EMOTIONS: THEORETICAL MODELS AND CLINICAL INDICATIONS

Sophie Baudic *
Gérard H. E. Duchamp †

Abstract

The confrontation between theory and clinical practice contributes to improve our knowledge of emotions. In the present paper, theoretical models both cognitive and biological are briefly reviewed in the first part. Interactions between research and clinical practice are examined across two separate approaches in the second part. The first, neuropsychological approach considers Alzheimer's disease to further explore relationships between emotions and memory disturbance. The second considers panic disorder to explore cognitive and behaviour theories of fear. Finally, we address therapeutic applications for each approach which come directly from the theory.[0]

1 INTRODUCTION

The aim of this paper is to show that researchers and clinicians can interact together rather than exist side by side without any real contact. This interaction is essential for the evolution of the discipline and for patients. Theoretical models provide insights onto the structural brain and functional organization of human emotions and therefore their evolution is crucial for the management of patients. The therapeutic actions, the strategies of rehabilitation and the development of new tools are based on theory. This paper depicts in the first part cognitive and biological models and it considers in the second part two approaches which illustrate the implication of theory in the management of patients. The first is that of neuropsychology which analyses the dysfunctions caused by Alzheimer's disease to emotions and the second is that of cognitive and behaviour theories applied by therapists in the management of panic disorder which is a good model for studying fear in humans.

2 THEORETICAL MODELS

The study of emotions is hampered by several conceptual problems. The major one is the relationship between emotions and cognition which remains very controversial in

*Inserm U 792, Physiopathologie et Pharmacologie Clinique de la Douleur, Hôpital Ambroise Paré, 9 av Charles de Gaulle, 92104 Boulogne Cedex *E-mail: baudic@im3.inserm.fr*

†Institut Galilée, LIPN (CNRS UMR 7030), 99 Av. J.-B. Clément, F-93430 Villetaneuse, France.

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the debate between biological (Zajonc 1980) and cognitive (Lazarus 1982) theorists of emotions. The former maintains that emotions and cognition are two independent systems whereas the latter argues that cognition plays an integral role in emotions (for an overview see Schorr 2001).
Before presenting the different theories of emotions, it is essential to define them, because there is a tendency to group together a number of different affective phenomena under the term of emotions.

2.1 How to define emotions precisely?

The exact nature of emotions remains controversial. Disagreements mainly stem from the fact that the sets of phenomena taken into account are very different. Some focus their attention on the simplest aspects of emotions as they appear in animals or in the early stages of the human development. Others are attracted by the complexity of emotional phenomena.
Emotions have two dimensions: expressive and cognitive. The expressive dimension concerns the production of facial emotions or expression of internal states of the brain whereas the cognitive dimension is related to the comprehension of facial emotions expressions and intentions to act. Emotions are feelings that accompany emergent "states of being" like happiness or despair.
According to Ekman (1984), emotions are reactions that last several seconds. They must be distinguished from very brief responses such as the reflex reactions or basic survival-related appetitive behaviours and from long-lasting affective schemata, such as affects or personality traits.
According to Scherer (1984), emotions are different from a simple state of the organism (behaviourists). They are processes, i.e. a dynamic sequence of different variables whose components are the following: cognitive evaluation of stimuli or situation, physiological activation, motor and facial expression, action draft or planning of behaviour and feelings (subjective states). In accordance with this point of view, the function of emotions is adaptive because it allows a large flexibility of behaviour thanks to an elaborate treatment of complex information. Examples of emotions are happiness, sadness, anger, fear, surprise, disgust and also despair, pride...

The next part of the present topic provides an overview of major theories of emotions. It resumes and combines several authors' works: Gainotti (2001), Scherer and Peper (2001).

2.2 Cognitive models of emotions

There are two major models; the componential and the hierarchical. See Scherer and Peper (2001) for an excellent review on emotion theories.

Componential models

Emotions have qualitatively different facets. According to Scherer and Peper (2001), most of the psychological theories postulate that subjective experience, peripheral physiological responses and motor expression are major components of the emotion construct. These three components have often been called the "emotional response triad". A few theorists include two other components: cognitive and motivational in the emotion process.

In Scherer and Peper's view (2001), componential models differ strongly with respect to the relative role assigned or the amount of attention paid to these different components. Systematic researches in this field are associated with Lazarus (1982) and Scherer (1984).

Hierarchical models

In Gainotti's view (2001), we can distinguish within this classification "structural from developmental" models. The structural models maintain that emotions are hierarchically organized with numerous discrete emotions at a basic level and very few emotional dimensions at a higher level. The basic emotions such as happiness, sadness or fear are viewed as the building blocks of the emotion system while the dimensions of valence, (pleasantness/unpleasantness) and arousal (rest or activation) take place on a higher level in the hierarchy. Emotions at the basic level have an important adaptive function and are devoted to the detection of stimuli that are crucial for the organism's well-being. They appear at the earliest stages of child development. Existence of differences between basic and complex emotions is at the origin of the construction of developmental models. The partisans of these models admit that complex emotions derive from the basic ones. According to Gainotti (2001) functioning of these developmental models is based on the activity of three functional levels: the sensorimotor, the schematic and the conceptual. The sensorimotor level is at the first grade of action or interpersonal communication, the basic interactive schemata of human species. It involves a set of expressive-motor programs that are innate and universal. The schematic level which is the second stage of emotional processing includes "emotional schemata". The latter are different for each individual as they spring from the association of the basic emotions of the sensorimotor level and the situations of individual experience. The schematic level corresponds to spontaneous and true emotions. The conceptual level is based on mechanisms of conscious declarative memory. It stores the abstract notions such as "what are emotions?" or "which situations provoke them?" or "how to deal appropriately with them according to the social rules?" (see Gainotti 2001 for a comprehensive description of hierarchical models).

2.3 Biological models of emotions

The biological models like their cognitive counterparts have either componential or hierarchical organization.

Componential models

Emotions are made up of several components which are subserved by different anatomical structures. With respect to motor expression, the hypothalamus appears to be involved in the generation of autonomic reactions at the more elementary level. Componential models in the neuropsychology of emotions (see clinical implications) prompted two main lines of research. The first considers relationships between disorders that affect different components of the emotional processing system. The second tries to clarify the interactions existing between specific components of emotions and well-defined structures of the brain (see the first case of part two).

Hierarchical models

The biological theories are in line with a phylogenetic perspective. As we saw, hierarchical models organization is based on the complexity of emotional computation performed

by different brain structures with control of the highest functional levels over the lowest ones. Brain structures subserving emotions may be based on complexity of operations performed at different levels. The highest brain structures inhibit, modulate and extend rather than replace the lowest and earliest functional systems (Gainotti 2001).
The most influential of these models was proposed by Papez (1937) who attempted to specify the role played by different brain structures in emotional processing. Emotions were subserved by an anatomical circuit (namely that of Papez) beginning and ending in the hippocampal formation. It included basic structures such as hippocampus, hypothalamus with its mamillary bodies, anterior nuclei of the thalamus, cingulated cortex and their interconnections. This description, which is not completely erroneous, is reconsidered today. New technologies showed that other structures were involved in the Papez's circuit such as the amygdala and the prefrontal cortex. LeDoux and colleagues (1984) showed that the amaygdala (and not the hypothalamus as supposed by Papez) is the structure where in-coming information acquires emotional signification.
Another example of a complex model was proposed by Gray and McNaughton (1996). They identified specific brain systems: the behavioral inhibition system (BIS), the behavioral approach system (BAS), and the fight-flight system as substrates of these emotional dimensions. According to these authors the BAS is supposed to regulate approach behavior, sensitivity to reward stimuli and active avoidance behaviour, whereas the BIS is supposed to inhibit instrumental and unconditioned behavior and to control orienting reactions. The authors further hypothesize that structures of different phylogenetic level might mobilize in situations of different complexity the same defensive fight-flight attitude, i.e. when the source of danger is very close and there is no time for analysis or when the situation involves more distant threats and there is more time for analysis. Moreover, the amygdala might mobilize defensive behaviour in light of potential rather than actual events (see for reviews Scherer and Peper 2001).
Each of the models presented above attempt to capture and explain emotion either as a basic or complex process. These models progress consistently over time and become more efficient. For a long time, emotions were considered as useless manifestations, irrational and a source of interference. So, the first cognitivists tried to eliminate emotional dimensions from their models. The former theorists also considered that emotions were undifferentiated. Indeed, it seems that each emotion corresponds to a distinct functional cerebral unit. Two basic emotions, fear and pleasure, were extensively examined over the past years and the studies showed they involve different circuits. The amygdala is involved in the circuit of fear while the accumbens is necessary for pleasure. However, functional neuroimagery studies (Zalla et al. 2000) show that the human amygdala can differentially respond to changes in magnitude of positive or negative reinforcement conveyed by lexical stimuli. Finally, the cognitive models did not take into account the biological constraints provided by the anatomical organization of the brain. Moreover, the adaptive systems subserved by the brain have undergone reorganizations during its phylogenetic history (Tucker et al. 2000). In the same way, the biological models of emotions reveal some limits: 1) most of the neuroanatomic models are based upon data obtained in animals, 2) divergent neural connectivity subsists across species, 3) furthermore, some controversies exist between the studies in animals and a generalization to humans is sometimes problematic (Scherer and Peper 2001).

3 CLINICAL IMPLICATIONS

Neuropsychology of emotions can be considered to be a very new field of inquiry. The first series of studies conducted in this area were almost exclusively devoted to the problem of hemispheric asymmetries in representation and control of emotions (Borod 1993). Today, the focus of attention is directed to a much wider array of problems.

3.1 In neuropsychology research (organic diseases)

Alzheimer's disease (AD) is a good model for studying the alteration of emotional disturbance because it involves the amygdala which plays an important role in emotions as demonstrated by functional neuroimaging studies (Cahill et al. 1996; Canli et al. 2000). In the present paper, we shall focus our attention on the relationship between emotions and memory disturbance in AD.

In normal controls, emotional items are associated with additional semantic or autobiographical indices when they learn new information. Emotions may serve as a retrieval cue. A person may initially remember how they felt about an event, and that cue may then allow them to generate additional features about the event.

Alzheimer's disease (AD) results in significant atrophy of the medial temporal lobe that leads to a dramatic memory deficit. At the early stage, impairment concerns mainly memory which is characterized by an inability to learn new information or to recall previously learned information. Memory decline is gradual and the disease produces other cognitive deficits as it progresses. Emotional influence on recall was studied by means of cognitive neuropsychology of memory (Tulving et al. 1972). This model postulates that memory can be considered in terms of dissociable systems, distinct processes, and neuroanatomical structures. Within long-term memory systems, episodic memory (i.e. knowledge of episodes and facts that can be consciously recalled and related) is typically severely impaired in early-stage of the disease. Semantic memory that underlies knowledge and language is less likely to be significantly affected, although impairments may be observed in some individuals. Procedural memory (i.e. ability to gradually acquire and retain motor, perceptual and cognitive skills) is preserved, as are some aspects of priming. Memory can also be considered in terms of the processes of encoding, storage and retrieval (Tulving and Thomson 1973).

Some studies (Kazui et al. 2000; Moayeri et al. 2000) showed that recall of AD patients is typically better for emotional than for neutral stimuli. Memory is also better for neutral stimuli embedded in an emotional context. Other studies, in contrast, concluded that AD disrupts memory enhancement for verbal emotional information (Hamann et al. 2000; Kensinger et al. 2004). AD patients also demonstrated impairments in emotionally mediated implicit memory (Padovan et al. 2002). Differences across studies are related to the heterogeneity of patient populations (disease severity), difference in the stimuli or the extent of amygdala atrophy (Mori et al. 1999).

Emotional arousal improves episodic memory in patients with AD and gives a clue to the management of people with dementia (Kazui et al. 2000). Rehabilitation of emotions is based on aspects of emotional communication such as prosody. A series of experiments (Thaut et al. 2005) investigated the effect of music as a mnemonic device on learning and memory. More researches are needed to develop a useful strategy for memory improvement.

3.2 In cognitive and behaviour therapy (functional disorders)

Panic disorder is relevant for studying fear, a basic emotion. It can be depicted as a profound blast of anxious affect. The physical symptoms are multiple: shortness of breath, rapid heart rate, dizziness, tingling, sweating. The cognitive symptoms involve automatic thoughts and mental images which tend to be catastrophic, i.e. there is a tendency to exaggerate the dangerousness of a situation and simultaneously to underestimate the control over the danger.

Therapeutic actions are based on classical conditioning theories (Pavlov 1928). Fear conditioning occurs when initially innocuous conditioned stimulus (CS) is associated with an aversive unconditioned stimulus (US) that activates unconditioned fear responses (URs). The CS comes to elicit various conditioned responses (CRs) that share similar characteristics to innate fear responses (Kim and Jung 2006). The best known example of fear conditioning reported by the authors is the little Albert case (Watson and Rayner 1920). As the authors recount: "Albert was an infant who initially exhibited curiosity over a white rat by touching and playing with it. As Albert's hand touched the rat, the experimenters triggered a big noise behind his head (US) causing him to startle and cry (UR). Afterwards, when the rat (CS) was placed near Albert's hand, he withdrew his hand and began to cry (CR). This exhibition of fear towards the rat was generalized to other white furry animals and objects."

Treatment of patients suffering from panic disorder involves exposure to fear cues (behaviour therapy) and cognitive restructuring (cognitive therapy). One powerful means of reducing anxiety problems is believed that of countering avoidance. Avoidance reduces anxiety in short term, but makes for more anxiety in the long term as avoidance increases over time. Exposure involves placing someone in the avoided situation until the anxiety decreases completely. The disappearance of anxiety is called Extinction. Cognitive restructuring is used to identify and counter fear of bodily sensations. Patients are encouraged to consider the evidence and think of alternative possible outcomes following the experience of bodily cues.

LeDoux's model (1986) provides a theoretical framework for therapeutic actions of cognitive and behavioural therapy as it establishes a relationship between emotions and cognitive factors. The model postulates the contribution of the thalamus and the amygdala in fear conditioning and anxious reactions. These structures form a circuit that involves immediate survival responses (i.e. flight or fight reactions). The connection between the thalamus and the amygdala is the most direct and therefore the fastest. In parallel, another pathway exists which includes the prefrontal cortex and the hippocampus in addition to the thalamus and the amygdala. The prefrontal cortex is involved in more complex conditioning that requiring planning actions. An individual takes more time for cognition to shift from reaction to action and he is seen as an emotional actor who copes with a cognitive plan of voluntary action rather than just a reactor to an involuntarily elicited emotional reaction.

In the light of this model, it is easier to understand the therapeutic actions of exposure to fear cues and cognitive restructuring. Exposure to fear cues seems to be mediated by the more direct circuit while cognitive restructuring, which explores several option-responses seems to be supported by the second longer pathway, which involves the prefrontal cortex. Extinction of anxiety is explained by the action of prefrontal cortex.

Bodily feedback of panic disorder (i.e. shortness of breath, rapid heart rate, sweating) is also taken into account in this model. Somatic responses are stored in amygdala together with their associated perceptual context. When patients experience bodily feedback of fear, they misinterpret them and tend to develop avoidance behaviour.

4 CONCLUSION

In conclusion, theories produce important results in the field of cognitive neuropsychology and provide a better understanding of brain functioning. This comprehension leads to profound changes in clinical practice in the evaluation of patients and produces new orientations in the rehabilitation. Neuropsychologists progressively changed their conception of the brain that is more theoretically and methodologically constructed. Single case studies have been crucial in the historical development of the discipline and they continue to lend consistent support to the development of existing models or in the elaboration of new ones. Models of cognitive and behaviour therapy give a theoretical framework for therapeutic actions and lead to new perspectives of treatment of patients. The exchange between theory and clinical practice allows progressive adjustment between the knowledge of the brain functioning and the management of patients.

References

[1] Borod, J. 1993. *Cerebral mechanisms underlying facial, prosodic, and lexical emotional expression: a review of neuropsychological studies and methodological issues.* Neuropsychology, 12, 2493-2503.

[2] Cahill, L.; R.J. Haier; J. Fallon; M.T. Alkire; C. Tang; D. Keator; J. Wu; and J.L. McGaugh. 1996. *Amygdala activity at encoding correlated with long-term, free recall of emotional information.* Processings of the National Academy of Sciences USA, 93, 8016-21.

[3] Canli, T.; Z. Zhao; J. Brewer; J.D. Gabrieli; and L. Cahill. 2000. *Event-related activation in the human amygdala associates with later memory for individual emotional experience.* Journal of Neuroscience, 20, 1-5.

[4] Ekman, P. 1984. *Expression and the nature of emotion.* In Approaches to Emotion, K.R. Scherer and P. Ekman (Eds.). Hillsdale, NJ: Erlbaum, 319-344.

[5] Gainotti, G. 2001. *Emotions as a biologically adaptive system: an introduction.* In Emotional behaviour and its disorders, F. Boller and J. Grafman (Eds.). Handbook of Neuropsychology, Elsevier, Amsterdam, 1-15.

[6] Gray J.A. and N. McNaughton. 1996. *The neuropsychology of anxiety: reprise.* In Nebraska Symposium on Motivation: Perspectives on Anxiety, Panic and Fear, D.A. Hope (Ed.). Lincoln, NE: University of Nebraska Press, 61-134.

[7] Hamann, S.B.; E.S. Monarch; and F.C. Goldstein. 2000. *Memory enhancement for emotional stimuli is impaired in early Alzheimer's disease.* Neuropsychology, 14, 82-92.

[8] Kazui, H; E. Mori; M. Hashimoto; N. Hirono; T. Imamura; S. Tanimukai; T. Hanihara; and L. Cahill. 2000. *Impact of emotion on memory. Controlled study of the influence of emotionally charged material on declarative memory in Alzheimer's disease.* British Journal of Psychiatry, 177, 343-7.

[9] Kensinger, E.A.; B. Brierley; N. Medford; J.H. Growdon; and S. Corkin. 2004. *Effects of Alzheimer's disease on memory for verbal emotional information.* Neuropsychologia, 42, 791-800.

[10] Kim, J.J.; and M.W. Jung. 2006. *Neural circuits and mechanisms involved in Pavlovian fear conditioning: a critical review.* Neuroscience and Biobehavioral Reviews, 30, 188-202.

[11] Lazarus, R.S. 1982. *Thoughts on relations between emotion and cognition.* American Psychologist, 37, 1014-1019.

[12] LeDoux, J.E.; A. Sakagachi; and D.J. Reis. 1984. *Subcortical efferent projections of the medial geniculate nucleus mediate emotional response conditioned by acoustic stimuli.* Journal of Neuroscience 4, 683-689.

[13] LeDoux, J.E. 1986. *Cognitive - emotional interactions in the brain.* Cognition and Emotion, 3, 267-289.

[14] Moayeri, S.E.; L. Cahill. Y. Jin; and S.G. Potkin. 2000. *Relative sparing of emotionally influenced memory in Alzheimer's disease.* Neuroreport, 11, 653-5.

[15] Mori, E.; M. Ikeda; N. Hirono; H. Kitagaki; T. Imamura; and T. Shimomura. 1999. *Amygdalar volume and emotional memory in Alzheimer's disease.* 156, 216-22.

[16] Padovan, C.; R. Versace; C. Thomas-Anterion; and B. Laurent. 2002. *Evidence for a selective deficit in automatic activation of positive information in patients with Alzheimer's disease in an affective priming paradigm.* Neuropsychologia, 40, 335-9.

[17] Papez, J.W. 1937. *A proposed mechanism of emotion.* Archives of Neurology and Psychiatry, 79, 217-224.

[18] Pavolv, I.P. 1928. *Lectures on conditoned reflexes.* New York International.

[19] Scherer, K.R. 1984. *On the nature and function of emotion. A component process.* In Approaches to Emotion, K.R. Scherer and P. Ekman (Eds.). Hillsdale, NJ: Erlbaum, 293-318.

[20] Scherer, K.R. and M; Peper. 2001. *Psychological theories of emotion and neuropsychology research.* In Emotional behaviour and its disorders, F. Boller and J. Grafman (Eds.). Handbook of Neuropsychology, Elsevier, Amsterdam, 17-48.

[21] Schorr, A. 2001. *Appraisal - the evolution of an idea.* In Appraisal Processes in Emotion: Theory, Methods, Reseach. K.R. Scherer, A. Schorr and T. Johnstone (Eds.). Oxford: Oxford Univeristy Press, 20-34.

[22] Thaut, M.H.; D.A. Peterson; and G.C. McIntosh. 2005. *Temporal entrainment of cognitive functions: musical mnemonics induce brain plasticity and oscillatory synchrony in neural networks underlying memory.* Annals of the New York Academy of Sciences, 1060, 243-54.

[23] Tucker, D.M.; D. Derryberry; and P. Luu. 2000. *Anatomy and physiology of human emotion: vertical integration of brainstem, limbic and cortical systems.* In The neuropsychology of emotion, J.C. Borod (Ed.). New York, Oxford University Press, 56-79.

[24] Tulving, E. 1972. *Episodic and semantic memory.* In: Organisation of memory, E. Tulving and W. Donaldson (Eds.). Academic Press, New York, 381-403.

[25] Tulving, E.; and D.M. Thomson. 1973. *Encoding specificity and retrieval processes in episodic memory.* Psychological Review, 80, 352-373.

[26] Watson, J.B. and R.R. Rayner. 1920. *Conditioned emotional reaction.* Journal of Experimental Psychology, 3, 1-14.

[27] Zajonc, R.B. 1980. *Feeling and thinking: preferences need no inferences.* American Psychologist, 2, 151-176.

[28] Zalla, T.; E. Koechlin; P. Pietrini; G. Basso; P. Aquino; A. Sirigu; and J. Grafman. 2000. *Differential amygdala responses to winning and losing: a functional magnetic resonance imaging study in humans.* European Journal of Neuroscience, 12, 1764-70.