The Evolution of “Enhanced” Cognitive Behavior Therapy for Eating Disorders: Learning From Treatment Nonresponse

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Abstract

In recent years there has been widespread acceptance that cognitive behavior therapy (CBT) is the treatment of choice for bulimia nervosa. The cognitive behavioral treatment of bulimia nervosa (CBT-BN) was first described in 1981. Over the past decades the theory and treatment have evolved in response to a variety of challenges. The treatment has been adapted to make it suitable for all forms of eating disorder—thereby making it “transdiagnostic” in its scope—and treatment procedures have been refined to improve outcome. The new version of the treatment, termed enhanced CBT (CBT-E) also addresses psychopathological processes “external” to the eating disorder, which, in certain subgroups of patients, interact with the disorder itself. In this paper we discuss how the development of this broader theory and treatment arose from focusing on those patients who did not respond well to earlier versions of the treatment.
The Cognitive Behavioral Theory of Bulimia Nervosa

According to the cognitive behavioral theory of bulimia nervosa, central to the maintenance of the disorder is the patient’s overevaluation of shape and weight, the so-called “core psychopathology” (see Fig. 1, which shows in schematic form the core eating disorder maintaining mechanisms). Most other features can be understood as stemming directly from this psychopathology, including the dietary restraint and restriction, the other forms of weight-control behavior, the various forms of body checking and avoidance, and the preoccupation with thoughts about shape, weight, and eating (see Fairburn, 2008).

The only feature of bulimia nervosa that is not obviously a direct expression of the core psychopathology is binge eating. The cognitive behavioral theory proposes that binge eating is largely a product of a form of dietary restraint (attempts to restrict eating), which may or may not be accompanied by dietary restriction (actual undereating). Rather than adopting general guidelines about how they should eat, patients try to adhere to multiple demanding, and highly specific, dietary rules and tend to react in an extreme and negative fashion to the (almost inevitable) breaking of these rules. Even minor dietary slips are viewed as evidence of lack of self-control. Patients respond to such rule-breaking by temporarily abandoning their dietary restraint, thus succumbing to the urge to eat that arises from the restraint (and any accompanying dietary restriction), the result being a short-lived period of uncontrolled eating (i.e., an episode of subjective or objective binge eating). This produces the distinctive pattern of eating that characterizes bulimia nervosa in which attempts to restrict eating are interrupted by repeated episodes of binge eating. The binge eating in turn maintains the core psychopathology by intensifying patients’ concerns about their ability to control their eating, shape, and weight. It also encourages yet greater dietary restraint, thereby increasing the risk of further binge eating. Patients’ dietary slips and binges are particularly prone to occur in response to adverse day-to-day events and negative moods, in part because it is difficult to maintain dietary restraint under such circumstances and in part because binge eating both temporarily ameliorates negative mood states and it distracts patients from thinking about their difficulties.

A further process maintains binge eating among those who practice compensatory purging (i.e., those who induce vomiting or take laxatives in response to specific episodes of binge eating). Patients’ belief that purging will minimize weight gain results in the undermining of a major deterrent to binge eating. They do not realize that vomiting only retrieves part of what has been eaten, and laxatives have little or no effect on energy absorption (see Fairburn, 1995).

This cognitive behavioral theory is supported by a variety of lines of evidence (for details, see Fairburn et al., 2003) and it has clear implications for treatment. It suggests that if treatment is to have a lasting impact on binge eating and purging, the one aspect of the disorder that most patients want to change, it also needs to address extreme dieting, overevaluation of shape and weight, and any tendency for a patient’s eating to change in response to adverse events and negative moods.

CBT of Bulimia Nervosa

CBT for bulimia nervosa is designed to address each of the maintaining processes outlined in the cognitive behavioral theory above. The treatment is outpatient-based and, as evaluated in treatment trials, involves 15 to 20 sessions over approximately 5 months. A range of cognitive behavioral procedures are used with the cornerstone being a specific sequence of cognitive behavioral tasks and “experiments” set within the context of a personalised version of the cognitive behavioral theory of maintenance.
As noted there is a substantial body of evidence supporting CBT-BN, and the findings indicate that CBT-BN is the leading treatment. However, at best, half the patients who start treatment make a full and lasting response. Between 30% and 50% of patients cease binge eating and purging, and a further proportion show some improvement while others drop out of treatment or fail to respond (Wilson & Fairburn, 2007). These findings led us to ask the question, “Why aren’t more people getting better?” (Fairburn et al., 2003).

**Why Aren’t More People Getting Better?**

We examined various explanations (for a detailed discussion of these see Fairburn et al., 2003) for why there has not been a greater response to CBT-BN and, on the basis of our clinical experience, concluded that the cognitive behavioral theory needed to be extended to embrace four additional maintaining mechanisms that, in certain subgroups of patients, contribute to the maintenance of the eating disorder: clinical perfectionism, mood intolerance, low self-esteem, and interpersonal difficulties. We provide two case illustrations of patients treated using the focused form of the intervention (see Table 1 for an outline of treatment) that highlight how failure to address these additional mechanisms might account for treatment nonresponse. This is followed by a brief description of the revised theory and the evidence that supports it.

**Two Case Illustrations**

**Patient A**

Patient A was a 27-year-old woman referred by her primary care physician for the treatment of an eating disorder. Initial assessment indicated that she was suffering from an eating disorder that met *DSM-IV* (American Psychiatric Association, 1994) diagnostic criteria for bulimia nervosa. She had been dissatisfied with her weight since adolescence and began dieting to lose weight during her last 2 years at high school. At this time she weighed 130 lbs (59 kg; BMI=22.5). She had always pushed herself to achieve and had done well academically, but felt under added pressure to perform well in her final school examinations. Adhering to a strict diet helped her to feel more confident and in control and she initially lost 15 lbs (7 kg). The weight loss and consequent change in shape also represented a valued achievement in that she felt that she had finally managed to attain some success in an important area of her life (achieving her desired shape and weight) that had previously eluded her. She continued to restrict her eating while at university and gradually lost more weight, particularly at times when she felt under pressure to perform well. In the year before her initial assessment she started a new demanding job, which she enjoyed. However, she again felt under pressure to do well and lost further weight, reducing her weight to 98 lbs (44.4 kg; BMI=16.9). At this stage she met criteria for a diagnosis of anorexia nervosa. By the time she was seen for assessment she had been experiencing episodes of binge eating for several months and her weight had risen to 103 lbs (46.7 kg; BMI=17.8).

The patient described her daily food intake as consisting of a small bowl of cereal in the morning, a salad for lunch, and a small helping of pasta and vegetables in the evening. On some occasions she would add a yoghurt or apple to her diet. She weighed all her food and her daily eating routine varied little. The patient described a range of rigid dietary rules concerning the type and quantity of food that she would allow herself to eat. Any breaking of these rules would lead to an episode of binge eating during which she would typically eat a large tub of ice cream and 4 to 5 large cookies. These occurred on average twice each week. Eating with others was problematic as it risked her breaking her rules and because she

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1Details about the cases have been modified to protect the identity of the clients.
disliked being seen while eating. Hence, she avoided eating out. The patient exercised each
day, alternating between running (at least 5 miles) and cycling (at least 15 miles), and felt
that she only deserved to eat if she had completed the requisite amount of exercise. She
recorded her exercising on a spreadsheet and was always trying to improve on her
performance.

The patient was extremely concerned about her weight and shape. She weighed herself daily
and she repeatedly checked her shape by scrutinizing parts of her body (to see if she was
“failing” and becoming fat) and by comparing her body with that of objectively thin women.
Her other prominent concern was the quality of her performance in two areas of life: her
work and her close friendships. In both of these areas she had goals that were as exacting as
those she pursued with regard to eating and exercise. She was striving to work harder than
all her colleagues and to meet certain targets about the exact amount of “quality” time she
spent with her various friends.

Initially the treatment of Patient A proceeded well. The therapist was able to engage the
patient in the process of treatment and provided education about the psychological, social,
and physical effects of maintaining a low weight. The patient showed interest in
understanding the eating disorder and what was maintaining it. She responded well to the
case formulation, was excellent at self-monitoring in real time, and appeared to understand
the benefits of change and weight regain. She had more difficulty reducing the frequency of
her weight checking and on relying instead on the weight recorded with the therapist at the
beginning of her treatment sessions, but did begin to manage this after several weeks.

Considerable difficulties were encountered in helping the patient to act consistently on her
decision not to undereat and thereby allow herself to regain weight. The introduction of
regular eating required her to add three snacks to her existing pattern of three meals. She did
this, albeit reluctantly, as she was able to incorporate the snacks without breaking her rules
regarding the type and quantity of food that she permitted herself. However, she was not
consistently able to increase the amount that she ate. When she did manage to increase her
food intake, her intense concerns about weight gain, and the loss of control that it
represented (to her), resulted in her weighing herself more frequently outside of sessions.
She reacted to any perceived increase in weight by thinking that she was becoming “fat” and
by reducing her eating. Thus, although there were small increases in her weight (recorded
during the weekly in-session weighing), these were counterbalanced by decreases and, over
time, her weight remained largely unchanged. She was also resistant to making any changes
in her exercise routine.

A review of the patient’s progress revealed that the major obstacle to change was her
overvaluation of the importance of controlling her shape, weight, and eating. An attempt to
reduce the importance attached to these domains of life was unsuccessful as she was
unwilling to do anything that would reduce the time she spent on work, exercise and her
close friendships (because she feared that this would risk her performing less well in these
areas). Equally, attempts to address the repeated body checking proved unsuccessful. It
appeared that the importance (to her self-evaluation) of striving to meet her standards in
multiple areas of life was proving an obstacle to change. She accepted this but was
nevertheless unable (as she saw it) to “lower her standards” with regard to controlling her
shape, weight, and eating, nor was she able to do so in the areas of exercise, work, and
friendships. This was due to the influence of what we term “clinical perfectionism.”

**Patient B**

Patient B was a 23-year-old college student who met DSM-IV diagnostic criteria for bulimia
erosa. She had been dissatisfied with her weight since early adolescence and began
dieting to lose weight at age 16. Initially she lost about 14 lbs (6 kg) in weight but then found it progressively more difficult to stick to her diet and began experiencing episodes of binge eating and vomiting. Over the subsequent 6 years her weight fluctuated between 117 lbs (53 kg; BMI=20) and 137 lbs (62 kg; BMI=24). At assessment the patient described her daily food intake as consisting of a very small bowl of cereal for breakfast and a small salad for lunch. She often tried to delay these meals or to skip them altogether. She reported episodes of binge eating on most evenings (a typical binge including a loaf of bread, a large family-size carton of ice cream, and a packet of cookies). During such evenings she would vomit up to five times. The patient described a range of rigid dietary rules about how much she should eat at each meal and she avoided eating foods that she regarded as fattening and likely to trigger binges. At assessment she weighed 143 lbs (65 kg; BMI=25).

Patient B found her body “repulsive” and had avoided weighing herself for the past 6 months. At times she checked her body in the mirror (to see “how fat she was”) and she sometimes measured herself, but at other times she would avoid seeing her body as far as possible. She always avoided wearing tight-fitting clothes.

In addition, to her weight and shape concerns, Patient B reported marked short-lived mood fluctuations characterized by anxiety or anger. In the past she had cut herself to relieve tension and more recently she had used large amounts of alcohol (a bottle or more of wine a night) to “numb” herself. She reported that binge eating also helped her to modulate her mood.

As with Patient A, this patient responded positively to the case formulation as she felt it “explained” her eating disorder. She was only partially successful at real-time self-monitoring as she abandoned recording when experiencing adverse mood states.

After 4 weeks of treatment the patient’s frequency of binge eating had decreased. Problem-solving helped her to address the events and circumstances that triggered binges, and this was successful on some occasions, but the patient continued to binge eat several times a week. A detailed review of the mechanisms responsible for these binges revealed that changes in mood were the usual proximal trigger. Indeed, it was concluded that a major process maintaining the patient’s eating disorder was her inability to tolerate certain emotional states, a phenomenon that we term “mood intolerance.” Instead of dealing appropriately with adverse mood states, she was using binge eating (and on occasions alcohol) to reduce her awareness of these states (and the associated cognitions) and to neutralize it.

The Extended Cognitive Behavioral Theory of the Maintenance of Bulimia Nervosa

In the light of our experience with patients such as those described, we proposed that in certain patients one or more of four additional maintaining processes interact with the core eating disorder maintaining mechanisms (shown in Fig. 1) and that when this occurs they constitute further obstacles to change. The first of these maintaining mechanisms concerns the influence of extreme perfectionism (“clinical perfectionism”) as shown by Patient A. The second concerns difficulty coping with intense mood states (“mood intolerance”), seen in Patient B. The two other mechanisms concern the impact of unconditional and pervasive low self-esteem (“core low self-esteem”), and marked interpersonal problems (“interpersonal difficulties”). Limitations on space prevent us from providing clinical illustrations of these latter two mechanisms.

This new theory represents an extension of the original theory illustrated in Fig. 1. Fig. 2 shows in schematic form both the core maintaining mechanisms and the four hypothesised additional mechanisms.
Clinical Perfectionism

We have defined “clinical perfectionism”—that is, perfectionism of clinical significance (Shafran, Cooper, & Fairburn, in 2002)—as the overevaluation of striving for, and achieving, personally demanding standards, despite adverse consequences. The suggestion is that at the heart of this psychopathology is a system for self-evaluation in which self-worth is judged largely on the basis of striving to achieve demanding goals and success at meeting them. Thus we view clinical perfectionism as an example of a dysfunctional system for self-evaluation much like the “core psychopathology” of eating disorders. We regard it as external to the eating disorder in the sense that it is not an inherent or necessary feature of the disorder. However, when perfectionism is present and personally demanding standards are applied to eating, weight, and shape and their control, they intensify aspects of the eating disorder. Patients may, for example, diet especially intensely and exercise particularly rigorously not only to control their weight and shape but also to achieve success at striving towards and meeting demanding personal goals.

Perfectionism is well-known to co-occur with eating disorders (see Shafran et al., 2002; Wonderlich, 2002). Under these circumstances, our clinical observations indicate that there is often an interaction between the two with the patients’ perfectionist standards being applied to their attempts to control eating, shape, and weight, as well as to other aspects of their life (e.g., their performance at work or sport). As in other expressions of clinical perfectionism (Shafran et al., 2002), there is fear of failure (i.e., with respect to eating disorder psychopathology, these are expressed as fears of overeating, “fatness,” and weight gain); frequent and selective attention to performance (such as frequent shape and weight checking, calorie counting, weighing food, etc.); and self-criticism arising from negatively biased appraisals of performance. The resulting secondary negative self-evaluation in turn encourages even more determined striving to meet valued goals—including, in this case, striving to meet goals in the domain of controlling eating, shape, and weight—thereby serving to maintain the eating disorder. In the case of Patient A, we hypothesized that this interaction was an obstacle to the patient ceasing to undereat and to modifying her weight and shape checking, both of which were contributing to the maintenance of her disorder. We predicted that, for patients such as A, were clinical perfectionism to be moderated, a potent additional network of maintaining mechanisms would be removed, thereby facilitating change. Details of how this might be achieved are provided in the comprehensive treatment guide (Fairburn, 2008).

Mood Intolerance

The original version of the cognitive behavioral theory recognized that adverse mood states can disrupt dietary restraint and constitute a trigger of binge eating (Fairburn et al., 1986). It subsequently became clear that in some patients there is a more complex relationship between emotional states and binge eating (e.g., Meyer, Waller, & Waters, 1998; Polivy & Herman, 1993; Steinberg, Tobin, & Johnson, 1990; Stice, 1994; Waller, 2002).

In our expanded conceptualization we proposed that a subgroup of patients with eating disorders (such as Patient B) have what may be termed “mood intolerance.” Usually this intolerance is of adverse mood states, such as anger, anxiety, or depression, but in some cases there is intolerance of all intense mood states including positive ones (e.g., excitement). Instead of accepting changes in mood and dealing appropriately with them, these patients engage in what may be termed “dysfunctional mood modulatory behavior.” This reduces their awareness of the triggering mood state (and the associated cognitions), and also neutralizes it, but at a personal cost. The dysfunctional mood modulatory behavior may take the form of self-injury (e.g., cutting, punching, or burning themselves), which has the effect of rapidly dissipating the initial mood state, or it may involve taking psychoactive...
substances (e.g., alcohol, tranquilizers) to directly modify how they feel. Both classes of behavior are not uncommon among patients with eating disorders (e.g., Claes, Vandereycken, & Vertommen, 2001; Holderness, Brooks-Gunn, & Warren, 1994; Paul, Schroeter, Dahme, & Nützinger, 2002).

It is often not clear whether patients with mood intolerance actually experience unusually intense mood states or whether they are especially sensitive to normal mood states. Often, in our experience, both appear to be the case. Either way, cognitive processes commonly contribute to the phenomenon; for example, such patients typically respond to incipient mood change by thinking that they will not be able to cope with the resulting feelings and thoughts, a reaction that can amplify the triggering mood state.

Among patients with an eating disorder and mood intolerance, binge eating, vomiting, and intense exercising can become habitual means of mood modulation. In the case of Patient B, we hypothesized that both the excessive alcohol intake and the binge eating had become ways of modulating her intense moods. We predicted that were mood intolerance to be addressed in such patients, a powerful additional process maintaining the disorder would be removed. A detailed account of the approach we use to address mood intolerance is provided in the complete treatment guide (Fairburn, 2008).

**Core Low Self-esteem**

While most patients with bulimia nervosa are self-critical as a result of their failure to achieve their goals, a form of negative self-evaluation that generally reverses with successful treatment, there is a subgroup whose self-evaluation is more pervasively negative. Thus, rather than simply thinking negatively about themselves as a result of their inability to control their eating, shape, and weight, these patients have, in our view, an unconditional negative view of themselves which contributes to their permanent identity. Their negative self-judgments are autonomous and largely independent of performance: In other words, they are neither affected much by events and circumstances nor by changes in the state of the eating disorder.

Such “core low self-esteem” tends to obstruct change in general. This is through two main mechanisms. First, it creates hopelessness about the capacity to change, thereby undermining patients’ compliance with treatment; and second, it results in their pursuing, with particular determination, achievement in valued domains (in this case control over eating, shape, and weight). The state is self-perpetuating since these patients show particularly pronounced negative cognitive processing biases coupled with overgeneralization, with the result that any perceived “failure” is interpreted as confirmation that they are failures as people, thereby reaffirming their overall negative view of themselves.

Given the many barriers to change that arise as a result of core low self-esteem, it is not surprising that clinical experience and some research findings have suggested that such patients respond particularly poorly to treatment (Fairburn, Kirk, O’Connor, Anastasiades, & Cooper, 1987; Fairburn Peveler, Jones, Hope, & Doll, 1993). On the other hand, were their core low self-esteem to be addressed, we predicted that these patients’ outcome would improve as a result.

**Interpersonal Difficulties**

The original cognitive behavioral account of the maintenance of bulimia nervosa paid little attention to patients’ circumstances—other than acknowledging that the proximal triggers of binge eating were commonly interpersonal in nature. The need to broaden this focus has been made especially clear by the research on interpersonal psychotherapy (IPT). This
indicates that an exclusively interpersonal treatment is about as effective as CBT in the longer-term (Agras, Walsh, et al., 2000; Fairburn, Jones, et al., 1993; Fairburn et al., 1995) despite the fact that it does not directly address any of the maintaining mechanisms identified by the cognitive behavioral theory (Fairburn, 1997b).

There can be no doubt that interpersonal processes contribute in a variety of ways to the maintenance of all forms of eating disorder. Four examples may be used to illustrate this point. First, in younger patients, family tensions often intensify resistance to eating. It can be argued that this reflects a short-term intensification of their need for a sense of “control,” a need that is displaced onto dietary self-control (Fairburn, Shafran, & Cooper, 1999). Second, it is obvious that certain interpersonal environments magnify concerns about controlling eating, shape, and weight. These include families in which there are other members with an eating disorder, and occupations in which there is pressure to be slim. Third, as already noted, adverse interpersonal events commonly precipitate episodes of binge eating, and there is evidence that patients with bulimia nervosa may be especially sensitive to social interactions (Steiger, Gauvin, Jabalpurwala, Seguin, & Stotland, 1999). Fourth, long-term interpersonal difficulties undermine self-esteem which, as noted earlier, is prone to result in patients striving even harder to achieve valued goals, such as success at controlling eating, shape, and weight. It is also relevant that there is evidence that disturbed interpersonal functioning predicts a poor response to treatment (Agras, Crow, et al., 2000; Steiger, Leung & Thibaudeau, 1993). In summary, we thought that there were good reasons to propose that interpersonal difficulties could perpetuate eating disorders and that their resolution might facilitate change.

An Evaluation of the New, Enhanced Form of CBT (CBT-E)

On the basis of this “extended” theory of the maintenance of bulimia nervosa, we developed two versions of CBT-E, a “focused” version that exclusively addresses the processes maintaining the eating disorder psychopathology (Fairburn, Cooper, Shafran, Bohn, Hawker, Murphy, et al., 2008) and a “broad” version that also addresses one or more of the four additional hypothesized maintaining processes (Fairburn Cooper, Shafran, Bohn, & Hawker, 2008). Both versions are transdiagnostic in scope. Treatment involves 20 individual treatment sessions over 20 weeks, the first 4 weeks being common to both forms of treatment (Fairburn, Cooper, & Shafran, 2008). We have recently compared these two forms of CBT-E in the context of a two-site randomized controlled trial (Fairburn et al., 2009). The trial was unusual in that focused on the treatment of outpatients with any form of eating disorder provided that their BMI was over 17.5. Therefore, by definition, these patients met DSM-IV diagnostic criteria for bulimia nervosa or eating disorder NOS. As those who are significantly underweight (BMI 17.5 and below) require a longer treatment, they were not included in this study.

One hundred and fifty-four patients were entered into the trial. Treatment was on an individual basis and it followed the CBT-E protocol of 20 treatment sessions over 20 weeks. Treatment was followed by a 60-week closed period of follow-up. After 4 weeks of treatment (the period when the two treatments are the same) the therapists rated the severity of each patient’s clinical perfectionism, mood intolerance, core low self-esteem, and interpersonal difficulties. These ratings were made prior to the therapist learning which treatment the patient was going on to receive and were used to identify patients with marked additional psychopathology of the type that the broad form of CBT-E is designed to target. The outcome of these patients was compared with that of the remainder in relation to the form of CBT-E that the patient actually received.
At the end of 20 weeks of treatment, over half the randomized patients (52.7% and 53.3% of those with bulimia nervosa and eating disorder NOS respectively) had a level of eating disorder features less than 1 SD above the community mean, and at 60-week follow-up the comparable figures were 61% and 46%. These figures suggest that CBT-E might be more effective than CBT-BN (see for comparison Agras, Walsh, et al., 2000), especially in light of the inclusive nature of the sample, although a direct comparison of CBT-BN and CBT-E would be needed to substantiate this conclusion. In the full sample there was no difference between the two forms of CBT-E and, not surprisingly, the subgroup with marked additional psychopathology (about 40% of the sample) responded less well in general than the remainder. However, within this subgroup, those who received the broad form of CBT-E did better than those who received the focused version, whereas, interestingly, the reverse pattern of findings was present within the remaining patients with the focused version being superior to the broad version.

Clinical and Research Implications

This program of work illustrates the value of focusing attention on those patients who benefit least from treatment. Doing so resulted in the enhanced form of CBT, which appears to be markedly more effective and more useful (in terms of the full range of patients treated) than its forerunner, CBT-BN.

However the finding regarding the relative effects of the two forms of CBT-E must be viewed as tentative. To determine its robustness, further comparisons of the two forms of CBT-E are needed. In the meantime, it is our view that it would seem reasonable to use the present findings to guide clinical practice. Thus the simpler focused form of the treatment should be viewed as the default form of CBT-E as it is easier to learn and implement, with the new, more complex, form being reserved for patients of the type that previously benefited least from treatment.

Acknowledgments

We are grateful to the Wellcome Trust. Without the Trust’s sustained support over the past 25 years this work would not have been possible. CGF is supported by a Principal Research Fellowship (046386) and ZC is supported by a programme grant (046386). The transdiagnostic trial (also funded by the Wellcome Trust) was conducted in collaboration with Professor Robert Palmer of the University of Leicester. Shawnee Basden helped prepare the manuscript.

References


Figure 1.
A schematic representation of the cognitive behavioral theory of the maintenance of bulimia nervosa (modified from Fairburn, Cooper, & Shafran, 2003).
Figure 2.
A schematic representation of the extended cognitive behavioral theory of the maintenance of bulimia nervosa. “Life” is shorthand for interpersonal life (from Fairburn, Cooper, & Shafran, 2003).
Table 1
The Core Elements of the Focused Form of Enhanced Cognitive Behavioral Treatment

Stage One
The aims are to engage the patient in treatment and change.
· Jointly creating a formulation of the processes maintaining the eating disorder
· Establishing real-time monitoring of eating and other relevant thoughts and behavior
· Providing education about body weight regulation and fluctuations, the physical complications and ineffectiveness of self-induced vomiting and laxative misuse as a means of weight control and the adverse effects of dieting
· Introducing weekly weighing
· Introducing a pattern of regular eating
· Involving significant others to facilitate treatment if appropriate

Stage Two
This is a transitional stage.
· Jointly reviewing progress
· Identifying barriers to change
· Modifying the formulation as needed
· Planning Stage Three.

Stage Three
The aim is to address the key mechanisms that are maintaining the patient’s eating disorder.
· Overvaluation of shape and weight
  - providing education about overvaluation and its consequences
  - reducing unhelpful body checking and avoidance
  - relabelling unhelpful thoughts or feelings such as “feeling fat”
  - developing previously marginalized domains of self-evaluation
  - exploring the origins of the overvaluation
· Dietary restraint
  - changing inflexible dietary rules into flexible guidelines
  - introducing previously avoided food
· Event triggered changes in eating
  - developing problem-solving skills to directly tackle such events
  - developing skills to accept and modulate intense moods

Stage Four
The aims are to ensure that progress made in treatment is maintained and that the risk of relapse is minimised.
· Providing education about realistic expectations
· Devising a short-term plan for the months following treatment
· Devising a long-term plan to minimise relapse in the future