the state of the world around one. But one's emotional perturbations may inform one of one's emotional attitudes. A pang of jealousy may indicate to one that one is falling in love with a person; a blush of embarrassment may bring home to one that one is ashamed of such-and-such; one's tears of grief may make one realise how much one loved the deceased. Far from one's emotions informing one of the state of one's body, the state of one's body informs one of one's emotions. Feeling grief does not inform one of the state of one's lachrymal glands, but one's hot tears may show one just how intensely one grieves for so-and-so.

8. Damasio's somatic marker hypothesis is misconceived. Bodily reactions are not ersatz guides to what to do, and do not inform us of good and evil. If one is indignant at a perceived injustice, what tells one that the object of one's indignation is an evil is not that one apprehends one's flushed face or racing pulses in association with one's thought (or 'image') of the unjust act. On the contrary, one feels indignant at the malefactor's action because it is unjust – not because one flushes in indignation when one hears about it. Indeed, the flush is a flush of indignation (and not of shame or guilt, for example) only because the object of one's feeling *is* an injustice of another (as opposed, for example, to an injustice one committed oneself). And, to be sure, one will feel indignant thus only because, and in so far as, one *cares* about the protection of the rights of others. The matter of *caring*, wholly disregarded by Damasio, brings us to our final point.
9. Damasio associates the capacity for effective practical reasoning and for pursuit of goals with the ability to feel emotions (hence the title of his book *Descartes's Error*). According to his somatic marker hypothesis, this is because the emotions are partly inherited and partly conditioned somatic responses to the beneficial and the harmful, and to good and evil – and, as such, they are efficient guides to action. It does indeed appear to be the case that damage to the ventromedial prefrontal cortex or even the amygdala in humans is accompanied by changes in the capacity to feel emotions, and by deterioration in the ability to pursue goals effectively. Since feeling an emotion is not a way of informing oneself of the state of one's body, since the emotions are misconstrued as somatic markers of good and evil, and since one's somatic responses to circumstances are not a litmus test for right and wrong, it is implausible to suppose that what is wrong with patients suffering from such lesions is that their somatic responses are awry or uninformative for them. A more plausible hypothesis, which would perhaps be worth investigating, is whether such brain damage affects their capacity to care or persist in caring about such circumstances as provide objects of standing emotional attitudes, on the one hand, and about goals, on the other. One feels no emotions concerning things about which one is indifferent, and one does not pursue goals efficiently unless one cares about achieving one's objective. Such deficiency in the ability to care would affect both the patients' emotions and their ability to pursue goals over time. This would indeed provide an integrative neuroscientific explanation linking emotion, motivation and actions.

References

Amaral, D., Price, J.L., Pitkanen, A., Carhael, S.T., 1992. Anatomical organization of the primate amygdaloid complex. The armygdala: neurobiological aspects of emotion. In: Aggleton, J.P. (Ed.), Memory and Mental Dysfunction. Wiley Liss, New York, pp. 1–66.

Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C., Damasio, A.R., 1995. Double dissociation of conditioning and declarative knowledge relative to the amygdale and hippocampus in humans. Science 269, 115–118.

Bechara, A., Tranel, D., Damasio, H., Damasio, A.R., 1996. Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. Cerebral Cortex 6, 215–225.

Bechara, A., Damasio, H., Damasio, A.K., 2000. Emotion, decision making and the orbitofrontal cortex. Cerebral Cortex 10, 295–307.

Bennett, M.R., Hacker, P.M.S., 2003. Philosophical Foundations of Neuroscience. Blackwell, Oxford.

Blair, R.J., Morris, J.S., Frith, C.D., Perrett, D.I., Dolan, R.J., 1999. Dissociable neural responses to facial expressions of sadness and anger. Brain 122, 883–893.

Braver, T.S., Cohen, J.D., Nystrom, L.E., Jonides, J., Smith, E.E., Noll, D.C., 1997. A parametric study of prefrontal cortex involvement in human working memory. Neuroimage 5, 49–62.

Breiter, H.C., Ectoff, N.L., Whalen, P.J., Kennedy, D.N., Rauch, S.L., Buckner, R.L., Strauss, M.M., Hyman, S.E., Rosen, B.X., 1996. Response and habituation of the human amygdale during visual processing of facial expression. Neuron 2, 875–887.

Brodmann, K., 1909. Vergleichende Lokalisation-lehre der Grosshirnrinde in ihren. Prinzipien dargestellt auf Grund des Zellengbaues, J.A. Barth, Leipzig.

Butter, C.M., Snyder, D.R., 1972. Alterations in aversive and aggressive behaviours following orbitofrontal lesions in rhesus monkeys. Acta Neurobiol. Exp. 32, 525–565.

Cahill, L., McGaugh, J.L., 1988. Mechanisms of emotional arousal and lasting declarative memory. TINS 21, 294–299.

Calder, A.L., Young, A.W., Rowland, D., Perrett, D.I., Hodges, J.R., Etcoff, N.L., 1996. Facial emotion recognition after bilateral amygdale damage differentially severe impairment of fear. Cog. Neuropsychol. 13, 699–745.

Carpenter, M., 1976. Human Anatomy. The Williams and Wilkins Company, Baltimore.

Carter, R.M., Hofstotter, C., Tsuchiya, N., Koch, C., 2003. Working memory and fear conditioning. Proc. Nat. Acad. Sci. U.S.A. 100, 1399–1404.

Christianson, S.A., 1989. Flashbulb memories: special, but not so special. Mem. Cog. 17 (4), 435–443.

Critchley, H.D., Rolls, E.T., 1996. Hunger and satiety modify the responses of olfactory and visual neurons in the primate orbitofrontal cortex. J. Neurophysiol. 75, 1673–1686.

Damasio, A., 1994. Descarte's Error: Emotion, reason and the human brain. Grosset/Putnam Book, New York.

Darwin, C., 1965. The Expression of the Emotions in Man and Animals. University of Chicago Press, Chicago.

Duchenne de Bologne, G.B., 1990. In: Cuthbertson, R.A. (Ed.), The Mechanism of Human Facial Expressions. Cambridge University Press, Cambridge (originally published in 1862).

Ekman, P., 1971. Universals and cultural differences in facial expressions of emotion. In: Cole, J.K. (Ed.), Proceedings of the Nebraska Symposium on Motivation. University of Nebraska Press, Lincoln, pp. 207–284.

Ekman, P., 1972. Universals and cultural differences in facial expressions of emotions. In: Cole, J.K. (Ed.), Proceedings of the Nebraska Symposium on Motivation. University of Nebraska Press, Lincoln, 19.

Ekman, P., Friesen, W.V., 1971. Constants across cultures in the face and emotion. J. Pers. Soc. Psychol. 17, 124–129.

Folkman, S., Lazarus, R.S., 1990. Coping and emotion. In: Stein, N.S., Leventhal, B.L., Trabasso, T. (Eds.), Psychological and Biological Approaches to Emotion. pp. 313–332.

Frankland, P.W., Cestari, V., Filipkowski, R.X., McDonald, R.J., Silva, A.J., 1998. The dorsal hippocampus is essential for context discrimination but not for contextual conditioning. Behav. Neurosci. 112, 863–874.

Fuster, J.M., 1998. Linkage at the top. Neuron 21, 1223–1224.

Goldman-Rakic, P.S., 1996. Memory: recording experience in cells and circuits: diversity in memory research. Proc. Nat. Acad. Sci. U.S.A. 93, 13435–13437.

Gratiolet, P., 1865. De la Physognomie et des Movements d' Expression. J. Hetzel, Paris.

Hornak, J., Rolls, E.T., Wade, D., 1996. Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal lobe damage. Neuropsychology 34, 247–261.

James, W., 1890. The Principles of Psychology. Henry Holt, New York.

James, W., 1922. The Emotions. Williams and Wilkins, Baltimore.

Killgore, W.D.S., Yurgelun-Todd, D.A., 2004. Activation of the amygdala and anterior cingulate during nonconscious processing of sad versus happy faces. Neuroimage 21, 1215–1223.

Kling, A.S., Steklis, H.D., 1976. A neural substrate for affiliative behaviour in non-human primates. Brain Behav. E 13, 216–238.

Kling, A.S., Steklis, H.D., Deutsch, S., 1979. Radiotelemetered activity from the amygdale during social interactions in the monkey. Exp. Neurol. 66, 88–96.

Kluver, H., Bucy, P.C., 1939. Preliminary analysis of functions of the temporal lobes in monkeys. Arch. Neurol. Psychol. 42, 979–1000.

LaBar, K.S., LeDoux, J.E., Spencer, D.D., Phelps, E.A., 1995. Impaired fear conditioning following unilateral temporal lobectomy. J. Neurosci. 15, 6846–6855.

Lazarus, R.S., 1982. Thoughts on the relationship between emotion and cognition. Am. Psychol. 37, 1019–1024.

Lazarus, R.S., 1991. Cognition and motivation in emotion. Am. Psychol. 46, 352–367.

LeDoux, J.E., 1992a. Emotion and the amygdala. In: Aggleton, L.P. (Ed.), The Amygdala. Wiley-Liss, New York, pp. 339–351.

LeDoux, J.E., 1992b. Brain mechanisms of emotion and emotional learning. Curr. Opin. Neurobiol. 2, 191–197.

LeDoux, J.E., 1993a. Emotional memory: in search of systems and synapses. Ann. N.Y. Acad. Sci. 702, 149–157.

LeDoux, J.E., 1993b. Emotional memory systems in the brain. Behav. Brain Res. 58, 69–79.

LeDoux, J.E., 1995. In search of an emotional system in the brain: leaping from fear to emotion consciousness. In: Gazzaniga, M. (Ed.), The Cognitive Neurosciences. MIT Press, Cambridge, MA, pp. 1019–1061.

LeDoux, J.E., 1996. The Emotional Brain. Simon and Schuster, New York.

LeDoux, J.E., 1998. The Emotional Brain. Simon and Schuster, London.

LeDoux, J.E., 2000. Emotion circuits in the brain. Ann. Rev. Neurosci. 23, 155–184.

LeDoux, J.E., Cicchetti, P., Xagoraris, A., Romanski, I.M., 1990. The lateral amygdaloid nucleus: sensory interface of the amygdale in fear conditioning. J. Neurosci. 10, 1062–1069.

Levy, R., Goldman-Rakic, P.S., 2000. Segregation of working memory functions within the dorsolateral prefrontal cortex. Exp. Brain Res. 133, 23–32.

Mora, R., Aurith, D.B., Phillips, A.G., Rolls, E.T., 1979. Effects of satiety on self- stimulation of the orbitofrontal cortex in the rhesus monkey. Neurosci. Lett. 13, 141–145.

Morris, J.S., Frith, C.D., Perrett, D.I., Rowland, D., Young, A.W., Caldes, A.J., Dolan, R.J., 1996. A differential neural response in the human amygdala to fearful and happy facial expressions. Nature 383, 812–815.

Murphy, S.T., Zajonc, R.B., 1993. Affect, cognition and awareness: affective priming with optimal and suboptimal stimulus exposures. J. Pers. Soc. Psych. 64 (5), 723–739.

Pavlov, I.P., 1927. Conditional Reflexes. Dover, New York.

Quirk, G.J., Armony, J.L., Repa, J.C., Li, X.F., LeDoux, J.E., 1996. Emotional memory: a search for sites of plasticity. Cold Spring Harb. Symp. Quant. Biol. LXI, 247–257.

Rolls, E.T., 1975. The neural basis of brain-stimulation reward. Prog. Neurobiol. 3, 73–160.

Rolls, E.T., 1992. Neurophysiology and functions of the primate amygdale. In: Aggleton, J.P. (Ed.), The Amygdala. Wiley-Liss, New York, pp. 143–165 (Chapter 5).

Rolls, E.T., 1993. The neural control of feeding in primates. In: Booth, D.A. (Ed.), Neurophysiology of Ingestion. Pergamon Press, Oxford, pp. 137–169 (Chapter 9).

Rolls, E.T., 1995. A theory of emotion and consciousness and its application to understanding the neural basis of emotion. In: Gazzaniga, M.S. (Ed.), The Cognitive Neurosciences. MIT Press, Cambridge, MA, pp. 1091–1106 (Chapter 72).

Rolls, E.T., 1999. The Brain and Emotion. Oxford University Press, Oxford.

Rolls, E.T., Baylis, L.L., 1994. Gustatory, olfactory, and visual convergence within the primate orbitofrontal cortex. J. Neurosci. 14 (9), 5437–5452.

Rolls, E.T., Treves, A., 1998. Neural Networks and Brain Function. Oxford University Press, Oxford.

Rolls, E.T., Burton, M.J., Mora, F., 1980. Neurophysiological analysis of brain-stimulation reward in the monkey. Brain Res. 194 (2), 339–357.

Rolls, E.T., Critchley, H.D., Browning, A., Hernadi, I., 1998. The neurophysiology of taste and affection in primates and unsami flavour. In: Murphy, C. (Ed.), Olfaction and Taste, vol. XII, Ann. N.Y. Acad. Sci.

Rolls, E.T., Sienkiewicz, Z.J., Yaxley, S., 1989. Hunger modulates the responses to gustatory stimuli of single neurones in the caudolateral orbitofrontal cortex of the macaque monkey. Eur. J. Neurosci. 1 (1), 53–60.

Sanghera, M.K., Rolls, E.T., Roper-Hall, A., 1979. Visual responses of neurones in the dorsolateral amygdala of the alert monkey. Exp. Neurol. 63 (3), 610–626.

Speisman, J.C., Lazarus, R.S., Mordkoff, A., Davison, L., 1964. Experimental reduction of stress based on ego-defense theory. J. Abnormal Social Psychol. 68 (4), 367–380.

Weiskrantz, L., 1956. Behavioural changes associated with ablation of the amygdaloid complex in monkeys. J. Comp. Physiol. Psychol. 49, 381–391.

Zajonc, R.B., 1984. On the primacy of affect. Am. Psychol. 39, 117–124.