COGNITION AND EMOTION, 1990, 4 (3), 161–190

A Theory of Emotion, and its Application to Understanding the Neural Basis of Emotion

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It is shown that emotions can usefully be considered as states produced by reinforcing stimuli. The ways in which a wide variety of emotions can be produced, and the functions of emotion, are considered. There is evidence that the amygdala is involved in the formation of stimulus-reinforcement associations, and the orbitofrontal cortex with correcting behavioural responses when these are no longer appropriate because previous reinforcement contingencies change. This evidence comes from the effects of damage to these structures, and from recording the activity of single neurons in these structures in the monkey during the formation and disconnection of stimulus-reinforcement associations. In so far as emotions can be defined as states produced by reinforcing stimuli, then the amygdala and orbitofrontal cortex are seen to be of great importance for emotions, in that they are involved respectively in the elicitation of learned emotional responses, and in the correction or adjustment of these emotional responses as the reinforcing value of environmental stimuli alters. One of the theses advanced is that the changes in emotional behaviour produced by damage to the brain can be analysed and understood by considering how different parts of it function in reinforcement and in the formation and disconnection of stimulus-reinforcement connections. Another thesis is that there is a population of neurons in the amygdala and parts of the temporal lobe visual cortex specialised to respond to faces, and that these neurons may be involved in social and emotional resposes to faces.

Some of the outputs of the amygdala and orbitofrontal cortex are directed to the hypothalamus, which not only provides one route for these

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The author has worked on some of the experiments described here with G. C. Baylis, M. J. Burton, M. E. Hasselmo, C. M. Leonard, F. Mora, D. I. Perrett, M. K. Sanghera, T. R. Scott, S. J. Thorpe, and F. A. W. Wilson, and their collaboration, and helpful discussions with or communications from C. C. W. Taylor (Corpus Christi College, Oxford), E. A. Wakeman, M. Dawkins, and N. S. Clayton, are sincerely acknowledged. Some of the research described was supported by the Medical Research Council.

reinforcing environmental events to produce autonomic responses, but also is implicated in the utilisation of such stimuli in motivational responses, such as feeding and drinking, and in emotional behaviour. Other outputs of the amygdala and orbitofrontal cortex which may enable them to influence behaviour are directed to the striatum, and also back towards some of the cortical regions from which they receive inputs. It is suggested that these latter projections are important in the effects which mood states have on cognitive processing.

INTRODUCTION

It is the purpose of this article to consider the neural bases of emotion, and to provide a theoretical basis for understanding how different neural systems are involved in emotion. Particular attention is paid to research in non-human primates, partly because the developments in primates in the structure and connections of neural systems involved in emotion, such as the amygdala and orbitofrontal cortex, make studies in primates particularly important for understanding emotion in humans.

A THEORY OF EMOTION

Emotions can usefully be defined as states produced by instrumental reinforcing stimuli (Millenson, 1967; Weiskrantz, 1968; Gray, 1975, Ch. 7; Gray, 1981; Strongman, 1987) (see below for an extension of this definition). (Instrumental reinforcers are stimuli which if their occurrence, termination, or omission is made contingent upon the making of a response, alter the probability of the future emission of that response.) Some stimuli are unlearned reinforcers (e.g. the taste of food if the animal is hungry, or pain); while others may become reinforcing by learning, because of their association with such primary reinforcers, thereby becoming "secondary reinforcers". This type of learning may thus be called "stimulus-reinforcement association", and probably occurs via the process of classical conditioning. If a reinforcer increases the probability of emission of a response on which it is contingent, it is said to be a "positive reinforcer" or "reward"; if it decreases the probability of such a response it is a "negative reinforcer" or "punishment". It also turns out to be the case that responses followed by termination or omission of a positive reinforcer go down in probability, the operations being termed "time out" and "extinction" respectively (and sometimes being described as "punishing"); and that responses followed by the termination or omission of a negative reinforcer increase in probability, this pair of negative reinforcement operations being termed "escape" and "active avoidance" respectively (see Gray, 1975, Ch. 4; and Mackintosh, 1983, pp. 19–21, for a further

discussion of this terminology). For example, fear is an emotional state which might be produced by a sound which has previously been associated with pain. Pain in this example is the primary negative reinforcer, and fear is the emotional state which occurs to the tone stimulus as a result of the learning of the stimulus (i.e. tone)—reinforcement (i.e. pain) association. The tone in this example is a conditioned stimulus because of classical conditioning, and has secondary reinforcing properties in that responses will be made to escape from it and thus avoid the primary reinforcement of pain.

The different emotions can be described and classified according to whether the reinforcer is positive or negative, and by the reinforcement contingency. An outline of the classification scheme thus created is shown in Fig. 1. Movement away from the centre of the diagram represents increasing intensity of emotion, on a continuous scale. The diagram shows

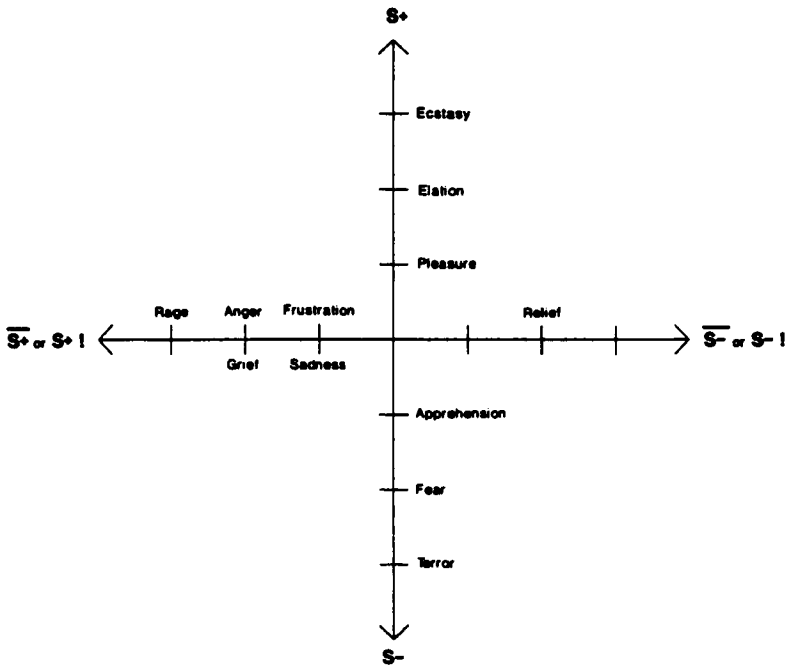

FIG. 1. Some of the emotions associated with different reinforcement contingencies are indicated. Intensity increases away from the centre of the diagram, on a continuous scale. The classification scheme created by the different reinforcement contingencies consists of: (1) the presentation of a positive reinforcer (S+); (2) the presentation of a negative reinforcer (S−); (3) the omission of a positive reinforcer ($\overline{S+}$) or the termination of a positive reinforcer (S+!); and (4) the omission of a negative reinforcer ($\overline{S-}$) or the termination of a negative reinforcer (S−!).

that emotions associated with the presentation of a positive reinforcer (S+) include pleasure, elation, and ecstasy. Of course, other emotional labels can be included along the same axis. Emotions associated with the presentation of a negative reinforcer (S−) include apprehension, fear, and terror (see Fig. 1). Emotions associated with the omission of a positive reinforcer ($\overline{S+}$) or the termination of a positive reinforcer (S+!) include frustration, anger, and rage. Emotions associated with the omission of a negative reinforcer ($\overline{S-}$) or the termination of a negative reinforcer (S−!) include relief. Although the classification of emotion presented here differs from earlier theories, the approach adopted here of defining and classifying emotions by reinforcing effects is one which has been developed in a number of earlier analyses (Millenson, 1967; Gray, 1975, 1981, 1987; see Strongman, 1987). It may be noted that along one of the axes of Fig. 1, the exact emotion which arises may also be influenced by the behaviour which is possible in a given environmental context. For example, if an active behavioural response can occur to the omission of an S+, then anger might be produced, but if only passive behaviour is possible, then sadness, depression or grief might occur.

The mechanisms described here would not be limited in the range of emotions for which they could account. First, different classes of emotion could arise because of different reinforcement contingencies, as described above. Secondly, different intensities within these categories or dimensions can produce different degrees of emotion (see above and Millenson, 1967). For example, as the strength of a positive reinforcer being presented increases, emotions might be labelled as pleasure, elation, and ecstasy. Similarly, as the strength of a negative reinforcer being presented increases, emotions might be labelled as apprehension, fear, and terror (see Fig. 1). It may be noted here that anxiety can refer to the state produced by stimuli associated with non-reward or punishment (Gray, 1987). Thirdly, any environmental stimulus might have a number of different reinforcement associations. For example, a stimulus might be associated both with the presentation of a reward and of a punishment, allowing states such as conflict and guilt to arise. The different possible combinations greatly increase the number of possible emotions. Fourthly, emotions elicited by stimuli associated with different reinforcers will be different even within a reinforcement category, because the original reinforcers are different. Thus, for example, the state elicited by a stimulus associated with a positive reinforcer, such as the taste of food, will be different from that elicited by a positive reinforcer such as grooming. Indeed, it is an important feature of the association memory mechanisms described here that when a stimulus is applied, it acts as a key which "looks up" or recalls the original reinforcer with which it was associated. Thus, emotional stimuli will differ from each other also in terms of the original reinforcers with

which they were associated. A fifth way in which emotions can be different from each other is in terms of the particular (conditioned) stimulus which elicits the emotion. Thus, even though the reinforcement contingency and even the unconditioned reinforcer may be identical, emotions will still be different cognitively, if the stimuli which give rise to the emotions are different. For example, the emotional state elicited by the sight of one person may be different from that elicited by the sight of another person because the people, and thus the cognitive evaluation associated with the perception of the stimuli, are different. A sixth possible way in which emotions can vary arises when the environment constrains the types of behavioural response which can be made. For example, it was noted above that different emotions can arise when the environment allows an active behavioural response, or constrains the behaviour to being passive. By realising that these six possibilities can be combined, it can be seen that it is possible to account for a very wide range of emotions, and this is believed to be one of the strengths of the approach described here. It is also the case that the degree to which a stimulus is reinforcing on a particular occasion (and thus the emotion produced) depends on the prior history of reinforcements (both recently and in the longer term), and that mood state can affect the degree to which a stimulus is reinforcing.

The definition of emotions given above, that they are states produced by reinforcing stimuli, is refined now. Only some of the states produced by reinforcing stimuli are emotional states. First, emotional states are normally initiated by external reinforcing stimuli, such as an (external) noise in the environment associated with pain (delivered by an external stimulus). In contrast, drive-related or motivational states, such as hunger and thirst, are normally initiated by internal stimuli, such as hypoglycemia (for hunger), or hyperosmolality (for thirst), and these states then make external stimuli, such as the taste of food or of water, become reinforcing (see Rolls, 1986c; Rolls & Rolls, 1982). It is remarked that if an external stimulus which is associated with a reinforcer is remembered, then this can produce an emotional state. (The remembered neuronal states are, it is thought, very similar to those produced by a real sensory input, in all but the early stages of sensory processing; see Rolls, 1989a). Further, the states elicited by reinforcing stimuli which are emotional states have certain functions (which are described below). It is also remarked that the stimulus which produces the emotional states does not have to be shown to be a reinforcer when producing the emotional state—it simply has to be capable of being shown to have reinforcing properties. The definition given provides great opportunity for cognitive processing (whether conscious or not) in emotions, for cognitive processes will often be required to determine whether an environmental stimulus or event is reinforcing. Normally an emotion consists of this cognitive processing which results in a decoded

signal that the environmental event is reinforcing, together with the mood state produced as a result. If the mood state is produced in the absence of the external sensory input and the cognitive decoding (for example, by direct electrical stimulation of the amygdala, see Rolls, 1975), then this is described only as a mood state, and is different from an emotion in that there is no object in the environment towards which the mood state is directed. It is suggested that, in order to produce some stability of mood, the firing rates of these neurons which are activated by external reinforcing stimuli to produce mood states must therefore have their spontaneous firing rates carefully controlled by the brain. (Many brain systems use lateral inhibition in order to maintain sensitivity to contrast constant, but this is not possible in the emotion system in which absolute levels of reinforcer must be reflected over moderately long time spans.) The difficulty of maintaining a constant absolute level of firing in neurons such as these may contribute to "spontaneous" mood swings, depression which occurs without a clear external cause, and the multiplicity of hormonal and transmitter systems which seem to be involved in the control of mood.

The approach described above shows that the learning of stimulus-reinforcement associations is the learning involved when emotional responses are learned. In so far as the majority of stimuli which produce our emotional responses do so as a result of learning, this type of learning, and the brain mechanisms which underlie it, are crucial to the majority of our emotions. This then provides a theoretical basis for understanding the functions of some brain systems in emotion. Ways in which this analytic approach can be applied are given below.

It also follows from this approach towards a theory of emotion that brain systems involved in disconnecting stimulus-reinforcement associations which are no longer appropriate will also be very important in emotion. Failure of this function would be expected to lead, for example, in frustrating situations to inappropriate perseveration of behaviour to stimuli no longer associated with positive reinforcement. The inability to correct behaviour when reinforcement contingencies change would be evident in a number of emotion-provoking situations, such as frustration (i.e. non-reward), and the punishment of previously rewarded behaviour. It will be shown below that this approach provides a basis for understanding the functions of some brain regions in emotion.

THE FUNCTIONS OF EMOTIONS

Before considering some of the main brain regions implicated in emotion in the context of this introduction, the functions of emotion will be considered, in order to provide further clarification of which states produced by reinforcing stimuli are emotional states, and of the nature of

emotions. Understanding the functions performed by emotions will be useful when considering the neural bases of emotion. (The following points are not necessarily mutually exclusive, but all I think help to clarify the functions of emotion.)

The first function proposed for emotion is the elicitation of autonomic and endocrine responses. It is of clear survival value to prepare the periphery, by, for example, increasing heart rate, so that actions which may be performed as a consequence of the reinforcing stimulus, such as running, can be performed more efficiently. The James–Lange theory (see Grossman, 1967; Schachter & Singer, 1962; Reisenzein, 1983), and theories which are closely related to it in supposing that feedback from parts of the periphery, such as the face (see Adelmann & Zajonc, 1989), leads to emotional feelings, have the major weakness that they do not give an adequate account of how the peripheral change is produced only by stimuli which happen to be emotion-provoking.

The second function proposed is of flexibility of behavioural responses to reinforcing stimuli. This can be seen by considering the Miller–Mowrer theory of avoidance learning (see Gray, 1975). The first stage or process in learning a behavioural response in order to avoid punishment is considered to be classical conditioning of an emotional response, for example, the elicitation of fear when the tone associated with shock sounds. The second stage or process is instrumental learning of an operant response, performed in order to terminate the fear-inducing stimulus. The advantage of having two processes is that if the particular operant response which has been learned becomes impossible (for example if a barrier prevents that response), then the organism is still in an emotional state of fear, when the tone sounds, and he can learn or perform another instrumental response to escape from or terminate the tone. This is preferable to a one-process method of avoidance learning, such as stimulus-motor response learning (e.g. hear tone–jump left), in that with such a one-factor theory, the organism would have to accept further experiences of the punishment until he learned another motor response by trial and error learning, and he might not survive the punishment a second time in real-life conditions in the natural habitat. Thus one function of emotion in the Miller–Mowrer theory of avoidance learning is that, once an emotional state has been produced by classical conditioning, then great flexibility of the behavioural response made as a consequence of that emotional state is possible. It is proposed that the allowance of flexibility of choice of the behavioural response to a reinforcing stimulus, so that the most appropriate response in any situation can be chosen, is a second function of emotion.

A third function of emotion is that it is motivating. For example, in the Miller–Mowrer theory (see Gray, 1975), the classically conditioned fear provides the motivation for the organism to perform the instrumental

response which enables him to avoid the shock. It is difficult to overemphasise the importance of this motivating function. It is one of the properties of reinforcers that if positive, the organism will work to obtain them, and that if negative, the organism will work to avoid them or will reduce responses on which they are made contingent. The state elicited by these reinforcers which motivates animals is an emotional state. In that reinforcers guide almost all our behaviour, the motivating properties of the emotional states elicited by reinforcers underlie almost all our behaviour. It is even likely that the inputs to our decision mechanisms include the motivational signals arising as part of the emotional state produced by reinforcers.

A fourth function of emotion is in communication. For example, monkeys may communicate their emotional state to others, by making an open-mouth threat, and this may influence the behaviour of other animals. Communicating emotional states may have survival value, for it may enhance the stability of social groups, by, for example, reducing fighting. It is, of course, possible in a small proportion of the population that the true emotional state may not be communicated and this may be an evolutionarily stable strategy (see Dawkins, 1989; Krebs & Dawkins, 1984).

A fifth function of emotion is in social bonding. Examples of this are the emotions associated with the attachment of the parents to their young (for example, the love of a mother for her child), with the attachment of the young to their parents, and with the attachment of the parents to each other. In the theory of the ways in which the genes affect behaviour ("selfish gene" theory, see Dawkins, 1989), it is held that (because, for example, of the advantages of parental care) all these forms of emotional attachment have the effect that genes for such attachment are more likely to survive into the next generation. Altruism can also be considered in these terms. (In order for such genes to be transmitted into the next generation, they must be resistant to invasion by other genes.)

A sixth function of emotion may be generalised from the above. It may be suggested that anything which feels pleasant to the organism, and is positively reinforcing, so that actions made to obtain it are performed, has survival value. One example of this is slight novelty, which may feel good and be positively reinforcing because it may lead to the discovery of better opportunities for survival in the environment (e.g. a new food). Another example is gregariousness, for behaviour as a member of a group may enhance gene survival in some species. (For many species, living in groups is an advantage—see Krebs & Davies, 1984.) Conversely, it is likely that natural selection acting on genes will lead to unpleasant feelings, and negative reinforcement, being associated with behaviour which does not have survival value, at least in cases where genes can influence matters. Of course, the genes may be misled sometimes and lead to behaviour which

does not have survival value, as when for example the non-nutritive sweetener saccharin is eaten by animals. This does not disprove the theory, but only points out that the genes cannot specify correctly for every possible event in the environment, but must only on average lead to behaviour appropriate for gene survival feeling pleasant. It is of interest that sensory-specific satiety, the phenomenon whereby prolonged normally reinforcing sensory stimulation may gradually become less pleasant (Rolls, 1981c, 1986c, 1989b), is probably a general adaptation to ensure that behaviour does eventually switch from one reinforcer to another.

A seventh effect of emotion is that the current mood state can affect the cognitive evaluation of events or memories (see Blaney, 1986), and this may have the function of facilitating continuity in the interpretation of the reinforcing value of events in the environment. A theory of how this occurs is presented in the section "Effects of emotions on cognitive processing".

An eighth function of emotion is that it may facilitate the storage of memories. One way in which this occurs is that episodic memory (i.e. one's memory of particular episodes) is facilitated by emotional states. Examples of this are the memories of where one was, with whom, etc. when emotion-provoking events are discovered, such as the death of a president. This enhancement of episodic memory may be advantageous in that storage of as many details as possible of the prevailing situation when a strong reinforcer is delivered may be useful in generating appropriate behaviour in situations with some similarities in the future. A second way in which emotion may affect the storage of memories is that the current emotional state may be stored with episodic memories, providing a mechanism for the current emotional state to affect which memories are recalled. A third way in which emotion may affect the storage of memories is by guiding the cerebral cortex in the representations of the world which are set up. For example, in the visual system, it may be useful rather than building perceptual representations or analysers based only on information received from the eyes, to guide the formation of representations in higher order parts of the visual system by feedback from the world. For example, if two physically similar visual stimuli are associated with different reinforcing consequences, then it may be important to build different representations of them, so that the visual system provides different outputs for these two stimuli. On the other hand, it may be possible to economise in the number of representations formed, by being less likely to build representations if there are no reinforcing consequences of stimuli. Ways in which back-projections from parts of the brain important in emotion (such as the amygdala) to parts of the brain important in storing perceptual representations (such as parts of the cerebral cortex) are discussed in the section "Effects of emotions on cognitive processing".

It may be noted that it might be possible to build a computer which

would perform the above functions of emotions, and yet we might not want to ascribe to it emotional feelings. This particular point in fact raises the general problem of consciousness and its functions. Although this will not be considered here, it is worth noting that one possible function of consciousness might be to enable prediction from an internal model of the world to be more accurate, in that the internal simulation or model of the world would have access (based on generalisation from personal experiences) to how another animal might feel, and thus behave, in different situations (see e.g. Humphrey, 1980).

THE NEURAL BASES OF EMOTION

Some of the main brain regions implicated in emotion will now be considered. These include the amygdala, prefrontal cortex, and hypothalamus. Some of these brain regions are indicated in Fig. 2, and reference to this may be helpful throughout this article. Particular attention is paid to the functions of these regions in primates, for in primates the neocortex undergoes great development and provides major inputs to these regions, in some cases to parts of these structures thought not be present in non-

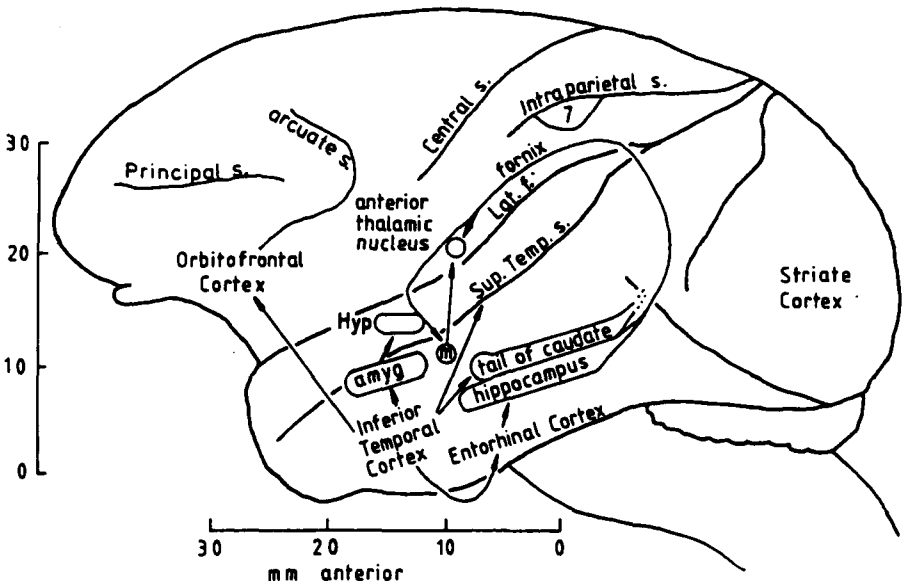

FIG. 2. Some of the pathways described in the text are shown on this lateral view of the rhesus monkey brain. amyg, amygdala; central s, central sulcus; Hyp, hypothalamus/substantia innominata/basal forebrain; Lat f, lateral (or Sylvian) fissure; m, mammillary body; Sup Temp s, superior temporal sulcus; 7, posterior parietal cortex, area 7.

primates. An example of this is the projection from the primate neocortex in the anterior part of the temporal lobe to the basal accessory nucleus of the amygdala (see below). Studies in primates are thus particularly relevant to understanding the neural basis of emotion in the human. Brain regions involved in an important primary reinforcer, pain, are discussed by Melzack and Wall (1988).

The Amygdala

The amygdala is a subcortical region in the anterior part of the temporal lobe. It receives massive projections in the primate from the overlying temporal lobe cortex (Aggleton, Burton, & Passingham, 1980; Herzog & Van Hoesen, 1976; Turner, 1981; Turner, Mishkin & Knapp, 1980; Van Hoesen, 1981). These come in the monkey to overlapping but partly separate regions of the lateral and basal amygdala from the inferior temporal visual cortex, the superior temporal auditory cortex, the cortex of the temporal pole, and the cortex in the superior temporal sulcus. Thus, the amygdala receives inputs from temporal lobe association cortex, but not from earlier stages of cortical visual information processing. It also receives projections from the posterior orbitofrontal cortex (see Fig. 2, areas 12 and 13). Subcortical inputs to the amygdala include projections from the midline thalamic nuclei, the subiculum and CA1 parts of the hippocampal formation, the hypothalamus, and substantia innominata, the nucleus of the solitary tract (which receives gustatory and visceral inputs), and from olfactory structures (Ben-Ari, 1981). Although there are some inputs from early on in some sensory pathways, for example auditory inputs from the medial geniculate nucleus (LeDoux, 1987), this route is unlikely to be involved in most emotions, for which cortical analysis of the stimulus is likely to be required. The outputs of the amygdala (Price, 1981; Ben-Ari, 1981) include the well-known projections to the hypothalamus, from the lateral amygdala via the ventral amygdalofugal pathway to the lateral hypothalamus; and from the medial amygdala, which is relatively small in the primate, via the stria terminalis to the medial hypothalamus. The ventral amygdalofugal pathway is now known to contain some long descending fibres that project to the autonomic centres in the medulla oblongata (Hopkins, McLean, & Takeuchi, 1981; Price, 1981; Schwaber, Kapp, Higgins & Rapp, 1982), and provide a route for cortically processed signals to reach the brainstem. A further interesting output of the amygdala is to the ventral striatum (Heimer, Switzer, & Van Hoesen, 1982), including the nucleus accumbens, for via this route information processed in the amygdala could gain access to the basal ganglia and thus influence motor output. The amygdala also projects to the medial part of the mediodorsal nucleus of the thalamus (Nauta, 1961; Price, 1981), which

projects to the orbitofrontal cortex and provides the amygdala with another output. In addition, the amygdala has direct projections back to many areas of the temporal, orbitofrontal, and insular cortices from which it receives inputs (Mufson, Mesulam & Pandya, 1981; Porrino, Crane, & Goldman-Rakic, 1981; Price, 1981; Amaral, 1986). It is suggested elsewhere (Rolls, 1989a) that the functions of these backprojections include the guidance of information representation and storage in the neocortex (when this is performed by reinforcing stimuli), and recall. Ways in which these functions of backprojections may be performed are discussed by Rolls (1989a). Another interesting set of output pathways of the amygdala projects to the entorhinal cortex, which provides the major input to the hippocampus and dentate gyrus, and to the ventral subiculum, which provides a major output of the hippocampus (Price, 1981; Amaral, 1986).

These anatomical connections of the amygdala indicate that it is placed to receive highly processed information from the cortex and to influence motor systems, autonomic systems, some of the cortical areas from which it receives inputs, and other limbic areas. The functions mediated through these connections will now be considered, using information available from the effects of damage to the amygdala and from the activity of neurons in the amygdala.

Bilateral removal of the amygdala in monkeys produces striking behavioural changes which include tameness, a lack of emotional responsiveness, excessive examination of objects, often with the mouth, and eating of previously rejected items such as meat (Weiskrantz, 1956). These behavioural changes comprise much of the Kluver–Bucy syndrome which is produced in monkeys by bilateral anterior temporal lobectomy (Kluver & Bucy, 1939). In analyses of the bases of these behavioural changes, it has been observed that there are deficits in some types of learning. For example, Weiskrantz (1956) found that bilateral ablation of the amygdala in the monkey produced a deficit in learning an active avoidance task. The monkeys failed to learn to make a response when a light signalled that shock would follow unless the response was made. He was perhaps the first to suggest that these monkeys had difficulty with forming associations between stimuli and reinforcement, when he suggested that "the effect of amygdalectomy is to make it difficult for reinforcing stimuli, whether positive or negative, to become established or to be recognised as such" (Weiskrantz, 1956). In this avoidance task, associations between a stimulus and negative reinforcement were impaired. Evidence soon became available that associations between stimuli and positive reinforcement (reward) were also impaired in, for example, serial reversals of a visual discrimination made to obtain food (Jones & Mishkin, 1972). In this task the monkey must learn that food is under one of two objects, and after he has learned this, he must then relearn (reverse) the association as the food is then

placed under the other object. Jones and Mishkin (1972) showed that the stages of this task which are particularly affected by damage to this region are those when the monkeys are responding at chance to the two visual stimuli or are starting to respond more to the currently rewarded stimuli, rather than the stage when the monkeys are continuing to make perseverative responses to the previously rewarded visual stimulus. They thus argued that the difficulty produced by this anterior temporal lobe damage is in learning to associate stimuli with reinforcement, in this case with food reward. Further more direct evidence for this is that amygdalectomised monkeys were impaired in a test in which they had to remember on the basis of a single presentation whether or not a trial-unique object had been paired with reward (Spiegler & Mishkin, 1981; Mishkin & Aggleton, 1981). In more recent experiments, Gaffan and Harrison (1987) and Gaffan, Gaffan, and Harrison (1988) have shown that the tasks which are impaired by amygdala lesions in monkeys typically involve a cross-modal association from a previously neutral stimulus to a primary reinforcing stimulus (such as the taste of food), consistent with the hypothesis that the amygdala is involved in learning associations between stimuli and primary reinforcers (see also Gaffan, Gaffan, & Harrison, 1989). Further evidence linking the amygdala to reinforcement mechanisms is that monkeys will work in order to obtain electrical stimulation of the amygdala, and that single neurons in the amygdala are activated by brain-stimulation reward at a number of different sites (Rolls, 1975; Rolls, Burton, & Mora, 1980).

Jones and Mishkin (1972) elaborated the hypothesis that many of the symptoms of the Kluver–Bucy syndrome, including the emotional changes, could be a result of this type of deficit in learning stimulus-reinforcement associations (see also Mishkin & Aggleton, 1981). For example the tameness, the hypoemotionality, the increased orality, and the altered responses to food would arise because of damage to the normal mechanism by which stimuli become associated with reward or punishment. Other evidence is also consistent with the hypothesis that there is a close relation between the learning deficit and the symptoms of the Kluver–Bucy syndrome. For example, in a study of subtotal lesions of the amygdala, Aggleton and Passingham (1981) found that in only those monkeys in which the lesions produced a serial reversal learning deficit was hypoemotionality present.

It may be noted here that the amygdala is well placed anatomically for such stimulus–reinforcement association learning, for not only does it receive highly processed visual and auditory inputs from the temporal lobe cortex, but it also receives inputs from the gustatory and somatosensory systems (via the insula in the primate—Mesulam & Mufson, 1982), and the olfactory and visceral systems (see Ben-Ari, 1981), so that a variety of well-processed stimuli and information about reinforcement should have access

to the amygdala. This type of anatomical convergence is appropriate for a system implicated in stimulus-reinforcement association formation, in which it may be necessary to associate stimuli from different modalities. Thus, in this function it may be necessary to "look up" (i.e. produce as output, or recall) for example, a taste or a pleasant or painful somatosensory event using for example a visual or auditory "key" (or conditioned) stimulus which was previously associated with the taste or somatosensory event. Moreover, the outputs of the amygdala include connections to the autonomic centres of the brainstem, hypothalamus, and other limbic as well as cortical structures, through some of which autonomic and others of which behavioural responses learned to stimuli which have been previously paired with reinforcement could be produced. In line with this, LeDoux, Iwata, Cichetti, and Reis (1988) were able to show that lesions of the lateral hypothalamus (which receives from the central nucleus of the amygdala) blocked conditioned heart rate (autonomic) responses but not the conditoned behavioural emotional response of freezing to an aversive conditioned stimulus. In contrast, lesions of the central gray (which also receives from the central nucleus of the amygdala) blocked the conditioned freezing but not the conditioned autonomic response to the aversive conditioned stimulus. Further, Cador, Robbins, and Everitt (1989) obtained evidence consistent with the hypothesis that the learned incentive (conditioned reinforcing) effects of previously neutral stimuli paired with rewards are mediated by the amygdala acting through the ventral striatum, in that amphetamine injections into the ventral striatum enhanced the effects of a conditioned reinforcing stimulus only if the amygdala was intact.

Although much evidence is thus consistent with the hypothesis that the amygdala is involved in responses made to stimuli associated with reinforcement, there is evidence that it may also be involved to some extent in behavioural responses made to novel, as opposed to familiar, stimuli, in a different type of memory. It has been found, for example, that the alteration in responses to foods in rats with damage to the amygdala is due in part to decreased neophobia; that is, the rats more quickly accept new foods (Rolls & Rolls, 1973; see also Dunn & Everitt, 1988; and Wilson & Rolls, 1990b).

Recordings from single neurons in the amygdala of the monkey have shown that some neurons do respond to visual stimuli, consistent with the inputs from the temporal lobe visual cortex (Sanghera, Rolls, & Roper-Hall, 1979). Other neurons responded to auditory, gustatory, olfactory, or somatosensory stimuli, or in relation to movements. In tests of whether the neurons responded on the basis of the association of stimuli with reinforcement, it was found that approximately 20% of the neurons with visual responses had responses which occurred primarily to stimuli associated

with reinforcement, for example, to a range of stimuli which the monkey had learned signified food (Sanghera et al., 1979; Rolls, 1981b). However, none of these neurons (in contrast to some neurons in the hypothalamus described below) responded only to rewarded stimuli, in that all responded at least partly to one or more neutral or aversive stimuli. Neurons with responses which are probably similar to these have also been described by Ono et al. (1980), and by Nishijo, Ono, and Nishino (1988). However, 10 of 11 neurons tested in the reversal of a visual discrimination (in which the visual stimulus associated with food reward delivery becomes associated with aversive saline delivery and vice versa) did not reverse their responses (and for the remaining neuron the evidence was not clear) (Sanghera et al., 1979; Wilson & Rolls, 1990b). Thus, these amygdaloid neurons had a preference for reinforcing stimuli, but did not alter their responses flexibly and rapidly when the reinforcement value of a visual stimulus was altered during reversal. (Nishijo et al., 1988, have tested four amygdala neurons in a rather simpler relearning situation in which salt was added to a piece of food such as a water-melon, and the neurons' responses to the sight of the water-melon diminished. This was an extinction rather than a reversal test, and it will be of interest in further studies to investigate whether the apparently altering neuronal responses were related to this simpler test situation or to other differences in the procedure.)

The failure of this population of amygdala neurons to respond only to reinforcing stimuli, and the difficulty in reversing their responses, are in contrast to the responses of certain populations of neurons in the caudal orbitofrontal cortex and in a region to which it projects, the basal forebrain, which do show very rapid (in one or two trials) reversals of their responses in visual discrimination reversal tasks (Thorpe, Rolls, & Maddison, 1983; Wilson & Rolls, 1990b; see below). On the basis of these findings, it is suggested that the orbitofrontal cortex is more involved than the amygdala in the rapid readjustments of behavioural responses made to stimuli when their reinforcement value is repeatedly changing, as in discrimination reversal tasks (Thorpe et al., 1983; Rolls, 1986b). The ability to flexibly alter responses to stimuli based on their changing reinforcement associations is important in motivated behaviour (such as feeding) and in emotional behaviour, and it is this flexibility which it is suggested the orbitofrontal cortex adds to a more basic capacity which the amygdala implements for stimulus-reinforcement learning (Rolls, 1986b).

Another interesting group of neurons in the amygdala responds primarily to faces (Rolls, 1981b; Leonard, Rolls, Wilson, & Baylis, 1985). It is probable that these neurons receive their inputs from a group of neurons in the cortex in the superior temporal sulcus which respond to faces, often on the basis of features present, such as eyes, hair, or mouth (Perrett, Rolls, & Caan, 1982). It has been suggested that this is part of a system which has

evolved for the rapid and reliable identification of individuals from their faces, because of the importance of this in primate social behaviour (Rolls, 1981b, 1984, 1985, 1990b; Perrett & Rolls, 1982; Leonard et al., 1985). The part of this system in the amygdala may be particularly involved in emotional and social responses to faces. According to one possibility, such emotional and social responses would be "looked up" by a "key" stimulus, which consisted of the face of a particular individual (Rolls, 1984, 1987, 1990b). Indeed, it is suggested that the tameness of the Kluver–Bucy syndrome, and the inability of amygdalectomised monkeys to interact normally in a social group (Kling & Steklis, 1976), arises because of damage to this system specialised for processing faces (Rolls, 1981a,b, 1984, 1985, 1990b).

Cortical cells found in certain of the temporal lobe regions (e.g. TEa, TEm, and TPO; Baylis, Rolls & Leonard, 1987) which project into the amygdala have properties which would enable them to provide useful inputs to such an associative mechanism in the amygdala (see Rolls, 1987, 1989a, 1990b,c). These cortical neurons in many cases respond differently to the faces of different individuals, so that information about face identity which would be useful in recognition is represented by some of the neurons. However, the face of each individual is coded by the pattern of firing across a subpopulation of neurons (Baylis, Rolls, & Leonard, 1985). That is, ensemble encoding rather than "grandmother cell" encoding is used. It is hypothesised that this type of tuning found is a delicate compromise between very fine tuning, which has the advantage of low interference in associative neuronal network operations but the disadvantage of losing the emergent properties of storage in such neuronal networks; and broad tuning, which has the advantage of allowing the emergent properties of neuronal networks to be realised but the disadvantage of leading to interference between the different memories stored in an associative network (Rolls, 1987, 1989a). Neurons in these areas are seen as filters which, as an ensemble, give a unique representation of a particular stimulus in the environment. There is evidence that the responses of some of these neurons are altered by experience so that new stimuli become incorporated in the network (Rolls, Baylis, Hasselmo, & Nalwa, 1989). Competition may play a role in the self-organisation of such networks (Rolls, 1989a,c). The representation which is built in temporal cortical areas shows considerable size, contrast, spatial frequency, and translation invariance (Rolls & Baylis, 1986; Azzopardi & Rolls, 1989; Rolls, 1990b). Thus, the representation is in a form which is particularly useful for storage and as an output from the visual system. A further advantage for an input to such an associative system in the amygdala, where associations may be made to primary reinforcers, is that for some of the cortical neurons the representation which is built is object-based rather than viewer-centred

(Hasselmo, Rolls, Baylis, & Nalwa, 1989b). In addition to the population of neurons which code for face identity, there is a separate population in the cortex which conveys information about facial expression (Hasselmo, Rolls, & Baylis, 1989a). These cortical neurons probably provide an input to the amygdaloid neurons which respond to faces, and one output from the amygdala for this information is probably via the ventral striatum, for a small population of neurons has been found in the ventral striatum with responses selective for faces (Rolls & Williams, 1987).

Although our understanding of the functions of the amygdala is based largely on research in animals, the evidence which is available for the human is consistent with the evidence described above suggesting that the amygdala is involved in emotion. Thus, electrical stimulation of the human amygdala can give rise to emotional feelings such as fear, anxiety, or in some cases pleasure (see Halgren, 1981). Conversely, amygdalectomy in humans has been claimed to reduce emotional tension, to make fear and aggression harder to provoke, and to enhance emotional control, resulting in better concentration, a steadier mood, and more rewarding social interactions (see review by Halgren, 1981). However, the requirements for adequate controls, adequate and independent assessment of the behavioural changes, and long-term follow-up studies are difficult to meet in this type of work, and these claims must be regarded as being only tentative (see Halgren, 1981; Valenstein, 1973, 1980). Robin and Macdonald (1975) and Valenstein (1973, 1980) have discussed many of the interpretive and major ethical problems endemic to this type of work and to psychosurgery in man.

The evidence described above implicates the amygdala in the learning of associations between stimuli and reinforcement. This means that it must be important in learned emotional responses, and at least part of the importance of the amygdala in emotion appears to be that it is involved in this type of emotional learning. This analysis provides a theoretical basis for understanding how the amygdala is involved in emotion.

The Orbitofrontal Cortex

The prefrontal cortex is the cortex that receives projections from the mediodorsal nucleus of the thalamus and is situated in front of the motor and premotor cortices (Areas 4 and 6) in the frontal lobe. Based on the divisions of the mediodorsal nucleus, the prefrontal cortex may be divided into three main regions (Fuster, 1989). First, the magnocellular, medial, part of the mediodorsal nucleus projects to the orbital (ventral) surface of the prefrontal cortex (which includes Areas 13 and 12). It is called the orbitofrontal cortex, and is the part of the primate prefrontal cortex that appears to be primarily involved in emotion. Secondly, the parvocellular,

lateral, part of the mediodorsal nucleus projects to the dorsolateral prefrontal cortex. This part of the prefrontal cortex is involved in cognitive tasks such as spatial short-term memory tasks (Fuster, 1989; Rolls, 1985; Rosenkilde, 1979). Thirdly, the pars paralamellaris (most lateral) part of the mediodorsal nucleus projects to the frontal eye fields (Area 8) in the anterior bank of the arcuate sulcus (Wurtz, Goldberg, & Robinson, 1980).

The orbitofrontal cortex will be considered in the rest of this section. The cortex on the orbital surface of the frontal lobe includes Area 13 caudally, and Area 14 medially, and the cortex on the inferior convexity includes Area 12 caudally and Area 11 anteriorly.

The orbitofrontal cortex receives inputs via the mediodorsal nucleus of the thalamus, pars magnocellularis, which itself receives afferents from temporal lobe structures, such as the prepyriform (olfactory) cortex, amygdala, and inferior temporal cortex (Nauta, 1972; Krettek & Price, 1974, 1977). Another set of inputs reaches the orbitofrontal cortex directly from the inferior temporal cortex, the cortex in the superior temporal sulcus, and the temporal pole (Jones & Powell, 1970; Van Hoesen, Pandya, & Butters, 1975; Chavis & Pandya, 1976; Seltzer & Pandya, 1989; Petrides & Pandya, 1988; Barbas, 1988); and a third set comprises the at least partly dopaminergic projection from the ventral tegmental area. The orbitofrontal cortex projects back to temporal lobe areas such as the inferior temporal cortex, and, in addition, to the entorhinal cortex (or "gateway to the hippocampus") and cingulate cortex (Nauta, 1964; Van Hoesen et al., 1975). The orbitofrontal cortex also projects to the preoptic region and lateral hypothalamus, to the ventral tegmental area (Nauta, 1964), and to the head of the caudate nucleus (Kemp & Powell, 1970). Damage to the caudal orbitofrontal cortex in the monkey produces emotional changes. These include decreased aggression to humans and to stimuli such as a snake and a doll, and a reduced tendency to reject foods such as meat (Butter, Snyder, & McDonald, 1970; Butter & Snyder, 1972; Butter, McDonald, & Snyder, 1969). In the human, euphoria, irresponsibility, and lack of affect can follow frontal lobe damage (see Kolb & Whishaw, 1985).

These changes which follow frontal lobe damage may be related to a failure to react normally to non-reward in a number of different situations. This failure is evident as a tendency to respond when responses are inappropriate, e.g. no longer rewarded. For example, monkeys with orbitofrontal damage are impaired on go/no-go task performance, in that they go on the no-go trials (Iversen & Mishkin, 1970), in an object reversal task in that they respond to the object which was formerly rewarded with food, and in extinction in that they continue to respond to an object which is no longer rewarded (Butter, 1969; Jones & Mishkin, 1972). There is some evidence for dissociation of function within the orbitofrontal cortex, in that lesions to the inferior convexity produce the go/no-go and object

reversal deficits, whereas damage to the caudal orbitofrontal cortex, Area 13, produces the extinction deficit (Rosenkilde, 1979).

The hypothesis that the orbitofrontal cortex is involved in correcting responses made to stimuli previously associated with reinforcement has been investigated by making recordings from single neurons in the orbitofrontal cortex while monkeys performed these tasks known to be impaired by damage to the orbitofrontal cortex (Thorpe et al., 1983). It has been found that one class of neurons in the orbitofrontal cortex of the monkey responds in certain non-reward situations (Thorpe et al., 1983). For example, some neurons responded in extinction, immediately after a lick had been made to a visual stimulus which had previously been associated with fruit juice reward. Some neurons responded in a reversal task, immediately after the monkey had responded to the previously rewarded visual stimulus, but had obtained punishment rather than reward. These neurons did not respond simply as a function of arousal, and usually responded in only some non-reward situations. It is thus suggested that their responses reflect information that expected reward has not been given in a certain situation. This information appears to be necessary for the primate to alter behavioural responses when reinforcement contingencies are changed, as shown by the effects of damage to the orbitofrontal cortex described above.

It was of interest that only a relatively small proportion (approximately 3.5%) of orbitofrontal neurons responded in this way, but that many of the other neurons had responses which suggested that they were involved in this computation that a particular environmental stimulus was no longer associated with reward. Thus, another class of orbitofrontal neurons responded in relation to the sensory aspects of visual stimuli, consistent with the projections to the orbitofrontal cortex from the inferior temporal visual cortex (see Thorpe et al., 1983). Other neurons responded to particular visual stimuli only if they were associated with reward. Thus these neurons conveyed information that a particular visual stimulus had been seen, and that it was currently associated with reward. Another class of neurons conveyed information about whether a reward had been given, responding for example to the taste of sucrose, or for other neurons of saline (Thorpe et al., 1983). These orbitofrontal neurons with gustatory responses have now been analysed further (Rolls, 1989b), and can be tuned quite finely to gustatory stimuli such as a sweet taste (Rolls, Yaxley, & Sienkiewicz, in press; Rolls, 1989b). Moreover, their activity is related to reward, in that those which respond to the taste of food do so only if the monkey is hungry (Rolls, Sienkiewicz, & Yaxley, 1989). It is likely that these neurons receive their input from the primary gustatory cortex, in the frontal operculum (Scott, Yaxley, Sienkiewicz, & Rolls, 1986; Rolls, 1989b). Moreover, in part of this orbitofrontal region, some neurons

combine taste and olfactory inputs, in that they are bimodal (Rolls, 1989b). It is suggested that these types of information are represented in the responses of orbitofrontal neurons because they are part of a mechanism which evaluates whether a reward is expected, and generates a mismatch (evident as firing of the non-reward neurons) if reward is not obtained when it is expected. This neurophysiological analysis thus provides evidence that neurons with responses appropriate for a system involved in correcting behavioural responses when these are no longer associated with reinforcement are present in the orbitofrontal cortex (Thorpe et al., 1983). This is further evidence that the orbitofrontal cortex is involved in emotional responses, particularly when these involve correcting previously learned reinforcement contingencies, in situations which include those usually described as involving frustration.

It is of interest that a number of the symptoms of frontal lobe damage in man appear to be related to this type of function, of altering behaviour when particular responses become inappropriate, as described next. Thus, humans with frontal lobe damage can show impairments in a number of tasks in which an alteration of behavioural strategy is required in response to a change in environmental reinforcement contingencies (see Goodglass & Kaplan, 1979; Jouandet & Gazzaniga, 1979; Kolb & Whishaw, 1985). For example, Milner (1963) showed that on the Wisconsin Card Sorting Task (in which cards are to be sorted according to the colour, shape, or number of items on each card depending on whether the examiner says "right" or "wrong" to each placement), frontal patients either had difficulty in determining the first sorting principle, or in shifting to a second principle when required to. Also, in stylus mazes, frontal patients have difficulty in changing direction when a sound indicates that the correct path has been left (see Milner, 1982). It is of interest that, in both types of test, frontal patients may be able to verbalise the correct rules, yet may be unable to correct their behavioural sets of strategies appropriately. Some of the personality changes which can follow frontal lobe damage may be related to a similar type of dysfunction. For example, the euphoria, irresponsibility, lack of affect, and lack of concern for the present or future which can follow frontal lobe damage (see Hécaen & Albert, 1978) may also be related to a dysfunction in altering behaviour appropriately in response to a change in reinforcement contingencies. Indeed, in so far as the orbitofrontal cortex is involved in the disconnection of stimulus reinforcement associations, and such associations are important in learned emotional responses (see above), then it follows that the orbitofrontal cortex is involved in emotional responses by correcting stimulus-reinforcement associations when they become inappropriate.

The Hypothalamus

There is evidence that the hypothalamus is involved in emotion. For example, Bard (1928) showed that transection of the brain caudal to the hypothalamus resulted in unintegrated fragments of rage behaviour in a cat when it was provoked. In contrast, transection rostral to the hypothalamus resulted in more integrated emotional behaviour, in which, for example, hissing and purring did not occur simultaneously (also see Grossman, 1967, Chapter 9). These observations may be interpreted as showing that neural structures at the level of the hypothalamus are important in integrating behavioural responses and thus in integrated expressions of emotional reactions. Hess in the 1930s (Hess, 1954) showed that electrical stimulation of the hypothalamus can lead to rage responses, and this also suggested that neural systems involved in emotions were in or had connections through the hypothalamus. Further evidence relating the hypothalamus to reinforcement was found when Olds and Milner, in 1954, demonstrated that electrical stimulation of the hypothalamus could provide reward (see Rolls, 1975). Grossman (1966) also emphasised the function of the hypothalamus in emotion, observing that rats with ventromedial hypothalamic lesions became vicious and that active avoidance was facilitated.

Further evidence implicating the hypothalamus in emotional responses was found by Rolls and his colleagues. When analysing the role of the hypothalamus in feeding, they described a population of hypothalamic neurons that responded to real foods, but came to respond, as a result of stimulus-reinforcement association formation, to visual stimuli associated with food and only responded to these stimuli when the monkey was hungry (Rolls, 1975, 1981a,c, 1982, 1986; Rolls, Burton, & Mora, 1976; Burton, Rolls, & Mora, 1976; Mora, Rolls, & Burton, 1976; Wilson & Rolls, 1990). In that these neurons responded to stimuli associated with reward, their responses reflected the operation of the type of learning involved in learned emotional responses, that is, stimulus-reinforcement association formation. Further evidence for this is that other neurons in the hypothalamus responded only to stimuli associated with punishment, that is to aversive visual stimuli (Rolls, Sanghera, & Roper-Hall, 1979). In that the responses of these neurons occurred to any visual stimuli associated with food reward, or in other cases to any aversive visual stimuli, the responses reflected the look-up by an associative memory mechanism to produce an output appropriate for a motivational or emotional response. This neuronal output would be appropriate for producing autonomic responses to emotional stimuli, via pathways which descend from the hypothalamus towards the brainstem autonomic motor nuclei (Saper, Loewy, Swanson, & Cowan, 1976; Schwaber et al., 1982). It is also possible that these outputs could influence emotional behaviour, through, for example, the

connections from the hypothalamus to the amygdala (Aggleton et al., 1980), to the substantia nigra (Nauta & Domesick, 1978), or even by the connections to the neocortex (Divac, 1975; Kievit & Kuypers, 1975). Indeed, it is suggested that the latter projection, by releasing acetylcholine in the cerebral cortex when emotional stimuli (including reinforcing and novel stimuli) are seen, provides one way in which emotion can influence the storage of memories in the cerebral cortex (Rolls, 1987; Wilson & Rolls, 1990c). In this case, the basal forebrain magnocellular neurons may act as a "strobe" which facilitates memory processing.

Inputs to the hypothalamus that enable the neurons in it to respond in these ways reach the hypothalamus through its limbic and frontal connections. Thus the amygdala provides a route for highly processed visual information from the inferior temporal visual cortex to reach the hypothalamus (see also Fukuda, Ono, & Nakamura, 1987). It is also likely that the amygdala is involved in the learning process by which only food-related visual stimuli come to influence this population of hypothalamic neurons. Similarly, the connections from the orbitofrontal cortex to the hypothalamus may be important in ensuring that only visual and other stimuli that are still associated with reward continue to activate hypothalamic neurons, thus maintaining their responses specific to the emotional and motivational significance of environmental stimuli (see above and Thorpe et al., 1983).

EFFECTS OF EMOTION ON COGNITIVE PROCESSING

The analyses above of the neural mechanisms of emotion have been concerned primarily with how stimuli are decoded to produce emotional states, and with how these states can influence behaviour. We have seen, for example, that the amygdala and orbitofrontal cortex are important in selecting those stimuli which have been associated with primary reinforcers in the past, and in producing emotional responses to these stimuli. We have seen that the tuning of neurons in these structures, and in the structures which send projections to them such as the inferior temporal cortex, can be understood as providing appropriate inputs to associative neuronal networks which perform the stimulus-reinforcement learning which is necessary for learned emotions. To finish this article, I wish to make a suggestion about how emotional states may influence cognitive processing.

Current mood state can affect the cognitive evaluation of events or memories (see Blaney, 1986). For example, happy memories are more likely to be recalled when happy. Why does this occur? It is suggested that whenever memories are stored, part of the context is stored with the memory. This is very likely to happen in associative neuronal networks

such as those in the hippocampus (Rolls, 1987, 1989a, 1990a). In such networks, memories of particular events are stored by increasing the strengths of active synapses which make connections to strongly activated neurons. Consider the points that each neuron has many inputs (approximately 10,000 in the rat hippocampus), and that the networks in the CA3 part of the hippocampus appear to operate as an auto-associative memory capable of linking together almost arbitrary co-occurrences of inputs. It is therefore very likely that some of the input axons will be carrying information about the current emotional state, and that these synapses as well as those conveying the particular event to be remembered will be enhanced. Now, recall of a memory occurs best in such networks when the input key to the memory is nearest to the original input pattern of activity which was stored. (Technically, optimal recall occurs when the key has a correlation of 1.0 with the original input to the memory—see Rolls, 1987, 1989a; Jordan, 1986.) It thus follows that a memory of, for example, a happy episode is recalled best when in a happy mood state, for then the key to the memory (the input pattern vector of axonal activity to each neuron) most closely resembles the pattern of modified synapses, so that the activation of those neurons with the matching synaptic pattern is optimal. This is a general theory of how context is stored with a memory, and of how context influences recall. (The "context" is simply part of what is stored, and it would be difficult in associative neural networks to arrange for no context to be stored, given that in principle the network should have considerable flexibility about what is connected to what.) The effect of emotional state on cognitive processing and memory is thus suggested to be a particular case of a more general way in which context can affect the storage and retrieval of memories, or can affect cognitive processing.

There are a number of sites in the brain where these effects of mood on storage and recall could be instantiated. One place is the hippocampus. The hippocampus receives projections (via the entorhinal cortex) from the amygdala, so that emotional states could easily gain access to the autoassociative network implemented by the CA3 recurrent collateral axons, and become linked into episodic memories (see Rolls, 1989a, 1990a). Another brain system where mood state could be stored with memories is in the backprojections from structures important in emotion such as the amygdala and orbitofrontal cortex to parts of the cerebral cortex important in the representation of objects, such as the inferior temporal visual cortex. It is suggested (Rolls, 1989a) that co-activity between forward inputs and backprojecting inputs to strongly activated cortical pyramidal cells would lead to both sets of synapses being modified (see Fig. 3). This may be part of a mechanism for allowing higher, often multimodal, stages of processing to influence the representations built in earlier cortical stages (see Rolls, 1989a). In the case of the amygdala backprojections, this could mean that

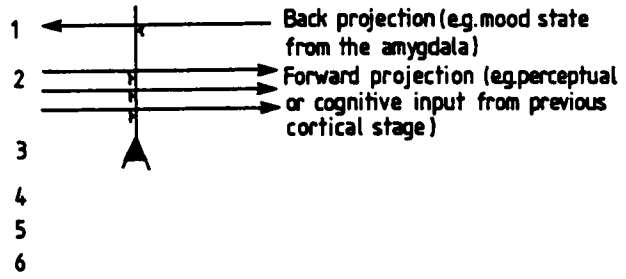

FIG. 3. Pyramidal cells in, for example, layers 2 and 3 of the temporal lobe association cortex receive forward inputs from preceding cortical stages of processing, and also backprojections from the amygdala. It is suggested that the backprojections from the amygdala make modifiable synapses on the apical dendrites of cortical pyramidal cells during learning when amygdala neurons are active in relation to a mood state; and that the backprojections from the amygdala via these modified synapses allow mood state to influence later cognitive processing, for example by facilitating some perceptual representations.

the current emotion is stored by modification of the active backprojecting synapses onto cortical pyramidal cells activated via their forward inputs by, for example, a particular face or expression. Then later recognition of that stimulus by the forward activation of cortical pyramidal cells will be better when the mood-carrying backprojecting neurons are active with the pattern specifying the appropriate mood state, for then the amygdala backprojections will tend to make those cortical pyramidal cells with the matching modified synapses from backprojecting axons more easily activatable by the forward input.

Thus, emotional states may affect whether or how strongly memories are stored using the basal forebrain memory strobe (see section on the hypothalamus); be stored as part of many memories; and may influence both the recall of such memories, and the operation of cognitive processing, in the way described in the preceding paragraph.

Manuscript received 13 December 1989
Revised manuscript received 4 May 1990

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