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Anxiety and its Regulation: Neural Mechanisms and Regulation Techniques According to the Experiential-Dynamic Approach

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Additional information is available at the end of the chapter

Abstract

Although anxiety is not necessarily a pathological phenomenon, it can become dysregulated, causing suffering. Indeed, emotion dysregulation lies at the core of many psychopathologies. Thus, anxiety regulation is central to all effective psychological treatment. The predominant perspective on emotion regulation and dysregulation is appraisal theory, which proposes that the cognitive appraisal of an event generates an emotional response. According to Gross's process model, any emotion can become dysregulated when the patient lacks or fails to use an appropriate regulatory strategy. Therefore, the clinician must teach the patient better regulatory strategies. The perspective we put forward departs from Gross's model based on appraisal theory. The experiential-dynamic emotion-regulation model, EDER, grounded in affective neuroscience and modern psychodynamic psychotherapy proposes that (1) emotions precede cognition (temporal and neuroanatomical primacy), (2) emotions are not inherently dysregulated (they have specific properties of time and strength proportional to the quality of the stimulus), and (3) dysregulation derives from the combination of emotions plus conditioned anxiety, or from secondary-defensive affects, both leading to dysregulated-affective states (DASs). To regulate DAS, the clinician must regulate the dysregulating anxiety or restructure the defenses, which create defensive affects, and then help the client to fully express the underlying emotions that elicit anxiety and defenses. In this chapter, we specifically focus on dysregulated anxiety, its neural bases, and how to regulate it according to the EDER model. First, we present hypotheses and data to show the neural bases of anxiety. Then, specific strategies and techniques to regulate anxiety are explained and clinical excerpts illustrate their application.

Keywords: anxiety, emotion regulation, affective neuroscience, psychotherapy

1. Introduction

We must study the mechanisms for regulating emotions when considering dysregulated emotions and emotionally driven behaviors in conditions such as personality or anxiety disorders [1–4]. The term emotion regulation refers to the neurocognitive mechanisms by which we regulate the onset, strength, and expression of our emotions [5]. According to Gross's process model of emotion regulation, emotions are generated through the following sequence: (1) an individual, exposed to a situation engages with it; (2) attends to a particular aspect of the situation; (3) interprets the event; (4) experiences an emotional response with a feeling, physiological arousal, and adaptive action tendency; and (5) the individual modulates that response. In this model, every emotion can in principle become dysregulated if the patient lacks or fails to use an appropriate regulatory strategy. Thus, the clinician must teach the patient better regulatory strategies. Cognitive-behavioral therapies (CBTs) follow these principles (see **Table 1**). Most of these strategies act at the level of attention and cognition and are consciously applied [4] by individuals to the experience of their emotions such as fear, anger, or sadness.

	Emotion generation and regulation in normality	Emotion dysregulation in psychopathology	Implications for treatment
Cognitive Emotion-Regulation model, CER (Gross [5] and further developments)	Cognitive appraisal produces emotions. The individual adopts cognitive top-down regulatory strategies at different levels (situation selection, situation modification, etc.) to regulate them	The individual fails to use regulatory strategies (deficit mechanism) and every emotion can become dysregulated	The clinician teaches the patient emotion-regulation strategies (therapeutic model: family of cognitive-behavioral therapies)
Experiential-Dynamic Emotion-Regulation model, EDER (Grecucci [3]; Frederickson and Grecucci [36]; Grecucci et al. [4])	Emotion is automatically generated by subcortical structures with certain properties (duration, intensity). The brain self-regulates emotions through a biological mechanism	After emotion is generated, dysregulatory mechanisms intervene that stop self-regulatory mechanisms and cause dysregulated-affective states (DASs)	The clinician helps the patient to remove dysregulatory mechanisms and downregulates DAS (therapeutic model: family of experiential- dynamic therapies)

Table 1. Two models of emotion regulation (modified from Grecucci et al. [4]).

Experiments studying emotion regulation show that individuals can learn to regulate their emotions, and their neural bases have been uncovered (see [6] for a review of basic findings). Of these, the dorsolateral prefrontal cortex, dlPFC, and the inferior parietal cortex, IPC, are commonly believed to control attention and working memory [7]. The anterior cingulate

cortex, ACC, is associated with monitoring and control of ongoing processes [8]. The ventro-lateral prefrontal cortex (v1PFC) appears to be responsible for selecting goal-appropriate responses [9, 10] and inhibiting inappropriate ones [11]. The target region of regulation is commonly believed to be the amygdala, a structure that supports the elaboration of external and internal emotional stimuli [12, 13] and negative stimuli [14].

In another line of research, Grecucci et al. [15–17] evaluated the regulation of socially cued emotions in real interactions. These studies showed that mentalizing (reappraising the intentions of the partner as less negative) changes emotional reactions, interpersonal behaviors, and neural responses. The tasks used in one of these experiments were derived from economic game theory (e.g., the ultimatum game and dictator game). One study showed that participants had weaker emotional reactions, used less rejection behavior, and had less neural activity if they downregulated their emotions while receiving unfair offers. This modulation of emotion was visible in an area of the brain involved in aversive reactions elicited by unfair offers, namely the insula. The insula has been found to represent visceral-affective experience [18–20], sensory experience [19], moral disgust, and anger [21].

A third line of research explored the interpersonal regulation of others' emotions. In line with [22, 23], we define this as regulation that occurs within interactions between two people. Despite the relevance of this type of regulation for clinical situations, we found only one attempt to study it in a laboratory setting. In a recent study [24], participants were asked to regulate their own (intrapersonal condition) and other persons' (interpersonal condition) emotional states. In the interpersonal condition, participants watched videos of people watching and reacting to the same emotional video they were watching. Participants were instructed to tell the person in the video how to interpret (reappraisal strategy) or suppress (suppression strategy) the emotional content of the video. Participants using interpersonal regulation showed decreased activation of the insula, the temporal-parietal junction, the temporal pole, and the medial prefrontal cortex similar to previous studies on emotion regulation of socially cued emotions [15, 17]. Despite methodological limitations, this study showed that regulating others' emotions interpersonally is possible.

1.1. Problems with cognitive regulation models

Cognitive theories of emotion regulation rely on the assumption that cognitive appraisals occur before emotional reactions, an assumption not supported by several neuroscientific studies of affect (see [25, 26], for a discussion). Emotion has a neurobiological primacy over cognition in terms of temporal dynamics (emotional stimuli are elaborated a few milliseconds before cognitive information [27]). The amygdala is activated and, in turn, activates the body before a later signal goes to the prefrontal cortex. Second, emotions have a neurobiological primacy over cognition in terms of anatomical circuitry, in that direct links exist between perceptual systems and emotional structures but not between perceptual systems and cognitive structures [25, 26].

Further, if cognition is primary, then cognitive strategies should be strong in the face of secondary emotional responses. However, research findings show that cognition-based strategies are not fully available for regulating emotions when emotion activation is high. For

instance, experiments of emotion-regulation choice (how we choose which strategy to adopt in a given situation) [28] demonstrated that participants used reappraisal to regulate only low-intensity emotional stimuli and used distraction for high-intensity stimuli. This result reduces the importance of cognitive regulation strategies during stressful events that are more emotional than experimental stimuli used in the laboratory [26]. From a neurobiological point of view, a decrease in BOLD signal during induced emotional states in the prefrontal cortex (known to implement regulatory strategies) has been reported [29, 30].

2. Mechanisms of emotion generation, regulation, and dysregulation

Following the appraisal theory of emotion [31, 32], Gross' [5] emotion-regulation model holds that the cognitive appraisal of an event generates an emotional response. Based on this theory, cognitive-behavioral therapies focus on discrete cognitive, attentive, and behavioral factors to foster emotion regulation. In this view, emotion dysregulation occurs due to the failure to apply appropriate cognitive, attentive, and behavioral regulatory strategies (see [33–35]). Behavioral strategies (exposure to appropriate situations or adaptive modification of the situation), attentional strategies (increasing attentional flexibility or developing awareness to internal and external situational cues), and cognitive strategies (cognitive restructuring) are applied.

In this chapter, we depart from appraisal theory and cognitive emotion regulation, and present the experiential-dynamic model of emotion regulation [4, 36] that is grounded in affective neuroscience findings [25, 37, 38] and modern psychodynamic and experiential psychotherapy [39–43].

The experiential-dynamic emotion-regulation model [4] holds that events trigger (1) emotional responses prewired at birth [38] with inborn adaptive action tendencies [44] and facial expressions [45] which (2) precede cognition (temporal and neuroanatomical primacy) [46, 38]. According to neuroscientific research [25, 37, 38], emotions are automatically and implicitly generated by mainly subcortical brain structures with certain properties (duration and intensity proportional to the event) to give a sense of what is happening [46]. In normal conditions, the brain regulates emotions through a biological mechanism. They rise in intensity, peak, and then go flat with a Gaussian-like shape once the emotion's adaptive action tendency has been expressed in adaptive action.

Once elicited, emotions have a duration and intensity proportional to the stimulus and automatically self-regulate [26]. The conscious control or the use of a specific strategy is therefore not required to regulate emotions [4, 36]. Emotions are generated, expressed, and channeled into healthy actions and return to baseline [43] once the resulting adaptive action has served its evolutionary function. Thus, emotions are not inherently dysregulated [36].

Dysregulation derives from the combination of emotions plus conditioned anxiety, or from secondary-defensive affects, both leading to dysregulated-affective states (DASs). To regulate DAS, the clinician must regulate the dysregulating anxiety or restructure the defenses, which create defensive affects, and then help the client to fully express the underlying emotions that elicit anxiety and defenses (see **Table 1**).

But if emotions are not inherently dysregulated, what causes emotions to *become* dysregulated? Dysregulation results from (1) emotions paired with excessive levels of conditioned anxiety or (2) emotions that are triggered not by a discrete stimulus in reality but by an ongoing defense [43]. For instance, a patient projects that the therapist is criticizing him (imaginary and continual stimulus), and becomes afraid of the supposed criticism. Here, the patient is not afraid of the therapist; he is afraid of the image he places on the therapist (his projection). Thus, his ongoing anxiety results from his ongoing defense. Anxiety resulting from the defense of projection would be considered a defensive affect. We differentiate defensive affect (response to an imaginary stimulus) from true affect (response to a real stimulus).

The therapist following EDER model treats emotion dysregulation by applying experiential anxiety regulation techniques so that feelings can be explored without excessive anxiety or by restructuring the defenses that cause defensive affects. As a last step, the therapist helps the patient experience the underlying emotion fully without excessive anxiety or defenses so it can be channeled into adaptive action [39–43, 47]. Once the patient experiences his feelings deeply and channels them effectively into action, they no longer trigger anxiety, defenses, and symptoms instead (see [48]).

2.1. Why we regulate

Excessive anxiety causes painful physical symptoms due to activation of the somatic and autonomic nervous systems [49]. It compromises higher brain functions due to the release of neurohormones, which shut down the prefrontal cortex and hippocampus [43]. It triggers the use of defenses that create the patient's symptoms and presenting problems [33–35, 50, 51]. The compromised mental functioning and automatically triggered behavioral patterns prevent the patient from seeing better options, thinking about them, or being able to act on them. Excessive anxiety is a painful condition that Meltzer defines as unbearable mental pain that the mind wants to get rid of [52]. Thus, anxiety regulation is essential for any change to occur in psychotherapy.

To understand why and how anxiety is regulated in our brain and why "emotion regulation" does exist in the brain, we use an analogy from statistical mechanics (see [3] for a complete description). Statistical mechanics is the application of the theory of probability to the thermodynamic behavior of systems composed of a large number of particles. This branch of physics provides a model to link the microscopic properties of the individual elements to the macroscopic properties of the system made by them. Since the brain is one of the most complex systems in the universe (the brain is composed of 10^{12} interacting neurons), statistical mechanics can be fruitfully used to describe why and how mechanisms exist to regulate emotions. As the Boltzmann distribution suggests [53], the probability (P^1) that a system (the brain) converges toward a desired energetic state (E^1) (affective state) is negatively proportional to the temperature T^1 (level of dysregulated anxiety) of the system:

$$P^1 = Ke^{(-E1/T1)} \tag{1}$$

In every complex system, given two states E^1 and E^2, respectively, associated with temperatures T^1 and T^2, where T^2 is higher than T^1, the system will settle into state E^1, where the temperature is lower:

$$\frac{P^1}{P^2} = \frac{Ke^{(-E1/T1)}}{Ke^{(-E2/T2)}} \tag{2}$$

In other words, when emotion is activated, the mind can settle into that state (E^1 with its given T^1). However, if emotion is associated with excessive anxiety (DAS), the temperature overcomes the threshold of tolerability (say E^2 with T^2, where $T^2 > T^1$) and must be downregulated by the system. In our terms, for the brain to work, anxiety must be kept at acceptable levels (temperature T^1). The brain has several prewired mechanisms to self-regulate our emotions under normal conditions. However, if these mechanisms are interrupted by dysregulatory mechanisms, such as excessive anxiety (DAS), the temperature of the system will exceed the capacity of the mind to bear it. If this is the case, the system (in a state E^2 with T^2) will adopt strategies to abruptly lower it (defense mechanisms in psychodynamic terms that lower anxiety). However, the strategies to lower anxiety may have costs for the system (e.g., progressive cognitive, affective, behavioral distortions, and consequent symptom formation). One of the clinician's tasks is to help patients to regulate anxiety so they can experience progressively increasing levels of emotions without relying on defense mechanisms that create their presenting problems. After having examined **why** the brain needs regulatory mechanisms (excessive anxiety is painful), and **how** this is possible from a complex system perspective (the system constantly tries to move toward the lower energetic state), we focus on **what** is regulated.

2.2. What we regulate: anxiety

Emotions are evolutionary products designed to prime adaptive action [38, 54]. They become dysregulated by being paired with conditioned anxiety or they are created by dysregulating defenses [43]. If the therapist downregulates anxiety, the paired affect will become regulated. If the therapist restructures the defense that creates the dysregulated affect, the affect will disappear. For instance, if a patient can see the therapist accurately, the feelings toward the therapist based on the projection will disappear. After all, the defensive affects were an illusion based on the illusion of projection.

When an objective danger is detected, fear arises [50] to motivate us to deal adaptively with an objective threat. When a feeling arises, anxiety rises if it was dangerous to have this feeling in a previous relationship. Thus, anxiety is a signal that a feeling is rising that is potentially dangerous for this relationship [55]. All children in their development learn which feelings are allowed in their primary relationships and which feelings make caretakers anxious [56], thus threatening a relationship necessary for the child's survival [57–59]. To adapt [60], the child learns to ward off emotions that would threaten the relationship [56]. Thus, whenever a

forbidden feeling arises, anxiety automatically signals danger [61]. The anxiety occurs out of the patient's awareness because it is generated nonconsciously in the brain [46].

The symptoms of anxiety in the body are created by the activation of the somatic and autonomic nervous systems. These systems are activated principally by several subcortical brain regions such as the cingulate and parahippocampal gyri, the amygdaloidal complex (the amygdala and bed nucleus of stria terminalis), septal nuclei, hypothalamus, some portions of the thalamus (inferior thalamic nuclei), and some parts of the basal ganglia [62]. Other brain areas involved in the anxiety circuit are the bed nucleus of the stria terminalis, hippocampus, hypothalamus, prefrontal cortex, periacqueductal gray matter, and the locus coeruleus.

The amygdala plays a fundamental role in the experience of anxiety by evaluating the valence (unpleasantness) and novelty of the stimulus [63–65]. We can divide the amygdala into three neural subgroups: the antero-central, the baso-lateral, and the medial. The baso-lateral group connects with the prefrontal orbital and medial cortex in the frontal lobe, and the associative cortex in the antero-temporal lobe. It is also hardwired with the bed nucleus of the stria terminalis, ventral hippocampus, and central amygdala. The antero-central group is hard-wired with the hypothalamus and brain stem, including the parabrachial nucleus and the solitary tract nucleus [64–66]. Recent research shows that activating projection neurons in the baso-lateral amygdala (BLA) increases anxiety, while selective activation of axons from the BLA to the lateral central amygdala reduces anxiety. Activating the monosynaptic glutamatergic projection from the BLA to the ventral hippocampus also increases anxiety [67]. Sensory information enters the baso-lateral portion, and then relevant information goes to the central portion. The central amygdala modulates behavioral, physiological, and cognitive activity through its connection with the cortex and the hypothalamus. The bed nucleus of the stria terminalis, BNST, is responsible for linking anxiety to emotional stimuli. This area is connected to the amygdala, hypothalamus, and parabrachial nucleus. Indirectly, it is connected with the prefrontal cortex, hippocampus, hypothalamus, locus coeruleus, raphe nuclei, lateral septum, and periaqueductal gray matter. The BNST can be functionally divided into the anterodorsal (adBNST), ventral (vBNST), and oval (ovBNST) portions. Inhibiting the ovBNST, or exciting the adBNST reduces anxiety. By contrast, activating the ovBNST and inhibiting the adBNST cause anxiety [68]. Perhaps, in the absence of threatening stimuli, ovBNST is under the inhibitory control of the adBNST, or the adBNST is uninhibited by the inhibition of ovBNST. Inhibition of BLA neurons, connected to the adBNST, triggers anxious behavior. Conversely, their activation reduces anxiety as does selective activation of axons terminating in the lateral hypothalamus. The connection of the adBNST with the parabrachial nucleus mediates autonomic responses of anxiety [68]. vBNST has both glutamatergic excitatory synapses and GABAergic inhibitory ones with non-dopaminergic cells in the ventro-tegmental area (VTA) [68]. Glutamatergic inputs trigger avoidance and anxiety; the GABAergic inputs produce reward and reduce anxiety [69]. Glutamatergic vBNST cells show an increase of activity during a foot-shock session, while GABAergic cells are inhibited [70]. Septal nuclei in the anteromedial portion of forebrain receive inputs from the hippocampus and amygdaloidal complex and make synapses with the thalamus, hypothalamus, and brain stem. The septo-hippocampal system may mediate stress-induced anxiety [71].

2.3. Dysregulated anxiety

Emotions may be accompanied by excessive anxiety as a result of conditioning in previous attachments. Anxiety becomes a conditioned response indicating that a rising feeling could endanger a relationship [50, 58]. In turn, anxiety triggers defense mechanisms (cognitive distortion, behavioral avoidance) that cause patients' symptoms and presenting problems [72].

The combination of emotion and excessive anxiety creates a dysregulated-affect state [36]. An internal feeling is perceived as a threat in a relationship. The somatic and autonomic systems are activated [49, 54], creating symptoms of anxiety [49, 73]. This activation of the amygdala occurs before the message gets to the prefrontal cortex and becomes conscious [37].

The amygdala activates the somatic and autonomic nervous systems, which create a variety of anxiety symptoms in the body [42, 43]. When anxiety is discharged in the striated muscles (somatic nervous system), patients experience symptoms caused by tension in the striated muscles (tension in voluntary muscles, clenched hands, and sighing). When anxiety is discharged into the somatic nervous system, it is at a healthy level, so feelings can be expressed and experienced deeply [43].
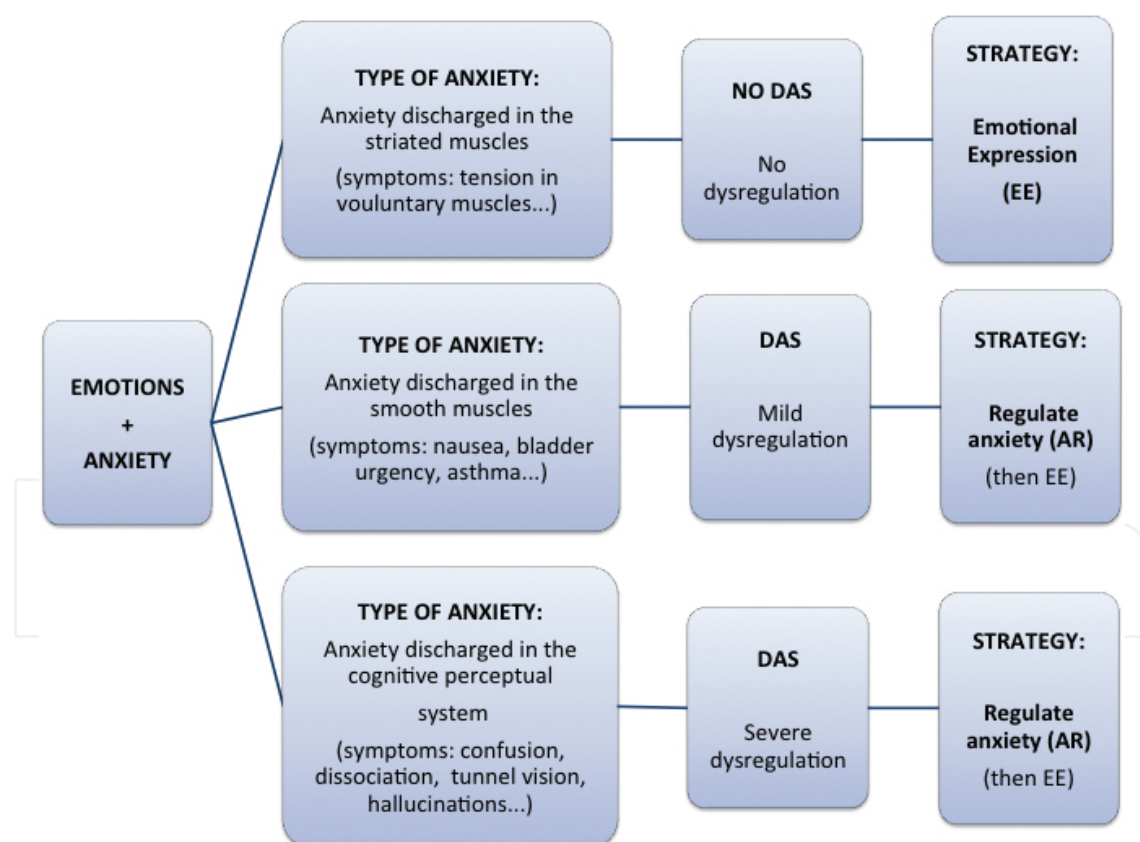

Figure 1. Decision tree for anxiety regulation.

When anxiety becomes too high, it shifts into the parasympathetic branch of the autonomic nervous system. Now, patients experience anxiety in the smooth muscles (nausea, diarrhea,

migraines, sick to stomach, and need to use the bathroom), resulting in moderate DAS [74]. Hence, anxiety regulation becomes imperative [43].

When anxiety becomes even stronger, the anxiety is discharged even more into the parasympathetic nervous system, causing cognitive perceptual disruption, due to hypoperfusion of the prefrontal cortex (problems thinking, loss of reality testing, blurry vision, ringing in the ears, dizziness, fainting, and blanking out) [43]. The clinician should stop exploring emotions and regulate anxiety until it is discharged again into the striated muscles. See **Figure 1** for a summary of the proposed method (see [43] for a more detailed discussion).

When anxiety is too high, it triggers defenses, which can distort the perceptual/cognitive processes. For instance, denial can prevent a woman from seeing the danger of remaining with an abusive husband ("He didn't mean it.") Her defense prevents her from accurately seeing the stimulus to her feelings. Or she might deny that she has any negative feelings toward him for hitting her ("I love him."). Here, the defense prevents her from seeing her responses to his abuse, keeping emotions out of consciousness.

Likewise, defenses can prevent the patient from being aware of her anxiety and, thus, from regulating it. For instance, a patient might ignore her anxiety ("I always talk quickly. I'm just a fast talker."). Unable to see her anxiety, she cannot regulate it. We might hypothesize that defenses interfere with optimal communication between the prefrontal cortex and the amygdala. For anxiety regulation to occur, the patient must be able to observe her anxiety (the effects of the amygdala on the body). A recent study suggested that anxious behavior can occur because reduced functionality between the prefrontal cortex and the amygdala can disinhibit the amygdala [75].

3. Techniques to regulate anxiety

3.1. Problems with CBT techniques to regulate anxiety

Thinking can affect our emotions. But our emotional impulses can also channel and influence our thoughts [25, 46]. While modifying emotions gives rise to changes in cognitions, working primarily on changing cognitions may not necessarily cause a shift in emotion [25]. The CBT framework fails to understand that most feelings are not triggered by conscious thoughts but by nonconscious neural processes. In fact, most thoughts are defenses triggered by feelings. Cognitive interventions ask the patient to cognitively manage emotions based on willpower and repeated practice of a technique. The patient is being asked to consciously control anxiety and dysregulated-affect states while not being helped to address the cause, the unconscious feelings triggering the anxiety and defenses.

This problem results from the fact that CBT theory fails to recognize that core emotions (an evolutionary response to environmental and interpersonal cues) trigger anxiety, based on conditioned responses [55]. While anxiety can be regulated through attentive focus and self-soothing, it will continue to be triggered each time feelings arise. Unless the patient is helped to bear those previously warded-off feelings, the patient will not be helped to cope with the

cause of his anxiety. In the following section, we outline regulatory strategies that are more consistent with affective neuroscience and the nonconscious processes that create emotions.

The CBTs are based on the assumption that the therapist can regulate the patient's anxiety. Unfortunately, this assumes that the patient is relating to the therapist instead of a projection placed upon the therapist. Just because the therapist wants to help does not mean the patient perceives her that way. In fact, if the patient perceives the therapist as critical, this perception will drive not only the patient's resistance to therapy, it will increase the patient's fear of the therapist. Anxiety cannot be regulated and the impasse cannot be resolved unless the therapist addresses the patient's projection.

Another potential drawback of the cognitive-behavioral framework is that techniques focusing on positive reappraisal may encourage some patients to obsessively ruminate about the thoughts that need reframing or pursue endless reframes without significant relief [76]. While the third-wave CBTs address this with cognitive defusion and mindfulness methods, this assumes that conscious top-down regulation can work with nonconscious bottom-up activation. See [4] for a more detailed discussion and a comparison between CBT and experiential-dynamic therapy (EDT) techniques.

3.2. Principles of experiential-dynamic techniques for regulating anxiety

Within the field of experiential-dynamic therapies, true feelings are understood to be generated through subcortical neuroperception of the environment, the experience of stimuli in reality [25, 37–39, 41–43, 46, 54, 55, 77]. DASs are created through the pairing of emotions with excessive conditioned anxiety or by defenses which create defensive affects [39, 41–43]. With this dual theory of causation, we differentiate true feelings generated by real stimuli from defensive affects generated by imaginary stimuli.

In the course of therapy, the patient describes relationships where his problem occurs. The therapist explores the feelings triggered toward a specific person for doing a specific thing to the patient. As feelings rise, feelings trigger anxiety and defenses. If necessary, the therapist regulates anxiety and then explores the feeling. Otherwise, the therapist helps the patient see the defenses that create his symptoms and presenting problems and then encourages the patient to face his feelings rather than use his defenses.

Sometimes, anxiety can be regulated fairly easily by helping the patient pay attention to the physical experience of anxiety in the body. Or the patient may be helped to feel less anxious just by understanding the process of the session: as he explores his feelings, feelings trigger anxiety, anxiety triggers defenses, and the defenses create his symptoms and presenting problems. Understanding causality orients the patient, reducing anxiety.

Another way to regulate anxiety is to block defenses that perpetuate or escalate anxiety. For instance, rumination about past events or future fantasies will perpetuate anxiety. Blocking those defenses will block the rise of anxiety based on fantasies. Then, the therapist directs the patient's attention to the feeling in this moment that triggers anxiety in this moment. Keeping the focus in the here and now maintains an effective focus that will be inherently anxiety regulating. The therapist needs to ask himself: (1) is the patient focusing

on experience in the past, future, or present? (2) Is this a real stimulus or an imaginary one? (3) Is this a specific example or a vague one? And (4) what is the patient feeling toward whom for doing what? To explore feelings and regulate anxiety, we need to examine what the patient feels now regarding a real stimulus toward a real person for a real deed. This clarity and focus is inherently regulating.

Every time anxiety moves into the smooth muscles or cognitive/perceptual disruption, the combination of feeling and anxiety creates a DAS. The therapist stops exploring feeling and regulates anxiety until it returns to the striated muscles. The feeling, without dysregulating anxiety, is inherently regulated. If the therapist fails to regulate the anxiety until it is in the striated muscles, the patient will suffer from more severe somatic symptoms and will shift into more primitive defenses that lead to a loss of reality testing, which will perpetuate his anxiety. While assessing the patient's anxiety, the therapist will monitor how rapidly it rises, how slowly it drops as a result of regulation, and the pathway of anxiety discharge in the patient's body (somatic or autonomic nervous system). This assessment allows the therapist to ensure that anxiety has been regulated enough that it is safe to explore feelings to build the patient's capacity for affect tolerance.

Every story the patient tells triggers feeling and anxiety, allowing the therapist to assess what issues and feelings trigger the most feelings, anxiety, and defenses. Obviously, whatever issue triggers the most anxiety is where the patient needs the most help. Each time anxiety rises, the therapist helps the patient see the issue he has, the anxiety it triggers in his body, the defenses he uses, and how those defenses create the patient's symptoms and presenting problems. If the patient can observe his feelings, his anxiety, his defenses, and how those defenses create his presenting problems, he can understand what causes his suffering and the therapeutic task: let go of the defenses causing his suffering and face the feelings he has been avoiding.

When anxiety moves out of the striated muscles into the smooth muscles or cognitive/perceptual disruption, the therapist should regulate the anxiety and then explore feeling gradually, what we call the "graded format" [39, 40, 43]. In the graded format, the therapist explores feelings gradually until anxiety moves into the smooth muscles or cognitive/perceptual disruption. Then, he stops exploring feelings and regulates anxiety until it returns to the striated muscles. Then, the therapist explores feelings again. Each time the therapist and patient explore feelings at progressively higher levels until the patient can experience the full extent of his feelings without his anxiety moving out of the striated muscles and without using defenses that cause DAS.

We have described earlier how affects by themselves are not dysregulated. They are adaptive responses that guide mammalian and human behavior, proportional to the stimulus. When affects are dysregulated by excessive anxiety, we regulate anxiety and, thereby, end the affect dysregulation. However, with mention of defenses that cause DAS, we shift to a second source of dysregulated affects: affects that are triggered by defenses, what we call defensive affects.

Simply put, if a person criticizes you unfairly (real stimulus), you will feel anger toward that person. However, if you *imagine* a person is critical and wants to hurt you (imaginary stimulus), you will feel angry also. You would not be angry at what that person really did but at the

projection you place on that person. Now your feeling results from a defense: projection. That is why we call it a defensive affect. It is the result of an imaginary stimulus, not a real stimulus.

Likewise, you might become afraid, fearing at any moment that this imaginary critic will attack you. This is not the fear of an objective danger, this is anxiety triggered by a projection. That is why we call it projective anxiety [43, 78]. So, if you project that someone wants to criticize you, you might become angry with that person or afraid of that person. In either case, the anger or anxiety would be defensive affects, feelings that result from the defense of projection.

Let us clarify causality more deeply. A patient is angry with his boss. This anger triggers anxiety. But he denies that he is angry and projects that his boss is angry with him. In response to this projection, he becomes afraid of a supposedly angry boss. Now, anxiety regulation will do no good. We can regulate anxiety that is caused by a genuine feeling toward a real person for a real deed. But we cannot regulate anxiety that is caused by an *ongoing* projection. For as long as he projects onto the boss, he will be afraid of the boss, or, more precisely, the projection he places on the boss. First, we must help the patient see how he is projecting onto his boss.

What impact can this insight have on therapy? To deal with the anxiety of having feelings or desires in therapy, the patient may project those feelings or desires onto the therapist. For instance, an angry patient may project that the therapist is angry. The patient who wants to look at her inner life may project that the therapist wants to look at her inner life. In each case, the patient becomes afraid of the therapist's imagined feeling or desire. This is projective anxiety: fear of the projection.

The therapist cannot regulate a patient's anxiety if the patient is projecting onto the therapist. If the patient projects that the therapist is critical, a critic cannot regulate anxiety. The therapist must deactivate the projection that is perpetuating the patient's high anxiety. Once the patient sees the therapist realistically, the anxiety due to projection will disappear. Then the therapist can explore the patient's inner feelings and desires she formerly projected onto the therapist.

The reader may be puzzled since it is well known that defenses are unconscious mechanisms for avoiding forbidden feelings and the anxiety they trigger [50, 51]. Defenses are supposed to *reduce* anxiety. That is true for some defenses. The EDER therapist focuses on the avoided feelings that trigger the patient's anxiety. If anxiety rises, the therapist knows he is approaching the issues the patient avoids. Thus, anxiety is a good sign: the therapist is approaching important therapeutic material. If anxiety becomes too high, it must be regulated. If anxiety remains in the striated muscles, it does not need to be regulated.

When feelings trigger anxiety, patients use defenses to ward off feelings and the anxiety. For instance, when the patient uses a defense, such as intellectualization, his awareness of feeling drops and his anxiety will drop. If the therapist identifies the defense for the patient and invites him to face the feelings underneath his intellectualization, the patient's anxiety will rise because the patient and therapist are going toward the avoided feelings that trigger anxiety.

However, certain defenses do not reduce anxiety. For example, the patient may project his anger onto the therapist to avoid feeling it within himself. However, once he projects his anger onto the therapist, he can become afraid of the therapist, resulting in even more anxiety, creating a DAS.

Suppose a man assaults a woman (stimulus in reality). This triggers anger (true feeling) in her and, as a result, she is able to fight him off. However, in the therapist's office she is terrified of the therapist, imagining that he is angry. She projects her anger upon the therapist (defense). This defense of projection creates fear (defensive affect), the result of projecting upon the therapist. Or suppose this patient criticizes herself for how she handled the assault. She turns the anger toward the assailant onto herself (defense) and becomes sad (defensive affect).

Understanding what causes a given feeling allows us to intervene effectively. If the patient's fear results from projection, we need to deactivate the projection [42, 43, 79] so that the anxiety resulting from projection will drop. Here, cognitive and experiential-dynamic therapists agree. Likewise, if the patient is sad due to self-attack, we need to help the patient see the defense and relinquish it, so that her defense-caused sadness (defensive affect) will drop. Again, cognitive and experiential-dynamic therapists agree.

Clinically, one form of dysregulated emotion is true feeling plus excessive anxiety paired through conditioning. Classic anxiety regulation techniques shared by cognitive and experiential-dynamic therapists bring the patient's anxiety down until the patient can bear her underlying feeling without anxiety [39, 43, 79]. However, to prevent future relapse, a risk in cognitive therapies (see [80, 81]), once the defense has been relinquished, experiential-dynamic therapists will explore the true feeling underneath, which triggered the anxiety and defenses [39, 41–43].

Unless the therapist builds the amount of feeling the patient can bear, DAS will occur with each subsequent activation of feeling (see [82] for a review illustrating the relationship between the degree of emotional experience and the level of long-term outcome.) To prevent further DAS in the future, the experiential-dynamic therapist will explore the feeling at progressively higher levels. Each time anxiety gets too high, the therapist will regulate anxiety (downregulation), and then explore the feeling at a higher level.

In this gradual stepwise exposure method, the therapist builds the patient's capacity to bear the full extent of her feelings without becoming dysregulated by anxiety or defensive affects. In this model, excessive anxiety is determined by whether the patient's anxiety shifts into the parasympathetic branch of the autonomic nervous system (see [43] for a fuller discussion of the symptoms, which indicate that the patient has gone over the threshold of anxiety tolerance, and signs of cognitive impairment due to neurohormonal discharge).

This graded exposure to feelings helps the patient develop the capacity to bear her feelings without anxiety, so she can channel them into effective action [40, 43]. Once she can bear her feeling to the fullest extent, relapse into DAS can be prevented.

A second form of emotion dysregulation occurs when the patient's defenses cause a defensive affect [39–43]. For instance, a patient who is irritated with the therapist may use the defense of self-attack and become depressed in session. The therapist will help the patient face the feeling toward him without using the defense of self-attack, which is causing the DAS. "If we look under these critical thoughts, I wonder what feelings might be coming up here with me?" The therapist helps build the patient's capacity to face and label her feelings without using the

defense of self-attack creating her DAS. See [36] for a more detailed discussion on how to deal with DAS due to defensive affects.

Type of affect	Strategy	Technique
Emotions	Emotional expression	-Identify and label the emotions
		-Help the patient pay attention to the emotions in the body
		-Differentiate feelings from anxiety
		-Differentiate feelings from defense
		-Experience feelings physically in the body
		-Feel the impulse physically in the body
		-Portray the impulse
DAS due to excessive anxiety	Regulation of anxiety	-Identify the symptoms of anxiety in the body
		-Mobilize self-observing capacity
		-Pay attention to anxiety in the present moment
		-Differentiate anxiety from the stimulus that generated it
		-Show causality: feelings in this moment trigger anxiety that triggers symptoms
		-Differentiate the symptoms of anxiety from the experience of feelings
		-Interrupt defenses that prevent anxiety regulation (ignoring, avoiding the present moment)
		- Interrupt defenses that perpetuate anxiety (self-attack, projective anxiety, symbolic equation)
		- Address spatial and temporal distortions
		-Shift the resistance system to isolation of affect
		-Restructure the pathway of anxiety discharge from the smooth muscles or cognitive/perceptual disruption into the striated muscles (the graded format)
		-Interrupt projective anxiety

Table 2. Strategies and techniques to regulate anxiety in the EDER model.

When the patient responds with feeling, the therapist will encourage the patient to experience her feeling more deeply, "How do you experience that anger physically in your body?" Or if the patient becomes sick to her stomach (a sign of anxiety), the therapist will intervene immediately. "That's a sign of anxiety. If we look under the anxiety, could we take a look under your anxiety and see what feelings are coming up here toward me?" If the patient becomes

irritated toward the therapist and then shifts into a DAS of weepiness, the therapist will interrupt the defense immediately causing her DAS. "Notice how these tears come in to wash away your anger? Could they be making you depressed? Could they be protecting me? If you don't protect me, could we look underneath those tears and see how you experience the anger that's underneath those tears?"

In these examples, we see how different defenses create different defensive affects. We either restructure the defense (e.g., projection) to eliminate the anxiety or defensive affects resulting from projection, or we identify the defense (e.g., self-attack) and DAS and then help the patient face the feelings which the defense and DAS are covering. In these ways, we build the patient's capacity to identify, experience, and bear her feelings without anxiety or defenses. Then, she can channel those feelings into effective action so they can fulfill their original evolutionary purpose: adaptation (see **Table 2**).

4. Conclusion

The EDER model presented in this chapter departs from other emotion-regulating psycho-therapies (see, e.g., [34, 35, 83, 84]). One of the assumptions is that emotion activation is a nonconscious physiological process that occurs initially without conscious awareness. The brain is programmed to generate emotions in response to internal and external stimuli. Emotions have physical properties with intensity and duration proportional to the intensity of the stimulus. Emotions, as evolutionary products, are not inherently dysregulated. They are activated so they can be channeled into adaptive action.

Moreover, emotion dysregulation is conceptualized as the result of dysregulating anxiety and defenses, not by the lack or failure of regulatory strategies, thus departing from a CBT view. From this perspective, dysregulation results from (1) emotions paired with excessive conditioned anxiety in the parasympathetic branch of the autonomic nervous system and (2) affects created not by a stimulus in reality but by defenses such as projection and self-attack. Frederickson and Grecucci [48] define feeling plus excessive anxiety and feelings covered by defensive affects as dysregulated-affective states. DAS due to excessive anxiety needs to be regulated [3, 4, 48]. If anxiety is too high, it impairs reality testing, and cognition is impaired due to a shutdown of the prefrontal cortex. Once anxiety is regulated and returns to the striated muscles and reality testing is restored, cognitive reworking (in a psychodynamic or CBT fashion) can be done. We use two basic overarching strategies: (1) emotion expression for true feelings triggered by real stimuli and (2) regulation of anxiety (and restructuring of defenses) for DAS due to emotions paired with excessive anxiety (see [43], for more details). We then explore true feelings as deeply as the patient can bear. The therapist upregulates true emotions while deactivating DAS until the patient fully experiences the previously avoided emotions without the DAS caused by anxiety (or by defenses). Now, the patient feels relief and can channel emotions into effective, adaptive action.

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