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Feeling States in Emotion

Functional Imaging Evidence

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ABSTRACT

An essential feature of emotional states is their association with change in autonomic function. The importance of these changes lies in the fact that in many theoretical accounts of emotion the realization of autonomic states is a primary means through which feeling states are realized. The issue addressed in this chapter is how the brain generates and represents autonomic states of the organism and their importance in feeling states. A distinction will be drawn between brain systems that mediate relatively automatic responses to emotional stimuli and systems that are involved in what can be termed conscious feeling states. These distinctions will be illustrated by observations from functional neuroimaging and patients with focal brain lesions and pathology of the autonomic nervous system.

Despite the impact of William James at the end of the nineteenth century, for the best part of several decades in the middle of the twentieth century, neuroscientists treated "emotion" and "feeling" as interchangeable terms. This approach to the language of emotion, coupled with the behaviorist movement prevalent in twentieth century psychology, meant that the neuroscientific study of emotion was grossly neglected, often seen as conceptually ragged and its practitioners as pursuing "soft science" (Damasio, 1999; Damasio, 1994; Damasio, 1998; LeDoux, 1996; LeDoux, 2000). Recent efforts, however, have put the study of emotion center stage in understanding the workings of the brain, proposing roles in cognition (Zajonc, 1980), decision making (Damasio, 1994), perception (Anderson & Phelps, 2001), and even consciousness (Panksepp, 1998; Damasio, 1999). Among the reasons that emotion has assumed center stage in neuroscience is a rediscovery of the importance of a dissociation between emotion and feeling (LeDoux, 1996; LeDoux, 2000), which potentially offers a more tractable approach to the neuroscientific study of emotion (Damasio, 2001). Indeed, Antonio Damasio has extended this distinction to formal definitions. Specifically, emotion is defined as "a patterned collection of chemical and neural

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responses that is produced by the brain when it detects the presence of an emotionally competent stimulus . . . the responses are engendered *automatically*" (emphasis added). This contrasts with feelings that "are the mental representation of the physiological changes that characterise emotions. Unlike emotions . . . feelings are indeed private, although no more subjective than any other aspect of the mind" (Damasio, 2001, this volume).

Though not universally accepted, this distinction between automatically elicited emotional states and experiential (and necessarily subsequent) feeling states is a useful heuristic in reviewing work in the relatively new field of functional neuroimaging of emotion.

AUTOMATICITY AND THE AMYGDALA

A substantial number of neuroimaging studies on emotion have explored the neural responses to emotional faces, often under conditions wherein volunteer subjects are unaware that the emotional aspect of the stimulus constitutes the experimental variable of interest. For example, Morris and colleagues (Morris et al., 1996) and Breiter and colleagues (Breiter et al., 1996) have provided findings implicating the amygdala in automatic processing of fearful faces. In the former study, using PET and involving a block design, subjects were presented with a series of morphed faces from the Ekman and Friesen series (Ekman & Friesen, 1975). The study showed that regional cerebral blood flow (rCBF) in the left amygdala was specifically enhanced to fear and increased monotonically with increasing degree of fear expressed in the face. Debriefing confirmed that subjects were unaware that emotion was the variable of interest in the study. Breiter and colleagues (Breiter et al., 1996), using functional magnetic resonance imaging (fMRI) and a blocked experimental design, showed a response in amygdala to both fearful and happy faces relative to neutral faces. An additional observation was attenuation in amygdala response with repetition of fearful faces. Again, subjects' task in the scanner was designated as gender discrimination, so avoiding any explicit reference to the emotional component of the experimental design. This basic result of an amygdala response to fearful faces has been widely replicated in subsequent studies (Dolan et al., 2001; Hariri et al., 2000; Phillips et al., 1997; Vuilleumier et al., 2001; Whalen et al., 1998).

Neuroimaging of human fear conditioning also implicates the amygdala in fear processing, as might be predicted from the animal literature (for review, see Davis, 1992; LeDoux, 2000) and case reports in humans with discrete brain lesions (Bechara et al., 1995; LaBar et al., 1995). As Buchel and Dolan point out in a recent review of the literature on fear conditioning (2000), the consistent finding in studies of classical aversive conditioning is that the amygdala is a central unit in the processing of CS-US contingency. In other words, the amygdala is the key locus in associating previously

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neutral stimuli with acquired aversive value. Commonly in fear conditioning experiments, subjects are unaware of the contingency, or only become aware of the contingency some time into the experiment. The response in amygdala, which tends to habituate rapidly (Buchel et al., 1999; Buchel et al., 1998; LaBar et al., 1998), would consequently seem to take an opposite time – course to subjects' reported awareness of CS+/UCS contingencies. Again, we note an apparent automaticity of amygdala responses, regardless of the awareness of the contingency. Indeed, in one study, Morris et al. (1986b) demonstrate that awareness of the conditioned stimulus itself is unnecessary for an amygdala response.

In a recent study on fearful faces and spatial attention, Vuilleumier and colleagues (Vuilleumier et al., 2001) addressed the issue of automaticity in emotional processing in a carefully controlled fashion. These authors demonstrated that while responses to faces, including fearful faces, in the fusiform gyrus (an area on the ventral surface of the brain anterior to visual cortex known to respond to faces) *are* modulated by attention, amygdala responses to fearful faces are not. In this study, subjects were instructed to attend to either a horizontal pair or vertical pair of stimuli, which could be either pairs of houses or faces (Figure 13.1a). Faces were either fearful or neutral. Pairs of faces or houses appeared at either task-relevant or task-irrelevant locations. The task was a same/different judgment across the task-relevant location. Regardless of whether fearful faces appeared at task-relevant or task-irrelevant locations, amygdala responses were of similar magnitude (Figure 13.1 b, c). In contrast, responses to fearful faces in the fusiform gyrus were modulated by both attention to the face and the emotive nature of faces (Figure 13.1 b, d). In a parallel behavioral study, the authors additionally demonstrate the effectiveness of their experimental manipulation – subjects were unable to state whether a face was emotional or neutral immediately after trials in which they had been attending to houses. In other words the experimental design had induced an inattentional amnesia for nonattended stimuli. Additional evidence that subjects need not be consciously aware of threatening stimuli in order to generate amygdala responses to these stimuli has come from a study using masking of fearful faces (Whalen et al., 1998) and recent studies on a single patient with blindsight (Morris et al., 2001).

The above leads to the conclusion that the amygdala links pre-perceptual or pre-attentive sensory processing with emotion. Notably, there is considerable evidence that the amygdala might also be involved in the reverse process, namely the heightening of perceptual processing in relation to emotionally salient stimuli. For example, an analysis of functional connectivity, derived from neuroimaging data, between amygdala and extrastriate cortex in the context of subjects being presented with fearful and happy faces has revealed increased coupling of activity in these areas when the target face was fearful rather than happy (Morris et al., 1998a). The authors

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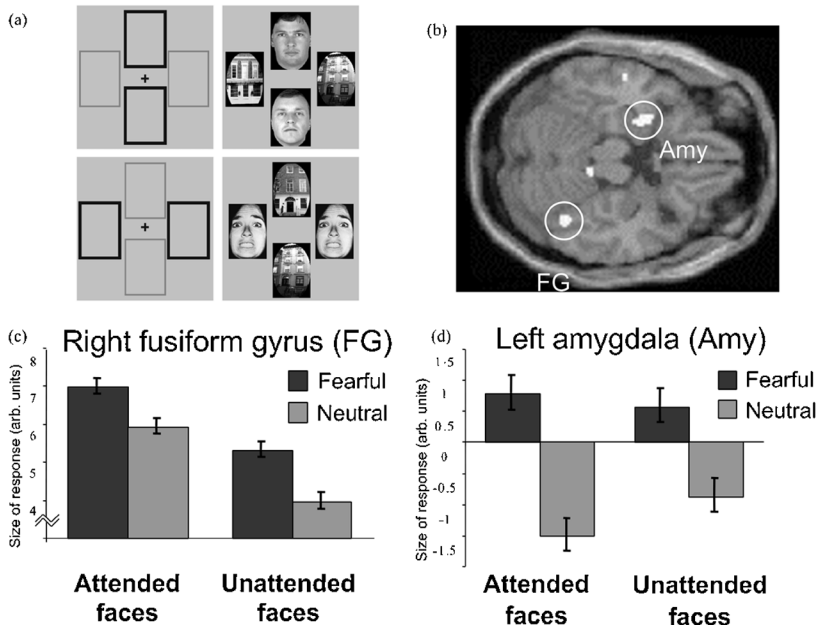


FIGURE 13.1. Differential responses in amygdala and fusiform gyrus dependent on attention and emotion (see Vuilleumier et al., 2001). (*a*) Task design: Faces and houses appeared paired at task-relevant or task-irrelevant locations. The task was to decide whether the pair of stimuli in the task-relevant locations were the same or different. In addition, faces could be fearful or neutral. Subjects were instructed which were the task-relevant locations before they were given a block of trials by bold outlines of the target stimuli locations. (*b*) Main effect of fearful faces relative to neutral faces. Statistical map overlaid onto a horizontal slice from single subject's structural scan demonstrating areas showing greater responses to fearful than to neutral faces. Both left amygdala and right fusiform gyrus exhibit such responses. (*c*) Measures of task- and stimulus-dependent responses in right fusiform gyrus. Note that there is modulation of fusiform responses by both emotion (greater responses to fearful faces) and attention (greater responses when faces were at task-relevant locations). (*d*) Measures of task- and stimulus-dependent responses in left amygdala. Note that there is an effect of emotion (greater responses to fearful faces) but no effect of attention: responses to fearful faces are independent of direction of attention. This suggests that the amygdala is automatically processing the fearful faces.

interpreted this alteration of activity in extrastriate cortex to the presentation of fearful faces as mediated by anatomical back-projections from amygdala (Amaral & Price, 1984). A more recent study, using expressionally transfigured face stimuli (the "Thatcher Illusion"), has replicated this finding of altered coupling between amygdala and lateral occipital cortex in association with emotionally valenced stimuli (Rotshtein et al., 2001). We

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note also that a recent human lesion study demonstrated that enhanced perception of emotional relative to neutral words during the attentional blink is absent in patients with bilateral amygdala lesions (Anderson & Phelps, 2001). This phenomenon of enhanced perception of emotional items may be the functional consequence of reafferent feedback from amygdala to extrastriate cortical regions.

OTHER AREAS INVOLVED IN GENERATING EMOTIONS

Although the amygdala is clearly important in bridging a gap between perception (or lower-level processes such as object recognition) and emotion (at least for the cases of fear discussed above), there are many other brain areas involved in the generation of the the chemical and neuronal changes that underlie emotional behaviour (Damasio, 1994). Among the key regions are hypothalamic autonomic centers, basal forebrain, and brainstem nuclei. It is of interest that the primate basolateral amygdala has extensive interconnections with all of these areas (Amaral et al., 1992), and it is more than likely that the entire landscape of an emotion includes important components mediated through outputs from these regions. However, relatively few imaging studies on emotion report activations in these structures (for a notable exception, see the recent study by Damasio et al., 2000). The relative absence of activation in these regions may, in part, relate to limitations in current neuroimaging methodologies: these include limited spatial resolutions of a few millimeters; a restricted resolution in the temporal domain of seconds; regional variability in time-courses of neuronal activation; the inability to account for regional variations in neurovascular coupling; and signal dropout in inferior temporal, inferior-medial prefrontal, and brainstem regions (particularly with fMRI).

EMOTIONS OTHER THAN FEAR

It remains unclear whether the human amygdala is a structure devoted more to the processing of fear than to the processing of other so-called basic emotions. Imaging studies of perception of disgust have emphasized activations in the insular cortex and basal ganglia (Gorno-Tempini et al., 2001; Phillips et al., 1998; Phillips et al., 1997; Sprengelmeyer et al., 1998). This distinct pattern of activation in relation to disgust would accord with a more general a priori hypothesis that proposes functional specialization devoted to processing distinct basic emotions. It should also be noted that there is converging evidence from neuropsychology that supports imaging findings in relation to disgust, in that patients with basal ganglia and insula damage have specific impairments in recognition of facial expressions of disgust (Calder et al., 2000; Gray et al., 1997). However, an important caveat with respect to the above neuroimaging data is that, unless

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critical comparisons are made between fearful- and disgust-evoked activation conditions, one cannot conclude that disgust activates the insula significantly more than fear, nor that fear activates the amygdala significantly more than disgust. To the best of our knowledge, in only one study is such a critical comparison actually reported (Phillips et al., 1998). In this study, using data derived from six subjects, a comparison of evoked responses to fearful minus neutral faces and disgusted minus neutral faces yielded a single point located in amygdala as more significantly activated in response to fearful faces. We note that the coordinate reported for this locus is probably too posterior and lateral for amygdala, and may correspond better to hippocampus. Two separate foci in the left anterior insula were more activated in response to disgusted faces.

This result does suggest a dissociation within regions subserving emotional processing in terms of preferential responses to specific basic emotions. However, we stress that it is a single result in a small number of subjects, and would caution against any strong inference of distinct neural bases for perception of different emotions (Calder et al., 2001) pending confirmatory evidence. Indeed, our considered view is that current findings from functional imaging studies of emotion are unable to resolve the issue of whether there are distinct neural substrates for "basic emotions" (or, indeed, whether there are "basic emotions" at all). At least one study has reported an amygdala response to sad faces (though not to angry ones) (Blair et al., 1999) and, notably, studies have indicated that happy faces activate amygdala relative to neutral faces (Breiter et al., 1996), as well as showing amygdala responses to affectively positive physiological stimuli such as tastes (O'Doherty et al., 2001), suggesting that its role in emotion may not even be limited to negative emotions, as has been suggested by others (Adolphs et al., 1999). The human lesion literature concerning the role of amygdala in recognition of emotion suggests that there is minimally an impairment in recognition of fear subsequent to bilateral amygdala damage, though individual cases with impairments in recognition of anger, sadness, and disgust have also been reported (Adolphs et al., 1994; Adolphs et al., 1995; Adolphs et al., 1999; Broks et al., 1998; Fine and Blair, 2000; Schmolck and Squire, 2001; Young et al., 1996). We anticipate that further neuroimaging and neuropsychological studies may clarify this topic.

THE NEURONAL BASIS OF FEELING STATES

A key component of many classical and modern hypotheses of emotion and feeling has been the crucial role ascribed to the autonomic nervous system in generating feelings. Thus, as James might have elaborated it, we *feel* scared partly because our heart beats faster, and our heart beating faster is itself an index of an emotional response. Though such autonomic activity

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is only one aspect of the somatic feedback hypothesized to compose emotional responses by James (1884) and Damasio (1994), it has been more extensively studied than other modes of somatic feedback, and we limit our discussion here to studies of the generation and mapping of autonomic states. A series of recent experiments using PET and fMRI have attempted to address the functional neuroanatomy of the autonomic nervous system. Using PET, with simultaneous recording of skin conductance changes elicited by a variety of stimuli, Fredrikson and colleagues (Fredrikson et al., 1998) demonstrated positive correlations between electrodermal activity (EDA) and blood flow in left primary motor, posterior cingulate, and anterior cingulate cortices. One of the strengths of this study was the authors' ability to analyze the association between EDA and blood flow independent of the task effects that generated the conductance changes. Using PET, and different measures of autonomic activity that included heart rate and blood pressure changes, Critchley and colleagues (Critchley et al., 2000a) demonstrated increased activity in cerebellum, pons, right anterior cingulate, and right insula covarying with heart rate and activity in right anterior cingulate and right insula covarying with mean arterial blood pressure. In this study, strenuous exercise and mental stressor tasks were used to generate cardiovascular arousal where an independent baseline for each condition enabled a conjunction analysis allowing identification of regions commonly activated across experimental conditions.

A well-documented problem with PET experiments is the limited temporal resolution. Cerebral blood flow measurements are usually a reflection of integrated activity in the brain typically taken over the time-course of approximately one minute. This makes it impossible to separate cause-and-effect influences on measures of peripheral autonomic arousal. Thus, in the experiments described, the increased blood flow in anterior cingulate reported in both studies might be the cause of the autonomic arousal, or an effect arising out of the central mapping of peripheral autonomic effects. To dissociate these possibilities, Critchley and colleagues used event-related fMRI, with its greatly improved temporal resolution of several seconds (Critchley et al., 2000b). In this study, a variant of a gambling task was undertaken by six subjects while skin conductance measurements and fMRI were simultaneously acquired. Skin conductance responses (SCR – an index of peripheral bodily arousal) were used in the subsequent analysis of the fMRI data by designating peaks in SCR as "events" in the analysis. The authors thus attempted to explain the responses recorded by fMRI by means of events preceding or succeeding peaks in SCR. This allowed the authors to attribute differential brain activity to the generation of SCRs as well as the mapping of neuronal changes consequent upon the occurrence of SCRs. In this study, activity preceding SCR events was evident in cerebellum, lingual gyrus, left medial prefrontal lobe, and fusiform gyrus.

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By contrast, activity subsequent to SCR events was demonstrated in right medial prefrontal cortex. Finally, activity both preceding and subsequent to SCR events was evident in regions such as orbitofrontal, bilateral medial prefrontal, and posterior cingulate cortex. The interpretation of these results is that they accorded strongly with a suggested role for ventromedial prefrontal cortex in generating and decoding the significance of "somatic markers" (Bechara et al., 1997; Bechara et al., 1996; Damasio, 1994). Thus, orbitofrontal and medial prefrontal cortices generate and map bodily states, a role that may facilitate guidance of behavior, as suggested by Damasio (1994).

In an experiment addressing the issue of self-modulation of autonomic activity, Critchley and colleagues (Critchley et al., 2001c) trained subjects with biofeedback techniques. Subjects then underwent PET scans in four conditions: instructed to relax using accurate biofeedback information based upon SCR; instructed to relax but provided with inaccurate biofeedback information; and instructed not to relax with both accurate and inaccurate biofeedback. Subjects were informed whether biofeedback information was accurate or inaccurate. Activity in anterior cingulate, left inferior parietal lobule, and globus pallidus was associated with subjects' attempts to relax. Additionally, and importantly, activity in anterior cingulate showed an interaction between between feedback and task, being higher when subjects were instructed to relax and were provided with accurate biofeedback. This suggests that anterior cingulate is involved in the intentional modulation of arousal.

In a study pertaining directly to autonomic arousal and feelings, Critchley and colleagues (Critchley et al., 2001a) used event-related fMRI with simultaneous GSR recording while subjects played a card-gambling game (Figure 13.2a). Eight subjects made decisions about whether the next card in a sequence would be higher or lower than the previous one. There was an objective measure of the risk involved in a given decision, namely the degree of certainty about the value of the next card (certainty was higher when the previous card took an extreme value). Additionally, the recording of SCRs allowed a measure of autonomic arousal association with anticipation of the outcome of each trial. The key finding in this study was the demonstration that a portion of the anterior cingulate cortex responded both to the uncertainty of decisions and to their associated arousal (Figure 13.2b, c). This, in the view of the authors, provided support for the view that "anterior cingulate function reflects integration of cognitive states with adaptive changes in bodily states mediated by the autonomic nervous system."

Some of the hypotheses derived from this work on autonomic regulation have been tested by Critchley and colleagues (Critchley et al., 2001b) in a patient group with pure autonomic failure (PAF). These patients suffer

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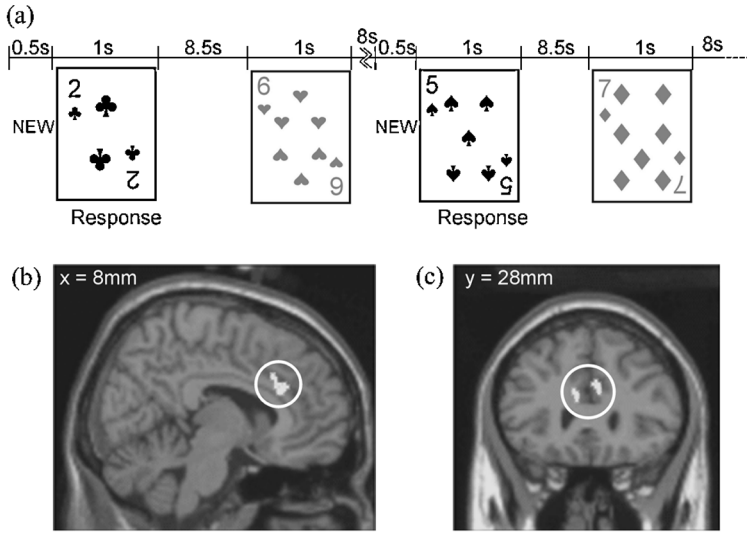


FIGURE 13.2. Anterior cingulate cortex integrates arousal and uncertainty (see (Critchley et al., 2001a). (*a*) Experimental design: The task was a gambling game in which subjects had to predict whether the next card would be higher or lower than the previous one. Card values ranged from 1 to 10. New trials were indicated by the word "new" appearing on screen before the first card. Cards appeared for one second, after which time subjects had to decide whether the next card would be higher or lower. There was then a gap before the next card was revealed. The last four seconds of this gap was the time during which anticipatory arousal was measured. The uncertainty of a given trial was a function of the first card that appeared. In the example in the figure, the first trial is relatively certain, as there is little chance of a card less than 2. The second trial is relatively uncertain, as there is an almost equal chance of a card higher or lower than 6. (*b, c*) Activity in anterior cingulate was modulated by both risk and arousal. Parasagittal (*b*) and coronal (*c*) sections through a template brain with statistical map showing regions where there was significant modulation of the delay period activity by both arousal and uncertainty.

peripheral autonomic denervation in the absence of any other neurological deficit and where there is no known central pathology. Patients thus lack peripheral autonomic change and its associated central representation. Comparing nine PAF patients to eight age-matched healthy controls (using PET) in an experiment where the tasks involved either mental or physical stress (and control tasks), Critchley and colleagues noted that patients showed increased anterior cingulate cortex activity associated with increased exertion. As expected from their medical condition, however, the patients showed no evidence of actual autonomic changes, as indexed by heart rate and blood pressure. This is suggestive of a conceptualization whereby anterior cingulate modulates autonomic function and integrates

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it with feedback information about ongoing autonomic changes. In the absence of the expected autonomic changes, cingulate activity is increased as it attempts to generate a task-appropriate autonomic tone. Crucially, the authors also documented alterations in emotional experience within the key patient group relative to Parkinsonian patients with similar physical disability, suggesting that an absence of autonomic arousal does impair their ability to experience feeling states.

STUDIES ADDRESSING "FEELINGS"

There have been few attempts since the inception of functional neuroimaging to address the neuronal basis of feelings. The difficulty of this undertaking must be acknowledged, given that the definition of feelings necessarily involves private subjective states. The predominant experimental approach has been to use visual or verbal stimuli as mood inducers and to scan subjects as they report alteration in feeling states. One problem with such study designs is that feelings are necessarily secondary to emotions in most models of feelings and emotions. This renders it difficult to separate the functional neuroanatomy of the feeling from that of the emotion. A fully factorial design, in which feelings occurred in the absence of emotions, might address this issue, but this would seem impossible within the definitions of emotions and feelings provided above. The best alternative would be experiments in which emotions are elicited in both the absence and presence of feelings (even here, one would be incurring the assumption of "pure insertion" [Friston et al., 1996]) but too little research seems to be carried out even along such lines.

Thus, in our view, the bulk of experiments to date confound emotions and feelings. Additionally, experiments on feelings are difficult to quantify because of the subjective nature of the experimental variable. We are reliant on subjects understanding what experimenters mean when they ask them to re-create a feeling of sadness, and to accurately report the intensity and quality of the feeling. Furthermore, the use of a "neutral feeling" baseline is less appropriate than in other types of experiments, as, for instance, in the case of experiments utilizing emotional faces. Re-creating a "neutral" feeling might rely upon the suspension of mnemonic activity, which will confound contrasts of emotional (induced by memory strategies) versus neutral conditions. An approach that can circumvent this problem might involve direct comparison of different emotional conditions, perhaps using post-scanning subjective ratings of experienced emotional valence as covariates of no interest.

In one of the earliest reported studies on emotional feelings, George and colleagues (George et al., 1995) used happy, sad, and neutral faces as well as recall of life events to induce feelings in eleven female subjects. These authors reported widespread increases and decreases in regional CBF in

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relation to transient sadness and happiness. In particular, they emphasized frontal, temporal, and cingulate as areas in which significant changes in blood flow were seen, depending upon the emotion elicited. A strong point of this study is that a direct comparison of blood flow in the happiness and sadness conditions was made. In this contrast, rCBF increase during sadness in putamen and anterior cingulate, which contrasted with increased during happiness in largely visual areas such as cuneus and occipital cortex. In a similar study by Lane et al. (1997b) subjects underwent ^{15}O PET scans while recalling happy, sad, or disgusting personal events or watching film clips designed to elicit happy, sad, or disgusting feelings. Neutral recall and film conditions were additionally included in the experimental design. In a subtraction analysis, whereby each emotion constituted one activation condition against its own neutral baseline, the authors reported common and distinct patterns of activation for different emotions. However, in failing to contrast the emotional conditions themselves, the authors could make no strong inferences regarding differences between the distinct feeling states.

More recently, Damasio and colleagues (Damasio et al., 2000) have undertaken a large PET study addressing the functional neuroanatomy of feelings involving thirty-nine subjects. A limitation of this study wherein distinct groups of subjects were required to experience sadness and happiness, fear and anger, sadness and anger, and fear and happiness, was a failure to fully counterbalance the pairings of emotions. Consequently, no subject was required to experience both sadness and fear, for example. As with the study of Lane and colleagues (Lane et al., 1997b), the authors describe distinct patterns of activation in relation to different feeling states when contrasted with independent baseline conditions. Note that there are no direct comparisons of activation conditions. An interesting finding was the increase in CBF in midbrain, pontine, and hypothalamic areas regardless of which emotion was experienced, which is supportive of the idea that emotions leading to feelings are associated with changes in autoregulatory (autonomic) functions controlled by such areas (Damasio, 1999; Damasio, 1994). In cortical regions, orbitofrontal, anterior and posterior cingulate, and secondary somatosensory cortices all displayed rCBF increases in relation to the experience of feeling states. This pattern of activity is compatible with the authors' interpretation as reflecting mapping of first-order changes in bodily states. However, in a PET study with limited temporal resolution, and in particular where feelings are self-generated rather than stimulus-driven, it is very difficult to separate cause and effect. Thus, it is possible that a component of activity reported is actually associated with subjects' deliberate modulation of their own bodily states to produce the emotion required. Supportive of this interpretation are data from Critchley and colleagues (Critchley et al., 2001b; Critchley et al., 2001c) indicating that anterior cingulate (for example) is involved in intentional modulation of

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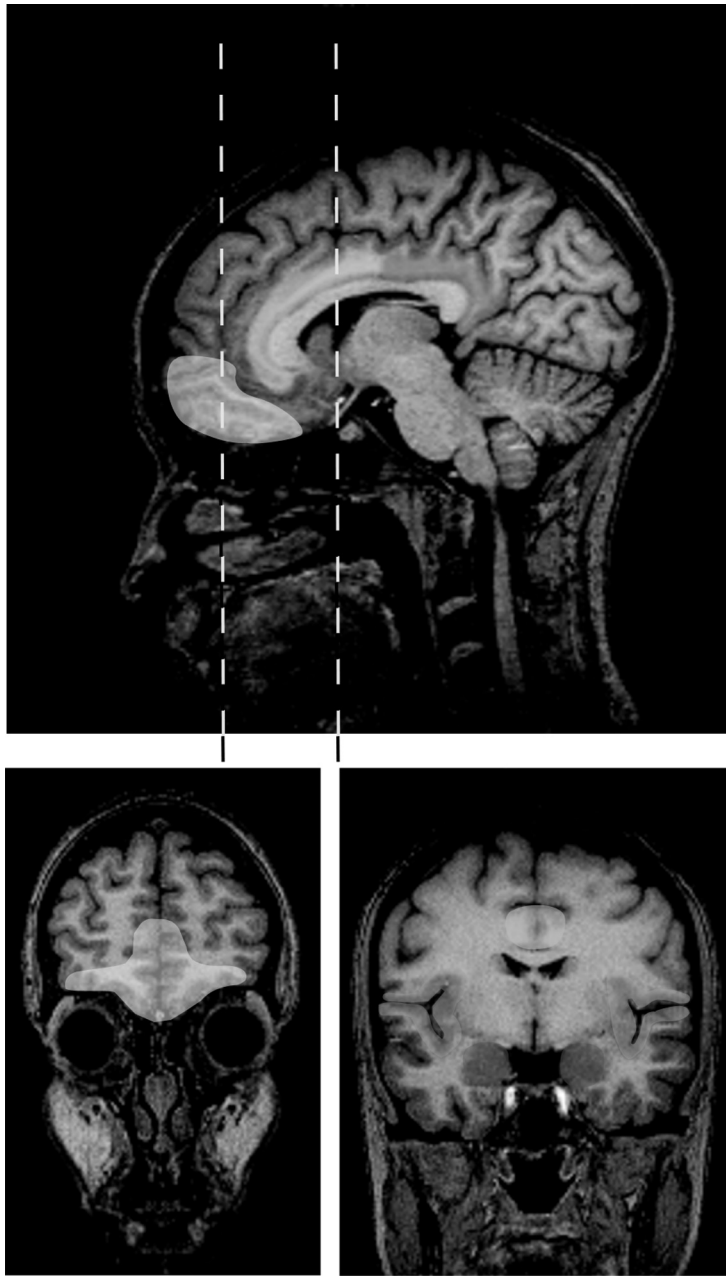


FIGURE 13.3. Brain regions demonstrated to be important in representing emotions and feelings. (*See color insert*)

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body states. However, in a study that addressed the attentional modulation of emotional responses, Lane et al. (1997a) demonstrated that anterior cingulate cortex is activated when subjects are required to pay attention to their own emotional responses rather that to external features of emotion-provoking stimuli. Either of these two effects might account for the observations of several authors that anterior cingulate cortex is invoked in feeling states.

CONCLUSION

In this chapter we have briefly surveyed some of the burgeoning functional imaging literature on the topic of feelings and emotions. We have noted that a defining characteristic of emotional responses is their automaticity, and that a number of studies report obligatory amygdala responses to emotive stimuli independent of subjects' awareness of the stimuli (Morris et al., 1998b; Whalen et al., 1998), of the emotional nature of the stimuli (Morris et al., 1996), or of the contingency between the stimulus and its emotive properties (e.g., early in conditioning studies, before subjects are aware of CS-US contingencies). These data are highly suggestive that the human amygdala is a crucial locus in associating stimuli with their appropriate emotive value. Additional evidence that the amygdala influences neural responses in perceptual areas (Morris et al., 1998a; Rotshtein et al., 2001) supports this view. The currently available evidence suggests that the anterior cingulate cortex in particular is involved in generating and mapping autonomic changes that are central components to feeling states. However, the empirical data also indicate that widespread cortical areas may provide the neural basis of feeling states (see Figure 13.3 for summary). What is also evident is that a distinction between what is termed "emotion" and "feelings" may provide an important conceptual framework for unraveling the neurobiology of what is generically referred to as emotion.

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