On the Dynamical Interplay of Positive and Negative Affects

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Emotional disorders and psychological flourishing are the result of complex interactions between positive and negative affects that depend on external events and the subject’s internal representations. Based on psychological data, we mathematically model the dynamical balance between positive and negative affects as a function of the response to external positive and negative events. This modeling allows the investigation of the relative impact of two leading forms of therapy on affect balance. The model uses a delay differential equation to analytically study the bifurcation diagram of the system. We compare the results of the model to psychological data on a single, recurrently depressed patient who was administered the two types of therapies considered (coping focused versus affect focused). The model leads to the prediction that stabilization at a normal state may rely on evaluating one’s emotional state through a historical ongoing emotional state rather than in a narrow present window. The simple mathematical model proposed here offers a theoretical framework for investigating the temporal process of change and parameters of resilience to relapse.

1 Introduction

Human functioning is regulated by a dialectical tension between both positive and negative states. Traditional psychology focused primarily on the...
negative dimension (Beck & Alford, 2009), whereas positive psychology has recently shifted the emphasis to include positive experience (Lopez & Snyder, 2009). Rather than developing these two dimensions along independent lines, psychology needs theories that systematically integrate both concurrently. Describing the process of change during psychotherapy thus requires the development of theoretically based models that capture the dynamical interaction of positive and negative dimensions, along with mathematical tools to analyze the evolution of these states.

First efforts in this direction were undertaken with the balanced state of mind model (BSOM; see Schwartz & Garamoni, 1989; Schwartz, 1997), an integrative model of positive $P$ and negative $N$ cognition and affect. This static model has demonstrated that distinct ratios differentiate psychopathological, normal, and optimal states. Drawing on Lefebvre’s (1992) mathematically based theory of consciousness, the BSOM model uses a ratio, the emotional balance $EB = P/(P + N)$, to define emotional and cognitive balance. Numerous studies have shown that clients progress from low pretreatment balances to normal or optimal balances depending on the success of the therapy (Bruch, Heimberg, & Hope, 1991; Haaga, Davison, McDermut, Hillis, & Twomey, 1993; Schwartz et al., 2002).

The dynamical evolution of these variables is extremely important but still largely ignored. Recent work has nevertheless reported naturally occurring rhythms in daily and weekly mood (Bisconti, Bergeman, & Boker, 2004; Chow, Ram, Boker, Fujita, & Clore, 2005; Deboeck, Boker, & Bergeman, 2008) in response to stresses: the model predicts a temporary increase in amplitude of the oscillations as the person fluctuates between more intense negative emotions dealing with loss and a more positive orientation of restoring normal adjustment (Bisconti et al., 2004; Stroebe & Schut, 1999; Ong, Bergeman, Bisconti, & Wallace, 2006), and as the loss is gradually integrated, the trajectory of emotional expression is dampened to return to a steady state. Moreover, it was shown that during effective psychotherapy, especially early in treatment, affect trajectories were characterized by extreme and uneven fluctuations rather than smooth oscillations. Using a dynamic systems model of change to investigate cognitive therapy of depression, Hayes and Strauss (1998) found that greater “destabilization” of depressive patterns and increased affect intensity early in treatment predicted superior treatment outcome. Although relative amplitudes of oscillations and their damping may adequately describe affective responses to normally occurring stressors, identifying additional phases of variability and stability may be important to better understand psychopathology and the process of change.

Fredrikson and Losada (2005) proposed a mathematical model for the emergence of these ratios. The model was based on a classical, chaotic dynamical system, the Lorenz equation, and opened an interesting and inflamed debate in the community. This model was decisively criticized
and retracted by Fredrickson because it was shown to be an inappropriate extrapolation from physics to psychology and failed to meet any of the criteria required to apply a mathematical model to data (Fredrickson, 2013). But despite these criticisms, the attempt was fundamental in promoting mathematical modeling to explain the well-established phenomenon of quantitatively precise ratios in psychology. Although this particular model of affect balance was flawed, mathematical modeling of the laws of human psychology remains essential to advance the theoretical understanding of emotional dynamics and mood disorders.

This is precisely the topic of this letter. We introduce a mathematical model of the evolution in time of positive and negative affect levels and how they evolve according to external events. From the model, we can evaluate a quantity analogous to the emotional balance that clinicians can evaluate on patients. We compared the dynamics of the model to the evolution of the balance between positive and negative affect during psychotherapy to ascertain whether the emotional trajectory contained distinct patterns that characterize different phases of treatment. Following the growing trend that focuses on intraindividual structures and dynamics or personality architecture, we adopted an idiographic, single-case quasi-experimental design to perform a detailed analysis of a patient’s change trajectory (Cervone, 2004; Molenar, 2004). The experimental data we consider in this letter show the evolution in time of the emotional balance of a recurrently depressed individual who was sequentially administered three increasingly intensive forms of therapy. The typical evolution of the emotional balance was examined in relationship to the type of treatment, stage of therapy, and critical events occurring in the patient’s life. Our mathematical model accurately reproduces the qualitative features observed clinically.

The development of a mathematical theory and its clinical confirmation is a complex task. A complete application of a theory to psychological data would require extensive experiments that are not yet available. As an initial step in this direction, this letter focuses primarily on introducing the modeling and mathematical analysis of positive and negative states with illustrative, empirical data that both supported and guided our investigations.

These results illuminate the dynamical process associated with phase transitions and optimal outcome during therapy, and this dynamical analysis refines the BSOM conclusions: reaching healthy levels of emotional balance may not be a stable, steady state, and deeper therapies building up on self-image may lead to small-amplitude, low-frequency oscillations of the EB that show increased stability. In other words, EB flexibility and the emergence of oscillations lead to more stable outcomes than rigid steady states or, as Confucius put it, “The green reed which bends in the wind is stronger than the mighty oak which breaks in a storm.”
2 Material and Methods

2.1 Balanced States-of-Mind Model. The balanced states-of-mind model (BSOM; Schwartz, 1997) is a framework relating ratios of positive and negative affects (the emotional balance, EB) to psychopathology and optimal functioning. The model draws on a psychological theory of consciousness and self-awareness developed by Lefebvre (1992). Theoretically derived predictions of levels of positive effects in distinct situations were compared to empirical scores derived from cognitive and affective measurement instruments (Lefebvre, Lefebvre, & Adams-Webber, 1986; Adams-Webber & Rodney, 1983), allowing a definition of specific ratios associated with a spectrum of emotional states ranging from depression to optimal functioning.

In Schwartz (1997) and Schwartz et al. (2002), the model and empirical results describe five emotional states associated with specific ranges of EB: Superoptimal $EB = 0.88$, Optimal $EB = 0.81$, Normal $EB = 0.72$, Subnormal or Coping $EB = 0.62$, and Pathological $EB = 0.50$, or below. With the exception of the extreme positive SOMs, which, because they rarely occur, are difficult to monitor, the model’s quantitative parameters have received considerable empirical support. For instance, it was shown that among a group of depressed men following a cognitive and pharmacological treatment, a subgroup a priori defined as average responders achieved, after treatment, an $EB = 0.70$, close to the predicted normal ratio. The same study showed that a predefined group of optimal responders reached after treatment the ratio $EB = 0.81$, theoretically defined as optimal. Similarly, Oishi, Diener, and Lucas (2007) reported that people who rated their happiness at 80% were more successful on measures of income, education, and political involvement than those who rated themselves either lower or higher (see also Schwartz & Garamoni, 1989; Fredrickson, 2013).

This theory is based on static evaluations of the emotional state. These states, however, are the result of a dynamical equilibrium building up as a response to external positive and negative events occurring in the individuals’ lives. Here, we address the nonlinear dynamical evolution of the state of mind using both empirical data and mathematical modeling. We start by a stochastic differential equation modeling the evolution of the emotional balance and investigate its dynamics. We then confront the outcomes of the model to new empirical data reporting the evolution of the emotional balance of a recurrently depressed individual subject to different kinds of treatment.

2.2 Mathematical Model of Emotional Balance Dynamics. This section is devoted to introducing our novel mathematical model characterizing the evolution in time of the psychological state of an individual. The model is based on the level of positive and negative affects, two quantities directly observed by the clinicians during therapy and on which the BSOM model
is grounded. These variables evolve depending on external events that may be positive or negative and that occur randomly in time at a rate denoted $\lambda_p$ and $\lambda_n$ and on the individual’s response depending on his or her emotional balance level. Our model is based on the simple psychological observation schematically depicted in Figure 1: depending on the emotional balance of a patient, positive and negative events distinctively affect that person’s state of mind. Specifically, depressed patients are strongly affected by negative events that can affect their emotional state for longer periods of time than that of nondepressed persons; in contrast, positive events barely affect their mood and are effective for only a brief period of time (Horner et al., 2014). The opposite arises for nondepressed people, who are able to deal with negative events and better sustain pleasant events. This variable integration of positive and negative events is central in understanding psychological resilience.

From a mathematical viewpoint, we model the evolution of self-assessed intensity of positive $P(t)$ and negative affects $N(t)$ in relationship to the EB ratio:

$$EB(t) = \frac{P(t)}{P(t) + N(t)}.$$  

The time evolution of the variables $P$ and $N$ is characterized by two main features:

1. It is driven by random positive and negative events occurring in the patient’s life. In the absence of determinism, we consider such events occurring as two independent Poisson processes $\Pi_P$ and $\Pi_N$ with intensity $\lambda_P$ and $\lambda_N$, respectively.
2. The way these events are integrated in the patient emotional state. If a positive or negative event occurs at time \( t \), two main quantities will describe its effect on the variables:

- The amplitude of the modification of the variables \( P \) and \( N \) subsequent to this event. These amplitudes depend on the emotional balance at time \( t \). We denote these amplitudes by \( q_P(EB(t)) \) and \( q_N(EB(t)) \), respectively, corresponding to the effect of a positive (negative) event on \( P(t) \) (\( N(t) \)) for a patient in a current emotional balance \( EB(t) \).

- The timescale characterizing the impact in time of this event. These also depend on the EB level of the patient at time \( t \) and are denoted \( \tau_P(EB(t)) \) and \( \tau_N(EB(t)) \).

Typically, depressed patients are affected by negative events in a more dramatic way and for longer durations than are nondepressed patients. Moreover, positive events affect them less intensely and for shorter durations than for nondepressed patients. In our model, this means that both \( q_P \) and \( \tau_P \) are increasing functions of the emotional balance and both \( q_N \) and \( \tau_N \) decreasing functions taking values in a bounded interval. For simplicity, we choose \( q_P \) and \( q_N \) as smooth error functions and constant \( \tau_P \) and \( \tau_N \).

The dynamics of the positive and negative affect levels therefore satisfies the system of stochastic differential equations with jumps (here, a piecewise deterministic Markov process; Davis, 1984):

\[
\begin{align*}
\frac{dP(t)}{dt} &= -\frac{1}{\tau_P(EB(t))}P(t)dt + q_P(EB(t))d\Pi_P(t) \\
\frac{dN(t)}{dt} &= -\frac{1}{\tau_N(EB(t))}N(t)dt + q_N(EB(t))d\Pi_N(t) \\
EB(t) &= \frac{P(t)}{P(t) + N(t)}
\end{align*}
\]

This model makes the implicit assumption that the individuals instantaneously evaluate their state of mind. A more realistic model would be that there exists an internal representation of the EB, the internal balance, \( IB(t) \), emerging from an internal representation of positivity \( IP \) and negativity \( IN \), and which governs the way individuals see themselves and feel external events. In addition to biasing the integration of external events, the internal balance creates self-induced positivity when the emotional balance is above the internal balance (corresponding to the feeling of being better than we thought) or self-induced negativity of being below our expectations. This
leads to the model:

\[
\begin{align*}
\frac{dP}{dt} &= \left( -\frac{1}{\tau_p(IB(t))} P(t) + g' (P(t) - IP(t)) \right) dt + q_p(IB(t))d\Pi_p(t), \\
\frac{dN}{dt} &= \left( -\frac{1}{\tau_N(IB(t))} N(t) + g' (N(t) - IN(t)) \right) dt + q_N(IB(t))d\Pi_N(t), \\
IB(t) &= \frac{IP(t)}{IP(t) + IN(t)}.
\end{align*}
\]

(2.1)

where the additional parameter \(g'\) denotes the bias strength toward internal affects.\(^1\) To complete the model, one needs to model the evolution of the internal representations as a function of the actual positive and negative affects. Essentially the internal positive and negative affect levels follow the positive and negative affect levels, but with a delay depending on the time needed to incorporate these modifications in our self-image. For simplicity, we will simply consider \(IP(t) = P(t - t_d)\) and \(IN(t) = N(t - t_d)\), where \(t_d\) is the typical time needed to take into account emotional changes in our representations. This delay is seen as a parameter of the model. In particular, it can be altered after a therapy that helps an individual to evaluate his or her emotional state from a broader perspective (Garland et al., 2010; see section 4.3.2). In a fluid limit approximation (Kurtz, 1970, 1971; Kurtz & Protter, 1991; Robert, 2013), one obtains that the system has an averaged behavior given by the simple system of deterministic nonlinear delay differential equations:

\[
\begin{align*}
\frac{dP}{dt} &= -\frac{1}{\tau_p(EB(t-t_d))} P(t) + g' (P(t) - P(t-t_d)) + \lambda_p q_p(EB(t-t_d)) \\
\frac{dN}{dt} &= -\frac{1}{\tau_N(EB(t-t_d))} N(t) + g' (N(t) - N(t-t_d)) + \lambda_N q_N(EB(t-t_d)) \\
EB(t) &= \frac{P(t)}{P(t) + N(t)}.
\end{align*}
\]

(2.2)

To fix ideas, we choose for \(q_p\) the three parameters sigmoid function,

\[q_p: x \mapsto \frac{\alpha x^2}{1 + \beta x^2} + c,\]

(2.3)

\(^1\)Here, we consider equal coupling on positive and negative affects for simplicity. Data are not available to evaluate this parameter yet, and in the case where distinct couplings emerge, this can be easily integrated to the model.
where \( c \) is the value at zero, \( \alpha \) is a scale parameter, and \( \beta \) controls the slope of the sigmoid at the inflection point—the sharpness of the changes between the way depressed or nondepressed patients integrate positive and negative events.

In order to uncover the role of different parameters in the dynamics and the effect of therapy, we use new psychological data.

2.3 Therapeutical Data and Analysis. We analyze the dynamics of emotional balance data from a recurrently depressed patient. The first investigations of this individual appeared in Schwartz (1997) and used visual examination of the affect balance trajectory to identify different therapy phases. In this work, we add up two data sets and, moreover, developed and employed qualitative methods validated by mathematical and statistical tools.

2.3.1 Participant. Our study focuses on an individual patient (JR) who was treated on three separate occasions over a 10-year period during which he twice relapsed. JR presented as a bright, overideational, 41-year-old Caucasian male, married with two boys, and the son of a renowned scientist. He was moderately depressed and anxious, and exhibited time urgency and strained interpersonal relationships because he was constantly competing to prove his superiority. His mother was chronically depressed, and he lived under the shadow of his renowned father. Because intellectual efficiency was central to his self-esteem, he felt distressed by rumination that inhibited productivity and generated fear of failure.

- **First period of treatment: Coping-focused therapy (therapy 1).** The first instance of cognitive-dynamic therapy administered to JR, described in Schwartz (1997), lasted five months and focused on developing coping strategies with minimal emphasis on psychodynamic exploration. JR learned anxiety management techniques to deal with stress, cognitive strategies to reduce worry, and communication skills to enhance interpersonal functioning. Later stages of therapy addressed the theme of JR being driven by competitive needs to surpass his unusually successful father. When his symptoms abated, JR prematurely terminated the therapy.

- **Second period of treatment: Mixed therapy.** Three years after the initial treatment, JR experienced a recurrence of mood disorder and work inhibition. The second therapy lasted for two years, expanded the coping strategies, and shifted toward the psychodynamic spectrum. The treatment uncovered anger at abusive peers and his depressed mother because of her “gray moods” and inability to protect him. Deeper dynamic issues surfaced with dream themes of deprivation, rage, and mortality fears. After two years of therapy and although JR was still struggling with these conflicts and exhibited dependency
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and interpersonal problems, he was no longer depressed or anxious. At this stage, he somewhat abruptly terminated his therapy.

- **Third period of treatment: Dynamic-focused therapy (therapy 2).** Five years later, JR experienced the deepest level of anxiety and depression when he saw that his grandiose expectations would not be realized. He recognized that he needed to fundamentally “reinvent himself professionally and personally.” Although his previous treatments provided reasonably enduring symptomatic relief, they ended prematurely and did not fully address his underlying personality structures that predisposed him to mood disturbances. Thus, we agreed to use a more psychodynamic focus from the start, to penetrate to a deeper level and work through these issues to a mutually agreed conclusion. This therapy lasted 18 months and began with a prolonged period of emotionally charged sessions of intense grieving about his mother’s death and not being as successful as he thought she expected him to be. He became aware of his narcissistic personality structure, compulsive achievement striving, and interpersonal conflicts. JR worked through emotionally charged dreams with classical psychodynamic themes of oral deprivation, Oedipal content (killing father and flirting with mother), and raw images of dehumanization. The end stage of therapy was different in that when JR became asymptomatic, he continued consolidating treatment gains by working through personality issues and dream material. Termination was not hastened, occurring at a time considered ripe by both patient and therapist.

For the last therapy, a log was produced from the clinical notes to identify session content and critical events of the patient’s life. For all three therapies, we monitored the emotional balance through the course of therapy from a 36-item inventory (18 positive and 18 negative mood terms), and the patient indicated how frequently he felt each emotion during the past week on a 5-point Likert scale (Likert, 1932): 0 = Not All to 4 = Almost Always (see Schwartz, 1997).

### 3 Theory

In this section we investigate the dynamics and bifurcations of the system, in order to characterize the possible states and transitions between these. Throughout the letter, we assume for simplicity that the rates of positive and negative events are equal ($\lambda_p = \lambda_n = \lambda'$).

The first step is to characterize the possible stationary solutions. At the equilibria of system 2.2, the emotional balance $EB^*$ necessarily satisfies the implicit equation:

$$EB^* = \frac{q_p(EB^*)\tau_P(EB^*)}{q_p(EB^*)\tau_P(EB^*) + q_n(EB^*)\tau_N(EB^*)}.$$
Heuristically, equilibria of the emotional balance do not depend on the precise frequency of occurrence of positive and negative events but only on their ratio (assumed to be equal to 1 here) and the amplitude of emotions integration. The right-hand side of the equation is a strictly increasing function of $EB^*$. Depending on its slope, it can have either one or three fixed points. We recall our choice $q_p(x) = \frac{ax^2}{1+\beta x^2} + c$.

In order to precisely understand the dynamics of the system as a function of the different parameters, we now slightly simplify the model to reduce it to a system that can be analytically solved. To this end, we make the simplifying assumption that the total amount of affect, $P + N$, is almost constant in time in both simulations of the full system and psychological data. This assumption, together with fixing constant timescales $\tau_P(x) = \tau_N(x) = \tau$, allows deriving a simplified version of the model by reducing it to one differential equation. Starting from the original equation for $P$ and denoting $T$ the constant value of $P + N$, we have

$$\frac{dP}{dt} = -\frac{P}{\tau} + \lambda q_P \left( \frac{P(t-t_d)}{T} \right) + g'(P(t) - P(t-t_d)).$$  \hspace{1cm} (3.1)$$

Defining $p(s) = \frac{p(ts)}{t}, \lambda = \frac{\lambda' \tau}{t}, \lambda = g' \tau$, and $t_0 = t_0 \tau, c = 0$, we obtain

$$\frac{dp}{ds} = (g - 1)p + \lambda \frac{p_d^2}{1 + \beta p_d^2} - gp_d,$$  \hspace{1cm} (3.2)$$

where $p_d(s) = p(s - t_0)$. We recall that $0 \leq p, p_d \leq 1$.

In this simplified model, we can characterize analytically the behavior of the system and its bifurcations as a function of the different parameters.

### 3.1 Steady States and Stability

Steady states are independent of the value of $g$ and depend only on the mutual relation between $\lambda$ and $\beta$. Simple algebra shows that the possible fixed points are given by (see Figure 2)

$$p_0 = 0,$$

$$p_{\pm} = \frac{\lambda \pm \sqrt{\lambda^2 - 4\beta}}{2\beta}.$$  \hspace{1cm} (3.3)$$

Taking into account the constraint that $p_+ \leq 1$, we have:

1. For $\lambda < 2, \beta > \lambda - 1$ and for $\lambda \geq 2, \beta > \frac{\lambda^2}{4}$: $p_0$ is the unique equilibrium.
2. For $\lambda < 2, \beta \leq \lambda - 1$ and for $\lambda \geq 2, \beta < \lambda - 1$: The system has two equilibria, $p_0, p_-$. 


Figure 2: Parameter space. (A) Parameter space regions for equilibrium states. The solid line corresponds to $p_+ = 1$, the dashed line to $p_0 = p_-$, and the dotted line to $p_0 = p_+$. (B) Parameter space regions for Hopf bifurcation, for a fixed value $\lambda = 4$. The solid line corresponds to $\beta = \lambda - 1$ and the dashed line to the combination of $g = 1 - \frac{\sqrt{\lambda^2 - 4\beta}}{2\lambda}$ (for $g < 1$) and $\beta = \frac{\lambda^2}{4}$ (for $g \geq 1$).

3. For $\lambda \geq 2$, $\lambda - 1 \leq \beta \leq \frac{\lambda^2}{4}$: The system has three equilibria, $p_0$, $p_-$, and $p_+$. Moreover, in the absence of delays, we have that $p_0$ and $p_+$ are always stable for $\beta \neq \frac{\lambda^2}{4}$, whereas $p_-$ is always unstable. However, for $\beta = \frac{\lambda^2}{4}$, $p_+ = p_- = 2/\lambda$ is left-unstable saddle point.

Let us now discuss briefly the transitions between the fixed-points regions 1 to 3. The boundary between regions 1 and 3 corresponds to the parabola $\{\lambda \geq 2, \beta = \frac{\lambda^2}{4}\}$, and along this line, we have $p_- = p_+$, and both values are in the interval $[0, 1]$. This hints toward a possible saddle-node bifurcation curve. It is not hard to show that along this line, the system satisfies the classical genericity and transversality conditions of that bifurcation (see, e.g., Guckenheimer & Holmes, 2013). At the line $\beta = \lambda - 1$, one of the equilibria crosses the value 1 and thus is no longer considered as a solution in our model. For $\lambda < 2$ and along that line, $p_- = 1$, and for $\lambda > 1$, we have $p_+ = 1$. This line does not correspond to a generic bifurcation, since virtual equilibria persist beyond this line (but lose their interpretation in our model).

3.2 Delay-Induced Hopf Instability. We now characterize the stability of the equilibrium $p_+$ in the presence of delays. We therefore concentrate on parameters within region 3. The result of the section can be summarized as follows.

For any parameters satisfying the inequalities

$$\lambda > 2,$$

$$\lambda - 1 < \beta < \frac{\lambda^2}{4},$$
there exists a unique value \( t_d \) of the delay for which the system undergoes a Hopf bifurcation. Along the boundaries of this domain, the Hopf bifurcation disappears through two different scenarios:

1. At \( \beta = \frac{\lambda^2}{4} \) and \((g - 1) t_0 = 1\): The system undergoes a Bogdanov-Takens bifurcation.\(^2\) The Hopf bifurcation curve tangentially reaches the saddle node bifurcation, and at this point, in particular, \( p_- = p_+ \).

2. At \( g = 1 - \frac{\sqrt{\lambda^2 - 4\beta}}{2\lambda} \), the delay associated with the Hopf bifurcation escapes the relevant boundaries by becoming either negative \((k = 0)\) or infinite \((k \neq 0)\).

Moreover, in order to show that the periodic orbits associated with the Hopf bifurcation are stable, we investigated the first Lyapunov exponent at the Hopf and Bogdanov-Takens bifurcations (see appendix D). We derive a very complex, but explicit, formula for this coefficient as a function of the parameters. In the case \( g = 1 \), several terms disappear and we show rigorously in section D.2 that the bifurcation is always supercritical. In the general case, the complexity of the formula prevents it from handling it analytically. We extensively evaluated this expression numerically (see Figure 12) and found that this coefficient is negative at all points tested, leading to the conjecture that the bifurcation is supercritical in the whole parameter space; thus, associated periodic orbits are stable.

In order to characterize the changes of stability of \( p_+ \), we study the linearized system,

\[
\frac{dx}{ds} = (g - 1)x(s) - \left[(g - 1) + \frac{\sqrt{\lambda^2 - 4\beta}}{\lambda}\right]x(s - t_0),
\]  

(3.5)

where we used the notation \( x(t) = p(t) - p_+ \). The dispersion relationship is obtained when looking for solutions of the form \( Xe^{\zeta t} \):

\[
\zeta = (g - 1) - \left[(g - 1) + \frac{\sqrt{\lambda^2 - 4\beta}}{\lambda}\right]e^{-\zeta t_0}.
\]  

(3.6)

The possible Hopf bifurcations correspond to purely imaginary values of \( \zeta = \pm i\omega \) (fixing, for example, \( \omega > 0 \)). Equation 3.6 can then be solved

\(^2\)The linearized equation at this point, \( \dot{u} = g(u(s) - u(s - t_0))\), is very close to the general case investigated by Faria and Magalhaes (1995a). The developments of that paper shall allow proving an analogous result here.
Figure 3: Time delay at the Hopf bifurcation for a fixed value of the parameter $\lambda = 4$. (A) Time delay (for $k = 0$) as a function of the parameters $\beta$ and $g$. (B) Sections of panel A as a function of $\beta$, along $g = 0.8$ (solid line), $g = 1$ (dashed line), and $g = 2$ (dotted line). (C) Sections of panel A as a function of $g$, along $\beta = 0.99\lambda^2/4 = 2.23$ (solid line) and $\beta = \lambda - 1 = 3$ (dotted line).

equating the real and imaginary part. Indeed, one obtains

$$\omega = \left[ \frac{\sqrt{\lambda^2 - 4\beta} \left( \sqrt{\lambda^2 - 4\beta} + 2\lambda(g-1) \right)}{\lambda^2} \right]^{1/2}$$

(3.7)

$$t_0 = \tan^{-1}\left( \frac{\omega}{g-1} \right) + 2k\pi$$

(3.8)

with $k = 0, 1, 2, \ldots$ These equations hence provide values of the parameters $(\lambda, \beta, g, t_0)$ related to possible Hopf bifurcations (see Figure 3). In particular, it is easy to see that the conditions the parameters have to satisfy to allow for Hopf bifurcations at $p_+ < 1$ (then for $\omega$ to be real and nonvanishing) are given by the announced set of relationships 3.4.

4 Results

We have introduced a mathematical dynamical model aimed at reproducing the dynamics of the emotional balance EB. We have investigated its dynamics, and specifically its equilibria and their stability. The existence of these equilibria depends on the way the individual processes the emotions and, in particular, the shape of the map $q_P$ modeling the way emotions...
are integrated (see Figure 4). This map depends on the parameter $\beta$ that
controls in particular the sharpness of emotional integration: the larger $\beta$
is, the more sensitive to $EB$ the affect integration.

The bifurcation diagram displays a clear hysteresis phenomenon: there
exists a range of $\beta$ for which the system displays two stable equilibria,
normal and depressed, and transitions between these two arise sharply.
The patient may thus switch from a normal state to a depressed state (resp.
from a depressed to a nondepressed state) if many negative (resp., positive)
events occur. As positive and negative inputs return to normal, the patient
will remain in the state he or she has reached, and it is then very hard to
escape this equilibrium without any external intervention. This is where
the therapy takes place.

In the following sections, we specifically describe the therapies used in
the treatment of JR and the associated evolution of its emotional balance.
This leads us to propose a way to integrate these therapies into our math-
ematical model. We then investigate the resulting system, with a specific
focus on the question of how to stabilize patients in a normal or optimal
state.

Figure 4: (A) Solid lines represent the sigmoid functions $q_p(EB)$ (blue) and
$q_N(EB)$ (red) used for simulations and analytical results. Dotted lines rep-
resent the corresponding functions when evaluated at a translated $EB$ value
($EB \rightarrow EB + 0.2$ in this example), illustrating the mathematical implementation
in the model of therapy 1 (coping-focused therapy) as discussed in section 4.2.2.
(B) Bifurcation diagram as a function of parameter $\beta$. Modifications in the shape
of the map as $\beta$ is varied are shown in the inset: the map becomes sharper (up)
and is globally scaled (bottom). Red solid lines correspond to a stable fixed
point, while the black dashed line indicates an unstable fixed point. This un-
stable point is the separatrix between depressed and nondepressed states (blue
arrows). The gray-shaded zone corresponds to the range of parameter $\beta$ investi-
gated within simulations shown in Figure 8 and discussed in section 4. For both
panels, parameters are fixed as $\alpha = 10$, $c = 0.2$, $\lambda = 4$, $\tau_p = \tau_N = 10$. In panel A,$\beta = 2.7$, corresponding to the vertical black solid line in panel B.
4.1 The Three Phases of Depression Recovery. We will see that the dynamics of the emotional balance during therapy can be well described by three phases, each characterized by local noise levels and the presence of oscillations (see appendix B):

- Phase 1: Variability, defined by large EB fluctuations
- Phase 2: Stability, defined in contrast by low levels of EB fluctuations
- Phase 3: Oscillations, defined by the presence of statistically significant periodic oscillations of EB

4.2 Therapy 1: Coping-Focused Therapy. We describe here the evolution of the EB during the course of JR’s first treatment, before modeling these dynamics.

4.2.1 Experimental Data. The dynamics of JR’s emotional balance during the coping-focused treatment presents two consecutive sequences of phase 1 (variability) and phase 2 (stability; see Figure 5). The first phase 1 (noted as 1a), which lasted five weeks, shows a rapid increase in emotional balance from a negative ratio ($EB = 0.34$) to a Successful Coping ratio ($EB = 0.65$), and despite high variability, it achieves an overall mean ratio for the phase of $EB = 0.49$, close to the Conflicted set point of $EB = 0.50$ associated with mild psychopathology. This rapid initial increase of the emotional balance indicates the likelihood of successful cognitive therapy, as established in Tang and DeRubeis (1999). The patient then stabilized at the Successful Coping ratio of $EB = 0.62$ and sustained this ratio during four weeks of phase 2 (noted 2a). Then another phase 1 (noted 1b) started afresh with the patient dropping into a Conflicted ratio ($EB = 0.48$), but rapidly rebounding to a Positive ratio of $EB = 0.72$, associated with normal (but not optimal) functioning. Finally, he stabilized at this level in another occurrence of phase 2 (noted 2b). At this stage, the emotional balance reached the values $EB = 0.72 \pm 0.05$. We tested if the segmentation obtained by this trajectory analysis is statistically significant. To this end, we used the classical $F$-test of equality of variance and obtained $F = 13.017$, which corresponds to a $p$-value of $p < 0.01$, thus validating that the fluctuations in the first and second phases are significantly different.

The therapeutic content and the life events corresponding to this dynamics are summarized in section E.1.

4.2.2 Mathematical Model. Depression induces an imbalance in the perception of positive and negative events that the coping-focused therapy aims at counterbalancing. In the depressed state of low EB, the effect of positive events becomes very weak while negative events become prominent ($q_p(EB)$ small and $q_N(EB)$ large).

The coping-focused therapy intends to reestablish a balance in the affects perception by helping the patient savor positive events and stop focusing
Figure 5: Therapy 1. (A) EB segmented into phases 1 and 2. Red = raw emotional balance data; Black = sliding mean; blue = standard deviation (multiplied by two for legibility). (B) Sliding Lomb-Scargle (LS) transform shows no significant oscillatory activity (the transform is at a 0.50 significance level for both phases) and (C) nonsliding LS transform on the whole phase 1a (purple) and 2b (blue).

on negative events. This brings the values of $q_P$ and $q_N$ back to those corresponding to the normal range, thus bringing the patient in the region where the normal state is the only stable state.

In order to emulate these effects in our model, we consider that the therapy alters the reduced impact of a positive event, $q_P(EB)$ by an enhanced amplitude $q_P(EB + a)$ with $a > 0$ (see Figure 4), and the excessive impact of negative affects $q_N(EB)$ by a level corresponding more to normal levels, $q_N(EB + a)$. Numerical simulations show that even small shifts $a$ are sufficient to induce the desired changes in the dynamics and bring a depressed patient to normal states. For instance, Figure 7 shows simulation results with $a = 0.2$ applied during a week to a month consistently results in a stabilization of the patient’s emotional state. Of course, stopping therapy at this point (by resetting $a$ to 0 and therefore bringing back $q_P$ and $q_N$ to their original shape) does not destabilize the patient’s emotional state.
that remains in the normal range, since the nondepressed state is a stable attractor. Important perturbations such as an accumulation of negative events can nevertheless destabilize this attractor and bring the patient back to depressed levels, as we show in section 4.4.

4.3 Therapy 2: Dynamic Affect-Focused Therapy. Three years after the first therapy, JR relapsed and was administered a mixed coping-focused, affect-focused therapy. We describe the therapeutical content and evolution of the emotional balance in appendix A. The time course essentially concatenates the typical features seen in therapies 1 and 2. We concentrate here on a third instance of treatment that was essentially psychodynamic, after the patient relapsed five years after the mixed therapy and returned to the clinician with an emotional balance at the lowest level. We again start by discussing the time course of the emotional balance during this therapy before discussing its modeling.

4.3.1 Experimental Data. The dynamic-focused therapy resulted in a trajectory that differed from the other treatments. The initial stage lasted longer than the previous treatments (five months) and presented a highly variable but globally increasing emotional balance that gradually reached the normal ratio of $EB = 0.72$ (see Figure 6). The emotional balance stabilized in phase 2 (note the sharp decrease in variability) and smoothly climbed to a ratio of $EB = 0.79$, close to the optimal set point of $EB = 0.81$. Phase 3 followed with moderate variability, intermediate between phases 1 and 2. The emotional balance began oscillating smoothly between the normal ($EB = 0.72$) and optimal ($EB = 0.814$) ratios with a period cycle of seven weeks and a high statistical significance ($p < 0.01$). This pattern was sustained for seven months during treatment and later confirmed at the six-month and one-year follow-up assessments (see Figure 6).

The Brown-Forsythe test for equality of variance on the segmentation obtained finds a phase 1 with a standard deviation of 0.0142 (sample size, 22) and a phase 2 with a standard deviation of 0.020 (sample size, 11). The statistical test confirms that the difference in variance between the two phases is significant ($F$-test, $F = 6.36$; p-value, $p = 0.017$).

The therapeutical content and the life events corresponding to this dynamics are provided in section E.2.

4.3.2 Mathematical Model. The dynamic therapy encourages the patient to reexperience and reconsider the origins of his troubles so he can put current events in perspective. In other words, the therapy will lead the patient to consider his emotional balance in a broader perspective. From the mathematical model point of view, this motivates us to consider a nonvanishing parameter $t_d$ in equation 2.2 that may vary and, in particular, increase throughout affect-focused therapy.
Figure 6: Emotional balance trajectory for JR’s third period of treatment (dynamic-focused therapy). (A) Depicts a single sequence of phase 1–phase 2–phase 3. (B) Lomb-Scargle transform identifies a prolonged phase 3 of oscillations with a six-week period sustained until the end of therapy. (C) Nonsliding LS transform on the whole phase 1 (purple), phase 2 (blue), and phase 3 (black) that presents a statistically significance level ($p < 0.01$).

The net effect on the dynamics is the appearance of oscillations around a stable fixed point, for $t_d \geq t_{d}^{c}$, where $t_{d}^{c}$ is a critical value determined by all the other parameters in the model. From a mathematical point of view, this corresponds to a Hopf bifurcation, as discussed in detail in section 3 and appendix D. This can be seen in the two examples shown in Figure 7. After the period of coping-focused therapy (gray zone) that allowed a progression from the lower fixed point to the upper one, when the delay is turned on, the emotional balance value oscillates around that value. Notice how by changing the parameters (see Figures 7A and 7B), oscillations can be more or less evident and distinguishable from the fluctuations due to the random positive and negative affects.
Figure 7: Numerical simulations modeling the dynamics of emotional balance during 5 years. After an initial period of 3 months, with low EB initial conditions, we administer a 3-month period of therapy 1 (gray shaded zone). (A) Four different cases are discussed. Blue and red (mostly hidden by the magenta) lines correspond to the different treatment, without or with therapy 2 ($t_d = 0$ or $t_d = 10.5$). Cyan and magenta correspond to the same cases when a stress period (green shaded zone, number of negatives equal to three times the usual one, fixed by $\lambda'$) of 10 days is taken into account after 2.5 years. The parameters of the model of equation 2.2 are fixed as $\alpha = 10$, $\beta = 2.7$, $c = 0.2$, $\lambda' = 4$, $\tau_P = \tau_N = 20$, $g' = 13$. (B) A different dynamics when parameters are changed and an equivalent stress period is taken into account: $\alpha = 1.5$, $\beta = 2.5$, $c = 0.04$, $\lambda' = 20$, $\tau_P = \tau_N = 14$, $g' = 11$. The blue and red solid lines respectively correspond to delays $t_d = 0$ and $t_d = 10.5$ days.

4.4 Stability. The models predict and reproduce with accuracy the time course of the emotional balance of a patient undergoing therapy. We have noted that more than five years after the end of the last affect-focused treatment, the patient did not relapse into depression, which may indicate that therapy 2 leads to a more stable state. This is a counterintuitive prediction: indeed, the emotional balance oscillations visible in the experimental data tend to indicate a destabilization of the normal state and also lead the individual to lower emotional balances. Moreover, if the advantage brought by the first coping-focused therapy is clear, it is much less so in the second treatment. In fact, as shown, for example, by the blue and red lines in Figure 7A or by the left part (from 0 to 2.5 years) of Figure 7B, the two possibilities (only therapy 1 or both therapies) can have very similar trajectories and the first therapy alone stabilizes the nondepressed state.

In order to test whether therapy 2 can have an impact on the stability on the nondepressed state, we incorporated in the model a stress period in which the frequency of negative events is increased. Two instances are
Figure 8: Statistical numerical analysis of the effect of therapy 2 on the resistance to a 1-week stress period. Fraction of final states near the normal stable fixed point, for different values of the parameter $\beta$, as a function of $j$, the ratio between the average number of negative events during the stress period and that of the normal one ($\lambda'$). The scenario studied is the same as in Figure 7A, with all other parameters fixed at $\alpha = 10$, $\beta = 2.7$, $c = 0.2$, $\lambda' = 4$, $\tau_P = \tau_N = 20$, $g' = 13$.

provided in Figure 7 (cyan and magenta lines for panel A and blue and red for panel B). In our simulations, we consider a period of 10 days in that the negative events have a rate of occurrence that is three times larger than the frequency of positive events. Such a stress period significantly decreases the emotional balance in the depression range. But the response to this stress period is very different when therapy 2 has occurred. The delays and, particularly, the presence of oscillations seem in fact to stabilize the normal state by producing increased resistance to drifting away from an originally stable state. And the greater the delay $t_d$, the longer and stronger period of exceptional negative events can be overtaken. In the same way, the specific shape of the sigmoid function, equation 2.3, can determine the limits toward which this therapy also becomes inefficient. In order to have an idea of this effect, we have simulated several trajectories from random depressed initial conditions by varying the parameter $\beta$ of equation 2.3 and a parameter $j = \frac{\lambda'_{Stress}}{\lambda'}$, where $\lambda'_{Stress}$ ($\lambda'$) is the rate of negative processes during the stressful (normal) period. The results are shown in Figure 8.

For each value of the parameter $\beta$ and $j$, we have considered a set of 50 numerical simulations of random Poisson processes and plotted the number of final states around the positive state-of-mind value of $EB$. The stabilizing effect of the second therapy is therefore manifest, especially for small values of $\beta$ and high values of $j$, the extreme situations (in terms of patient and in terms of negative events) in which depression is more likely or, in other words, when the basin of attraction of the lower stable point is more important than that of the upper one.
Discussion

This letter introduced a theoretical framework to mathematically model and empirically investigate the complex interplay of positive and negative affects in normal and depressed situations. As an illustrative example of the model’s potential, we provided a quasi-experimental study of a recurrently depressed patient undergoing multiple treatments, demonstrating that the most effective therapy progressed from an initial period of extreme mood fluctuation to a smooth, enduring oscillation in emotional balance. The model is based on a few basic observations of reactivity to positive and negative life events and on how these vary with the emotional state of the individual.

The findings build on a research tradition demonstrating the effects of positive and negative events on depressed individuals (Gotlib, Krasnoperova, Yue, & Joormann, 2004; Kuiper & Derry, 1982) and more recently delineating the differential temporal course of sustaining positive and negative affect in normal and depressed persons (Horner et al., 2014). Our contribution is to go beyond the classical static approach of emotional ratios. We showed that not only ratios but also fluctuations convey information on the emotional state and the possible outcome of a therapy.

At a critical juncture when mathematical modeling of psychological dynamics has come under attack (Brown, Sokal, & Friedman, 2013), our theoretical model offers a viable alternative for understanding mathematically the dynamics of emotions that can be fitted to experimental data. From the model naturally coexist two stable states that we associate to normal and depressed ratios. Fluctuations and therapy effects were modeled to understand how a depressed individual can stabilize to a normal state. This was not previously addressed in the context of psychotherapy, which has focused more on cross-sectional data to demonstrate, for example, that psychotherapy clients progressed from a specific negative balance prior to treatment to a normal or optimal balance at posttreatment (Schwartz, 1997; Schwartz et al., 2002). In order to confront the model with actual data, we investigated the EB trajectories during treatment for one individual and showed that three distinct phases characterize its dynamics: variability, stability, and oscillation. Moreover, these phases correlated in meaningful ways with the type of therapy, stage of treatment, critical external events, and inner states of the client. We found that a more intensive and effective treatment yielded end-state oscillations in emotional balance and reasoned that these oscillations may be the source of resilience to depression after stressful events.

In order to investigate this hypothesis, we used extensive numerical simulations of the model and quantified that in regimes during which the emotional balance shows low-magnitude oscillations, the nondepressed state is more stable and resists periods of intense stress. In the model, these oscillations are related to the fact that the individual evaluates his
current state not only at the present time, but within a longer window of ongoing history. These considerations suggest that phase 2, characterized by nonsustained periods of relatively high stability, can be designated as a stage of consolidation when prior gains of the therapy are being integrated rather than an ultimate treatment outcome. Overall, the stability phase was not maintained for very long and was terminated by the occurrence of positive or negative internal states or life events. When learning a new skill, a novice may initially prefer to maintain a more fixed set of circumstances until acquiring the confidence and flexibility that allows engaging more varied situations.

In contrast to the stability phase, the oscillation phase, designated as resilience, is associated with well-being and flourishing. This is supported by the patient’s psychological progress to an alleviation of mood disorder, enhanced interpersonal sensitivity, and heightened spiritual awareness. Since the oscillations of the emotional balance are bounded by the normal and optimal set points, no striking change was observed in the patient’s state during these smooth, moderate shifts in mood. This capability to oscillate allows one to maintain a positive state while flexibly reacting to both good and bad events. This adaptive property can be likened to a reed that remains intact despite severe wind. Unlike the thicker but rigid branches of a tree that might snap, the thin and seemingly fragile reed will bend and oscillate with the breeze. It is this flexibility that enables it to smoothly survive life’s vicissitudes.

To the best of our knowledge, our study is the first to mathematically demonstrate oscillation in affect as a posttherapy outcome. The results join the increasingly observed phenomenon of normally occurring damped oscillations in mood during natural stressors (Bisconti et al., 2004; Chow et al., 2005), as well as in biological systems such as cardiovascular heart rate and brain rhythms (Strogatz, 2003). The use of dynamic models to study the evolution of cognitive and affective states is relatively new, so it is likely that different trajectories will define diverse disorders and processes such as bereavement, unnatural trauma, or change during psychotherapy. The data from the therapies presented here indicate that during treatment, emotions do not evolve as a transformation in only the magnitude of the emotional balance, but rather that progress through distinct phases of variability early in therapy, (nonoscillating) stability during midtherapy, and oscillation only at the final stage of intensive therapy.

Many questions remain about what constitutes an optimal trajectory for various life events. For example, Stroebe and Schut’s (1999) dual process model suggests that “optimal adjustment” in coping with stress is associated with a moderate level of oscillation between loss (negative) and restoration (positive) responses. But Bonanno, Goorin, and Coifman (2008) contend that chronic and dysfunctional loss reactions are associated with “more extreme and unregulated forms of oscillation” and more enduring negative affect (p. 803). These approaches differentiate between stability.
and oscillation, but do not distinguish true oscillation from random fluctuation or what we have termed “variability” (Coifman, Bonanno, & Rafaeli, 2007). We believe that some confusion may be caused by not distinguishing between the nontechnical meaning of oscillation as “wavering between conflicting courses of action” and its meaning in physics as “an effect expressible as a quantity that repeatedly and regularly fluctuates above and below some mean value” (Stein & Urdang, 1966). If we limit our scientific use of the term oscillation to its meaning in physics, then Bonanno et al.’s (2008) use of the term refers to fluctuations. A more precise rendering based on this clarification suggests that a moderate level of oscillation represents the optimal trajectory and that more extreme and unregulated fluctuations or variability, not necessarily larger oscillations, are associated with dysfunctional adaptation.

In sum, this study has quantified the fact that a high emotional balance or positive ratio alone is not always a sufficient indicator that treatment has achieved a sustainable, optimal result. Psychological resilience and resistance to relapse depend on the ability to sustain this high level of positive mood, and the presence of oscillations in the system provides the flexibility needed to accomplish this. Presumably the person in an oscillating state has cultivated the necessary strategies to monitor and regulate his or her state so it remains optimally balanced. The mathematical tools introduced here allow a more precise assessment of the levels and dynamics of affect that can be used in future studies that delineate optimal treatment outcomes and predict the likelihood of relapse.

The strength of this approach that allows a detailed analysis of the complexity of the change process also brings corresponding limitations. The question that arises, as in any idiographic study, is the universality of this discovery. Although our findings statistically demonstrate an oscillatory phenomenon in the end-state functioning of a highly successful treatment, these observations need to be further investigated on additional patients and with different therapists. Since the dynamic-focused treatment temporally followed two less intensive therapies, the presence of oscillation in affect and general flourishing cannot be conclusively attributed to the greater depth and duration of the final treatment, as opposed to its position as the culmination of a long process of self-development. Although the current data are consistent with this conclusion, only group design studies can confirm the connection between type of therapy and the generation of end-state oscillation in affect. Larger-scale studies need to recruit more individuals and devise simpler, more accessible data recording systems. The timescale of the oscillations (on the order of 7 weeks) represents a practical limitation for large-scale experimentation that complicates further analysis of this “macro” oscillatory phenomenon.

This initial, intensive study raises new questions with implications for positive and classical psychology about the dialectical effects of positive and negative affects considered separately: Are the oscillations driven by
positive or negative affects, or are they an emerging property of both? Are
phase switches induced more by positive versus negative states? Do differ-
ent content categories of positive versus negative events (e.g., bereavement,
job loss, relationship enhancement, spiritual uplifts) have differential im-
 pact on phase transitions in depressed and normal persons? We encourage
further intraindividual studies to delineate how variability, stability, and
oscillation in human systems evolve during different stages of therapy
with different disorders and personality types. The study reported in this
latter provides an illustrative example of how using a dynamical approach
and mathematical tools within a theoretical framework of positive-negative
balance can illuminate the process of cognitive and affective change during
psychotherapy.

Appendix A: JR Mixed Therapy

Three years after the first (coping-focused) therapy, JR came back to con-
sultation. His therapy started with a coping-focused phase followed by an
affect-focused phase, and this is why we refer to this therapy as mixed.

When JR returned after a three-year hiatus, he began with an emotional
balance $EB = 0.64$, placing him within the subnormal but Successful Coping
range. Although not clinically depressed, he was struggling with negative
mood, work inhibition, worry, and sleep disturbance (see Table 1). His tra-
jectory was moderately variable (phase 1a), progressing gradually to the
normal range, at which point he entered a prolonged period of reduced
variability and stabilization around the normal ratio of $EB = 0.72$ (phase
2a). As phase 2a progressed, he entered a phase that evinced the highest
variability of the treatment and some of the lowest emotional balances that
fluctuated between the Conflicted and Successful Coping ranges, with oc-
casional peaks into the low Normal range. During this four-month phase,
he displayed no oscillation. The variability then dropped to its lowest level,
and the emotional balance stabilized in phase 2b for one and a half months.
Finally, he completed the iterative process by (presumably) entering phase
3 for the first time. During this phase lasting six and one-half months, the
variability increased to a moderate level, and the emotional balance began
oscillating between the Optimal ($EB = 0.81$) and Superoptimal ($EB = 0.88$)
levels with a 7-week period. The premature end of therapy interrupted

Table 1: Emotional Balance Data for JR: Mixed Therapy.

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Figure 9: Emotional balance trajectory for JR’s second period of treatment (mixed therapy). (A) Depicts two iterations of phase 1–phase 2 and a trend toward a phase 3 sequence (see panels B and C). (B) Note that the short duration of the recorded oscillating phases prevents the Lomb-Scargle transform to reach significant levels. (C) Nonsliding LS transform on the whole phase 1b (purple), phase 2b (blue), and phase 3 (black).

the two periods of putative, visually detected oscillations. Thus, the Lomb-Scargle transform, though presenting a clear peak compared to the rest of the signal (see Figure 9), did not achieve significance ($p = 0.17$). The Brown-Forsythe test corresponding to this segmentation shows a variance of 0.0249 for phase 1 (merged data from 1a and 1b) and 0.0027 for phase 2 (merged data from 2a and 2b) that yields a significant difference ($F$-test: $F = 5.79$, $p = 0.025$), thus statistically validating the phase segmentation for this treatment. The analysis of JR’s data is exemplary of how visual inspection can be used with statistical tools. When analyzing the full data set, our automatic segmentation algorithms failed to segment the therapy into a single phase 1 (variability) followed by phase 2 (stability), which is confirmed by the visual inspection of the data. Instead, the data are characterized by a
phase of high variability, followed by a second phase where the signal is not oscillating but the variability is slowly increasing to a standard deviation between typical values of phase 1 and phase 2. This sequence is then followed by another variability phase, a second stability phase of short duration, and finally a brief period of oscillation. We are therefore able to visually detect the segmentation proposed in Figure 9. This visually detected segmentation was tested and statistically validated using the Brown-Forsythe indicator.

The therapeutic content and the life events corresponding to this dynamics can be summarized:

- **Phase 1a.** Therapy focused initially on a review of cognitive-behavioral coping strategies. After three months, the client explored interpersonal themes and engaged in emotional and dream expression (e.g., themes of food, need for unconditional approval, and disturbing images of a primitive nature), but no deeper psychodynamic or dream analysis was done. The transition from phase 1a to 2a is marked by a stabilization in mood and self-image noted in the clinical log. Themes were emerging of oral deprivation and wishful thinking that others would “read his mind” so they could satisfy his needs.

- **Phase 2a.** The client no longer reported dreams during most of this phase. Apparently picking up on the themes from earlier dreams of oral (maternal) deprivation, the patient worked on childhood loss because of his mother’s depression and her current illness, as well as marital conflicts. The emotional and optimism balances reached an optimal level as he coped better with frustration and reduced his compulsion. Otherwise free of dream activity, at midphase he reported dreams in four successive sessions with themes of two dying people presumed to be his parents, childhood yearnings for attention from his self-centered mother, and fear of world destruction. After this flurry of dream work ceased, JR shifted to a prolonged focus on here-and-now issues of ambivalence in current peer relationships. The phase transition 2a to 1b, unlike the prior phase transition into a positive and stable state, regressed to an extended period of variability and low emotional balance. It was not preceded by a dream but was instead triggered by a verbal lashing from a colleague and the client’s awareness that his interpersonal insensitivity provoked conflict.

- **Phase 1b.** During this second phase of high variability, the client worked on current interpersonal conflicts and mounting anxiety, depression, and sleep problems caused by his realization that his lifelong, grandiose ambitions were unlikely to be realized. The transition from phase 1b to 2b was triggered during the last week of phase 1b when the client’s mood reached the subnormal but Successful Coping level of \( EB = 0.62 \) and he recalled a dream about his wife’s salary increase that made him feel diminished. Also, a critical event occurred that the client experienced as transformational. His son had a significant accident, but his survival and recovery led the client...
to become more attuned to the needs of his family (especially his wife), to slowing down his hectic pace, and softening his competitiveness and interpersonal brusqueness.

- **Phase 2b.** This stable phase of normal emotional balance \((EB = 0.72)\) finds the client engaging in less “name calling” when he makes mistakes, enjoying family vacations more because he is less self-centered and more flexible, and communicating better with his wife. Two weeks prior to the phase transition from 2b to 3, the client reported successive weeks of dreams. The first is of two dogs dying that the patient related to fear about the death of his aging parents and to conflicts with his mother as a “suffocating, amorphous and ill-defined problem.” The second dream is of a woman falling through a dam and his not being able to rescue her, reflecting his mother’s precarious moods and his inability to save her.

- **Phase 3.** Dream recall and processing continued throughout this final phase of oscillating affect. The clinical log notes decreased self-focus, increased ability to live in the moment, and better connection with his wife. Paradoxically, these improvements were accompanied by dreams of disconnection from his mother, perhaps reflecting work toward resolution. He was able to express his emotions more directly regarding grief about his grandmother’s recent death. For the first time, his wife commented appreciatively about his progress.

JR requested a May termination that appeared influenced more by the academic year than by his psychological state. Despite significant gains, the client had a dream revealing oral rage about maternal deprivation and parental inattention. Such recurrences of old themes when terminating therapy are not unusual, but the overall termination summary raised questions about the need to continue working on self-confidence, dependency issues, and interpersonal style, as well as marital counseling. Interestingly, his final emotional balance of \(EB = 0.74\) was near the normal (not optimal) ratio of \(EB = 0.72\), but his Happiness subscore was one-half lower than the other positive scales for Vitality and Friendliness. Thus, he achieved a normal affect balance, but was still not optimally balanced and remained deficient in happiness.

**Appendix B: Automatic Segmentation of Therapy Phases**

The emotional balance trajectory presents three phases, described by the concepts of emotional balance trend, local variability, and oscillation. Patients were instructed to complete the balance inventories weekly and freely choose the day of the monitoring between sessions, which made the data unevenly sampled in time. Missed sessions and periodic extended vacations further increased the irregularity in the data collection. The interval between two consecutive measurements for JR ranged from 3 to 27 days, with a mean of 8.6 days and a mode of 7 days, thus approximating weekly
ratings. In order to evaluate the dimensions of interest (trend, variability and oscillation), we used the following data analysis tools:

- The emotional balance trend was evaluated using a sliding mean. Specifically, at time $t$, this quantity is equal to the local mean of the emotional balance in a time window centered on $t$, with a range of 7 weeks, including the three previous and the three subsequent sessions. The choice of this window size was a compromise between the need to have sufficient points in the signal to compute a meaningful value and few enough points to track with sufficient sensitivity the trend of the signal. Note that the results obtained do not depend significantly on the choice of this time window.

- The fluctuation level is estimated from the signal by computing a sliding standard deviation (similar to the sliding mean).

- The automatic segmentation method distinguishing a high-variability phase and a low-variability (i.e., relative stability) phase is based on optimizing the $p$-value across the possible segmentations using the standard Brown-Forsythe test for equality (or homogeneity) of variance. If the resulting $p$-value of the Brown-Forsythe test is less than some critical value (e.g., 0.05), the obtained difference in sample variances is unlikely to have occurred based on random sampling; thus, the hypothesis of equal variances is rejected, and it is concluded that there is a difference between the variances in the population (Brown & Forsythe, 1974). The following automatic segmentation algorithm was used: Considering a sample of $N$ values $(x_i, i = 1, \ldots, N)$, we aim at detecting whether this sample is composed of two distinct subgroups characterized by different variances: phase 1 corresponding to $i = 1, \ldots, t$ and phase 2 corresponding to $i = t + 1, \ldots, N$. We want to find this time $t$ and check if the segmentation obtained presents a significant difference of variance. For each time $t$ considered, we perform the Brown-Forsythe test and compute the $p$-value. The segmentation time $t$ is chosen as the value that minimizes this $p$-value. The $p$-value corresponding to this optimal segmentation is then compared to our threshold (0.05) to assess the statistical significance of the segmentation.

- The presence of oscillations was assessed by computing a sliding local Lomb-Scargle transform on a neighboring window around the time point of interest (Lomb, 1976; Scargle, 1982). Because of the timescale involved in the oscillations (around 7 weeks) and the need to have around three cycles in a time window to assess the presence of oscillations, we chose a window of 20 weeks. The Lomb-Scargle transform derives from the classical Fourier transform that is widely used for signal analysis applications. It extends the Fourier transform to unevenly spaced data and aims at revealing the frequencies that are present in a signal. It is associated with statistical significance tests of the detected oscillation (see the details in appendix C). The significance level evaluated assumes that each frequency constitutes an independent test and corrects the significance level by the number of tests performed in order to control for type 1 error. The fact that the Lomb-Scargle
handles unevenly spaced data makes it the method most appropriate for our clinically derived data, and our ability to automatically compute significance levels provides an objective criterion for ascertaining the presence of oscillations in the signal. The sliding transform allows identifying the onset of significant oscillations in the signal in the time window considered and highlights a period when the signal presents significant oscillations in each time window. In order to fully assess the presence of significant oscillations throughout the identified segment of the signal, we compute a standard (nonsliding) Lomb-Scargle transform on the whole phase and derive from this test the actual statistical significance of these oscillations.

Note that visual inspection of a noisy signal in the raw data can sometimes produce the spurious impression that the signal is oscillating. The analysis of the amplitude of Lomb-Scargle transforms and the application of the present tests differentiate objectively the two phenomena (see appendix C).

Appendix C: The Lomb-Scargle Transform

Since the clinical protocol allowed patients to freely monitor their emotional balance between consultation sessions, the obtained assessments were not evenly spaced. In the case of irregularly sampled data, the classical Fourier transform (see Gottman, 1994; Ong & van Dulmen, 2006) fails to provide the frequency content of the signal. To handle such cases, we used the Lomb-Scargle transform, a very efficient method that was developed for the study of astrophysical data (Lomb, 1976; Scargle, 1982; Vanček, 1971). This method is based on the following principles we now make explicit. Consider that we observe a continuous phenomenon though a given scalar measurement $h$. The continuous time phenomenon produces a continuous time measure $h(t)$, but we only have access to a discrete set of $N$ values of this function sampled at unevenly spaced times $\{t_i, i = 1, \ldots, N\}$. For this set of $N$ measurements $\{h_i := h(t_i), i = 1, \ldots, N\}$, of mean denoted by $\bar{h}$, and of standard deviation denoted $\sigma$, the Lomb-Scargle transform performs a projection on sines and cosines evaluated only at times $t_i$ where data are actually measured. In detail, the Lomb normalized periodogram is defined by

$$
P_N(\omega) = \frac{1}{\sigma^2} \left\{ \frac{\left( \sum_{j=1}^{N} (h_j - \bar{h}) \cos(\omega(t_j - t)) \right)^2}{\sum_{j=1}^{N} \cos^2(\omega(t_j - \tau(\omega)))} + \frac{\left( \sum_{j=1}^{N} (h_j - \bar{h}) \sin(\omega(t_j - t)) \right)^2}{\sum_{j=1}^{N} \sin^2(\omega(t_j - \tau(\omega)))} \right\}, \quad (C.1)
$$
Figure 10: Lomb-Scargle transforms. (A) On oscillating data, the Lomb-Scargle transform presents a peak at a frequency $\omega$ related to the period of oscillations, and whose amplitude is related to the statistical significance of the observed oscillations (data of JR, phase 3; see section 4). (B) Nonoscillating data present a shuffled Lomb-Scargle transform with small amplitude, corresponding to low levels of significance. (For legibility, the scale of the two images is different.)

where $\tau$ is defined by the relation

$$\tau(\omega) := \frac{1}{2\omega} \arctan \left( \frac{\sum_{j=1}^{N} \sin(2\omega t_j)}{\sum_{j=1}^{N} \sin(2\omega t_j)} \right). \quad (C.2)$$

The amplitude of the transform $P_N(\omega)$ gives access to the oscillatory content of the signal. A peak in the transform at frequency $\omega$ indicates that the signal presents oscillations at this frequency, and the bigger the amplitude of the peak, the more significant the oscillations are. Oscillating signals present highly peaked transforms, whereas nonoscillating signals produce flat, generally noisy periodograms (see Figure 10).

Peaks are therefore related to oscillations and indicate the potential frequencies in a signal. The statistical significance of these oscillations can be rigorously evaluated under the assumption that the data are samples of a periodic signal perturbed by a gaussian white noise. This estimator has a closed form; a formula provides levels of $P_N(\omega)$ directly related to statistical significance levels of the observed oscillation. More precisely, the probability of a peak with amplitude $z$ to be a false alarm of oscillation detection can be written $P(> z) = 1 - (1 - e^{-z})^M$, where $M$ is the number of independent frequencies considered, usually chosen to be equal to $2N$ (i.e., twice the number of observations).

To evidence the appearance of oscillations in the course of treatment, we performed a sliding Lomb-Scargle transform (instead of a Lomb-Scargle transform on the full time series). This means that for each time $t$, we compute the Lomb-Scargle transform of the recorded data in a time interval...
Figure 11: Sliding Lomb-Scargle transform, 3D representation. Sliding Lomb-Scargle transform, on the function $f$ defined piecewise by $f(t) = \sin(\phi t)$ with $\phi = \omega_1 = 2\pi$ for $t \leq t_1$ and $\phi = \omega_2 = 4\pi$ for $t > t_1$. We can clearly see the transition at time $t = t_1$, from oscillations with frequency $\omega_1$ to oscillations at frequency $\omega_2$. Note the imprecision in the observed frequencies and the decreased amplitude of the transform at the transition, linked with the presence of multiple frequencies in the signal.

Appendix D: Reduction to Normal Form at the Hopf Bifurcation

The type of the Hopf bifurcation is characterized by the sign of the first Lyapunov exponent. This coefficient can be computed using reduction to normal form (or at least the computation of a few coefficients of this normal form). The theory for computing these coefficients is well developed in the case of delay differential equations, and relatively classical methods are available to perform these reductions (Faria & Magalhães, 1995b; Diekmann, van Gils, Lunel, & Walther, 1995; Campbell, 2009). We review this methodology below and instantiate it to our particular case. This will provide us with a complex but explicit formula for the coefficient, allowing for extensive evaluations.
D.1 General Derivation of the Lyapunov Exponent. We start by re-writing equation 3.2 as in the classical theory by recentering the system at the fixed point \( p_+ \) and separating between linear and nonlinear terms. We denote \( x(t) = p(t) - p_+ \). We have

\[
\frac{dx}{dt} = A_0 x(t) + A_1 x(t-t_0) + f(x(t), x_i(t-t_0)) \tag{D.1}
\]

with

\[
A_0 = g - 1 \\
A_1 = -(g - 1) - \frac{\sqrt{\lambda^2 - 4\beta}}{\lambda} \\
f(x, y) = \frac{\lambda (p_+ + y)^2}{1 + \beta(p_+ + y)^2} - (1 - \frac{\sqrt{\lambda^2 - 4\beta}}{\lambda})y. \tag{D.2}
\]

For our purposes, we will need only a few terms of the expansion of the nonlinear part \( f(x(0), x(t_0)) \) near the origin. In particular, we can write

\[
f(x, y) = B_2 y^2 + B_3 y^3 + O(y^4)
\]

\[
B_2 = \frac{4\beta^2 \left( 8\beta - 3\lambda \left( \sqrt{\lambda^2 - 4\beta} + \lambda \right) \right)}{\lambda^2 \left( \sqrt{\lambda^2 - 4\beta} + \lambda \right)^3}
\]

\[
B_3 = \frac{2\beta \left( \lambda^2 \left( \sqrt{\lambda^2 - 4\beta} - \lambda \right) - 2\beta \left( \sqrt{\lambda^2 - 4\beta} - 2\lambda \right) \right)}{\lambda^3}. \tag{D.3}
\]

The theory treats these delay equations as functional differential equations on the space of continuous functions. They thus see the linear part of equation D.1 as a linear operator. At the Hopf bifurcation, this operator has two eigenvalues with zero real part \( \pm i\omega \); one can compute eigenvectors and associated projectors to reduce the system on the center manifold associated with these eigenvalues. Here, this technique yields the following equation for the coordinates \( u \) of the system on the center manifold,

\[
\dot{u}(t) = B u(t) + F(u(t)), \tag{D.4}
\]

where \( F \) has no affine component at 0 and

\[
B = \begin{pmatrix} 0 & \omega \\ -\omega & 0 \end{pmatrix}.
\]
Figure 12: Scatter plot for the evaluation of the coefficient $\alpha_{\text{Lyap}}$ as a function of the delay $t_0$ at the bifurcation. One million points are distributed in the parameter space for $2.01 \leq \lambda \leq 20$ and $g \leq 10$. A zoom of the region near the origin is shown in the inset.

Following the standard approach in center manifold theory, we assume that $F$ can be expanded in power series:

$$
\begin{align*}
\dot{u}_1 &= \omega u_2 + c_{20}^1 u_1^2 + c_{11}^1 u_1 u_2 + c_{02}^1 u_2^2 + c_{30}^1 u_1^3 + c_{21}^1 u_1^2 u_2 + c_{12}^1 u_1 u_2^2 + c_{03}^1 u_2^3, \\
\dot{u}_2 &= -\omega u_1 + c_{20}^2 u_1^2 + c_{11}^2 u_1 u_2 + c_{02}^2 u_2^2 + c_{30}^2 u_1^3 + c_{21}^2 u_1^2 u_2 + c_{12}^2 u_1 u_2^2 + c_{03}^2 u_2^3,
\end{align*}
$$

The first Lyapunov coefficient is given by

$$
\alpha_{\text{Lyap}} = \frac{1}{8} \left( 3c_{30}^1 + c_{12}^1 + c_{21}^2 + 3c_{03}^2 \right) - \frac{1}{8\omega} \left( c_{11}^1 (c_{20}^1 + c_{02}^1) - c_{11}^2 (c_{20}^2 + c_{02}^2) - 2c_{20}^1 c_{20}^2 - 2c_{02}^1 c_{02}^2 \right). \quad (D.5)
$$

Following the method clearly outlined in Campbell (2009), we obtain an explicit expression depending only on the parameters $\lambda$, $\beta$, and $g$ of the model of equation 3.2, using Mathematica. This very long formula (it would take more than a page to write up) would be difficult to handle analytically, except in the particular case of $g = 1$ treated in section D.2. The formula is, however, very useful for numerical evaluations. In Figure 12, we display the result of extensive numerical evaluations of the Lyapunov coefficient for 1 million points uniformly distributed in the $(\lambda, \beta, g)$ parameter space, for $2.01 \leq \lambda \leq 20$, $1.01 \ast (\lambda - 1) < \beta < 0.99 \ast \left( \frac{\lambda^2}{4} \right)$, and $1 - \sqrt{\frac{\lambda^2 - 4\beta}{2\lambda}} < g \leq 10$, as a function of the delay $t_0$ at the bifurcation (for $k = 0$). The boundaries for the coefficient $\beta$ have been chosen in order to avoid numerical errors and
instabilities (see section 3). All values obtained are negative, indicating that in the parameter region of equation 3.4, the Hopf bifurcation is supercritical.

D.2 Case $g = 1$. The formulas obtained for the curve of Hopf bifurcations and the Lyapunov coefficients appear relatively complex at this level of generality. We make it explicit in the specific case $g = 1$.

The constraints of equation 3.4 are satisfied as long as

$$\lambda > 2, \quad 0 < \gamma = \sqrt{\lambda^2 - 4\beta} < \lambda - 2. \quad (D.6)$$

In this case, the differential equation depends only on the delayed variable

$$\frac{dp}{ds} = \left( -1 + \frac{4\lambda p_d}{4 + (\lambda^2 - \gamma^2)p_d^2} \right) p_d; \quad (D.7)$$

the positive stable fixed point is now given by

$$p_+ = \frac{2}{\lambda - \gamma}; \quad (D.8)$$

and the dispersion relation reads

$$\zeta = -\frac{\gamma}{\lambda} e^{-\zeta t_0}. \quad (D.9)$$

In this case, the corresponding frequencies (substituting $\zeta = \pm i\omega$, $\omega > 0$) and delays associated with the Hopf bifurcation are

$$\omega = \frac{\gamma}{\lambda}, \quad (D.10)$$

$$t_0 = \frac{(4k + 1)\pi}{2\omega} = \frac{(4k + 1)\pi\lambda}{2\gamma}, \quad k = 0, 1, 2, \ldots. \quad (D.11)$$

The equations for the Lyapunov coefficient largely simplify, and an easy formula can be written:

$$\alpha_{Lyap}^{g=1} = \frac{1}{80(4 + \pi^2)\gamma^3\lambda^3}(\lambda - \gamma)^3(2(7\pi(4k + 1) - 8)\gamma^3 - 30\pi(4k + 1)\gamma^2\lambda$$

$$+ 3(4 - 11\pi(4k + 1))\gamma\lambda^2 + (4 - 11\pi(4k + 1))\lambda^3)). \quad (D.12)$$

In particular, for $k = 0$, it easy to see that

$$\alpha_{Lyap}^{g=1} = a_0(\lambda - \gamma)^3(\gamma - a_1\lambda)(\gamma^2 - 2a_2\gamma\lambda + (a_2^2 + a_3^2)\lambda^2), \quad (D.13)$$
Table 2: Emotional Balance Data for JR, Therapy 1.

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Table 3: Values of the EB during the Course of Therapy 2.

<p>| | | | | | | | |</p>
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<td>0.80</td>
<td>0.81</td>
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</tr>
<tr>
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<td>0.72</td>
<td>0.91</td>
<td>0.78</td>
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</table>

with \( a_0 = \frac{(7\pi - 8)}{40(4 + \pi^2)} \gamma \), \( a_1 \simeq 4.2 \), \( a_2 \simeq -0.42 \), and \( a_3 \simeq 0.29 \). In particular, imposing the constraints of equation D.6, it is straightforward to see that \( (\lambda - \gamma) > 0 \), \( (\gamma - a_1 \lambda) < 0 \), and therefore \( \alpha_{g=1} < 0 \). Similar results are obtained for the general \( k \neq 0 \) case.

### Appendix E: Experimental Data and Logs

The emotional balance values corresponding to the three therapies are provided for each therapy in Tables 2, 1, and 3, together with an analysis of the therapeutical content associated with each EB phase.

#### E.1 Therapy 1.

Therapy 1 was characterized by two repetitions of phase 1–phase 2, progressively leading the patient to a normal level of EB. The therapeutical log provides information on event that may be associated with the phases and the switches between those:

- **Phase 1a.** The initial increase in emotional balance was attributed in our earlier study (Schwartz, 1997) to the process of “remoralization” that often characterizes the first stage of treatment (Howard, Lueger, Maling, & Martinovich, 1993). The transition from phase 1a to 2a was not triggered by any identifiable critical events, but with a change in the patient’s attitude. Specifically, the patient started to constructively analyze the source of his problems rather than ruminate about them.

- **Phase 2a.** During this phase, the patient stabilized his affect by applying anxiety management techniques, communication skills, and cognitive strategies to reduce worry. No dreams were reported. Mounting job pressures and interpersonal conflicts at work triggered the transition from phase 2a to 1b.

- **Phase 1b.** The EB shows a sudden destabilization that did not appear to be triggered by external life events. Therapy indicates that this
stage was characterized by growing self-confidence resulting from cognitive restructuring of his attitudes toward his critical father and teasing from peers.

- **Phase 2b.** The EB stabilized at a normal level \( (EB = 0.72) \) as JR consolidated his new coping strategies and improved mood.

Follow-up assessments conducted at 3, 4, and 5 months showed a sustained, normal emotional balance, but with an increasing standard deviation, probably associated with destabilization of the steady state reached at the end of treatment and a possible return to phase 1.

**E.2 Therapy 2.** Therapy 2 was characterized by a progressive evolution of the EB dynamics through phases 1, 2, and 3. The following analysis of the therapeutical content provides information on life and therapy events during the phases and at the switch:

- **Phase 1.** The client engaged in more emotional expression than in earlier treatments, with intense sobbing about his mother’s death and not succeeding at the level he thought she expected of him. A proliferation of emotionally charged dreams occurred with themes of maternal deprivation, conflict with father, and awareness of narcissistic strivings to succeed. As can be seen in Figure 6, this phase was characterized by extreme variability in emotional balance, with the sliding mean showing a gradual, steady increase. Several weeks prior to the transition from phase 1 to 2, the client worked through dreams, sometimes twice weekly, that progressed from female figures who were inconsistently present and associated with bad food to recovery themes of eating steak (good food) to get into shape. JR made progress in shifting from external and uncontrollable sources of self-esteem to becoming more self-validating, yielding a more stable sense of self. Several weeks prior to the phase transition, he experienced stressful events, including ambivalence about his father’s remarriage (which he did not attend) that led to a precipitous drop in emotional balance to \( EB = 0.38 \). A dramatic surge in optimism to an optimal level \( (EB = 0.81) \) triggered a recovery in mood that then stabilized at the normal ratio \( (EB = 0.72) \).

- **Phase 2.** JR engaged in increased positive activities and a shift from dependence on validation from others to self-validation. Mourning losses continued, but were diminished in intensity, and JR focused less on mother and more on working through his dependence on his wife. He developed more insight into his narcissistic preoccupation with self-esteem management that diminished his sensitivity to others. Dreams revealed early concerns about loneliness as a child and throughout college, as well as peer rejection. The transition from phase 2 to 3 (Oscillation) was marked by a startling experience: JR announced in the session prior to the transition that he had a
transformative spiritual experience of increased God awareness following the inspiring story of a colleague who faced his death with tranquility and positive attitude. He dreamed that his mother was alive and that he felt greater acceptance of his parents being in the process of dying. Immediately after this, his overall emotional balance began consistently oscillating between the normal ($EB = 0.72$) and optimal ($EB = 0.81$) ratios (see Figure 6).

- **Phase 3.** With his emotional balance oscillating, JR continued to work on resolving residual issues of narcissism, deprivation, and childhood anger. He was less stressed by work and learned to maintain some joy even while engaged in the more thankless aspects of his job. Importantly, JR reported less envy, increased humility, social graciousness, acceptance of self and others, and spiritual transcendence. Although he continued to recall and process similar dream themes during this phase, his mood remained positive and his interpersonal functioning was vastly improved, with the exception of some residual tensions in his marriage. The Happiness subscale peaked at its highest level ever, and he felt less constricted and more creative in his approach to work. With his Beck Depression and Anxiety Inventory scores at zero, we worked toward a planned termination. Follow-up at six months and one year showed a sustained pattern of oscillation between the normal and optimal ratios, with all measures remaining at similar levels, indicating a resilient treatment outcome thus far.

**References**


On the Dynamical Interplay of Positive and Negative Affects


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