



## Chapter 11

# Emotion and delusion: Seeking common ground between neuroscience and the psychotherapies

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### Abstract

This chapter explores areas of common ground between psychoanalysis and neuroscience—of which many areas exist. Emotion is an area of obvious overlap between the two fields and is especially important for any discussion of delusional beliefs. The chapter highlights such links and particularly suggests ways in which developments in neuroscience can potentially enrich psychoanalysis. We focus especially on the issue of emotional regulation, and the many ways in which false beliefs might be emotionally driven, in doing so focusing on the topics of self-deception, emotion and stereotyping, emotion in decision making, dreams and psychosis, and neurological disorders such as confabulation and anosognosia. The chapter concludes by highlighting the need to develop a strong two-way relationship between the two fields.

*Keywords:* emotion regulation; decision making; anosognosia; confabulation; psychoanalysis; neuroscience.

### Introduction

A central driving force in the recent reunification of psychoanalysis and neuroscience has been a shared interest in emotion, and a growing interest in the potential role of emotion and delusional beliefs (Solms and Turnbull, 2002; Turnbull and Solms, 2007). Cognitive neuroscience had, for many years, not been especially focused on the issue of emotion. However, the recent development of affective neuroscience (e.g. Panksepp, 1998) has made it clear that an understanding of basic emotions and their role in shaping high-level cognition opens a great deal of common ground with psychoanalysis.

The question of the number and nature of basic emotion systems has generated a large and complicated literature in recent years (Damasio, 1999; Panksepp, 1998; Rolls, 1999). In part, work has attempted to establish what the number of basic emotions might be (see Panksepp, 1998; Solms and Turnbull, 2002, Chapter 4, for a brief review). The literature suggests that there are a number of different ways of attempting to measure the phenomena of emotion (Johnson-Laird and Oatley, 2000; Panksepp, 2000; Stearns, 2000). The field does, however, seem to generally agree on a shortlist of at least four basic emotions. These are firstly ‘happiness’, or positive



## Box 11.1 Term definitions

*Anosognosia:* A condition in which neurological patients hold delusional beliefs, typically about their bodies, and deny that they are disabled. Prototypically, they tend to be paralysed on the left side of their body, after large right-sided strokes.

*Confabulation:* A term used to describe a condition seen in neurological patients who hold false beliefs—who believe, for example, that they are living in a hotel not a hospital, or that their spouse is an imposter.

*Defence:* Psychological processes that organize and maintain mental life in a way that protects the individual from aversive emotional experiences.

1 emotion of one description or another (Panksepp and Burgdorf, 2000; Schultz, 1998). In addition,  
2 at least three negative emotions most notably those of ‘sadness’, ‘anger’, and ‘fear’ have been  
3 identified in the literature (Calder et al., 2001; Panksepp, 1998). Each basic emotion appears to  
4 have relative chemical and anatomical independence (Panksepp, 1998). These are common to all  
5 classes of mammal species, and distributed across a range of subcortical structures, overlapping  
6 in the upper brain stem (periaqueductal grey)—(see Watt, 2000; Solms and Turnbull, 2002,  
7 Chapter 4, for a review).

8 This anatomical distribution is also demonstrable in humans, as in the work of Damasio in the  
9 functional imaging of human beings experiencing powerful emotional states in the scanner (Damasio  
10 et al., 2000). Thus, in the sadness condition, we see symmetrical activation of subcortical structures,  
11 especially in the upper brainstem, hypothalamus, and anterior cingulate. At the same time there is  
12 almost no change to cortical brain regions, or indeed in some cases there is a degree of *deactivation*.  
13 Thus, participants in the sadness condition (some of whom were reported to be able to cry in the  
14 scanner) are experiencing powerful emotional states, activating a range of phylogenetically ancient  
15 brain structures, but not activating the cortex (McManus, 2002; Springer and Deutch, 1998).

16 This represents a general survey of the current state of the field, when focusing on the *experience*  
17 of basis emotions. However, had we been writing this chapter 20 or 30 years ago on the topic of  
18 the neuropsychology of emotion, we would have stressed an entirely *different* class of evidence  
19 (see Borod, 2000 for a review). Several decades ago the theme of hemispherical asymmetry of  
20 function in relation to emotion was a fairly substantial research topic (Borod, 1992; Borod and  
21 Caron, 1980; Borod et al., 1983; Davidson, 1993; Sackheim et al., 1978; Schiff and MacDonald,  
22 1990). A number of studies demonstrated that the *right* cerebral hemisphere has selective advan-  
23 tage in, for example, perceiving the emotion in speech (i.e. speech prosody) and in recognizing  
24 the facial expressions associated with emotions even controlling the musculature experience of  
25 emotion (see Borod, 2000 for a review).


26 So the question, then, is how to square this older literature in neuropsychology (which suggests  
27 a right hemisphere cortical advantage for emotion) with the neuroscientific evidence which  
28 suggests a subcortical (and laterally symmetrical) role for a number of basic emotional systems  
29 (see Gainotti, 1997 for a review). This argument seems to hang on the question of the ‘cognitive’  
30 versus the ‘visceral’ aspects of the emotional state (see Solms and Turnbull, 2002, Chapter 4, for a  
31 brief review). That is, the cortical systems (which tend to be rightward lateralized) appear to  
32 underpin the more visuospatial and cognitive aspects of emotion, for example recognizing that  
33 someone has a sad face (Bruce and Young, 1986; Ellis and Young, 1990; Parkin, 1996). This  
34 requires a complex computation about the way in which the musculature expresses itself across  
35 the face. This includes calculations of the relative position of the mouth, the shape and tension of



1 the musculature underpinning the cheek, the position and tension of extraorbital muscles, etc.  
 2 (see Keltner and Ekman, 2000, for a review). These complex visuospatial calculations unquestion-  
 3 ably require a great deal of cognitive skill. However, recognizing that a face is ‘sad’ or ‘happy’, for  
 4 example, is very different from *experiencing* the profound feelings of sadness that overcome us  
 5 when we are depressed, or the terror that overcomes us when we are anxious. It appears that  
 6 the *cortical* systems are associated with the more cognitive aspects of the emotional state, whereas  
 7 the visceral *experience* of those emotions are mediated by phylogenetically ancient subcortical  
 8 systems. The neurobiological basis of these systems is the basis of the review above (see also  
 9 Panksepp, 1998; Panksepp and Biven (Chapter 9), this volume).

## 10 Emotion regulation

11 In this chapter we will focus on some of these cortical aspects of emotion, and especially the way  
 12 in which emotion might be managed or controlled. Thus, the interaction between these subcortical  
 13 emotion systems and, for example, prefrontal cortex is potentially of great interest. Research by  
 14 our group (Fotopoulou et al., 2004; Nardone et al., 2007; Tondowski et al., 2007; Turnbull et al.,  
 15 2002, 2004a,b, 2005a) and others (e.g. Feinberg, 2001; Ramachandran and Blaklee, 1998) has  
 16 suggested that there are at least two classes of cortical systems that are important for managing  
 17 emotion, both of which appear to produce false beliefs when damaged.

18 However, before reviewing this material, it seems appropriate to discuss the approach that most  
 19 cognitive neuroscientists have adopted towards emotion regulation. This speaks to the question of  
 20 having a ‘common language’, which forms the central theme of the present book: how one might  
 21 reach out from the psychodynamic perspective to cognitive neuroscience colleagues, to make the  
 22 language of the two fields more compatible. One area of common ground is the management of  
 23 emotion. Cognitive neuroscientists not only clearly understand the importance of cognition, but  
 24 they also recognize the *existence* of emotion—including, to some extent, its anatomical and phar-  
 25 macological basis (Lane and Nadel, 2000). By and large, they do not deny the existence of emotion,  
 26 in the way that the  behaviourists might once have done (see Skinner, 1953).

27 When one discusses emotion with the cognitive community, they also understand at least many  
 28 aspects of emotion *regulation* and its importance in mental life (see Koole, 2009, for review). One  
 29 would particularly think, for example, about work with children: the way in which powerful  
 30 childhood events are experienced, and that it is very difficult for children to be able to manage and  
 31 regulate their emotions appropriately (Buss and Goldsmith, 1998; Guttentag and Ferrell, 2008;  
 32 Mangelsdorf et al., 2008). Neurologically and psychiatrically intact adults have, of course, a much  
 33 greater capacity of to experience emotion in a regulated manner—and the developmental psy-  
 34 chology literature has a clear awareness of this issue. However, many cognitive neuroscientists  
 35 appear to disagree with the psychodynamic community on the *relative* importance of emotion  
 36 and cognition as systems. Some imagine, for example, that emotion and cognition are relatively  
 37 independent in their operation (Leventhal and Scherer, 1987; Zajonc, 1980, 1984). Alternatively,  
 38 some hold the opinion that cognition is the ‘dominant’ system of the two, for example, Frijda  
 39 et al. (2000) discuss the role of cognition and emotion through ‘cognitive emotion theory’  
 40 (e.g. Lazarus, 1991). For example:

41 Emotions *result from* how the individual believes the world to be, how events are believed to have come  
 42 about, and what implications events are believed to have. Beliefs thus are regarded as one of the major  
 43 *determinants* of emotion, and therefore an important part of the study of emotion can properly be seen  
 44 as falling *under the umbrella* of cognitive psychology. Oddly enough, however, the reverse direction of  
 45 influence in the relation between emotion and cognition has received scant attention.

Frijda et al. (2000, p. 1, emphasis added)

1 The approach that emotion is ‘determined’ by belief (and not vice versa) would be regarded as  
 2 extraordinary by anyone viewing the question of emotion and cognition from a psychodynamic  
 3 perspective. One reason for this unusual perspective may be that the roots of cognitive psychology  
 4 are in work with adults, with people who are emotionally well adjusted, with people who have  
 5 intact levels of executive function, and of course in situations that are not powerfully emotionally  
 6 charged. The average laboratory setting does not typically involve the sorts of powerful affective  
 7 states that human beings commonly find themselves in, and which of course have very important  
 8 influences on the mind. For mainstream cognitive psychology there has been little investigation  
 9 of thinking in settings of powerful feelings of love, hate, fear, or despair—though these are central  
 10 to our experience of being human. Indeed, for the psychotherapy which most directly developed  
 11 from this perspective, cognitive behavioural therapy (CBT), cognitive states determine emotions  
 12 (Beck, 1976; Butler et al., 2006). The psychodynamic perspective holds the opposite position, and  
 13 it is this issue of the ‘primacy of affects’ that one would most want to persuade ‘cognitive’ theorists  
 14 of (Turnbull and Solms, 2007).


15 The ‘primacy of affects’ is made most clear under two classes of circumstance. The first, as sug-  
 16 gested above, are situations where emotions are extremely powerful, and second in situations  
 17 where we have poor executive control—poor management of these affective states. This is the  
 18 claim which we aspire to have our neuroscientific colleagues move towards, and which we are  
 19 attempting in the literature to persuade them of (Fotopoulou et al., 2004; Nardone et al., 2007;  
 20 Tondowski et al., 2007; Turnbull and Solms, 2007; Turnbull et al., 2002, 2004a,b, 2005a). That is,  
 21 in circumstances where *powerful* affective states might overwhelm ‘normal’ cognition, or situa-  
 22 tions where executive functions are impaired—we appear to be able to ‘use’ our intellectual  
 23 capacities wisely in the service of correctly perceiving reality (Westen, 2007). Indeed, there are  
 24 times when we not only *allow* the perception of reality to be distorted, but there are also times in  
 25 which we use our intellectual resources to actively conspire *against* a clear understanding of real-  
 26 ity. Thus, those with more substantial cognitive capacity are (in some respects) *better* at holding  
 27 false beliefs than people with more modest cognitive abilities, because they have more ‘intellectual  
 28 horsepower’ to deploy in settings where there is something in which they really *want* to believe,  
 29 for powerful emotional reasons (see Shermer, 2007).

30 In affective neuroscience and neuropsychanalysis we have been able to focus with some  
 31 success on the core emotions (for reviews, see Panksepp, 1998, 1999; Solms and Nersessian, 1999;  
 32 Panksepp and Biven (Chapter 9), Watt (Chapter 6), and Pfaff and Fisher (Chapter 5), this  
 33 volume). We are on perfectly legitimate grounds in doing so, because basic emotions remain a  
 34 centrally important question in our understanding of the architecture of the mind. However, we  
 35 would also like to emphasize the importance of emotion *regulation* in any coherent model of  
 36 mental life (Koole, 2009). A primary reason for this emphasis is that the entire question of  
 37 ‘emotion distorting cognition’ speaks to the issue of *conflict* in the mind. One suspects that every-  
 38 one who has a psychodynamic perspective on mental science understands that the issue of con-  
 39 flict, and the difficulty in managing *competing* mental demands, is central to mental life (Freud,  
 40 1911). Successful management of the conflict between drives and reality is vital for maintaining  
 41 reasonable mental health, and its dysregulation forms the basis of neuroses, and for a variety of  
 42 false-belief states. Indeed the existence and nature of our civilization is (arguably) underpinned  
 43 by the fact that we are able to manage and control our feelings (Freud, 1930)—a point also made  
 44 by a number of later analytic theorists.

45 A further reason for stressing the importance of emotion regulation relates to the issue of treat-  
 46 ment. The last century of work in the treatment of mental health has been sharply polarized between  
 47 pharmacological treatments (of ‘neurochemical imbalances’), and treatments focusing on psy-  
 48 chologically driven approaches to dealing with distress and dysfunctional personal circumstances.



1 This has led to virtually parallel streams of treatment for disorders of mental health (which can,  
2 of course, take many forms). The psycho-pharmacological (organic psychiatry) approach has  
3 clearly gained substantial success through the pharmacological modification of the basic emotion  
4 systems discussed above (Panksepp, 1998). However, it is likely that the *management* of emotion  
5 systems will always be intractable to pharmacological interventions, which serve only to up- or  
6 down-regulate basic emotion systems.

7 In contrast, it seems likely that the primary mechanism by which the psychotherapies treat  
8 mental distress, in its various forms, is through the modification of *cortical* systems, and more  
9 generally the dynamic management of emotion. This is, of course an argument that may  
10 well apply to other forms of psychotherapy (CBT, dialectical behaviour therapy, acceptance and  
11 commitment therapy, mindfulness) as well as psychoanalysis. Indeed, it is likely to be true not  
12 only for the psychotherapies, but also for all other classes of psychosocial intervention, such as  
13 activities to enhance play opportunities in children (Panksepp, 2002, 2007), and improved train-  
14  ~~ing emotion management of children by their parents and/or teachers~~ (Bywater et al., 2009;  
15 Hutchings, 1996; Hutchings et al., 2004, 2007). In sum, it is important to understand the notion  
16 of emotion regulation and its neurobiological basis—but a sound understanding of the neuro-  
17 science of such issues remains a substantial topic for future research. The extent to which cortical  
18 emotion-regulatory systems are involved in psychotherapies remains wide open for future  
19 research. This research domain will also be able to address the ways in which various forms of  
20 psychotherapy differ in their regulatory mechanisms. To take one, rather polarized, example, the  
21 psychoanalytic approach versus that of CBT differ strikingly in terms of their focus on emotion,  
22 and also on the role of conscious awareness and voluntary action in treatment (e.g. Beck, 1976;  
23 Lemma, 2003; Roth and Fonagy, 2005).

24 This chapter addresses the way in which two different approaches have tackled the question of  
25 emotion regulation in mental science. Bearing in mind that there is only one mental apparatus to  
26 study, it is of some interest to observe whether cognitive psychologists have identified concepts that  
27 are relevant for psychoanalysis in this domain. In a brief survey, this chapter offers four domains  
28 in which there is potential overlap on the issue of how emotional life interacts with high-level  
29 cognition.

### 30 **Links between cognitive science and psychoanalysis**

31 First, there is a developing literature on emotion regulation that is not driven by the psychody-  
32 namic literature, but by cognitive psychologists who are migrating towards studying emotion.  
33 One example is the growing interest on the way in which emotions are hidden in social circum-  
34 stances so as to better serve us in the interpersonal world (see Butler and Gross, 2004, for a  
35 review). Examples might include avoiding breakdowns of intimacy and enhancing long-term  
36 marital satisfaction (Gottman and Levenson, 1992), and the interpersonal advantages of deception  
37 (Hrubes et al., 2004; Shiota et al., 2004). This includes a substantial literature on self-deception  
38 (e.g. Gur and Sackeim, 1979; Robinson et al., 2009; Tavis and Aronson, 2007; Trivers, 2000),  
39 which will be discussed later in the chapter. There is also a growing interest in the neurological  
40 basis of social pain (social loss) and rejection, including evidence that physical and social pain  
41 share the same neural circuitry (Eisenberger, 2006; Eisenberger and Lieberman, 2004; Eisenberger  
42 et al., 2003, 2006; Panksepp, 1998).

43 A related domain of cognitive research on the relationship between emotion and cognition is  
44 that of cognitive dissonance, in many ways related to the psychoanalytic concept of defence—  
45 psychological processes that organize and maintain mental life in a way that protects the  
46 individual from aversive emotional experiences (Aronson, 2007; Greenwald and Ronis, 1978;

1 Pyszczynski et al., 1993; Sherwood, 1981). Festinger's (1957) original argument relates to holding  
 2 two contradictory 'cognitions': the classic example being the Aesop's fable of the fox and the  
 3 grapes, where the fox sees the grapes seem tempting ('cognition 1' as Festinger would describe it), but as  
 4 soon as the fox realizes that he cannot access them (cognition 2), they are dismissed as being sour.  
 5 The fox thus deploys a classic rationalization. There has been a substantial literature on cognitive  
 6 dissonance (e.g. Cooper, 2007; Egan et al., 2007; Elliot and Devine, 1994; Festinger, 1957;  
 7 Greenwald and Ronis, 1978; Harmon-Jones and Mills, 1999; Kay et al., 2002; Pyszczynski et al.,  
 8 1993; Tavris and Aronson, 2007), and there is clear potential for overlap between a psychody-  
 9 namic and a cognitive perspective. However, there is an important difference in the interpretation  
 10 of the findings. In the cognitive dissonance literature there is far less focus on motivation, and the  
 11 *emotional* consequences of a thought—where (psychodynamically) the 'sour grapes' worldview  
 12 protects the fox from feelings of loss.

13 Emotions also influence mental life in various other ways, which have been investigated by  
 14 psychologists through a literature, social psychology, that is far distant from that of emotion regu-  
 15 lation and the defences. Nevertheless, in the social psychology literature on stereotyping, one can  
 16 see a similar trend towards understanding the way in which emotion can shape beliefs. Stereotypes  
 17 are, of course, *generalized* beliefs about the psychological characteristics of a specific group or  
 18 class of people, that might not be accurate of that person. For example the effects of being blonde,  
 19 black, gay, or tattooed (Burns et al., 2008; Sinclair and Kunda, 1999; Swami and Furnham, 2007;  
 20 Takeda et al., 2006; Weir and Fine-Davis, 1989).

21 We also know that forming stereotypes is a commonplace activity, which *all* humans engage in,  
 22 often occurring outside of conscious control (Aronson, 2007; Devine and Monteith, 1999; Macrae  
 23 and Bodenhausen, 2000; Operario and Fiske, 2001). This is especially true under situations of  
 24 conflict (Sinclair and Kunda, 1999). For example, Kunda et al. (2002) found that when white  
 25 participants were in disagreement with black individuals, these participants would perform faster  
 26 on a reaction time task when stereotypical words (e.g. rap, crime, drugs) were presented. There  
 27 are many theories on why humans use stereotypes, including their use as a time-reducing cogni-  
 28 tive process to help us to simplify and categorize the world we live in (Aronson, 2007; Macrae  
 29 and Bodenhausen, 2000), or to support goal-directed actions by building self-justification or self-  
 30 esteem (Fein and Spencer, 1997; van den Bos and Stapel, 2009).

31 Given the prevalence of emotional reactions that accompany stereotyping, it is not surprising  
 32 that there exists a body of research examining how *emotion* might be involved in the formation of  
 33 stereotypical behaviour (e.g. Davies, 2008; Esses and Zanna, 1995; van den Bos and Stapel, 2009).  
 34 For instance, emotional stress has been shown to activate stereotypes (e.g. Maner et al., 2005), and  
 35 stereotyping also plays a role in 'self-enhancement' (see van den Bos and Stapel, 2009 for a  
 36 review). However, the link between emotion and stereotyping has never been a *core* topic of  
 37 investigation for the social psychology literature. Nevertheless, there are several lines of evidence  
 38 to support the link. Notably, neuroscientists have begun to map the brain correlates associated  
 39 with perceiving and applying stereotypes, showing substantial right frontal activation (Mitchell  
 40 et al., 2008; Quadflieg et al., 2008). A related finding comes from the Harris and Fiske (2006)  
 41 study, which demonstrated medial frontal activation when viewing others for whom there was  
 42 empathy, but showing no activation for 'out-groups' such as the homeless (i.e. perhaps suggesting  
 43 that the viewers do not regard such out-group members as fully 'human'). These findings  
 44 are consistent with an emotion-orientated view of stereotyping, given the role of the medial and  
 45 right frontal cortex in emotion and emotion regulation (Kim and Hamann, 2007; Solms and  
 46 Turnbull, 2002).

47 A further topic of 'common ground' between the analytic and cognitive literature is the substantial  
 48 literature built up from the 1970s on the nature of decision-making biases (e.g. Kahneman, 2003;

1 Kahneman and Tversky, 1979; Tversky and Kahneman, 1974). Again, many of these so-called  
 2 ‘cognitive’ biases probably have their basis in powerful affective states. To take one example,  
 3 ‘post-purchase rationalization’ (Aronson, 2007; Cohen and Goldberg, 1970) focuses on the way  
 4 in which the purchaser seeks to argue (after the fact) that they have made a financially sound deci-  
 5 sion. This is typically cited as a ‘cognitive’ bias, but of course speaks powerfully to motivational  
 6 issues: where the emotional consequences of believing that you wasted your money, make it in  
 7 your best interests to deploy intellectual resources explaining why this may have actually been a  
 8 very good decision. Importantly for psychoanalysis, the cognitive literature on this topic suggests  
 9 strong evidence that many motivational states have their origins generated outside of conscious  
 10 awareness (Bos et al., 2008; Custers and Aarts, 2005, 2007, 2010; Dijksterhuis and Aarts, 2010).

11 A further example of topics outside psychoanalysis that may be of relevance is the literature on  
 12 self-deception. There is, for example, sound evidence that we are unexpectedly poor in making  
 13 judgements about ourselves—for example from non-verbal cues (Hofmann et al., 2009). One  
 14 strand of this work comes from evolutionary psychology, most notably the work of Robert  
 15 Trivers (Trivers, 2000, also see Mele, 1997), which emphasizes the possible evolutionary advan-  
 16 tages of self-deception. Trivers’ argument has been that if we can find a way in which we can  
 17 convince *ourselves* of a lie (so that we ourselves think that it is true), then this confers a powerful  
 18 evolutionary advantage, in that we are better able to deceive others. Again, we think there is an  
 19 interesting opportunity here for the world of self-deception in evolutionary psychology to map  
 20 onto things that are of interest to the psychoanalytic community.

21 These examples cited above show how the cognitive literature is relevant, but limited, in its  
 22 applicability and usefulness to psychoanalysis. Any limitations are primarily because of the focus  
 23 on *cognitive* accounts, and avoidance of psychoanalytically common terms such as ‘defence’, with  
 24 its implications of reality distortion for *emotional* gain. There are further limitations to the rele-  
 25 vance of the cognitive psychology literature for neuropsychanalysis, in that cognitive psychology  
 26 has been mapped imperfectly onto its neurobiological foundation. It is therefore worthwhile for  
 27 us to consider why a neuroscientific perspective might add value to work in the psychological  
 28 sciences.

## 29 The benefits of neuroscience

30 Neuroscience has a history of being helpful in psychological science for at least two reasons. First,  
 31 because neuroscience offers a second ‘viewpoint’ (c.f. Solms and Turnbull, 2002) on all psycho-  
 32 logical issues, from memory to language. Importantly, *all* of the mind is mediated by brain  
 33 processes—so that brain injury can disrupt psychological abilities that may not have been ‘fash-  
 34 ionable’ for psychological scientists to investigate, uncovering material that the field had not  
 35 previously encountered. For example, the literature on semantic memory remained relatively  
 36 obscure until work in the 1970/1980s on patients with loss of semantic knowledge (especially  
 37 ‘semantic dementia’) transformed the field (Hodges et al., 1992; Snowden et al., 1989; Warrington,  
 38 1975). Similarly, the fields of decision making and problem solving have been (and are being)  
 39 transformed by the study of patients with frontal lobe lesions, which make it clear that a substan-  
 40 tial fraction of the forebrain is dedicated to executive functions (Daum and Mayes, 2000; Rodrigues  
 41 Gouveia et al., 2007). However, the early history of cognitive science shows that there was a  
 42 disproportionate interest in ‘foundational’ skills such as language and episodic memory—with  
 43 limited emphasis on synthetic/executive ability (see Finger, 1994 for a review). Moreover the  
 44 study of neurological patients makes it clear that executive function is a multi-component and  
 45 fractionable skill (Baddeley, 1998, 2002; Baddeley and Della Sala, 1996; Baddeley et al., 1986;  
 46 Shallice, 2002), including processes which the cognitive literature had not previously focused on,

1 such as action initiation (as lost in patients who are profoundly adynamic), and the capacity to  
 2 shift-set (as in patients with perseveration) (Kimberg et al., 1997; Rolls, 2002).

3 More importantly neuroscience (and neuropsychology in particular) is useful because it pro-  
 4 vides scientific clarity by the investigation of ‘extreme’ cases. Thus, studying patients with selective  
 5 deficits, in which the disorder suffered by the patient is strikingly obvious in comparison with  
 6 preserved psychological abilities (Shallice, 1988), reduces the reliance on subtle reaction-time  
 7 effects to make a phenomenon clear. One obvious example is the case of H.M. (Scoville and  
 8 Milner, 1957), whose profound recent episodic memory impairment (with intact procedural and  
 9 immediate memory) greatly clarified the extent to which several independent memory systems  
 10 exist in the brain, and transformed memory research (see Schacter and Scarry, 2000 for a review).

11 The fact that cognitive processes (for example, memory and executive function) have been  
 12 greatly clarified by the study of brain-lesioned patients, stands in clear analogy to phenomena of  
 13 psychoanalytic interest, such as studying the experience of strong emotion, and the way that is  
 14 managed. Thus, for example it has been possible to study the selective loss of particular cases of  
 15 emotion (Calder et al., 2001; Damasio, 1999, pp. 62–67). Also it has been possible to demonstrate  
 16 preservation of emotion and emotion-based memory in patients with profound episodic memory  
 17 impairments (Claparede, 1951; Evans-Roberts and Turnbull, 2011; Tranel, and Damasio, 1993;  
 18 Turnbull and Evans, 2006; Turnbull et al., 2006). In addition, it is important also to identify  
 19 which brain areas can be damaged and yet still underpin important psychological processes. For  
 20 example, the demonstration of preserved patterns of emotional experience after right convexity  
 21 lesions (Tondowski et al., 2007; Turnbull et al., 2002, 2005a) demonstrate that cortical brain areas  
 22 sometimes cited as being important for emotion (Borod, 2000; Davidson, 2001; Davidson and  
 23 Irwin, 1999) are not centrally involved in emotional *experience* (which is, as discussed above,  
 24 likely to be mediated subcortically—Damasio et al., 2000; Panksepp, 1998; Watt, 2000). Recent  
 25 findings of this sort suggest that the capacity to ‘carve cognition at its seams’ (McCarthy and  
 26 Warrington, 1990, p. 20) also applies to the emotional world. Thus, the move which psychoanaly-  
 27 sis has been making towards neuropsychology offers it all sorts of opportunities for verifying and  
 28 identifying the building blocks of the parts of the mental apparatus that have long been of interest  
 29 to psychoanalysis.

### 30 Emotion in decision making


31 We finish this section by focusing on some of the examples in which researchers have studied the  
 32 neurobiology of disorders to understand the way in which they change or distort the mind.  
 33 A range of settings in which emotion influences decision making have been investigated (Bechara  
 34 et al., 1994, 2000; Bowman and Turnbull, 2004; Dunn et al., 2006; Turnbull et al., 2005b). These  
 35 findings clarify the important role of emotion in mental life, which has long been central to  
 36 psychodynamic thinking (Freud, 1911).

37 Of course, for the past several thousand years, philosophers have informed us that in order to  
 38 make good choices we need to be entirely rational, and exclude emotions from the decision mak-  
 39 ing process (Kant, 1781/2004; Plato, 360BC/1956). However, over the past two decades, research  
 40 has shown us that emotion is often fundamental for human decision making (Bechara et al.,  
 41 2000; Koole, 2009; Turnbull and Solms, 2007). This is especially true under two clear settings:  
 42 situations of high levels of complexity, and circumstances which are rather uncertain, ambigu-  
 43 ous, or unpredictable (Bechara et al., 2000; Kahneman, 2003; Turnbull and Evans, 2006; Turnbull  
 44 et al., 2007). Under these circumstances, if emotion is *not* involved in decision making, humans  
 45 tend to make poor choices, and often make catastrophically unfortunate errors. Indeed, it has  
 46 become increasingly clear that inaccuracies/biases in decision making result from the influence of

1 emotion-related brain areas (De Martino et al., 2006; de Gelder et al., 2005; Pessiglione et al.,  
 2 2007, 2008).

3 The classic examples of these phenomena comes from the neuropsychological literature of  
 4 patients who have lesions to the ventromesial frontal lobes, key to the way in which emotion  
 5 influences high-level cognition. The prototype example is that of Phineas Gage (Harlow, 1848),  
 6 who suffered an extraordinary injury that damaged, probably bilaterally (Damasio et al., 1994),  
 7 the ventromedial surfaces of his frontal lobes. Like all the modern cases of patients with similar  
 8 lesions, Gage experienced three noticeable classes of change after his brain injury. First, like many  
 9 such patients, he made very poor real-world decision choices. He struggled to hold down a job,  
 10 managed his finances poorly, and operated poorly in the interpersonal world (Harlow, 1868;  
 11 Macmillan, 2000, 2004). Second, like many of these patients, he also remained by and large intel-  
 12 lectually ‘intact’. Several such modern patients perform well on measures of conventional cogni-  
 13 tive function (Bechara et al., 1997, 2000; Clark et al., 2003, 2004; Eslinger and Damasio, 1985).  
 14 Certainly they often perform well in the structured settings of many intelligence tests, for example,  
 15 probably because they do not require the sorts of knowledge that emotions add to the decision-  
 16 making process—given that many task decisions are not inherently complex or uncertain. Finally,  
 17 of course, such patients show substantial personality change. In the classical case of Phineas Gage,  
 18 his physician described him as ‘no longer Gage’ (Harlow, 1868, p. 327)—he had become a different  
 19 person. Presumably this is because the interpersonal world is the most complicated and uncertain  
 20 of all of the settings in which we find ourselves in.

## 21 Dreams and psychosis

22 An additional literature, in relation to the question of affect and our perception of reality, relates  
 23 to the role of emotion in dreams. Here work with brain-lesioned patients has been seminal in  
 24 transforming the dream literature, making it abundantly clear that emotion has a role to play in  
 25 the delusional beliefs seen in these sleeping states (see especially Solms, 1997, 2000, 2002). Notably,  
 26 lesions to the medial parts of the frontal lobe appear to completely terminate the dream process,  
 27 primarily because such lesions disrupt a core emotion system, mediated by dopamine. Dopamine  
 28 pathways, which travel from the upper brainstem to a range of ventral and mesial frontal fore-  
 29 brain sites, are the substrate for a  motion system (Panksepp, 1985; Robbins and Everitt,  
 30 1992). Various referred to over the years as a ‘reward’ (Schultz, 2001), a ‘preparation’ (Hobell,  
 31 1997), or a ‘SEEKING’ system (Ikemoto and Panksepp, 1999), whose chemistry appears to  
 32 consistently activate ventromesial frontal structures during tasks involving reward and punish-  
 33 ment (Dias et al., 1996; O’Doherty et al., 2001; Schultz, 2001; Turnbull et al., 2007). It appears  
 34 especially apt to motivate us to *investigate* the environment, search for rewards (e.g. Robbins and  
 35 Everitt, 1992), and to construct causal relationships between events in the perceived world  
 36 (Schultz, 2001).

37 What then of a quite different set of findings relating to dopamine? It has been known since Arvid  
 38 Carlsson’s work in the 1960s, and especially since the 1970s (see Snyder, 1976), that pharmacological  
 39 management of the positive symptoms of schizophrenia (the delusions and hallucinations) targets  
 40 one or more of these dopamine systems in the brain. This ‘dopamine’ theory of schizophrenia  
 41 remains (through a range of modifications) by far the most robust account of the neurochemical  
 42 basis of the disorder—originally emphasizing the simple overactivation of the dopamine system,  
 43 but more recently uncovering roles for other members of the D2 dopamine family, and revealing  
 44 the extent to which cortical mechanisms regulate these forebrain dopamine systems (see Carlsson  
 45 and Carlsson, 1990; Egan and Weinberger, 1997; Moore et al., 1999 for a review).


46 Most intriguing is the observation that dreaming stops completely when fibres in the ventromesial  
 47 frontal lobes are severed (Solms, 1997, 2000); a symptom that coincides with a general reduction

1 in motivated behaviour. The lesion producing this syndrome is the same as that which was delib-  
 2 erately produced in prefrontal leucotomy (see Solms, 1997), which was of course replaced in the  
 3 1960s by drugs that dampened activity in the same dopaminergic pathways discussed above in  
 4 relation to schizophrenia.

5 There is therefore robust support for the claim that the system that mediates powerful positive  
 6 emotions (Berridge, 2003; Panksepp, 1998; Schultz, 1998, 2001), and is *also* centrally implicated  
 7 in hallucinations and delusions (Silbersweig and Stern, 1996; Silbersweig et al., 1995), is at the  
 8 heart of the ‘false-belief’ states that generate the dream process (Solms, 1997, 2000, 2002). Thus,  
 9 dreams—the delusional beliefs we *all* experience, appear to have a shared neurobiological basis  
 10 with the psychotic states seen in psychiatric disorders, suggesting that emotion is to be centrally  
 11 placed in any argument about the casual basis of delusional beliefs.


## 12 Confabulation

13 Emotion also appears to be centrally involved in delusional ideation in the false beliefs of neuro-  
 14 logical patients who confabulate (where confabulate refers to a false-belief state in neurological  
 15 patients—who believe, for example, that they are living in a hotel not a hospital, or that their  
 16 spouse is an imposter). Such delusional states are found in patients who suffer large bilateral  
 17 medial frontal lesions (Schnider, 2001, 2003). However, the cognitive psychology literature has  
 18 tended to ignore the issue of motivation in such cases (Fotopoulou et al., 2004; Turnbull et al.,  
 19 2004a,b) and focus mainly on cognitive issues.

20 In particular, the cognitive literature app  regard impairments of general *executive* func-  
 21 tion as being at the core of these patients’ confabulations (Baddeley and Wilson, 1986; Benson  
 22 et al., 1996; Kapur and Couchlan, 1980; Papagno and Baddeley, 1997; Stuss et al., 1978). However,  
 23 the argument that impairments of executive function are the sole cause of confabulation appears  
 24 to be flawed, for a number of reasons (see Turnbull and Solms, 2007, for a review). Most impor-  
 25 tantly, such patients can have substantial impairments of general executive function without  
 26 becoming confabulatory—neurological wards contain many patients with substantially impaired  
 27 executive functions, but confabulation is actually rather rare. Confabulation is also rather selec-  
 28 tive in its nature. Most notably, when you speak to a patient who confabulates, they are typically  
 29 sensible with regard to most issues, but have areas in their mental life, often associated with family  
 30 or work issues, in which they suddenly become rather delusional (some notable examples are  
 31 Burgess and McNeil, 1999; Conway and Tacchi, 1996; Villiers et al., 1996). More recent accounts  
 32 of confabulation seek to explain such specificity by focusing on alternative cognitive explanations,  
 33 such as monitoring errors (Gilboa et al., 2006; Schnider, 2003).

34 The potential role of emotion has long been noted anecdotally, but has now been studied more  
 35 systematically in confabulatory patients. In a series of studies (Fotopoulou, 2010; Fotopoulou  
 36 et al., 2004, 2008b; Turnbull et al., 2004a,b) we demonstrated that the vast majority of delusional  
 37 beliefs experienced by these patients are positively biased. Naturally, it is difficult to classify peo-  
 38 ple’s false beliefs in terms of valence, as the experimenter cannot always be certain whether the  
 39 situation being described would be pleasant or pleasurable for that individual; an obvious exam-  
 40 ple is of Capgras delusions (Ellis and Young, 1990), where the patient believes that a family mem-  
 41 ber (say her husband?) is an imposter—who looks, sounds, and dresses like her actual husband.  
 42 Whether this delusion is pleasurable or not depends entirely on how she feels about her husband.  
 43 If he was a charming and much-loved partner, the delusion has unpleasurable consequences. If he  
 44 was nasty and uncaring, the delusion is potentially an ingenious method for distancing the patient  
 45 from this difficult man.

46 In summary, it is virtually impossible to be certain about the question of emotional valance for  
 47 false beliefs about a *person*. However, one way of studying this issue is to investigate confabulations

1 about *place*. In doing so the experimenter can study with relative certainty the pleasantness of the  
 2 confabulations, as they know both where the patient *is* in reality, and the *confabulated* location.  
 3 These locations can then be independently rated with greater certainty. Turnbull et al. (2004a)  
 4 investigated the pleasantness of confabulations in this manner in the cases of the 16 patients  
 5 reported in the neuroscientific literature between 1980 and 2000, who provided *actual* and  
 6 *confabulated* locations. They found that individuals universally believed themselves to be in *more*  
 7 affectively positive situations than in reality. The confabulated locations are themselves psycho-  
 8 analytically interesting. One substantial group (roughly half of the cases) goes to very ‘exotic’  
 9 locations, for example, a bistro in the south of France, or a ferry in the Caribbean. The other class  
 10 of confabulators  do not go to exotic locations but to ‘safe’ places. These confabulators go home,  
 11 to their parents’ home, or to their old university. The sorts of personality predisposition that  
 12 might predict this choice is incompletely understood, but the work of our group suggests that  
 13 patients in low mood states are more likely to produce positive confabulations, that is, delusional  
 14 beliefs serve as a form of defence (Fotopoulou et al., 2007a,b, 2008a,b; Turnbull et al., 2004b).

## 15 **Anosognosia**

16 Another interesting class of neurological patients with false beliefs includes the anosognosics—  
 17 who hold striking delusional beliefs, typically about their bodies, and denying that they are disa-  
 18 bled. Prototypically, they tend to be paralysed on the left side of their body, after large right-sided  
 19 strokes. In extreme cases, for example, the patient denies they are in any way paralysed, and pro-  
 20 duces bizarre arguments to counter the questions of the examiner—for example, if ‘a patient who  
 21 claims that she is able to run is asked why she is in a wheelchair, she might respond: “There was  
 22 nowhere else to sit.” If asked why she was not moving her left arm, she may say something like:  
 23 “I exercised it a lot earlier today, so I’m resting it.”’ (Solms and Turnbull, 2002, p. 262). Nevertheless  
 24 they appear to not be aware that they are paralysed. There is a growing literature (primed by  
 25 Kaplan-Solms and Solms, 2000, Chapter 8) on the fluctuating nature of these patients’ delusions,  
 26 and the way in which psychological variables can change the presentation of these patients (Marcel  
 27 et al., 2004; Nardone et al., 2007; Tondowski et al., 2007; Turnbull et al., 2002, 2005a). That is,  
 28 they deny their disorder under *most* circumstances, especially when they are formally asked to say  
 29 whether they are paralysed or not. However, if one discusses things in a safer setting, they tend to  
 30 become far more aware of their disorder. Indeed, this awareness often leads to episodes of sad-  
 31 ness, as they begin the process of mourning their disability. There are also reports of patients  
 32 making quite a striking recovery of awareness (e.g. Fotopoulou et al., 2009)—which may have  
 33 substantial effects on the patient’s mood.

34 In more rigorous experimental investigations of the extent of their awareness, it can be demon-  
 35 strated that these patients *do* have some implicit knowledge of their paralysis, even if they for-  
 36 mally deny this (Fotopoulou et al., 2010). Nardone et al. (2007) found that the patients who  
 37 showed the *greatest* magnitude of denial of deficit were also those who found their attention best  
 38 captured by deficit-related words (e.g. paralysis). An illuminating clinical example of the fluctuat-  
 39 ing nature of these delusional states is a patient who was paralysed (but denied paralysis) described  
 40 by Ramachandran and Blakslée (1998, p. 151), in whom the denial deficit could be completely  
 41 removed by offering a circumstance in which the patient could be paralysed without it being  
 42 emotionally threatening. To produce such a situation, Ramachandran spoke to the patient, saying  
 43 that he was terribly sorry about troubling them, but as part of the neurological examination he  
 44 needed to deliver an injection into their arm. This would be a slight inconvenience as he presented  
 45 it, for it meant that for a few minutes their left arm would be paralysed. Naturally, Ramachandran  
 46 gave the patient a *saline* injection, with no paralysing effect. However, after the injection they



1 patient was asked about their arm and they reported 'it doesn't seem to want to do anything . . .  
 2 it's not moving' (Ramachandran and Blakslée, 1998, p. 151). Under these circumstances, the  
 3 patient has of course had the same neurological deficit (paralysis) as a few minutes earlier, but  
 4 now without the catastrophic negative emotional consequences. However, the lack of movement  
 5 is now explained as being simply a result of a temporary injection—an idea with much more  
 6 modest emotional consequences, and it now appears that the patient is quite able to tolerate the  
 7 paralysis.

8 Naturally, from a psychoanalytic perspective it is fascinating that psychological variables of this  
 9 sort should be able to distort the patient's perceived reality so dramatically. Indeed, Ramachandran  
 10 does not hesitate to describe these phenomena as classic defence mechanisms: including rationali-  
 11 zation, projection, and even reaction formation (Ramachandran and Blakslée, 1998). To better  
 12 understand the fluctuating emotional presentation, we have studied the emotional range of these  
 13 patients, which is the same as in non-anosognosic patient controls (Tondowski et al., 2007;  
 14 Turnbull et al., 2002, 2005a). However, the *object* to which the patients direct these powerful emo-  
 15 tions seems entirely different. They tend, almost exclusively to feel (for example) great sadness at  
 16 the loss of their spectacles, or the disability of others, but are remarkably indifferent or stoic about  
 17 their own difficulties—in a phenomenon akin to projection (Turnbull et al., 2002, 2005a).

## 18 Conclusion

19 This chapter has discussed a vast literature (see also Turnbull and Solms, 2007 for a review) sug-  
 20 gesting that there are a wide range of psychological processes, from stereotyping to false-belief  
 21 states, which have been investigated by the cognitive psychology, social psychology, and neu-  
 22ropsychological communities, and are also potentially of enormous importance to the psycho-  
 23analytic community, especially because they speak directly to the question of emotion and  
 24 motivation in mental life, which is of course what psychoanalysis, and related psychotherapies,  
 25 are centrally concerned with. These findings in particular suggest that the 'balance of power'  
 26 between emotion and cognition is such that emotion appears capable of acquiring the 'upper  
 27 hand' quite readily, at least in certain circumstances. This is perhaps clearest when we experience  
 28 powerful emotional states, and where a great deal is at stake for the individual. In such situations  
 29 we appear to have our cognitive processes distorted, or 'hijacked', by these powerful motivational  
 30 states. Nevertheless, this 'balance of power' issue, with emotion in the ascendant, is one domain  
 31 which seems especially difficult for neuroscientific colleagues to accept (Turnbull and Solms,  
 32 2007) and appears to be a central stumbling block in the narrowing gap between topics of interest  
 33 to neuroscience and psychoanalysis.


34 The relationship between psychoanalysis and neuroscience has, of course, been difficult for  
 35 much of the 20th century (Kandel, 1998). It has meant having an almost entirely separate literature  
 36 for psychoanalysis, which often appeared to be independent of neuroscientific publications.  
 37 Naturally, this is a non-optimal situation for any science. The fact that psychoanalysis has a pro-  
 38fessional terminology that can seem rather obscure to neuroscientific colleagues has not always  
 39 helped with this divide. There has also been a good deal of speculation-without-data in the field  
 40 and probably too much emphasis on clinical work in psychoanalysis rather than on research  
 41 (Kandel, 1998). This is not to suggest that clinical work is unimportant. It is extremely important—  
 42 but the field of psychoanalysis would be well served to dedicate resources to the question of  
 43 research as well.

44 Nevertheless, in spite of these difficulties, there are enormous prospects associated with bring-  
 45 ing together the two fields. It needs, of course, for the neuroscientific world to take emotion, and  
 46 the extent to which emotion can distort cognition, seriously. In contrast, the psychoanalytic





### Box 11.2 Questions for future study/research

1. To what extent are cortical emotion-regulatory systems involved in psychotherapies? This research domain will also be able to address the ways in which various forms of psychotherapy differ in their regulatory mechanisms.
2. Can psychoanalysis better understand the neurobiological basis of core psychoanalytic concepts such as  with the knowledge neuroscience can now offer?
3. How do psychoanalytic concepts of emotion-regulation differ from those of neuroscience? How does emotion regulation develop through the lifespan?

1 world should reach out to the neuroscientific community, and to be able to play according to the  
 2 rules of the game of mainstream science. This means publishing papers in neuroscientific journals,  
 3 and generating the kinds of experimental work that can survive peer review. Under these circum-  
 4 stances, neuroscientists are (in our experience) not resistant to engaging in communication on  
 5 topics of mutual interest (Turnbull and Solms, 2007), when you can demonstrate to them that  
 6 data have been collected using all of the conventional methods associated with experimental  
 7 science, and published in journals that they trust. Under these circumstances, the gap between  
 8 the two fields is narrowed enormously. Indeed there is more goodwill among neuroscientists  
 9 towards psychoanalysis than psychoanalysts may be aware of (Kandel, 1998), and not quite as  
 10 much resistance as we think we may fear.

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