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The Multidisciplinary Approach in the Treatment of Eating Disorders

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Table of Contents

Acronyms

ACT – acceptant and commitment therapy
ALT – alanine aminotransferase
AN-ERP – exposure response prevention for anorexia
APA – American Psychiatric Association
ARFID – avoidant/restrictive food intake disorder
AST – aspartate aminotransferase
AN – anorexia nervosa
BED – binge eating disorder
BMI – body mass index
BN – bulimia nervosa
BUN – Blood urea nitrogen
CBT – cognitive behavior therapy
CCK - cholecystokinin
DA – dopamine
DBT – dialectical behavior therapy
ED – eating disorders
ERP-B – exposure response prevention for binging
ERP-P – exposure response prevention for purging
FSH – follicle stimulating hormone
GAPS – Guidelines for Adolescent Preventative Services
HAES – health at every size
HVA – homovanillic acid
IBW – ideal body weight
IE – intuitive eating
IGF-1 – insulin-like growth factor 1
IOP – intensive outpatient program
IPT – interpersonal therapy
LH – luteinizing hormone
LOC – levels of care
MAOIs – monoamine oxidase inhibitors
MED- DBT – multi-diagnostic complex eating disorder dialectical behavior therapy
MRI – magnetic resonance imaging
NICE – National Institute for Health and Clinical Excellence
NPY – neuropeptide Y
OCD – obsessive compulsive disorder
OSFED – other specified feeding or eating disorder
PHP – partial hospitalization program
PYY – peptide tyrosine tyrosine
PTSD – post traumatic stress disorder
RANZCP – Royal Australian and New Zealand College of Psychiatrists

RD – registered dietitian
RMR – resting metabolic rate
RO-DBT – radically-open DBT
SUD – substance use disorder
SSRI – serotonin re-uptake inhibitor
TCAs - Tricyclic antidepressants
UFED – unspecified feeding or eating disorder

Introduction

Eating disorders (ED) are characterized by severe disturbances in eating behavior and body weight.[1] They encapsulate a broad range of compulsive food and eating related disturbances that are considered one of the more malicious psychiatric disorders.[2] Disturbances may include persistent dietary restraint, starvation, low weight, overvaluation of body shape, body dysmorphia, periodic overeating, purging, overweight, and aversion to food based on sensory and texture aversions.[2] The etiology of eating disorders is multi-factorial including genetic, biological, individual, family, psychological, and socio-cultural and as such require a multi-disciplinary treatment approach.[3]

EDIN: Education and Insight on Eating Disorders, is a local non-profit working to create awareness and provide education on eating disorders to those struggling with EDs, caregivers, and medical and therapeutic professionals in the metro Atlanta area. Currently, they offer a wide variety of educational resources including in-person and web-based presentations and educational curriculum. The purpose of this literature review is to explore the current research on eating disorders including, but not limited to, prevalence, etiology, metabolism, neurochemistry, and the multi-disciplinary roles of the treatment team. The ultimate goal of this capstone project is to provide a comprehensive and educational video aimed at improving ED knowledge in the professional community.

Eating Disorder Prevalence

National and global studies indicate an increase in the overall prevalence of ED over recent years.[1] It is estimated that up to 70 million individuals worldwide and 30 million individuals in the United States will have an eating disorder during their lifetime.[4-6] In Georgia, there are around 339,500 people who suffer from ED.[7] In both Canada and the United Kingdom, it is estimated that ED are third highest chronic condition among girls ages 15-19 years old.[2,8] Although ED are traditionally thought to be confined to Western countries, studies have highlighted a higher prevalence in Asia and developing Middle-Eastern countries.[1] ED affect all ages, genders, ethnicities, and sexualities. Approximately 95% of initial cases in both males and females occurred by the age of 25, with the highest mean annual prevalence around the age of 21 years.[9] Although the estimated annual prevalence of ED is lower comparative to other disease states, the cumulative lifetime burden is high at 14.3% for male and 19.7% for female individuals.[9] The stereotypical ED patient is an affluent, white female adolescent, but males can also suffer from greater body dissatisfaction as well as problematic eating behaviors.[3] Additionally, the LGBTQIA+ community have shown a higher prevalence than their straight counterparts.[3] Caucasians are the largest ethnic group affected by ED, but Mexican-American, African Americans, and Asians have been shown to be at risk as well.[3] African American women have been shown to be at equal risk as their Caucasian counterparts, specifically for bulimia nervosa.[3] As the second deadliest mental illness behind opiate addiction, one person dies every 62 minutes from an ED.[10] When the initial onset occurs during adolescence, it can result in up to a 25-year reduction in lifespan as well as impaired quality of life.[11] The most prevalent ED is other specified feeding or eating disorder (OSFED), followed by binge eating disorder (BED),

bulimia nervosa (BN), and anorexia nervosa (AN).[9] As the majority of research has focused on AN, BN, and BED, we will further detail prevalence by category.

Feeding & Eating Disorder Types

The American Psychiatric Association has outlined and defined diagnostic criteria for eight main ED in the Diagnostic and Statistical Manual of Mental disorders (DSM-V). As we review the diagnostic criteria for each category, it is important to note that individuals should be carefully evaluated for an eating disorder regardless of other criteria if they are unable to maintain or attain a healthy weight, height, body composition or sexual maturation for gender or age.[3]

AN is characterized by the restriction of energy intake resulting in significantly low body weight, intense fear of weight gain or the persistent behavior that interferes with weight gain despite low body weight, and a disturbance in body image, or persistent lack of recognition of the seriousness of their current low body weight.[12] There are two other subtypes of AN, atypical and binge-purge AN. Atypical AN is similar to the standard AN, but patients are not significantly underweight.[13] Roughly between 42.1%-56.2% of those with AN will meet at least one criteria for another DSM-5 disorder.[14] The most common being anxiety and major depressive disorders.[14] Substance abuse is also present at a rate between 13%-27%.[14] It is estimated that males represent between 10-25% of those patients diagnosed with anorexia nervosa and are at a higher risk of dying than their female counterparts partly due to a lack of education around diagnosing eating disorders and a lack of awareness of eating disorders in the male population.[15] Anorexia has one of the highest mortality rates of all ED with an approximate mortality rate of 20%.[12] More recent evidence supports that those suffering from AN will still meet full diagnostic criteria more than two decades after onset of AN.[2] Less than half of those with AN will completely recover.[16] Around 30% will show improvement while 25% maintain a chronic course.[16]

BN is detailed as recurrent episodes of binge eating and inappropriate compensatory behaviors to prevent weight gain with a frequency of at least once per week for three months.[12] Those that suffer from BN often express self-evaluation that is unduly influenced by body shape and weight. In this context, binge eating is described as eating an excessive amount of food in a discrete period of time along with a sense of lack of control.[12] Compensatory behaviors may include use of laxatives, diuretics, vomiting, fasting, or excessive exercise. Of particular note, BN occurs in the absence of AN.[12] The lifetime prevalence of BN is estimated between 1.7-2.9% with peak incidence between 16-20 years of age in females.[14] Although studies vary, the male lifetime prevalence is estimated to be between 0.13% and 1.34%.[17] Although BN is more prevalent than AN, it is generally diagnosed at a later stage.[3] This can partly be attributed to the tendency for those with BN to be at weight or overweight as well as poor health screening practices across disciplines.[3] The prognosis for BN is slightly better than those with AN.[16] Around 50% will recover, while 30% will maintain disordered behaviors.[16]

Newly added to the DSM-V is binge eating disorder (BED). Similar to BN, BED consists of recurrent episodes of binge eating that occur at least once per week for three months, however

without the additional compensatory behaviors as is the case in BN.[12] It is associated with symptoms such as eating more rapidly, feeling uncomfortably full, eating while not feeling hungry, and eating alone due to embarrassment and/or feelings of self-disgust.[12] Patients often note marked distress around binge eating. It occurs in the absence of AN and BN.[12] The lifetime prevalence of BED in males and females is around 2.0% and 3.5%, respectively.[5,17] As this has been newly added to the DSM-V, there is limited research available.

Other recently added ED to the DSM-V include, pica, rumination, avoidant/restrictive food intake disorder (ARFID), other specified feeding or eating disorder (OSFED), and unspecified feeding or eating disorder (UFED). A diagnosis of pica is classified as eating of one or more nonnutritive, nonfood substances on a persistent basis over a period of at least 1 month that is inappropriate to the developmental level of the individual.[18] It occurs in the absence to any cultural or socially normative practices.[18] Furthermore, if the eating behavior coincides with another mental disorder such as autism, a separate diagnosis of pica should only occur if severe enough to warrant additional clinical attention.[18] Rumination features repeated regurgitation of food over a period of at least one month and the regurgitated food may be re-chewed, re-swallowed or spit out.[18] Similar to pica, if the behavior coincides with another mental disorder, diagnosis of rumination should only be considered if it is severe enough to warrant the additional diagnosis.[18] It can only occur in the absence of BN, AN, BED, and ARFID.[18] The current prevalence data on both rumination and pica is inconclusive, however an increased prevalence in those with intellectual disabilities is noted.[18]

ARFID is noted when there is an eating or feeding disturbance with the persistent failure to meet nutritional needs associated with significant weight loss or growth failure, significant nutritional deficiency, dependency on enteral/supplemental feeding or marked with interference with psychosocial functioning.[12] These symptoms are not explained by lack of food availability, other medical illnesses, or cultural practices and occurs in the absence of AN, BN, or body image disturbances.[12] The OSFED category is for those eating disorders causing significant distress, but not meeting the criteria for the other diagnostic categories.[12] OSFED has a lifetime prevalence estimated around 7.6%.[1] Night eating syndrome, purging disorder, subthreshold binge eating disorder, low frequency/limited duration BN, and atypical AN would be classified under this category.[12] The last category, UFED covers eating disorders where there is presentation of clinically significant symptoms, but they do not meet the full criteria for other ED.[12] Additionally, subclinical eating disorder behaviors such as binge eating, purging, laxative abuse, and fasting for weight loss is nearly as common among males as to females.[15]

Risk Factors

There are several risk factors associated with the development of ED that cover a variety of genetic, physiological, environmental, psychological or temperamental factors. From a genetic or physiological standpoint, those that have a history of gastrointestinal problems, brain abnormalities, childhood obesity, and early pubertal maturation may be more susceptible.[3,18] From a familial side, those with a first-line relative with a history of an eating disorder, bipolar and depressive disorders, as well as a history of alcohol or substance abuse have an increased

risk. Additionally, those that have experienced family anxiety, are part of cultures or occupations that value thinness, or have had a history of neglect, physical, emotional, or sexual abuse are more predisposed.[18] From a temperamental view, there are some key personality characteristics. Those with BN are often characterized with increased impulsivity and risk-taking behavior, while patients diagnosed with AN often have a difficult time expressing negative feelings, exhibit low self-esteem, and show some degree of constraint.[3] Comorbid anxiety disorders, autism, obsessive-compulsive disorder, attention-deficit/hyperactivity disorder, or those that display obsessional or perfectionistic traits are associated with the development of ED.[18] Lastly, those with a history of dieting, obesity, and/or an increased focus on weight or body image either from themselves or from family members may be more vulnerable.[3]

Screening & Diagnosis

Being able to correctly diagnose ED is paramount in the treatment and prognosis of a patient with an eating disorder. Early intervention is associated with a better prognosis, so it is essential that health care professionals be familiar with the early signs and symptoms of ED.[16] Unfortunately, many cases of ED are missed by health practitioners. In fact only half of all cases of AN and one-third of BN cases are ever detected by the healthcare system.[14] According to Murray only 26% of those with ED will receive any consultation for symptoms let alone engage in supported treatments.[2] Approximately 20% of females and 13% of males will go into treatment.[2] Since the initial onset of ED is skewed towards adolescence and young adulthood, it suggests that this is a prime time for prevention efforts, while the higher prevalence later in life highlights the importance of identification and treatment for long term prognosis.[9]

ED are multi-factorial and as such require a multi-disciplinary team approach. At a minimum treatment teams include a physician, dietitian, and psychologist or therapist. Patients may initially present for treatment at any one of these team members and may not even realize they have an eating disorder.[16] Thus understanding how to screen and what to watch out for with regards to signs and symptoms is imperative among all practitioners. On a basic behavioral level, there are some symptoms that are associated with ED and should highlight to look further into identifying and diagnosing. If a patient exhibits a strong tendency for extreme physical activity, frequent meal skipping, restrictive eating patterns, thinness as a valued goal, unrealistic goals, poor coping skills related to life events, and/or withdrawal from friends then a deeper investigation should be warranted.[3] Children that have obsessive-compulsive traits such as perfectionism, having to follow rules, and concern about mistakes are much more likely to develop an ED than those that do not, so practitioners should be watchful.[19] There are a number of screening tools that can be utilized to assist in the detection and diagnosis of ED. The most widely known is the SCOFF questionnaire which only consists of 5 questions.[12] 1) Do you make yourself **sick** because you feel uncomfortably full?[12] 2) Do you worry that you have lost **control** over how much you have eat?[12] 3) Have you recently lost more than **one** stone (6.35 kg) in a 3-month period? 4) Do you believe yourself to be **fat** when others say you are thin?[12] 5) Would you say that **food** dominates your life?[12] If the client answers 2 or more questions with a yes, then a diagnosis of AN or BN may be likely.[12] Bright Futures and the

Guidelines for Adolescent Preventative Services (GAPS) are also useful evidenced-based screening forms.[3] The Bright Futures screening tool utilizes a variety of questions including how do you feel about the way you look, how do you feel about your weight, are you trying to change your weight, and do you ever fast, vomit, or take laxatives or diet pills to control your weight.[3] The GAPS guidelines recommends screening adolescents annually for ED and to inquire about body image and dieting patterns.[3] However, according to the National Institute for Health and Clinical Excellence (NICE) guidelines, the most effective screening tool probably remains the practitioner thinking about the possibility of an eating disorder.[12,20]

Warning Signs & Symptomology

In addition to screening tools, there are a number of warning signs or symptoms that can aid in the detection and identification of ED. A detailed client history is critical in the assessment process and in determining the level of care. Significant symptoms can be divided into five main categories: cardiovascular, gynecological, gastrointestinal, psychological, and other.[14] Common symptoms of someone with AN include hypotension with a systolic blood pressure below 99 mm HG and/or a diastolic blood pressure below 50 and bradycardia with a heart rate below 60 beats per minute.[3,14] It is important to note that patients, parents, and even physicians may link a slow heart rate as a sign of fitness, when it could be a sign of decompensation as is the case in malnutrition.[3] Additionally, around 30-50% of patients with AN have mitral valve prolapse. With severe malnutrition, cardiac muscle diminishes, but the size of the valve does not, leading to prolapse.[3,14] Another possible complication in those with AN who also purge via self-induced vomiting is the development of pneumomediastinum, a condition where air leaks into the mediastinum.[14] It is hypothesized that malnutrition causes thinning of the alveolar walls and connective tissue, which can predispose patients to pneumomediastinum when intrathoracic pressure increases such as is the case during vomiting.[14] Patients have also shown congestive heart failure, dysrhythmias, orthostatic hypotension, pericardial effusion, syncope, and electrocardiographic abnormalities.[3] In addition, patients with BN who abuse ipecac, which contains alkaloid emetine, are at risk for cardiomyopathy, which can result in heart failure and ventricular arrhythmias when taken in excess.[14]

From a gastrointestinal perspective, delayed gastric and colonic emptying, abdominal pain and distention are all common complaints in those with AN and BN.[3,14] Those with AN are higher at risk for this and are often diagnosed with gastroparesis, gastric distention, gastroesophageal reflux, constipation, and superior mesenteric artery syndrome.[3,14] BN patients who self-induce vomiting often complain of painful swallowing, hoarseness, dysphagia, and heartburn secondary to gastroesophageal reflux.[14] Patients who abuse laxatives may complain of diarrhea and abdominal cramping.[14] With regards to constipation, it is important to not treat with stimulant laxatives such as Senna-Gen and bisacodyl due to the potential for long-term abuse, which puts the patient at risk for developing colonic nerve cell damage causing cathartic colon syndrome.[14] Patients who have a history of abusing stimulant based laxatives may develop rebound constipation.[14] Other common gastrointestinal issues include acute pancreatitis, Barrett's esophagus, bloody diarrhea, esophagitis, esophageal rupture, gallstones, and

perforation or rupture of the stomach. Barret's esophagus occurs from chronic gastroesophageal reflux that affects the lining of the esophagus causing it to thicken and become red.[14] Mallory-Weiss tears and parotid hypertrophy are primarily seen in those with BN. Mallory-Weiss tears or lower esophageal tears that typically occur due to violent vomiting.[14] Parotid hypertrophy is the chronic enlargement of the parotid glands in those who purge via vomiting as seen in BN and atypical AN binge-purge type.[14,21] The tale-tale signs of enlarged parotid glands is swelling of the cheeks.[14] Additionally acid reflux from self-induced vomiting and damage to the esophageal sphincters can affect the pharynx and larynx and can lead to the development of laryngopharyngeal reflux.[22] The regurgitated acidic contents can come into contact with the vocal cords and surrounding areas resulting in hoarseness, dysphagia, chronic cough, and repeated sore throats.[22]

The most prominent gynecological symptoms among females is irregular menstrual cycles, amenorrhea, or delayed pubertal development in adolescents. In female patients with AN development of hypothalamic amenorrhea syndrome can occur resulting from variable reduction in pulsatile hypothalamic GnRH gonadostat signaling to the pituitary gland resulting in failure of ovulation.[23] As a result of this gonadotropin failure, individuals with AN have decreased levels of the sex hormones, estradiol, estrone, progesterone, and testosterone.[23] Menstrual irregularities are also common in BN although usually less severe and prevalent than in cases of AN.[23] Typically, this functional hypogonadotropic, hypogonadism-induced amenorrhea reflects a temporary and reversible disturbance in function.[23] According to Golden et al., menses can be expected to resume at a weight that exceeds 90-93% of ideal body weight.[24] However, other studies have demonstrated more variable weights than predicted weights.[23] Additionally, there is some evidence that weight loss may not be the only factor causing amenorrhea. Amenorrhea can precede significant weight loss in about one-quarter of women and can persist even after weight restoration, while other women may resume menstruation despite a low body weight.[23] It is thought that adaptive hormonal responses to sociocultural-psychic stress, excessive exercise and chronic nutritional energy deficiency along with reduced thyroid and leptin levels may also contribute.[23] From a medical standpoint, treatment efforts should focus on the overall treatment of the ED as there is little inherent value in administering female sex hormones (i.e. birth control) as withdrawal bleeding can promote a false sense of well-being and minimize the need for therapy.[23] Men with AN display a few abnormalities of reproductive hormones including low testosterone, LH, and FSH which may lead to poor semen quality, reproductive suppression, decreased sperm motility, and an increase in immature sperm number.[23,25]

It is important to note that pregnancy in women with an ED is still possible. At least 1 in 20 women experience some form of an eating disorder in pregnancy with the highest risk for those with a history of an eating disorder.[26] A 2013 study found that 7.5% of women receiving their first routine ultrasound scan during pregnancy met the criteria for an eating disorder.[27,28] Another study showed that 28% of subjects displayed disordered eating symptoms and psychological and behavioral traits associated with ED during pregnancy.[29] Unfortunately, 93.3% of the women in this study were not identified by their medical practitioners.[29] This is especially important as it is associated with a greater incidence of complications for mother and

child. Those with BN have 2x the risk for hyperemesis gravidarum, increased weight gain, and miscarriage.[26,30-32] Mothers with BED are at risk for increased weight gain and pre-term delivery. AN has been associated with low weight gain during pregnancy[33].[26,30,31] All mothers are at higher risk for gestational diabetes, hypertension, and pre-eclampsia.[26,30,31] The child on the other hand is at risk for being large or small for gestational age, smaller head circumference, stillbirth, low birth weight, low APGAR scores, breach, and cleft lip and/or palate.[26,31] Even 2-6 months post-partum, mother and child are at continued risk. The mother is more likely to stop breastfeeding, increase eating disorder behaviors including quick weight loss, and are 5X more likely to develop mood disorders such as depression.[31] The baby is more at risk for decreased child feeding, low mother-child attachment, and roughly 17% will have failure to thrive within the 1st year.[31]

Psychological co-morbidities and dual diagnosis are prominent in ED. The presence of comorbidity is a strong indicator of poorer long-term outcomes and is associated with more severe symptoms.[34] In a study of more than 2400 hospital patients with an eating disorder, researchers found that more than 97% had one or more co-occurring conditions. The study further found that 94% had co-occurring mood disorders such as major depression, 56% with anxiety disorders, 20% had obsessive-compulsive disorder, 22% with post-traumatic stress disorder (PTSD), and 22% with alcohol or substance abuse disorder.[19] In fact, two-thirds of people with AN had signs of an anxiety disorder before the onset of their eating disorder.[19] Further supporting the high prevalence of comorbid psychiatric disorders, a national representative survey found that 95% of respondents with BN, 79% with BED, and 56% with AN met the criteria for at least one other psychiatric disorder.[5] Among those BN respondents, they also noted that 64% met the criteria for three or more co-occurring psychiatric disorders.[5]

As one of the top 3 comorbid psychiatric disorders, PTSD can occur when a person is exposed to a traumatic event such as death, threatened death, actual or threatened serious injury or sexual violence.[18] This exposure can result from the direct experience with the traumatic event, witnessing the event in-person, learning of a traumatic event that occurred to a close family member or friend, or first-hand, repeated experiences or extreme exposure to adverse details of the traumatic event.[18] Symptoms of PTSD can be grouped into four main categories including intrusion symptoms, avoidance, negative alterations in mood, and alterations in arousal and reactivity.[18] Some of these symptoms may include flashbacks, nightmares, irritability, angry outbursts, exaggerated startle, problems concentrating, insomnia, being overly watchful, numbing, forgetting, partial amnesia, negative beliefs around oneself, others, or the world, and self-blame.[18] Evidence suggests that ED patients may be sensitive or vulnerable to stress and its consequences. Two national representative studies have shown that individuals with BN, BED, or binge eating have higher rates of PTSD symptoms than those without ED.[5,35] The lifetime prevalence of PTSD among BN groups range between 38-44%.[5,35] Additionally, women who were victims of assault were 1.86X more likely to develop BN than if they hadn't been victimized.[35,36] In another study looking at PTSD and AN, the researchers found that 13.7% of participants met the criteria for PTSD.[37] When they delved further into the results, they found that AN purging subtype had higher odds of PTSD than standard AN.[37] Another study found that 48% of females and 68% of males with AN, up to 41% of females and

24% of males with BN, and up to 35% of females and 16% of males with BED have experienced some form of sexual violence.[36] Unresolved trauma and/or PTSD may be an important factor in the maintenance of eating disorder symptoms. Similar to the way substance abuse may be used to self-medicate, binge eating and/or purging may also help reduce the hyperarousal or anxiety symptoms associated with trauma as well as help numb or avoid traumatic experiences.[38] These behaviors are reinforcing, making it difficult to break the cycle.[38] As a result, individuals with ED complicated by trauma and PTSD need treatment for both conditions utilizing a trauma-informed, integrated approach.[38] Of note, the best approach to address both PTSD and ED remains debated, but work so far primarily focusses on cognitive behavioral therapy with integrated treatment for the eating disorder.[38]

Additionally, personality disorders occur commonly in individuals with ED. In a meta-analysis examining the prevalence of personality disorders and ED, they found that avoidant personality disorder, dependent personality disorder, and obsessive-compulsive personality disorder (OCD) were commonly associated in both AN and BN, while borderline personality disorder, which is marked by impulsivity and instability is common among BED.[39] Additionally comorbid substance use disorders (SUD) are common with roughly 1 in 5 individuals developing a SUD at some point in their lifetime and roughly 1 in 10 meet current criteria for a SUD.[40] The lifetime prevalence of comorbid SUD among adults with AN, BN, BED, and subthreshold BED is estimated between 23-37%.[40] In a meta-analysis, researchers found that among the highest prevalence of comorbid SUD was tobacco (36.1 ± 23.1%), caffeine (23.8 ± 12.5%), alcohol (20.6 ± 16.0%), or any other illicit drug (19.8 ± 19.6%), cannabis (14.5 ± 16.0%), and cocaine (13.7 ± 23.4%).[40] Lifetime SUD comorbidity is higher among females, Caucasians, and individuals with BN and binge-purge behaviors.[40] It is estimated that roughly 20% of individuals being treated for a SUD report binge eating.[41] Of men with BED, 40.4% report having struggled with a SUD.[42] ED and SUD share many common risk factors including brain chemistry, family history, low-self-esteem, anxiety, and depression.[43] They also share similar characteristics such as compulsive behavior, social isolation, and risk for suicide.[43] Successful treatment of comorbid psychiatric diagnoses and SUD in patients with ED can be challenging when the patient is malnourished as starvation can have a significant impact on mood and cognition and some psychiatric symptoms may improve with weight restoration.[3]

Beyond the gastrointestinal, cardiovascular, reproductive, and psychological issues associated with ED, there is a vast array of other signs or symptoms. Other affected areas include dermatologic, endocrine, skeletal, hematologic, neurologic and fluid and electrolyte balances. Hematologic effects of ED include bone marrow suppression, mild anemia, leukopenia, thrombocytopenia, impaired cell-mediated immunity, and low sedimentation rate.[3] Neurologically speaking patients might have cortical atrophy, myopathy, peripheral neuropathy, and even seizures.[3] From a dermatological standpoint, patients often present with brittle nails, alopecia, lanugo on the arms and sides of their face, and dry skin and hair.[3,14] Acrocyanosis, alopecia, hypercarotenemia, and knuckle calluses also known as Russel's sign have been noted.[3] Russel's sign is indicative of self-induced vomiting in BN patients.[3] Hypercortisolism, growth retardation or short stature, low T3 syndrome and partial diabetes insipidus or an inability to concentrate urine has been noted in adolescents with ED.[3]

The skeletal ramifications are detrimental and include osteoporosis, osteopenia, and fractures.[3] As a disease characterized by low bone mass and deterioration of the microarchitectural structure of the bone, osteoporosis results in bone fragility and nontraumatic fractures due to low bone mass.[23] Osteoporosis is present in almost 40% of patients with AN, while osteopenia is present in 92%. This is particularly of concern as peak bone mass generally occurs between the ages of 17 and 22, which often coincides with the onset of AN.[23] Once amenorrhea is present, the estrogen-progesterone deficiency plays a role in hindering bone development and promoting bone resorption.[23] In addition to the hypothalamic hypogonadal state addressed above, excess hydrocortisone secretion, low insulin-like growth factor 1 (IGF-1), and low androgen levels are often present.[14] Low levels of IGF-1 reduce the levels of osteocalcin and can cause abnormalities in osteoblasts.[23] The elevated cortisol levels are also inversely related to levels of osteocalcin.[23] All of these factors together, are likely to promote the rapid and aggressive bone loss in individuals.[23] Other signs of ED include poor dentition in BN and complaints of fatigue, peripheral edema, and bloating.[3,14]

Fluid and electrolyte imbalances are a major concern for those with ED. Laboratory results such as blood count and electrolytes should be reviewed. In more severe cases of AN, anemia, leukopenia, hypoglycemia, and hypophosphatemia have been noted.[14] The more concerning electrolyte abnormalities occur in the early stages of refeeding in patients with AN.[14] Refeeding syndrome is the clinical complications that can occur as the result of fluid and electrolyte shifts during aggressive nutritional rehabilitation of malnourished patients and can potentially be fatal when not detected or treated early in nutritional rehabilitation.[44] When reintroducing glucose into the diet it stimulates insulin secretion to help promote uptake of glucose as well as potassium, magnesium, and phosphorus into the cells.[14] Phosphorus is then used for protein and glycogen synthesis and is quickly depleted.[14] This process leads to a state of hypophosphatemia that causes adenosine triphosphate depletion which impairs muscle contraction in the heart and diaphragm resulting in cardiopulmonary failure.[14] Additionally, the increased uptake of magnesium and potassium into cells further contribute to cardiac arrhythmias and muscle weakness.[14] In BN patients, lab results can point to metabolic alkalosis, hypochloremia, and hypokalemia.[14] The frequency of hypokalemia and hypochloremia in patients who purge through vomiting has been shown to be related to the frequency of daily purging.[14] In one study, more than 40% of patients who purged twice a day or more became hypokalemic.[14,45] Hypokalemia puts them at risk for atrial and ventricular arrhythmias. Both vomiting and diuretics produce a contraction alkalosis secondary to dehydration and subsequent activation of the renin-angiotensin system.[14] Pseudo-Bartter syndrome may develop due to purging and creating a chronic state of dehydration and hypokalemia.[14] When the renin-angiotensin system is stimulated, it causes an increased amount of aldosterone to be secreted, which then attempts to restore intravascular volume via increasing sodium absorption in the distal convoluted tubule, but at the expense of potassium and hydrogen excretion. This is important to note as aggressive fluid administration can lead to rapid and severe edema.[14] Other known fluid and electrolyte imbalance complications in ED patients include decreased glomerular filtration rate, elevated blood urea nitrogen (BUN), hyponatremia, and ketonuria.[3,14]

Suicidality & Eating Disorders

There are many factors that are associated with an increased risk of suicidality or self-injurious behaviors among individuals with ED. Comorbid psychiatric diagnoses, illness severity, substance abuse, excessive exercise, child abuse, and alexithymia all have been associated with an increased risk.[46] Additionally, among age groups, adolescents show a strong relationship between suicidal behavior and completed suicides.[46] Among the different subtypes, the overall mortality risk including by suicide appears to be significantly higher among individuals with AN when compared to BN and BED. Within the subtypes of AN, 7.4% of AN-restricting types reported at least one suicide attempt whereas 20-30% of other subtypes of AN reported at least one suicide attempt.[46] However, suicide attempts seem to be more prevalent in BN than in AN.[46] Women who suffer from BN and who were alcohol-dependent report a higher rate of suicide attempts than those not alcohol-dependent.[43]

Neurochemistry

Several studies have researched how neurotransmitters and hormones are involved in disturbances across the eating disorder spectrum. In particular, research has focused on the role of monoamine systems such as dopamine and serotonin.[47] Many AN patients have high levels of anxiety, obsessive-compulsive behaviors, and harm avoidance.[47] In acute AN, patients have displayed significantly lower levels of serotonin metabolites, reduced 3H-imipramine binding, and blunted prolactin responses to drugs with serotonin activity, which suggests reduced serotonin activity.[47] Serotonin is known to influence impulse control, obsession, mood, and appetite.[47] Additionally, AN patients may have higher levels of serotonin prior to the onset of symptoms leading to a dysphoric state, so significant dieting or starvation may actually make them feel better by reducing serotonin activity in the brain.[47] It is believed that starvation in AN leads to less consumption of the essential amino acid tryptophan, which acts as a precursor to serotonin.[47] With tryptophan depletion, the brain responds by increasing the number of serotonin receptors in an effort to utilize the remining serotonin more efficiently.[47] All of this leads to a cycle where in order to feel better patients need to reduce food consumption to reduce tryptophan.[47] When they start to eat more food with tryptophan, serotonin levels rise sharply causing anxiety and emotional chaos making it extremely difficult to recover.[47] Chronic stress may also play a role as it is may increase serotonin levels creating yet another reason to diet to help alleviate the dysphoric state.[47] Different from AN, BN sees serotonin levels drop even during short periods of fasting leading to mood irritability and binge eating episodes.[47] Similar to AN, there is some evidence that suggests that disruptions in the serotonergic system may exist prior to the onset of BN.[47] In contrast, the tryptophan depletion in BN lowers mood and causes the urge to binge.[47] Further supporting reduced serotonin production, studies have shown that the activation of the serotonin receptor helps reduce binge eating.[47] When SSRI medication is introduced, patients often experience a reduction to binge.[47]

The neurotransmitter, dopamine, plays a role in movement, attention, learning, reward processing, and may be related to novelty seeking.[48] In AN, patients have reduced

concentrations of cerebral spinal fluid homovanillic acid (HVA), the major metabolite of DA.[48] BN patients, however, appear to have normal HVA levels.[48] However, binge frequency may decrease HVA levels.[48] It is hypothesized that the low DA levels in AN patients could be related to the altered reward response to food, the low motivation to approach food, and the difficulties with behavioral change during treatment.[48] This could result in impaired cognitive flexibility.[48] The different balance between DA and serotonin activity in AN and BN may contribute to the differences in overt expression between patients.[48] Further research is needed to define the role of DA in BN.

Along with disruptions in neurotransmitters, many neuropeptides are altered including neuropeptide Y (NPY), peptide tyrosine (PYY), and cholecystokinin (CCK).[48] NPY exerts an appetite stimulant signal over the hypothalamic-pituitary-adrenocortical axis increasing the adrenocorticotropic hormone, cortisol, and prolactin release, which is known to be involved in appetite regulation.[49] In as study by Galusca et. al, patients with AN were unable to upregulate NPY to adapt to their energy demand when exposed to chronic undernutrition.[50] High levels of NPY are associated with high food intake.[49] The dysregulation of these peptides may account for the erratic consummatory patterns in AN and BN.[48] Luckily, these alterations seem to normalize with recovery. CCK is a peptide hormone of the gastrointestinal system that promotes satiety, but interestingly is also related to anxiety, panic, and hallucinations. In a study by Cuntz et al., they found that AN patients had similar plasma levels of CCK when compared to healthy individuals both pre- and post-prandially suggesting hormonal adaptation.[51] Although, results have been inconsistent in prior studies.[49] PYY, a gut peptide, is secreted in the ileum an colon and plays a role in loss of appetite. Batterham & Bloom conducted a study comparing serum levels of PYY hormone between patients with BN/BED and AN.[52] Unsurprisingly, serum levels of PYY hormone were decreased in BN/BD comparative to AN.[52]

Orexins are orexigenic neural hormones that are expressed and secreted in the lateral hypothalamus as well as in peripheral tissues such as the kidney, pancreas, ilium, and colon.[49] Orexins interact with leptin by regulating neural orexigenic pathways and indirectly modulating the activity of orexigenic neurons in the lateral hypothalamus.[49] In AN patients, orexins seem to be upregulated.[49] Although further research is necessary, it is postulated that this up-regulation might result from an adaptive mechanism to increase food intake in undernourished conditions or that this chronic increase could reinforce dopamine induced anxiety further increasing the aversion to eat.[49]

Ghrelin, also known as the hunger hormone, is a peptide hormone expressed in the stomach with lesser expression in the pancreas, gallbladder, colon, and brain.[49,53] In the brain it exerts a paracrine effect by activating orexigenic NPY neurons and inhibiting anorexigenic neurons increasing appetite.[49] When the stomach is empty, ghrelin increases gastric secretion and gastrointestinal motility.[49] Ghrelin binds to its receptor, the ghrelin/growth hormone secretagogue receptor, and triggers the synthesis of NPY leading to an increase in appetite.[49] Additionally, the effects of ghrelin also involve the reward system activation via dopamine pathways.[49] Interestingly, studies have actually reported elevated ghrelin levels in AN

patients.[49] The hormone, leptin, on the other hand promotes satiety.[49] It is involved in the regulation of energy balance both in the long and short term.[49] Leptin stimulates anorexigenic neurons expressing proopiomelanocortin and corticotropin release factor while inhibiting NPY neurons.[49] AN and BN patients exhibit low levels of plasma leptin in cerebrospinal fluid pointing to the potential importance of leptin homeostasis in ED.[49]

Several interesting findings have been noted in brain imaging studies of patients with ED. MRI studies have found reduced gray and white matter in symptomatic AN and BN patients.[48] Additionally, it has been reported that in the prefrontal cortex there is a lower ratio of N-acetyl aspartate to choline, a lower ratio of glutamate to glutamine, and lower myo-Inositol concentrations in AN patients.[48] This is of note as N-acetyl-aspartate is the precursor of neuronal peptides involved in brain myelin synthesis and choline is required for neurotransmitter activity.[48] PET studies have found resting state hypo-perfusion in the frontal, parietal, and temporal brain regions in those with AN.[48] These findings reveal the neuronal membrane damage and reduced hemodynamic brain activity secondary to malnutrition.[48]

Metabolism

Eating disorders lead to metabolic changes throughout the disease state. Loss of lean body mass has been associated with significant reductions in resting metabolic rate, low body temperature, and depletion of the brain, heart, and other organ tissues.[54] Undereating, fasting, starvation and malnutrition all lower metabolic needs as the body tries to compensate for these life-threatening consequences.[54] Additionally, in this state when caloric intake is inadequate, exercise can further compound and actually contribute to lowering RMR to conserve energy rather than increasing one's metabolism.[54] Metabolic studies have shown resting metabolic rates (RMR) between 50-70% of normal RMR in both AN and BN.[54,55] In this "famine" state the body lowers its metabolic rate so that it can store as many calories as fat so that it will be ready and able to survive the next time "famine" occurs.[54] In the famine state, your body first depletes its stores of carbohydrates. Since the brain's favorite energy source is carbohydrates or glucose, the body then switches to obtain glucose from alternative mechanisms or gluconeogenesis when stores are depleted.[54] During gluconeogenesis the body starts breaking down protein and fat and converting some of the by-products to glucose, even at the expense of breaking down other body tissues to obtain energy.[54]

When a patient starts the refeeding process, RMR can actually increase through the process of weight restoration making it difficult for some to gain weight.[54] In fact, RMR has been shown to increase disproportionately in comparison to body weight or lean body mass.[54] Krahn et al., found that patients with AN exhibited a rise of 25-30% above predicted values of a variety of predictive equations.[54] In a study by Wymelbeke et al., researchers measured resting energy expenditures in 87 AN patients and found that with a goal rate of weight gain of 1/3 lb. per day, would on average require around 75 kcal per kilogram of body weight.[56] It is theorized that this hypermetabolism is related to the high calorie costs of growing lean body mass tissue, protein synthesis, muscle mass, bone formation, heart rate, heat dissipation, and restoring fat stores.[54] This is particularly important when determining energy requirements in ED patients.[54] Over

time, improved caloric rate and weight gain can normalize metabolic rate.[54]

Determining Levels of Care (LOC)

Understanding the physiological, psychiatric, and nutritional implications of ED has important clinical implications as they can affect how treatment is approached, levels of care, prognosis, and mortality. Primary treatment of ED is multi-disciplinary and determination of levels of care will be dependent on how ill or affected the individual presents.[3] The continuum of care for ED includes outpatient care, intensive outpatient programs (IOP), partial hospital programs (PHP), residential programs, and inpatient hospitalization.[34] Determining the level of care is often not a straight forward process and practitioners should consider existing practice guidelines from reputable organizations while also taking into account individual differences with regards to predicting treatment response.[34]

The American Psychiatric Association (APA) defines several factors to be considered when determining levels of care including age of patient, symptom severity, course of illness, medical status, weight as a percentage of healthy body weight, suicidality, patient motivation, psychosocial or family support, regional availability of specialized inpatient or outpatient programs and associated psychiatric comorbidity (*See Appendix 1*).[12,34,57] According to the APA, patients should first try to receive treatment at facilities within a close distance to home if at all possible, but if that is not feasible admission to a residential or inpatient facility that provides housing should be seeked.[34] Other factors needing to be considered is the amount of structure needed for weight restoration, ability to control compulsive exercising, purging behaviors, and environmental stressors. Other factors that may signal a higher level of care such as inpatient, residential or PHP include the patient being a significant risk of suicide or severe self-harm.[34] The range and extent of comorbid psychopathology can also pose challenges to treatment. For comorbid disorders such as SUD, PTSD or mood disorders, it is important to consider whether to address both disorders simultaneously and, if not, which disorder is primary to treat first.[34] However, sequential treatment may lead to relapse in one disorder as the other disorder improves.[34]

At higher levels of care where treatment is more structured, providers frequently make critical decisions regarding patient care such as imposed treatment, enforced feeding, and determination of capacity when patients are unwilling or unable to consent to treatment.[34] These environments are critical to restoring health, but it can affect motivation and autonomy.[34] Regardless of treatment setting, patient autonomy can include respecting patient choice, refraining from controlling treatment with external consequences, and helping patients become engaged in treatment.[34] Autonomy has been shown to be a significant predictor of behavior change in eating disorder patients.[34] Treatment is more effective when it is collaborative and relies on patient autonomy.[34]

Inpatient Hospitalization

Inpatient hospitalization is the highest level of care and is designed for patients presenting

with medical instability.[34] All three organizations, APA, NICE, and the Royal Australian and New Zealand College of Psychiatrists (RANZCP) provide a clear outline for determining medical stability and recommend that patients not meeting outlined requirements should be considered for admission to an inpatient level of care.[34] In adults, medical instability is specified to include a heart rate <40 bpm, blood pressure <90/60 mmHg, fasting glucose <60 mg/dL, potassium <3 mEq/L, electrolyte imbalance, temperature <97.0 F, dehydration, poorly controlled diabetes, and indication of organ compromise or a BMI of <14 kg/m^2.[34] For children and adolescents, the APA and RANZCP indicates medical instability by a heart rate near 40 bpm, orthostatic blood pressure changes (<20 bpm increase or <10-20 mmHg drop, blood pressure of <80/50 mmHg, hypokalemia, hypophosphatemia or hypomagnesmia.[34] Additionally, inpatient treatment may be necessary if the patient's symptoms such as vomiting, laxative use, or compulsive exercise are uncontrolled and require 24-hour supervision.[34] Inpatient hospitalization has subspecialty medical consultation readily available, supervised meals, and one-on-one monitoring if necessary.[34] Traditionally, these programs have 24/7 skilled nursing care, daily physician visits, subspecialist physician visits as needed, and at a minimum 1 weekly dietitian visits to oversee the patient's nutritional rehabilitation.[23] A psychotherapist may be part of the team at this level, but provides supportive sessions rather than in-depth psychotherapy.[23] Often the patient is too weak or cognitively impaired by malnutrition to engage in this type of therapy.[23] The overall goal in inpatient hospitalization focuses on medical stabilization, beginning nutritional rehabilitation, effectuating weight gain to 65-70% of IBW and then referral to an outpatient team or another specialized facility among the continuum of care for continued nutritional and weight restoration.[23]

Residential Treatment

The next level down from inpatient hospitalization are residential treatment programs.[34] These programs house patients full-time in a non-hospital-based treatment setting where patients receive meal support and multidisciplinary treatment including individual and group therapy.[34] Residential treatment typically has 24/7 nursing supervision and a minimum of 2 weekly physician visits. Similar to inpatient, clients usually have twice weekly individual psychotherapy visits, 1 weekly family therapy visit, and 1 weekly dietitian visit. Other disciplines such as occupational therapy or educators may be involved depending on the facility.[23] The average length of stay in a residential treatment is approximately 83 days, however it can vary based on a variety of factors including insurance and patient motivation.[34]

Partial Hospitalization Program

The next step down the continuum is PHP. PHP settings typically offer treatment from 6 to 11 hours a day and between 3 to 7 days a week in more of an outpatient setting.[23,34] In this setting, patients spend nights and sometimes weekends at their home allowing them to maintain social relationships as well as utilize skills they have learned throughout treatment out in the "real-world".[34] During treatment patients receive daily meals, snacks, and group

therapy as well as regular meetings with a therapist, dietitian, and psychiatrist.[34] According to Mehler et al., typical treatment includes a minimum of 1 physician and/or nursing care visit, twice weekly individual therapy session, 1 weekly family therapy visit and 1 weekly dietitian session.[23] After initial assessment, medical follow-up is provided as needed including blood draws to monitor phosphorus levels.[23] Depending on the facility and patient other disciplines may be included such as activity and occupational therapists and educators for child and adolescent programs.[23] As patient's ED behavior symptoms lessen or stop, the program will often adjust or reduce the days and hours expected of the patient in the program.[23]

Intensive Outpatient Program

IOP programs offer outpatient treatment for approximately 3-4 hours a day and between 3 to 5 days per week.[34] Similar to PHP, IOP typically includes meal support, group therapy, individual therapy, dietary sessions, and medication management.[34] At this level, physician care and family therapy is typically as needed whereas weekly individual therapy and dietetic sessions are usual practice.[23]

Outpatient Treatment

Outpatient treatment teams can vary widely. At this level, the primary responsibility is on the client and/or family to manage and locate treatment providers. Typically, initial medical and psychiatric medication evaluations are recommended during the first outpatient visit with follow-up visits with physicians and psychiatrists as needed.[14] Psychotherapy sessions often are provided once to twice per week with family or couple therapy added if indicated.[23] Depending on the severity, weekly to monthly visits with a dietitian for nutritional counseling is necessary.[23] Weight monitoring is often provided by the registered dietitian or by the medical physician.[23] With treatment providers often not under the same location, one of the most challenging parts of the multi-disciplinary treatment team is maintaining communication and providing coordinated care.[23] Of note as medical and psychiatric symptoms are typically minimal at this level of treatment, team communication is often coordinated by the psychotherapist.[23] RANZCP and NICE differ slightly on guidelines for transition to higher levels of care in that they state that transfer to higher levels of care is recommended only when a patient demonstrates non-responsiveness to outpatient care.[34] For patients not needing immediate hospitalization, treatment should be individualized and initially take place in an outpatient setting if possible.[12,34]

Overall, patients may move through the various LOC based on severity of symptoms, medical status, motivation, treatment history, and financial limitations.[34] This movement can be bidirectional based on patient needs.[34] It is important to note that stepping between LOC can be destabilizing for patients.[34] It is preferable to continue with the same treatment team while moving through the differing levels.[34] Transition planning must be done carefully and communication between programs and disciplines should be clear and ongoing throughout treatment.[34]

The Multidisciplinary Team

As ED are multi-factorial and have physical, psychological and nutritional implications the treatment team often includes at a minimum a physician, psychologist, and dietitian, although many other disciplines may be involved. Each member of the treatment team possesses unique skills and responsibilities with respect to patient care. There can be considerable overlap in what each member of treatment team does to help promote recovery from ED.[16] Patients may initially present for treatment to any of the three primary team members and the severity of eating disorder dictates the LOC and the speed of referrals to other members of the multidisciplinary team.[16]

Physician

Physicians are responsible for the assessment and initial diagnosis, treatment of any medical complications, assessment of weight and nutritional status, referral to appropriate health professionals, education of the patient and their family, prescribing medication, and providing ongoing medical monitoring.[12,16] The initial assessment should specifically include height, weight, the patient's perceived ideal body weight (IBW), body mass index (BMI) or BMI percentile for age, heart rate and blood pressure with postural measurements, temperature, assessment of breathing, edema and peripheral circulation, skin color, hydration status, the examination of head, neck, skin, hair, and nails, and muscle power.[12]

Laboratory tests should include a full blood count, urea, electrolytes, creatinine, AST, ALT, alkaline phosphatase, blood glucose, B_{12}, folate, iron workup, calcium, magnesium, phosphate, thyroid function tests, follicle-stimulating hormone (FSH), luteinizing hormone (LH), estradiol, and prolactin.[12] Other tests may include urinalysis, electrocardiography, x-rays, and bone densitometry.[12] X-rays are useful for the identification of bone age where delayed growth may be of concern. Bone densitometry is recommended after 9-12 months of disease in the case of amenorrhea.[12]

Medical history should include questions around eating behavior such as a 24-hour recall of diet intake, the number of meals per day, eliminated food groups, and binging or purging behaviors.[16] A psychological history should be obtained and include questions about life stresses, relationship issues, and problems at work or school. Since the registered dietitian and psychologist will also collect some of this information, this allows for cross-referencing of data between disciplines to determine accuracy and content at team meetings.[16] Additionally, family medical history of obesity, ED, and mental illness should be covered.[16] The physician should pay particular attention to medications, such as diet pills, laxatives, oral contraceptives, and nonsteroidal anti-inflammatory drugs as they may indicate prior amenorrhea, purging behaviors, and history of overuse injuries resulting from exercise activity.[16]

Some patients may be in denial of having an eating disorder or the severity of the disease. To help overcome this denial, the physician may visually present the laboratory and radiology

results when communicating abnormalities.[14] Although rare, forced hospitalization may be indicated in a medically unstable patient who refuses treatment. Involuntary care should be considered if the patient has a psychiatric disorder, this disorder presents a substantial possibility of death by direct or indirect action of the patient, and the patient does not recognize the need for treatment despite the life-threatening illness.[14] It is recommended that a consultation with a psychiatrist is obtained to assess the patient's competence as legalities are complex.[3] If the patient is medically stable, then it is still recommended to consult social work and psychiatry to provide resources and referrals for outpatient treatment.[3]

Psychotherapist

The psychotherapist helps monitor mental health issues and process underlying issues.[16] Psychotherapy can be delivered by psychiatrists, clinical psychologists, social workers, and master's level counselors or psychotherapists.[23] The initial meeting is critical for the mental health provider to determine how to proceed with treating the client including what psychotherapy approaches, modalities, and the length and frequency of sessions.[23] The initial assessment should include an intake interview covering the presenting problem, psychosocial history, current mental status, diagnosis, and treatment plan.[16]

The primary role of the therapist is to build a trusting interpersonal relationship with the patient.[16] They should provide the client with a review of how the treatment team works together, alleviate any fears around trust, and determine treatment goals that are patient-directed.[16] Discussion around the nature of the eating disorder, the stigma associated and confronting any moral or self-blame issues are important.[16] Although destructive, eating disorder behaviors often serve valuable functions and may act as an effective distraction from stressors.[3] These behaviors may be a patient's attempt at distress tolerance and regulation.[3] The psychotherapist plays an important role in helping the client reframe change and develop alternatives for coping with stressors.[3] Additionally, the psychotherapist is responsible for providing the patient factual information, providing structure, but also the flexibility to develop their self-identity.[3] They need to create a safe atmosphere to let the client just be.[16]

Registered Dietitian

Registered dietitians (RD) assess the nutritional status of the patient including food attitudes, eating patterns and behaviors. Specifically, they provide and implement a treatment plan, monitor nutritional status and changes in eating patterns and behaviors.[16] Nutritional assessment should include a food frequency interview along with typical daily intake. When asking the client about food-frequency of a particular food, the dietitian is also focused on inquiring information around attitudes about food rather than the accuracy of intake of that food.[16] Asking about a typical good day versus a bad day may give the dietitian valuable insight into the patient's nutritional knowledge.[16] Since patients with ED often are not forthright about volunteering information, dietitians need to ask direct questions around past and current eating patterns, compensatory behaviors such as the use of diet pills, laxatives, diuretics, vomiting, ipecac syrup, enemas, food restriction, fasting, and excessive exercise.[16] Additionally,

obtaining a weight history is important. The dietitian should work with the client by having them discuss when the patient last remembers not being concerned about food, weight, or body and then moving chronologically forward.[16] Note that although the physician is also responsible for reviewing the physical status of the patient, the dietitian should also review those same points as patients may not have disclosed the same signs and symptoms to other providers.[16] This is also an important time to assess what support system the patient has in place. If this is the first point of contact in treatment, the dietitian will need to make referrals to other disciplinary team members.[3] Additionally, if the patient is working closely with a physician, the dietitian may need to weigh the importance of obtaining an additional weight measurement during their nutrition appointment.[16] Sometimes patients experience a great deal of anxiety around being weighed and not obtaining may help reduce anxiety connected to it and the nutrition session, by allowing them to focus on nutrition and not weight.[16] They then communicate and coordinate their findings and treatment regimen to the other members of the treatment team.[16]

During treatment, one of the first priorities is to develop trust between themselves and the patient, so that the patient can feel comfortable.[16] This helps the dietitian dispel nutritional myths and hopefully help the client comply with future plans.[16] Registered dietitians are responsible for challenging the patient in a safe and structured environment.[16] Depending on the eating disorder stage, the dietitian will either work on decreasing behaviors or improving nutritional intake first.[16] This nutritional rehabilitation stage may involve weight restoration, achieving energy balance, compensatory behavior withdrawal, and discovering hunger awareness, if possible.[16] Throughout the treatment process, the RD is responsible for providing nutritional education around weight regulation, energy balance, effects of starvation, dieting myths, consequences of purging, fluid retention, and emotional connections to food behaviors.[16] The dietitian should inquire about bloating, gastric emptying, fluid retention, fullness, diarrhea, and constipation as well as monitoring for refeeding syndrome.[16] Additionally, they will be monitoring signs and symptoms as related to nutritional rehabilitation, changes in patient's energy level, social environment, mood swings, fear of weight gain, food obsession or restriction, cognitive function, symptom change, preoccupation with food, body, or calories, and awareness of hunger and fullness throughout treatment.[16] The majority of patients will benefit from vitamin and mineral supplementation. More specifics on supplementation will be addressed below.

Other Coordinated Staff & Specialists

Beyond the traditional treatment team, there are a variety of other specialties that may be part of the treatment process. Nurses working in the field may function in a variety of capacities including case managers, clinic nurses or coordinators, specialty coordinators, primary care clinicians or discharge planners.[3]

Psychiatrists are typically responsible for medication management of psychiatric conditions.[23] It is recommended that psychiatrists are brought in early in the treatment process for evaluation and determination of the complexity of medications as well as the level of

treatment.[23] Depending on the psychiatrist's background they may also deliver psychotherapy in which they would be responsible for determining the frequency of sessions.[23] Other disciplines such as family practice physicians and pediatricians may fill the role of psychiatrist depending on location and individual circumstances.[23] Conversely, psychiatrists are informed about the medical management of ED patients and may assume that role, except for ED patients with the most severe form of illness.[58] There currently is not strong evidence for psychiatric medications for ED, but better evidence for treating the comorbid psychiatric diagnoses.[23] Other team members may include social workers, activities therapists, school teachers, and coaches.[3] Activity therapy may include art therapy, equine therapy, yoga among others.

Family Members

The Academy for Eating Disorders states that families should be involved routinely in the treatment of young people with ED.[6] How this is structured is going to vary by individual and family.[6] Research points to the significant and essential role that parents may play in the treatment of ED in children and adolescents. They act as key informants in the assessment of children and adolescents, especially since minimization or denial of symptoms is common.[59] Therapies, such as family-based therapy (discussed below), assign parents with a pivotal role in their child's recovery as well as provide parents with the support they need.[59] The involvement of parents in the treatment process has been linked to a reduction in symptom severity and treatment attrition rates.[59] At the same time, it is important to remember that family members caring for patients with ED often experience distress, anxiety, and diminished quality of life and as such it is key to keep that in mind as reducing caregiver strains may be important for improving outcomes.[60]

Treatment Approaches

There are a variety of common evidenced-based psychotherapy treatments for ED including cognitive-behavioral therapy (CBT), interpersonal therapy (IPT), dialectical behavioral therapy (DBT), acceptance and commitment therapy (ACT), family-based therapy (FBT), and narrative therapy.[23]

Cognitive Behavioral Therapy

CBT is a psychotherapeutic approach to solve problems concerning dysfunctional behaviors, emotions, and cognitions through a goal-oriented, systematic procedure.[61] It helps clients to analyze and test their existing patterns of thinking, emotional reactions, and behaviors through an assessment of current difficulties and then attempt to try out new approaches, monitoring those, and evaluating its effects.[62] Research has pointed to the effectiveness of CBT in preventing relapse after weight restoration in adults with AN.[63] Additionally, CBT is considered the most effective treatment for BN and BED, although treatment should be individualized and other psychotherapy treatments may be utilized.[64,65] Several studies have shown that CBT has helped reduce the number of binge eating episodes as well as ceasing binge eating overall.[66-68] For dietary restriction, the course of CBT treatment is deliberately sequential. For example, a

practitioner might first target developing a regular, more normal pattern of eating such as 3 meals and 2 or 3 snacks.[69] Then start to incorporate those "bad" or forbidden foods into the patients' meal patterns.[69] This is meant to help empower them and prove that normal eating won't lead to loss of control.[69] Treatment is designed for a slow and gradual increase in the overall amount of food consumed and thus caloric intake.[69]

Interpersonal Therapy

IPT is considered a time-limited (around 12-15 sessions), affect-, life event, and present-focused psychotherapy that emphasizes interpersonal relationships in the context of symptoms. Interpersonal problems are common in patients and may predate the onset of ED, which is one of the reasons IPT is thought to be beneficial in the treatment of ED. In the setting of IPT, the therapist and patient work together to define the central interpersonal problem which may fall into one or more categories related to lack of intimacy and interpersonal deficits, interpersonal role conflicts, role transitions, major life changes, complicated grief, and life goals that may be interfering with interpersonal functioning. In a multi-center study comparing CBT with IPT in BN patients, researchers found that CBT was superior at the end of the treatment with regards to normalizing eating attitudes and behaviors, however, in the long-term follow-up, there were no differences in results.[70,71] In another study, researchers compared IPT and CBT in BED patients and found that recovery was similar for both CBT and IBT at the end of treatment and at the one-year follow-up.[71,72] For patients with BN in which CBT has not seemed to be effective, the APA guidelines recommend IPT.[64]

Dialectical Behavioral Therapy

DBT works on the premise that maladaptive and impulsive behaviors function as a way to regulate unwanted emotions and focuses on developing adaptive skillful behaviors to help improve effective self-regulation.[73] It is believed eating disorder patients often present with issues related to emotional under control such as in BED and BN while others may present with emotional overcontrol (AN). With a wide range of behaviors such as binging/purging and food restriction, there is a growing interest in applying DBT as a way to develop adaptive skillful behaviors in place of those maladaptive behaviors.[74] There are three primary DBT models for eating disorders. The Stanford model proposes that binging and/or purging occur as a way to regulate painful affect specifically around emotional deficits found in patients with BN and BED.[74] The radically-open DBT (RO-DBT) theorizes that temperamental threat sensitivity interacts with feedback from the environment that emphasizes self-control and minimizes mistakes. This results in the inability of the individual to feel safe. Patients with AN restrictive type are associated with heightened threat sensitivity, low reward sensitivity, loneliness, and inhibited emotional recognition. As such, RO-DBT is based on openness, flexibility, and connectedness and as such teaches skills that target these issues of overcontrol that are thought to underlie their problematic behaviors, emotions, and cognitions.[74] The third model, multi-diagnostic complex ED DBT (MED-DBT), was created to address multi-diagnostic, highly complex individuals with ED that require higher levels of care.[74] This model combines CBT with DBT to manage multiple high-risk behaviors (i.e. suicide) in the context of emotional

dysregulation and therapy interfering behaviors.[74] This model focuses on patient responsibility and flexibility in defining and achieving treatment goals.[74] Overall, DBT is effective for the treatment of BED and initially effective with BN.[74] The evidence does point to a particularly good fit with complex presentations of ED and comorbid disorders.[74] However, although research has generally produced positive results, the extent does vary. As a general rule, it is recommended that DBT be considered when CBT has not been effective or emotional regulation is hindering treatment effectiveness in those with BED and BN.[74]

Acceptance & Commitment Therapy

ACT works towards training individuals to actively and openly contact their current experiences fully present without defense and either change or remain in the behavior as it serves the person's chosen values.[75] The purpose or goal of ACT is psychological flexibility leading to valued living.[75] It is comprised of six components including cognitive diffusion, experiential acceptance, transcendent self-awareness, valued living, and committed action.[75] ACT teaches patients to obtain psychological distance from distressing internal experiences through clarifying overarching personal values, creating goals that help patients live a more fulfilling and meaningful life, and increase their willingness to experience negative internal experiences in service of valued behavior.[76] ACT is thought to be effective, because, at the core, ED have a devastating impact on valued living.[75] Along those lines, the purpose of treating ED is to increase contact with the client's values.[75] ACT targets the broad behavior shift not in the sense of reducing behaviors, but by changing the dominant functions of behavioral repertoire and shifting life towards things that the client cares about.[75] It is important to note that ACT is not currently regarded as an evidence-based treatment for ED, however, some studies have suggested that it leads to symptom improvement in patients with AN.[77] Additionally, in the few studies on ACT in eating disorder patients, the effectiveness of treatment does not outperform other evidenced-based treatments such as CBT.[77]

Family-Based Therapy

Considered the gold standard intervention for adolescents with ED, FBT integrates techniques from a variety of psychotherapy approaches including systemic, strategic, narrative, and structural family therapy.[78] The theory highlights the importance of incorporating patients' interpersonal relationships into treatment approaches.[78] There are three phases to FBT. In phase 1, parents are assigned the responsibility of correcting the adolescents eating behaviors and low weight.[59,78] In the second phase, once behaviors have been reduced, control over food consumption is transferred back to the adolescent.[59,78] Once normal body weight is achieved and eating behaviors are normalized, the third phase begins focusing on more general issues of adolescent development such as creating a healthy adolescent-parental relationship, the adolescent's identity, and autonomy.[59,78] A key aspect of FBT is to separate the ED from the patient to enable the parents to take action against the illness rather than their child[78] Although FBT is individualized to the client and family, generally phase 1 includes a minimum of weekly therapy sessions that last around 3-4 months, phase 2 then reduces sessions to every second or third week, and phase 3 transitions to sessions every third week or monthly

intervals.[78] There has been particularly strong evidence for the effectiveness of FBT in the treatment of AN in adolescents.[63] However, as each patient, family, and illness are unique, FBT may not be effective in all cases. NICE recommends CBT when the treatment is ineffective or contraindicated.[78]

Exposure Therapy

Exposure therapy operates under the principle that stepwise exposure to the item promoting fear via introducing stimuli attempt to break the avoidance pattern, thereby allowing the patient to learn by experience that the feared consequences do not occur.[79] There are four common forms of exposure therapy: exposure with response prevention for purging (ERP-P), exposure with response prevention for binging (ERP-B), exposure-response prevention for AN (AN-ERP), and body image exposure.[79] ERP-P focuses on the prevention of binging or purging in a patient with BN or AN binge/purge.[79] Since eating elicits a fear of weight gain and purging is often used to help cope with the associated anxiety, the purging behavior reinforces the removal of fear of weight gain.[79] In ERP-P the client is gradually exposed to fear food cues and prevents the purging after consumption.[79] The idea is that this method helps the client's self-efficacy and bolsters their ability to cope with stressors.[79] Limitations of ERP-P include that it doesn't take into account any cues that might precede binging and doesn't emphasize binge eating as a maintaining factor in disordered eating.[79] Additionally, ERP-P can be logistically complicated and time consuming.[79]

ERP-B, also known as cue exposure, hypothesizes that if a conditioned stimulus predicts binging and elicits a physiological response such as craving, then repeated and/or prolonged exposure to the stimulus in absence of binging will result in less or complete removal of the craving.[79] For a patient with BED, professionals may expose the patient to binge foods via touch, smell, and small tastes while preventing the binge.[79] Studies have shown ERP-B to produce positive results in terms of reduction of symptoms for clients who had not improved with CBT or pharmacological treatments, thus making ERP-B a good second-line treatment for those individuals.[79] In a large RCT, researchers combined CBT sequentially with ERP-P, ERP-B, or a relaxation condition and found that at a 5-year follow-up both ERP groups showed greater improvements in BN symptoms than the combined CBT and relaxation treatment.[79] In AN-EXRP, patients with AN use graded food exposure to help reduce avoidance and facilitate habituation to anxiety around anticipation and ingestion of the "fear" food.[79] The aim is to increase food choice flexibility and caloric consumption leading to an improved ability to restore and maintain weight and prevent relapse.[79] Although a few limited preliminary studies on AN-ERP have shown positive results, many had very small sample sizes that confounded the results.[79] Body image exposure has been used to reduce body dissatisfaction, body checking, and avoidance in patients with ED.[79] This process often incorporates other modalities such as CBT for body image disturbance and cognitive dissonance-based mirror exposure.[79] This exposure therapy often uses mirrors or videos to encourage individuals to look at each part of their body systematically for an extended period of time.[79] This may occur while wearing tight-fitting clothing with additional exposure homework assignments.[79] Although research is limited, there is preliminary evidence in the short-term that clients experience improvement in

a low mood, self-esteem, body image, body checking, and weight or shape concerns.[79] Further supporting the effectiveness of body image work, Murray et al. suggest that increased psychological flexibility around body image is associated with reduced symptomology severity, greater quality of life, and improved mental well-being.[2] Overall, there is limited research on exposure therapy outside of inpatient or residential settings and this limits the ability of the research to decipher if the improved result is due to the food exposure therapy or other treatments.[79] Larger, more robust studies are needed to evaluate the treatment in these patients.

Narrative Therapy

Narrative therapy explores factors that have contributed to the meanings that the patient has constructed about their life experiences and identifies the knowledge held by the patient concerning the influence of the eating disorder in their life as well as the factors that support or undermine that influence.[80] At the same time, it allows the patient to reflect on ways of resisting or challenging the eating disorder and uses that as a foundation for further behavioral change.[80] It operates under the premise that the client holds insider knowledge about living with the eating disorder that other professionals do not possess.[80] It is a collaborative process between the client and the therapist, where the therapist is not seen as the "expert".[80] A major part of narrative therapy is the process of externalization. It challenges the internalizing or individual pathological language and argues that ED arise out of cultural, familial, and social relationships and experiences.[80] This process of externalization creates a space or separation between the patient and the eating disorder by engaging in conversations that position the self in relationship to thoughts, feelings, experiences, and actions.[80] This is key as many patients with ED view themselves as the problem rather than adopting an identity separate from the eating disorder leading to guilt and self-blame.[80]

Group Therapy

Group therapy incorporating various modalities such as CBT and IPT may be utilized throughout the treatment process. Group psychotherapies include peer interpersonal feedback, social learning, emotional expression, and group cohesion that may be particularly useful in targeting interpersonal problems for some individuals.[81] According to Grenon et al. found that participants who underwent group psychotherapy were 5.51 times more likely to abstain from binge eating and or purging than those not undergoing group psychotherapy.[81] The APA practice guidelines for the treatment of patients with eating disorders and the APA Task Force on Evidenced-Based Practice both include group CBT and IPT as the two psychotherapies that have shown strong evidence for BN and BED.[81]

Intuitive Eating

Considered the founders of intuitive eating (IE), Evelyn Tribole and Elyse Resch, state that intuitive eating is the dynamic mind-body integration of instinct, emotion, and rational thought.[82] As only you can be the expert of your own body, thoughts, feelings, and experiences, it relies on the personal process of honoring health by paying attention to what

our bodies tell us in order to meet our physical and emotional needs.[82] Intuitive eating is guided by 10 principles: 1) reject diet mentality; 2) honor your hunger; 3) make peace with food; 4) challenge the food police; 5) respect your fullness; 6) discover the satisfaction factor; 7) honor your feelings without food; 8) respect your body; 9) exercise-feel the difference; and 10) honor your health.[82,83] These principles were designed to help guide body attunement or the ability to hear and respond to physical sensations and by removing those obstacles standing in the way to body attunement.[82] Overall, intuitive eating is about building a healthy relationship with food, body, and mind while focusing on self-care and body appreciation regardless of body size.[82] The effectiveness of IE in the treatment of ED has shown some promising results. Research has found that IE has been associated with improved trust in one's body being able to monitor food requirements and that it may lead to closer adherence to recommended caloric requirements.[83] Although opinions vary, this may be important for those with AN as they present with restriction of energy intake relative to their requirements.[83] In a 2-year pilot study, researchers found that patients with AN have the ability to learn to intuitively eat in a highly structured treatment setting in conjunction with other medical, nutritional, and psychological considerations. Additionally, they found that IE was associated with reductions in symptomology, depression, anxiety, social conflict and improvements in body image and spiritual well-being.[83] Similar reductions in symptomology and improvements in attitude have been found in experimental studies around IE and disordered eating such as binge eating.[83-85] Patients with BN often have difficulty recognizing and responding to satiety cues making IE a particularly important intervention.[83] In a study of patients with BN, researchers found that patients were able to improve their ability to eat intuitively.[83] IE was linked with decreases in binging episodes and increased feelings of self-control.[83] However, research on the long-term efficacy is limited in the eating disorder population.

Health at Every Size

Health at Every Size is an interdisciplinary movement that focuses on health promotion through supporting health behaviors for all body sizes without using weight as a focus.[86] It focuses on encouraging body acceptance rather than weight loss or weight maintenance, utilizing IE to focus on our internal cues, and actively moving in a way that feels good.[86] Recently, HAES has gained momentum in the eating disorder community. Currently, the Academy for Eating Disorders, Binge Eating Disorder Association, Eating Disorder Coalition, National Eating Disorder Association, and the International Association for Eating Disorder Professionals support the HAES approach as standard practice in the ED setting.[86] Several evidence-based studies have shown that the HAES approach is associated with statistically and clinically relevant improvements in blood pressure, blood lipid levels, physical activity, eating disorder pathology, mood, self-esteem, and body image in overweight individuals and those that experience binge eating.[84,87-89] As prospective studies have linked body dissatisfaction with binge eating and other eating disordered behaviors, it is believed that by learning to accept your body, cueing into your internal hunger and fullness cues, and reducing the focus on weight that it will lead to more normalized eating pattern among patients.[86] However, there is a need for research on the effectiveness of HAES as it relates to the subtypes of eating disorders.

Pharmacology & Supplementation

Pharmacology

Many patients may benefit from medication so that they can more fully participate in treatment, although it is always important to individualize treatment.[14] Antidepressants are the most frequently prescribed followed by antipsychotics in children and adolescents with AN.[64] Pharmacological management is often utilized when comorbid psychiatric disorders such as depression, anxiety, and delusions.[64] The use of atypical antipsychotics in patients revolves around the rationale that patients with ED can have irrational beliefs or delusions around body shape.[64] Additionally, they can assist in managing anxiety and depressive symptoms also present in ED.[64] In AN, atypical psychotics, such as olanzapine, risperidone, and quetiapine, are generally regarded as safe and acceptable, however evidence of enhanced weight gain in AN is limited.[64] Olanzapine when compared against other antipsychotics was shown to promote more rapid weight gain and improved AN psychopathology including a reduction in depression, anxiety, obsessive-compulsive symptoms, and aggression.[64] The APA suggests the utilization of atypical antipsychotics may be useful in individuals with severe or treatment-resistant symptoms.[64]

Antidepressant medications have not been found overly effective in the treatment of AN. It is believed that this is due to the depletion of central serotonin that occurs with weight loss as well as the inadequate intake of tryptophan, a precursor of serotonin.[64] The symptoms of starvation may coincide with or mimic features of depression and these symptoms are often improved by improving nutritional status.[64] However, selective serotonin reuptake inhibitors (SSRIs) may play a role in treating the comorbid depression and anxiety symptoms after weight restoration.[64] Guidelines currently recommend a trial of SSRI antidepressants as an initial or alternative treatment in BN in conjunction with CBT.[16] SSRIs, including citalopram, escitalopram, and sertraline, have demonstrated the strongest efficacy in reducing binge eating episodes and improvement in other clinical indicators in the BED population.[64] These are indicated as a first-line pharmacotherapy by the World Federation of Societies of Biological Psychiatry for BED.[64] Tricyclic antidepressants (TCAs) are commonly prescribed medication for adolescents with AN to help with comorbid depression, obsessive-compulsive symptoms, self-harm.[64] However, there are some serious concerns in this population related to the potential for TCAs to cause arrhythmias at a low body weight.[64] In the cases of AN, there currently is no clear evidence on the addition of pharmacotherapy to psychotherapy, except in the cases of comorbid psychiatric conditions.[64] The TCAs, imipramine, amitriptyline, and desipramine, have displayed some efficacy in BN with a reduction in binge frequency, depression, and anxiety in RCT, however, they are not tolerated well.[64] Due to their adverse effects and overdose toxicity, they are not recommended as a first-line treatment for adults with BN and is discouraged in children and adults.[64] Fluoxetine is recommended as a first-line medication in BN as it has shown to prevent BN psychopathology, depression, and help to prevent relapse in large sample randomized control trials.[64] Although research is limited on TCAs in BED, there is some promise in the reduction of binging, however, they don't seem to promote weight loss, so they are not

recommended as a first-line treatment.[64] Another class of antidepressants that have shown some promise in the reduction of BN symptoms are monoamine oxidase inhibitors or MAOIs. However there are significant side effects and some dietary restraints associated.[64] When comparing TCAs, MAOIs, and SSRIs no one class outperformed in regard to efficacy and rarely lead to complete symptom remission and as such are inadequate as a monotherapy.[64]

Among several anti-epileptic drugs tested in RCT, topiramate was the only drug that has exhibited good efficacy as it relates to the reduction of binge and purge episodes.[64] However, it has several side effects such as sedation, dizziness, headache and paresthesia which limits its tolerability among patients.[64] Among the BED population, the anti-epileptics, topiramate, and zonisamide have been proven to reduce binge frequency and weight loss, but similar to the BN population, their side effects lead to a high discontinuation rate limiting their clinical effectiveness.[64] Research has also looked the effectiveness of utilizing the pancreatic lipase inhibitor, orlistat, in the BED population.[64] The evidence showed an overall decrease in binging and improved weight loss with mild to moderate gastrointestinal issues.[64] One dietary item to keep in mind is that Orlistat may affect the absorption of fat-soluble vitamins and negatively affect growth in adolescents.[64]

Overall, the use of medication in the treatment of ED should be used as part of a multi-modal treatment plan incorporating in nutritional, medical and psychosocial interventions and not solely as a monotherapy.[64] Studies on various medications have shown limited efficacy across ED.[64] Clinicians should use psychological and behavioral multidisciplinary approaches as the front line treatment in ED with medication as an additive treatment for those where more support is necessary. Close monitoring of medications is required and if deemed ineffective should be reviewed and adjusted as a priority.[64]

Supplementation

Food intake in eating disorder patients can be unpredictable and can lead to neurobiological dysfunction. Studies have highlighted several nutrient deficiencies, including electrolytes, vitamins, and minerals that may play a role in this dysfunction.[90] Ideally, a person should obtain adequate nutrients via a varied diet. In an eating disorder patient, one may need to offer nutrient supplementation in addition to the diet as consumption may be an issue. As always, close monitoring is recommended.

Patients with ED typically do not have deficiencies in the essential fatty acids, however, deficits in long-chain polyunsaturated fatty acids, such as omega 3 or omega 6 have been demonstrated.[90] Omega 3 fatty acids have demonstrated some control in inflammation and oxidative stress, so a deficiency in omega 3 fatty acids could be related to dysfunction.[90] This inflammatory dysfunction and increased oxidative stress have been shown to be a marker of ED.[49,90] As such, it is hypothesized that supplementation of polyunsaturated fatty acids may prevent neuronal damage and act as an anti-inflammatory agent.[90] Several studies have shown the usefulness of omega 3 fatty acid supplementation with regards to significant improvement in symptoms.[90-92] Although more research is needed in this area, most studies to recommend

treatment with omega 3 fatty acids.[90]

Electrolyte abnormalities can be a common consequence of purging behaviors such as vomiting or abuse of laxatives.[90] Excessive vomiting is known to cause hypokalemia and or hypochloremic alkalosis.[90] Laxative abuse has been linked to hypomagnesemia and hypophosphatemia.[90] Hypocalcemia is common in patients with ED.[90] Often blood plasma levels of calcium are not accurately representative of calcium levels as the body tries to maintain homeostasis via calcium from the bone to compensate for deficits leading to poor bone density, which is a better indicator of long-term calcium deficits.[90] For calcium deficiency, supplement with calcium carbonate up to a total of 1500 mg per day elemental calcium from food and supplement.[93] As these electrolytes are critical, emergency supplementation is recommended based on individual needs.[90]

The lack of nutrition in the diet often leads to abnormally low plasma iron levels.[90] In addition, erythrocyte hemolysis can contribute to iron deficiency in patients who are heavy exercisers.[90,93] Interestingly, amenorrhea may offer some protection from iron deficiency.[90,93] Replenishment of iron stores is a long-term process so supplementation and monitoring may be required even after food intake is normalized.[90,93]

Results have been mixed with regards to vitamin A supplementation.[90,93] Elevated serum levels of vitamin A have been attributed to inadequate intake of other nutrients that are required for vitamin A metabolism whereas decreased serum levels were associated with inadequate vitamin A intake.[90,93] Vitamin A is common in many fruits and vegetables that ED patients tend to eat, however, if the diet is low in fats, absorption can be hindered.[90,93] Inadequate zinc intake may also impair vitamin A absorption and metabolism.[90,93] Based on existing literature, it would be premature to recommend supplementation beyond the recommended daily allowance (RDA).[93]

Deficiency in several B vitamins is common mostly due to inadequate food intake although comorbid SUD in the form of alcohol may contribute to deficiency.[90,93] Food restriction can lead to low thiamine (B_1), riboflavin (B_2), pyridoxine (B_6), folic acid (B_9), and cobalamin (B_{12}).[90,93] Thiamin deficiency can cause varied neuropsychological symptoms including worsening of depression symptoms, a common comorbid condition.[90] B_6 deficiency may exacerbate ED because of its link to serotonin metabolism.[90] Similar to B_6, thiamin may also exacerbate psychiatric symptoms.[90,93] We also know that deficiency in cobalamin or B_{12} can produce symptoms of anemia and long-term damage can lead to brain damage.[90,93] Currently, no recommendation is available for supplementation of B_1, B_2, B_6, and B_9 beyond supplementation at the RDA.[93] Most commonly, Vitamin B_{12} is often supplemented via an intramuscular injection of 1 mg of cyanocobalamin per month.[93]

Vitamin C deficiency is rare in the ED population as it is available in a variety of fruits and vegetables.[90,93] Bleeding gums or poor wound healing may point to a deficiency as can over-exercise with poor dietary intake.[93] If a deficiency is detected, supplementation above the RDA (up to 400 mg per day) is appropriate until urinary excretion indicates tissues are saturated.[93]

Vitamin D deficiency is likely in patients when there is limited dairy intake and/or have little to no exposure to sunlight.[90,93] A study on AN patients found a strong link between vitamin D values and bone mineral density and as we know AN is associated with loss of bone mass. Currently, there is no consensus on supplement strategy in the ED population.[93]

Studies on zinc deficiency in eating disorder patients have been variable.[93] Zinc deficiency may be related to low dietary intake, especially in vegan patients or in the case of heavy exercise which can contribute to impaired zinc absorption.[90,93] Initial laboratory tests taken after a period of weight loss may not accurately reflect the status as zinc is released from catabolized tissue.[90,93] It can be particularly troublesome in eating disorder recovery as it can negatively affect an underweight patient from gaining weight, especially muscle mass.[90,93] Additionally, as mentioned above, zinc deficiency can inhibit vitamin A metabolism leading to high serum carotenoid levels.[93] It can also give a sense of depression and cause hypogeusia making it unappealing to eat.[93] Zinc supplementation may be helpful even for patients that have slightly low serum zinc levels.[93] In a double-blind trial, supplementation of 100 mg per day of zinc gluconate resulted in twice the rate of increase in BMI compared to the placebo.[93] It is important to monitor food intake, because as preferences change and patients incorporate zinc-rich foods, supplementation may no longer be advised.[93]

Tryptophan, an essential amino acid and precursor to serotonin, has become of interest in the treatment of ED. Since tryptophan is an essential amino acid, one must obtain it through their diet. When diet intake is inadequate a deficiency may occur leading to lower levels of serotonin.[90] Studies have suggested that response to antidepressants can be hindered if there is a deficiency in tryptophan.[90] Supplementation of tryptophan is not recommended as monotherapy but as an adjunctive treatment.[90] Supplementation aims to normalize the neurobiological mechanisms involved in the disorder and also make the pharmacological treatment of serotonergic drugs more effective.[90]

Along with tryptophan, some have started looking at the possibility of supplementing arginine, another essential amino acid that is often deficient when intake is low.[90] Arginine has particular relevance in the synthesis of nitrous oxide, which plays a role in vasodilation.[90] Since mortality of ED is sometimes related to cardiovascular issues, studies have focused on investigating the role of arginine in patients with ED.[90] Currently, arginine supplementation is lacking scientific backing.[90]

Other Medical & Nutritional Protocols

Long term goals for treatment revolve around normalized eating behaviors, nutritional adequacy, and medical stability.[3] Patients tend to respond better to short term goals and will vary based on the level of treatment and individual.[3] For instance, the first goal for a patient in an inpatient setting might be vital sign stabilization and to increase weight to >75% of their IBW.[3] With regards to determining body weight goals, many methods are used. In a survey spanning 100 treatment providers, a variety of approaches were used including consulting a

patients' growth curve history, utilizing BMI standards, relying on physiological markers such as the return of menses, consulting patients for their preferred goal weight, and making inferences based on behavioral symptoms.[2] It is imperative that each member of the treatment team be on the same page with regards to goal weight.[2] As patients work toward their goal weight, the typically acceptable weight gain in the outpatient setting for adolescents is 1 kg (2.2 lbs.) a month.[3] If there is a lack of progress in the outpatient setting, such as agreed to weight gain or continued purging, referral to a more specialized or higher level of care may be necessary.[3]

Throughout the weight restoration and refeeding process, there are a wide variety of treatment effects to be watchful of. First, as severe malnutrition can result in decreased cardiac muscle mass, the ability of the heart to contract is reduced.[14] As a consequence, intravenous fluids should be done carefully as to not overload the weakened heart and precipitate pulmonary edema.[14] Additionally, for those that have electrolyte imbalances as may be the case with purging, the first line of treatment should be to focus on the correction of dehydration.[58] This is essential to decrease aldosterone and limit renal potassium and hydrogen losses.[58] Note that in the cases of purging, completely turning of aldosterone may take a few weeks after cessation of purging.[58] To combat the propensity toward edema, spironolactone, a potassium-sparing diuretic may be used for up to 1 to 2 weeks.[58] To prevent refeeding syndrome, the current recommendations are to start the refeeding process slowly.[58] Intake levels usually begin at levels around 600-1000 kcal per day and are increased by 300-400 kcal every three or four days, with the caveat that it should be individualized based on the rate of weight gain.[58] The caloric requirements necessary for weight gain can vary between 1800-4500 kcal per day.[58] There are a few general rules to follow: 1) The TEE should never exceed BEE, 2) caloric intake should rarely exceed 70-80 kcal per kilogram of body weight, 3) for a severely malnourished patient begin a diet at 20-25 kcal per kg, 4) protein intake should not exceed 1.5-1.7 grams per kilogram of body weight and is generally in the 1-1.5 gram range, 5) if TPN or enteral feedings are required, carbohydrate intake should not exceed 7 mg per kilogram per minute, and 6) weight gain should be in the 2-3 pounds per week. Liquid supplementation may need to be prescribed to achieve the caloric goal needed to achieve weight gain.[58] Caloric intake must be modified throughout refeeding, because of changes in the REE.[58] Checking for edema is important as it can be a sign of refeeding syndrome.[58] In addition, in the early phases of refeeding, insulin secretion normally increases causing sodium retention by increasing kidney tubular sodium reabsorption.[58] If this is noted, a low-sodium diet should be part of the nutritional plan.[58] As always, frequent and close monitoring of a patient's blood chemistry including at a minimum potassium, phosphorus, magnesium, sodium, and glucose is a must.[58] It is recommended daily lab draws be obtained early on until they are more normalized.[58] Other metabolic complications may include elevated liver enzymes, thiamin deficiency, and hypoglycemia. Slight elevations in AST and ALT, two liver enzymes, are typically noted in the first few weeks of refeeding followed by increases in alkaline phosphatase and bilirubin.[58] There is little clinical significance usually and they typically resolve with a slowing of the rate of the refeeding process.[58] If elevations are significant, the RDN may need to reduce carbohydrate-dextrose calories.[58] Thiamin deficiency in the acute refeeding period is life-threatening as it may cause congestive heart failure or Wernicke's encephalopathy and must be monitored.[58] Additionally,

atrophy of intestinal mucosa and pancreatic impairment may cause severe diarrhea.[58] Hypoglycemia may be an issue as the large glucose loads in more aggressive refeeding regimens may stimulate insulin release from the pancreas that the depleted hepatic glycogen reserves cannot offset.[58] This is particularly important if the patient is on enteral feeds or TPN as abrupt cessation can cause dangerous hypoglycemia.[58] Lastly, delayed stomach emptying, and prolonged colonic transit time is often noted in AN.[58] A bowel regimen with a judicious amount of fiber and hydration may help with these symptoms.[58] For those with a history of laxative abuse, rebound constipation may occur. It is recommended to use a non-stimulant laxative such as polyethylene glycol and to abstain from fiber-based products as they may cause bloating.[14]

Coordination & Communication

Regardless of treatment setting, coordination, and communication of findings and treatment among disciplines is critical. Comparing information ensures comprehensive and cohesive care. It is essential to communicate the patient's level of motivation and participation in treatment, treatment goals, and progress or ambivalence about them.[16] Topics or general themes in psychotherapy and circumstances or events that could complicate progress may also be noted.[16] From a dietitian's perspective, gaining information on a patient's psychological state may highlight the potential for introducing nutritional concepts or changes at the right time.[16] Furthermore, when a food behavior is connected to a particular emotion, the dietitian can encourage the client to process it with their psychologist.[16] From a physician standpoint, communicating the patient's medical status and stability, weight, lab data, medical risks, current ED behaviors, medication adjustments, effectiveness, and any pertinent side effects to members of the team lets the team know where they are from a medical stability standpoint.[16]

If communication is not well coordinated and clear among all team members, it can lead to patient splitting.[16] Splitting is the phenomenon when a patient forms an alliance with a member of the multi-disciplinary treatment team in opposition to the other members threatening the overall effectiveness of treatment.[16] Differences in opinions or perspectives among disciplines are why the treatment approach is most effective.[16] It is common for patients to make progress on physical variables that the physician might see, but also show little progress in psychological behaviors.[16] Likewise, if a patient's physical and nutritional condition deteriorates, it may derail any positive psychological changes.[16]

Barriers to Treatment

Medical Perspective

Several barriers have been identified via qualitative studies. Linville et al highlighted seven themes for practitioners avoiding eating disorder screening during appointments.[94] Respondents comprised of physicians, nurse practitioners, and psychologists, expressed reluctance due to lack of time, interdisciplinary role confusion, lack of treatment options, perceived helplessness of provider, provider discomfort, education, and difficulty of treating

ED.[94] Seventy-eight percent of the respondents reported having ED patients in their practice, but they felt unsure how to properly screen and treat them.[94] Highlighting the need for more education, one participant noted that they had one lecture on ED in their psychiatry rotation. Further supporting the need, a national survey of university medical and psychology programs found didactic teaching hours among medical programs ranged from 1.94 – 5.25 hours, while general psychiatry and child and adolescent psychiatry ranged between 4-4.05 hours.[95] Early identification and diagnosis are affected by the competence of professionals in the eating disorder field and the earlier we identify the better for the prognosis.[12] There is a desire for more specifics about understanding appropriate diagnostic screening tools, prevalence rates, risk factors, height and weight norms for athletes and non-athletes, and eating disorder referral sources.[94] Additionally, clarification on the multi-disciplinary roles and boundaries is necessary. Role confusion between disciplines can result in back and forth referrals that can prevent or delay interventions.[94] Researchers highlighted that medical providers often think that ED are a mental health issue and mental health professionals can tend to view ED as a medical issue.[94] There are also several myths around ED that participants felt affected providers' ability to screen and provide an intervention. They include that ED are a mental issue and doctors are not part of the treatment team, ED are just a phase, individuals with ED have a personality disorder and doctors can't help anyway.[94] One participant noted that they were trained to think that there was nothing they can do for eating disorder patients and to just refer them to mental health practitioners.[94] Reasons noted for the difficulty of treating disorders were lack of patient motivation, family denial, patient discomfort, confusion with the treatment process, lack of referral sources, and frequent patient relapses and setbacks.[94] All respondents of the Linville et al. study noted a need for medical training programs to offer better training, continued education, and consultative forums on eating disorder topics and in particular recommended hands-on training, a brief screening tool, and a collaborative team.[94]

Another area affecting treatment is confusion around the role of the family in the treatment process in the child and adolescent population. Marcon et al. found that while physicians responded with a fair degree of accuracy regarding assessment, their responses regarding knowledge of treatment were not in line with best practice guidelines.[59] In particular, only 10.5% of participants correctly endorsed the statement that it is the parents' responsibility to bring their child to recovery than the child.[59] Additionally, 79.2% agreed that children with ED will never get better until they receive some sort of individual therapy.[59] Researchers noted that physicians lacked knowledge around the nature of the involvement of parents and provided conflicting messages to families involved in FBT.[59] This can be problematic when children and adolescents with ED are already not accepting of family involvement with meal support and the refeeding process.[59] This further supports the need for evidence-based protocols, curriculum development, and training. It also points to the need for some cross-training as it relates to the importance of FBT, specific ways for parents to become involved and education around effective family-orientated treatment strategies.[59]

Dietitian Perspective

A representative survey of US dietitians working in the ED field indicated a need for expanded

education on ED and disordered eating for RDs in pediatrics and other practice areas.[96] They noted educational gaps around the role of genetics in the development of ED, etiology, prevention, identification, and care for patients exhibiting distorted thoughts and maladaptive behaviors with food and body, and counseling processes.[96,97] The RDs surveyed noted that their formal education usually only included a brief introduction to ED with little information about how to treat them.[96] If treatment information was provided it was outside of their nutrition coursework and did not cover nutritional interventions.[96] This lack of education spanned undergraduate, coordinated programs, dietetic internships, and graduate coursework.[96] Additionally, they noted that greater experience in the field and education covering the psychological aspect of ED are needed across health professions including RDs counseling children and adolescents for weight management.[96] It's important to note that without training in nutrition-focused treatment modalities, the RDN may be less effective in changing eating behaviors, which may delay the initiation of more effective treatment with an ED specialist.[96] There is a need for research on the comparative effectiveness of nutrition therapies.[96] Other research gaps revolve around predictors of recovery and methods to facilitate early recognition and referral.[98] A study by Whisenant and Smith found that the reasons dietitians who do not work with ED and cannot identify them was attributed to lack of education, training, or experience, lack of awareness of psychological issues, denial of the eating disorder by the patient, orientation to the medical model of therapy that focuses on structured diets, and dietitians themselves having ED.[98] Moreover, psychological issues with patients and their families can present a major barrier to dietitians.[98] Knowledge of counseling techniques for patients and their families can be beneficial when educating about food and weight behavior.[98] The researchers also noted that universities and internship programs have a responsibility to ensure every dietitian is capable of properly screening for, identifying, and referring patients with ED to the appropriate specialists.[98]

Patient Barriers

Patients have reported that they are more likely to be blamed for their disorder and to be perceived as attention-seeking.[99] Studies among university students and adult women have highlighted that seeking treatment may be considered a weakness and that the perception from others is that ED are not real illnesses.[100,101] Also, patients report feelings of shame and embarrassment around their illness and treatment.[102] Another area of concern is the reported lack of family support or lack of understanding from their support circle as some prefer to seek help from informal sources such as family and friends.[103] Building this support system to encourage treatment may have a major impact on the treatment process.

Studies have also mentioned that individuals with ED do not always consider or understand the severity of their illness, which can prevent them from seeking help.[104] Some more practical barriers include the cost of treatment, transportation, and lack of time for treatment.[101] Rural areas may lack the necessary resources for treatment leading to the need to travel to find specialized treatment providers.[104] From more of an internal eating disorder belief system, patients report a fear of gaining weight, losing the positive aspects of their ED, and losing part of their identity.[105] Perceived positive aspects may include mood regulation, control, and

comfort.[105] For some patients, they perceive that their eating disorder is an integral part of their identity and that life without it is unimaginable.[105] Their eating disorder provides a sense of structure and meaning.[105] There is also a lack of knowledge around available resources. In a study by Cachelin et al., they highlighted that participants did not understand where to go to treatment nor what type of treatment exists for treating their eating problems.[101] There also is a sense of distrust of treatment professionals among various communities. Becker et al. highlighted how social stereotypes can act as a barrier to care. When compared to white participants, African Americans, Hispanic, and Native Americans were significantly less likely to receive a referral to care.[100] Additionally, participants reported that clinicians did not believe that ED occurred in certain ethnic groups such as African Americans.[100] This along with negative attitudes towards professional treatment, doubt, and fear around treatment, and personal characteristics of the provider such as language barriers or lack of providers with similar ethnic backgrounds can further impede treatment.[100,101,104]

Financial Cost & Insurance

Inpatient treatment for ED costs on average $30,000 per month while intensive outpatient varies between $500 to $2000 per day depending on demographics.[106] As of 2016, there were only 10 states that require private insurers to cover treatment for AN and BN but OSFED was left off coverage.[106] A study in 2010, by Horgan et al., found that 22.4% of plans did not cover services for ED.[107] Insurance carriers place restrictions on mental health care such as lower caps on physical health care and limiting the number of days covered in an inpatient treatment program.[106] Some insurers claim that inpatient and IOP treatment for BN and OSFED are not medically necessary and that there is not enough evidence on effective treatments pointing to the need for more research.[106] Additionally, many of the treatment facilities may be out of network leading to higher out of pocket expenses.[108]

Conclusion

With multiple physical, nutritional, and psychiatric implications, eating disorders significantly decrease the quality of life of those affected and require skilled treatment professionals. The etiology of eating disorders includes genetic, biological, individual, family, psychological, and socio-cultural factors.[3] Each discipline of the treatment team has unique skills and responsibilities concerning their area, however, there is overlap in what each member of the treatment team does to promote recovery. Additionally, the lack of formalized ED screening, education, and training among health practitioners potentially adds to continued misconceptions, poor diagnosis, and prognosis. All health practitioners should be educated and trained in screening, diagnosis, and referral resources. As health practitioners require years of education and didactic internships, there is an opportunity to address this gap in the university and collegiate systems as well as health facilities.

Methods

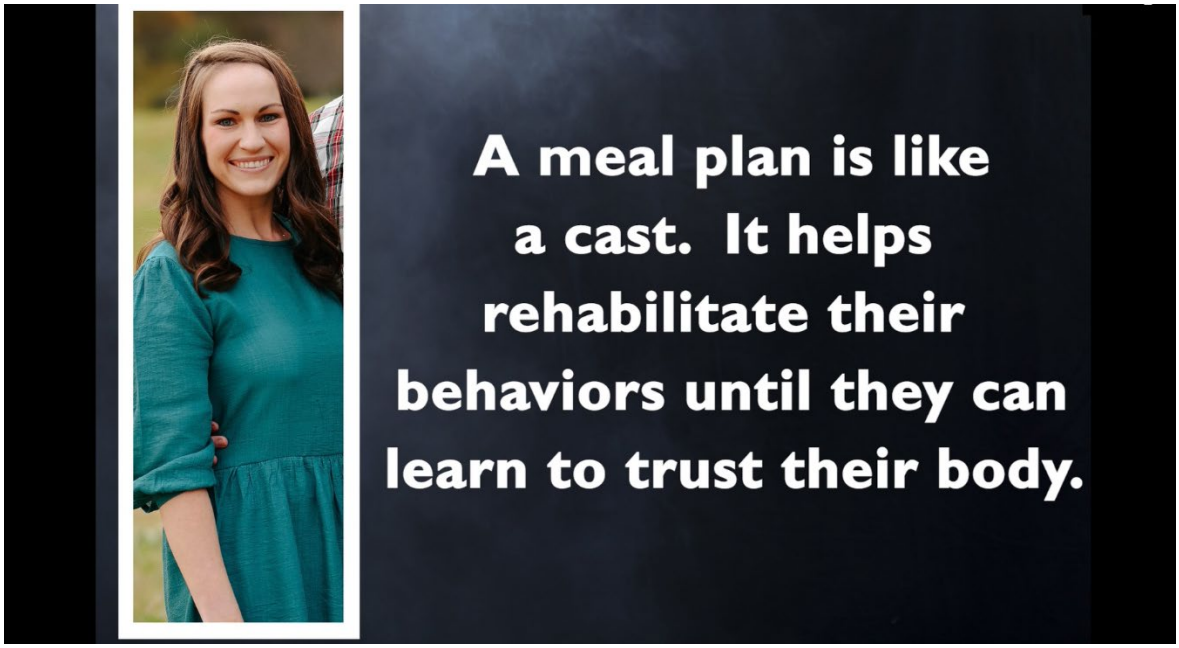
Video interviews were conducted of 12 healthcare providers, family members, and recovered patients in the eating disorder community either in person at EDIN's corporate office or over ZOOM. Each interview lasted between 30-60 minutes and contained between 10-20 questions. After interviews were completed, one video was created based on the feedback from the interviewees. Editing was completed utilizing Adobe Premiere Pro software. The video was approved by the Executive Director of EDIN and uploaded to their website.

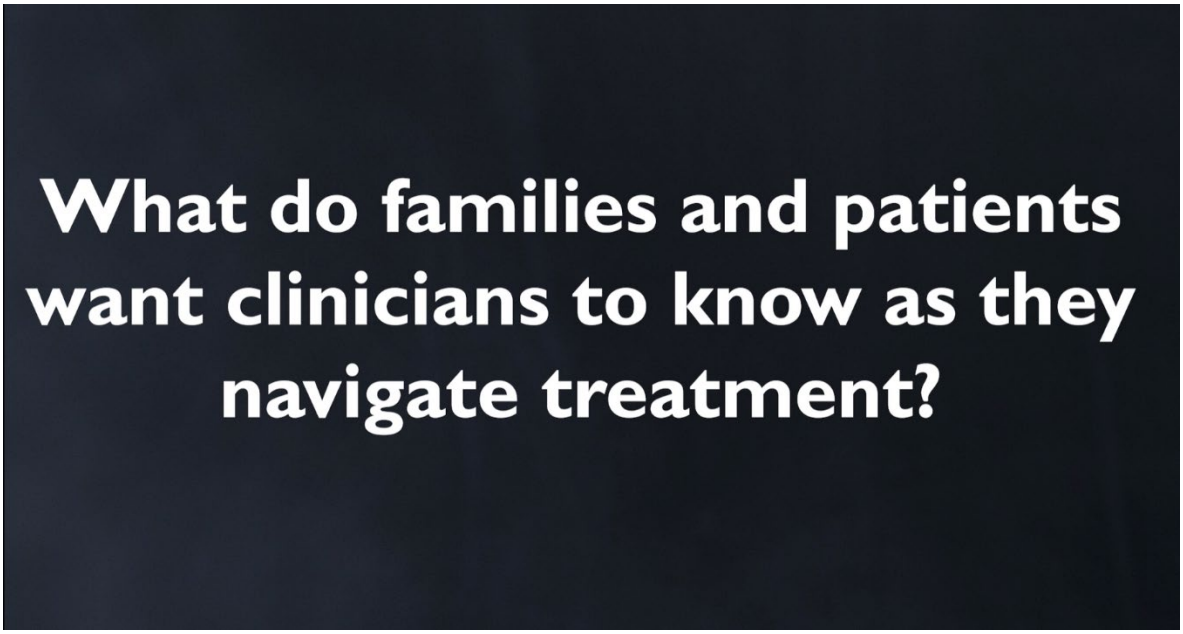
Appendix 1: LOC Guidelines

Guidelines for Levels of Care for Patients with Eating Disorders					
	Outpatient	**IOP**	**PHP**	**Residential**	**Inpatient**
Medical Status	Stable – extensive monitoring not needed			Medically stable (not requiring IV fluids, NG tube feedings, or multiple daily lab results	**Adults:** HR <40 beats/min; BP <90/60 mm HG; glucose < 60 mg/dL; potassium <3 mEq/L; electrolyte imbalance; temperature <97.0F; dehydration, hepatic, renal, or cardiovascular organ compromise, poorly controlled diabetes **Children and Adolescents:** HR near 40 beats/min; orthostatic blood pressure changes, BP <80/50 mm HG; hypokalemia, hypophosphatemia, or hypomagnesemia
Suicidality	If suicidality is present, inpatient monitoring and may be needed depending on level of risk				Plan with lethality or intent; admission of suicide attempt depending on presence of other factors modulating suicide risk.
Weight (based on % of healthy body weight)	>85%	>80%	>80%	<85%	<85%; acute weight decline with food refusal even if not <85%
Motivation	Fair to good	Fair	Partial; cooperative; preoccupied with intrusive repetitive thoughts >3hrs/day	Poor to fair; preoccupied with intrusive repetitive thoughts 4-6 hrs/day; cooperative with structured environment	Very poor to poor; preoccupied with intrusive repetitive thoughts; uncooperative with treatment or cooperative only in highly structured environment
Comorbid disorders	Presence may influence LOC				Any existing psychiatric disorder that would require hospitalization
Structure Needed	Self-sufficient		Some structure needed	Needs supervision at all meals or will restrict	Needs supervision during and after all meals or NG/special feeding needed
Ability to Control Compulsive Exercise	Ability to control	Can manage through self-control	Some degree of external structure required to prevent patient		
Purging Behavior	Can greatly reduce incidents in unstructured setting; no significant medical complications			Can ask for and use support from others or use CBT skills to inhibit purging	Needs supervision during and after all meals and in bathrooms; unable to control multiple daily episodes of purging that are severe and disabling, despite trial of outpatient care even if lab results are normal
Environmental Stress	Often able to provide adequate emotional and practical support and structure		Others able to provide at least limited support and structure	Severe family conflict or problems or absence of family so unable to receive structure environment at home, or patient lives alone without adequate support system	
Geographic Availability of treatment	Patient lives near treatment setting			Treatment programs are too distant for patient to participate from home	

HR = heart rate; BP = blood pressure; IV = intravenous; NG = Nasogastric; mm HG = millimeter of mercury; hrs = hours
Adapted from Anderson LK, Reilly EE, Berner L, et al. Treating Eating Disorders at Higher Levels of Care: Overview and Challenges. *Curr Psychiatry Rep.* 2017;19(8):48

Appendix 2: Video Screenshots





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