

Physical activity in patients with anorexia nervosa

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Anorexia nervosa (AN) is often associated with physical hyperactivity. Recent studies have established links between anorexia and hyperactivity, suggesting the existence of commonalities in neural pathways. How physical activity should be managed during the clinical care of patients with anorexia remains controversial. This review first focuses on the implication of hyperactivity in the pathophysiology of AN. Hyperactivity during refeeding of patients with AN has been associated with increased energy needs to achieve weight gain, poorer clinical outcome, longer hospitalization, and increased psychiatric comorbidity. This typically leads to the prescription of bed rest. However, current knowledge suggests that preserving some kind of physical activity during refeeding of patients with AN should be safe and beneficial for the restoration of body composition, the preservation of bone mineral density, and the management of mood and anxiety. In the absence of standardized guidelines, it is suggested here that physical activity during refeeding of patients with AN should be personalized according to the physical and mental status of each patient. More research is needed to assess whether programmed physical activity may be a beneficial part of the treatment of AN.

INTRODUCTION

Anorexia nervosa (AN) is characterized by difficulty maintaining minimal weight, fear of gaining weight, and a distorted body image; it is also often associated with denial. The diagnosis is based on criteria from the Diagnostic and Statistical Manual of Mental Disorders.¹ Two subtypes have been described: (1) the restricting subtype, with primarily loss of weight through significant reductions in caloric intake; and (2) the binge-eating subtype, with recurrent binge eating and purging through self-induced vomiting or laxative misuse. Heterogeneity in the clinical picture is the rule; patients can have mixed disease subtypes and they can transition from one type of eating disorder to another.^{2,3}

AN is a severe disease. Mortality rates of 5%–10% at 10 years have been reported, making it the psychiatric disorder with the highest mortality,⁴ although more

recent studies report less severe mortality rates.⁵ The prevalence of typical AN is approximately 0.2%–0.9% in the general population, and AN predominantly affects adolescent and young females.⁶ Atypical forms of AN are more prevalent but seem to share the same outcome.⁷ AN is associated with multiple and severe somatic complications related to malnutrition, including bradycardia, hypotension, anemia, hormonal imbalance, and low bone mass.⁸ AN is also associated with significant psychiatric comorbid conditions, including anxiety, depression, and obsessive-compulsive disorders, as well as considerable impairment in psychosocial functioning; this all contributes to the persistence and global burden of the disease. AN patients typically feature obsessive preoccupations about food, body shape, and body weight and engage in highly ritualized behaviors such as counting calories, frequent weighing,

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Key words: anorexia nervosa, eating disorders, physical activity.

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and excessive physical activity, referred to as hyperactivity.^{9,10}

To define hyperactivity related to AN, it might be useful to consider 3 dimensions of exercise behavior: (1) frequency or amount of exercise (>1 h/day, 6 days/wk during >1 mo), (2) obligatory exercise (feeling bad if unable to exercise up to a certain amount), and (3) motivation for exercise (control of body weight).^{11,12} There is no consistent cut-off used to define what is considered excessive physical activity in terms of duration, frequency, and intensity in contrast with healthy physical activity. It has been suggested that what makes exercise excessive in AN is not the amount but, rather, the obligatory nature of the activity.¹³ Hyperactivity in AN is characterized by a significant amount of physical activity combined with a compulsive need to exercise. Thus, the compulsive element of unhealthy physical activity has been reported to be a better predictor of disordered eating than excessive quantity of exercise.^{14–16} Furthermore, in the literature on AN, various terms are used, such as “excessive physical activity,” “compulsive exercise,” and “hyperactivity” to indicate unhealthy and excessive activity related to the disease.

“Physical activity” refers to any bodily movement produced by skeletal muscles that requires energy expenditure and includes recreational or leisure-time physical activity, transportation (walking or cycling), occupational activity (work), household chores, play, games, sports, or planned exercise in the context of daily, family, and community activities. It is well established that physical activity has substantial health benefits by reducing the risk of coronary heart disease, stroke, diabetes, hypertension, colon cancer, breast cancer, and depression.^{17,18} The World Health Organization has released evidence-based physical activity guidelines that recommend at least 150 minutes of moderate-intensity aerobic activity, or 75 minutes of vigorous-intensity aerobic activity per week, or an equivalent combination of both.¹⁹

“Exercise” is a subcategory of physical activity that is planned, structured, repetitive, and purposeful in the sense that the improvement or maintenance of one or more components of physical fitness is the objective. “Exercise” and “exercise training” frequently are used interchangeably and generally refer to physical activity performed during leisure time with the primary purpose of improving or maintaining physical fitness, physical performance, or health.

Physical activity is a key determinant of energy expenditure and, thus, is fundamental to energy balance and weight control. Swartz et al.²⁰ have quantified the total energy expenditure of 3 different durations of physical activity within a 30-minute sedentary period in a healthy population and examined the potential

benefits of interrupting sedentary behavior with physical activity for weight control. Results showed an additional expenditure of 24, 59, or 132 kilocalories/day if the participant stood up and walked at a normal, self-selected pace for 1, 2, or 5 minutes every hour, respectively, compared with sitting for the 8-hour period. Thus, when physical activity is increased, there is also an increase in the consumption of energy, which suggests that levels of physical activity should be matched with dietary intake. Most studies relating physical activity with diet have reported that being physically active is associated with healthy nutritional choices.^{21,22} However, AN is associated with both 1) extreme energy restriction with selective exclusion of fats and carbohydrates in the diet and 2) increased total amounts of physical activity to control body weight.

The pathophysiology of AN remains debated, but increasing evidence suggests that AN is a complex, neurobiologically based, psychiatric disorder. Studies have underlined the contribution of heritable genetic risk factors but failed to clearly identify susceptibility genotypes.^{23,24} Data have been accumulated to support a dysregulation of the serotonergic²⁵ and the neuropeptidergic pathways²⁶ and, more recently, a dysfunction of the gut–brain axis.²⁷

The first part of this review summarizes data regarding the common implication of hyperactivity in the anorectic process and the perpetuation of the disease. The second part opens the debate about the potential benefits of some physical activity during the course of the disease and its clinical management.

METHODS

Briefly, study identification began with an electronic search of the PubMed database. The subject headings used in the search were “anorexia nervosa,” “eating disorders,” “hyperactivity,” “physical activity,” and “exercise.” Secondary searches consisted of manually searching the reference lists of all identified articles.

HYPERACTIVITY AS A FEATURE AND RISK FACTOR IN ANOREXIA NERVOSA

Frequency of hyperactivity in anorexia nervosa

Hyperactivity is a common feature of eating disorders. Indeed, 31%–80% of patients with AN exhibit hyperactivity, depending on the phase (onset or acute) and the severity of the disease.²⁸ Whether excessive activity is related to a specific form of AN remains unclear: excessive activity was more frequent in the purging subtype of AN in 1 study,²⁹ whereas Dalle Grave et al.³⁰ found that hyperactivity was associated with the

restricting subtype of AN. Globally, hyperactivity is considered to be a compensatory behavior used to control weight, in association with or instead of dieting or purging.

Hyperactivity as an early symptom of anorexia nervosa

Hyperactivity is a frequent symptom in the course of AN.^{31–33} Moreover, premorbid activity levels have been shown to be predictive of later excessive activity.²⁸ Indeed, hyperactivity in women may be an important risk factor for developing an eating disorder.³⁴ Moreover, a large longitudinal study reported that the level of physical activity during AN treatment and after recovery was an important modulator of the body composition recovery mechanism.³⁵ This suggests that physical activity may play a central role in the pathogenesis and progression of AN.

Furthermore, recent experimental studies established a link between AN and hyperactivity, suggesting the existence of commonalities in neural pathways, most likely in the nucleus accumbens, a brain structure involved in the reward system and feeding behavior.³⁶ These data support the interest of clinical researchers in studying AN patients to investigate the possible contribution of hyperactivity to the pathophysiology of AN.

Negative impact of hyperactivity during the treatment of anorexia nervosa

Hyperactivity and outcomes in anorexia nervosa patients. There is some evidence that hyperactivity is associated with more physical problems, including osteoarticular or tendon trauma and increased energy needs to achieve weight gain.³⁷ Accordingly, routine clinical practice is based on the assumption that weight gain could be accelerated and, therefore, hospitalization length of stay decreased, by restricting activity during refeeding of in-patients with AN. In a 3-year inpatient hospital record analysis, increased hospitalization duration was found for AN patients with hyperactivity.³⁸ Beyond hospital stay, 2 prospective studies have reported that hyperactivity was associated with higher probability of chronic outcome.^{39,40} In a recent prospective study of adult female inpatients, reduced eating disorders psychopathology was correlated with reduced physical activity dependence scores.⁴¹ Finally, hyperactivity has been reported as one of the predictive factors of higher risk of relapse after recovery from AN.⁴²

Hyperactivity and obsessive-compulsive disorders. Hyperactivity during refeeding of patients with AN is frequently associated with other comorbid conditions

such as obsessive-compulsive disorders (OCDs) and with pronounced obsessive-compulsive personality traits such as rigidity, restraint in emotional expression, and greater impulse control.⁴³ Some authors even consider that AN with hyperactivity is close to an OCD phenotype.^{44,45} In a large group of patients with AN, the hyperactive group had greater OCD symptoms than the nonhyperactive group but did not differ on body mass index.⁴⁴ Davis et al.⁴⁶ proposed a model in which hyperactivity, starvation, and obsessive compulsiveness are reciprocally and dynamically related, with each factor creating a destructive bidirectional loop that is resistant to change and difficult to break, leading to a self-perpetuating circuit and maintenance of eating disorders in a certain subgroup of patients. Moreover, hyperactivity and OCDs share some neurochemical characteristics involving serotonin metabolism.^{47,48} This has important therapeutic implications in that targeting hyperactivity with adequate pharmacological, behavioral therapy, and/or programmed physical activity (PPA) might contribute significantly to the improvement of AN.⁴⁹

Impacts of hyperactivity on psychological status and quality of life. Some studies have reported that AN patients with hyperactivity report higher levels of anxiety,^{50,51} depression, and perfectionism,^{29,52} higher levels of anhedonia,⁵³ higher self-esteem but lower body esteem,⁵⁴ and lower levels of reward dependence and novelty seeking.³⁰ This raises the question whether hyperactivity induces anxiety or anhedonia, or the contrary. Some data suggest that hyperactivity might be an adaptive and addictive behavior set up by the patient to alleviate anxiety and depressive symptoms through the release of endogenous opiates or dopamine, among other neurobiochemical mediators elicited by hyperactivity.⁵¹ Finally, Keyes et al.⁵⁵ recently reported that the drive to exercise in AN patients is aimed more at improving body tonicity than health and enjoyment, which suggests that eating disorders pathology is also an important contributor to the drive to exercise in AN patients.

In conclusion, available data suggest that maintained hyperactivity during the course of AN is an indicator of poor outcome and can be related to OCD symptoms, leading to a self-perpetuating loop (Table 1).^{37–40,44} In this regard, hyperactivity represents a specific therapeutic target, similarly to other comorbid conditions. During active in-hospital treatment of AN, persistence of hyperactivity probably should be avoided; however, the routine suppression of any physical activity in hospitalized patients is not supported by the available clinical data.

Table 1 Negative impact of hyperactivity during treatment of anorexia nervosa

| Reference | Sample size | Age (years) | Protocol, duration | Negative effects of physical activity |
|---|-------------|-------------|--|--|
| Kaye et al. (1988) ³⁷ | n = 11 | 25 ± 3 | Acceleration-sensitive device attached to the belt Exercise allowed ad libitum during the hospitalization | Increased energy needs for weight gain Increased cost of hospitalization |
| Solenberger et al. (2001) ³⁸ | n = 115 | 20.6 ± 7 | Retrospective analysis, 3-y inpatients hospital record analysis | Increased hospitalization duration |
| Strober et al. (1997) ³⁹ | n = 95 | 15 ± 3 | Comprehensive face-to-face interviews at 6-mo intervals from the point of discharge through the first 5 y, annually thereafter until completion of follow-up | Higher probability of chronic outcome |
| Casper and Leslie (1996) ⁴⁰ | n = 75 | 19 ± 8 | 8 y follow-up Face-to-face interviews with a follow-up questionnaire | Factor in chronic evolution of anorexia nervosa |
| Davis et al. (2006) ⁴⁴ | n = 50 | 25.4 ± 9.1 | Excessive exercise: >4 h/wk Questionnaire both at admission and at discharge following weight restoration | Excessive exercisers (>6 h/week with patient's perception of being "out of control") had greater obsessive-compulsive disorder symptoms than nonexcessive exercisers |

POTENTIAL BENEFITS OF PROGRAMMED PHYSICAL ACTIVITY FOR ANOREXIA NERVOSA PATIENTS

Combined programmed physical activity and refeeding during disease-related malnutrition

Physical activity has repeatedly been shown to be associated with reductions in the risk of any-cause mortality and reductions in major causes of death, such as cardiovascular disease and cancers.^{17,18} There are currently recommendations regarding the practice of physical activity in patients with chronic disease, diabetes, obesity, and cardiovascular pathology. Furthermore, regular physical activity is also important in the prevention and treatment of mental disorders.^{56,57} In many conditions of malnutrition related to chronic organic diseases, such as chronic obstructive pulmonary disease, cancer, chronic renal failure, or cardiac cachexia, evidence has accumulated over recent years demonstrating that associating a physical rehabilitation program with refeeding is beneficial for achieving a significant weight and, moreover, functional gain.^{58–61} Indeed, preservation or restoration of lean body mass is a key determinant of outcome, performance, and quality of life. In cancer patients, a close link between sarcopenia (low lean body mass) and mortality, as well as between sarcopenia and chemotherapy toxicity, has been established.⁶² Globally, the purpose of any nutritional intervention should be to increase muscle anabolism and decrease proteolysis. Malnutrition results from complex and multifactorial mechanisms, including inflammation, anorexia/malnutrition, and alterations of protein and lipid metabolism leading to cachexia.⁶³ Its management requires,

therefore, a multimodal approach that addresses these different mechanisms and includes nutritional supplementation, pharmacological agents (anabolic hormones, anti-inflammatory drugs, specific nutrients), and an appropriate physical exercise program,⁶⁴ which will enable the nutritional supply to translate to lean body mass gain.

PPA is a supervised therapeutic program integrated into the multidisciplinary treatment of AN patients; it utilizes personalized schedules that aim to limit the harmful consequences of hyperactivity and optimize the expected benefits of physical activity. Little is known about the potential benefits of PPA during the treatment of AN, but this topic has raised some recent interest in the field of physiotherapy and sports medicine.^{65,66} The following paragraphs discuss prospectively which benefits might be expected of PPA, in analogy with nutritional strategies used in other malnutrition states, and how PPA could influence several determinants of AN and its consequences.

Impact of programmed physical activity on body composition in anorexia nervosa

Usually, the 2-compartment model used in studies to assess body composition in AN separates fat mass and fat-free mass, which requires the stability of body hydration level and bone mineral content.⁶⁷ AN has a severe impact on body composition.⁶⁸ Alterations are mainly represented by an extensive loss of fat mass but also by a loss in fat-free mass, depending on the level of physical activity maintained by the patient, vomiting, laxative abuse, and diet.^{69–71}

Clinical data indicate that controlled physical activity during refeeding of patients with AN is safe and does not impair weight gain.^{72,73} An early case report suggested that physical activity may even reinforce weight gain.⁷⁴ However, in a pilot study of a graded exercise program, there were no significant differences in weight gain and body fat in 5 exercising patients vs 7 nonexercising patients.⁷⁵ Another study reported a better increase of body mass index (BMI) in exercising patients at 1-year follow up (from $18.8 \pm 0.5 \text{ kg/m}^2$ at baseline to $21.7 \pm 0.5 \text{ kg/m}^2$; $P < .001$).⁷⁶ Chantler et al.⁷⁷ also demonstrated that adding low-intensity resistance training to the standard care of patients with AN over the course of 8 weeks resulted in a higher BMI ($+2.3 \text{ kg/m}^2$, $P < .006$ vs $+1.3 \text{ kg/m}^2$, $P < .001$) and fat mass percentage ($+2.6\%$, $P < .01$ vs $+1.9\%$, $P < .001$) compared with standard care alone. Szabo and Green⁷⁸ reported that after 8 weeks of treatment that either included or did not include resistance training, body mass, body mass index, and fat body mass increased significantly in both exercising and nonexercising groups, with no statistical difference in these parameters.⁷⁸ Interestingly, the increase of lean body mass was significantly higher in the exercising group ($4.2 \text{ kg} \pm 3.4 \text{ kg}$ vs $2.11 \text{ kg} \pm 1.00 \text{ kg}$; $P < .05$). Whether the gain in lean body mass during refeeding of patients with AN may be further increased by some specific nutritional intervention, as reported in studies of cancer-related malnutrition,⁷⁹ needs investigation.

Two studies reported that fat gain is mainly located in the visceral area in AN patients confined to bed rest during refeeding.^{80,81} This clinical observation is experienced adversely by the patients and contributes, in addition to frequent digestive disorders,⁸² to enhanced stress about abdominal discomfort and dissatisfaction about body shape, which can favor resistance to refeeding and relapse.^{42,83} Thus, it is hypothesized that the inclusion of physical activity during refeeding would influence the body fat distribution. However, clinical studies that include regional body composition measurements are needed to test this hypothesis.⁶⁵

Impact of programmed physical activity on exercise capacity in anorexia nervosa patients

Only one study has evaluated the effects of physical activity in AN patients on exercise capacity (endurance time, oxygen uptake, oxygen consumption, peak oxygen consumption, and heart rate) at baseline and at a 1-year follow-up in an exercising group ($n=9$) vs a control group ($n=8$).⁷⁶ Indices of exercise capacity at the 1-year follow-up were significantly improved in the exercising group as compared with baseline, whereas

the increase did not reach statistical significance in the control group.

Exercise capacity is dependent on the muscular-skeletal system, which is deeply affected in AN. Indeed, as previously reported by McLoughlin et al.,⁸⁴ muscular damage is reflected in reduced muscle mass and function, characterized by skeletal myopathy in patients with severe AN. Despite the importance of the recovery of muscle tissue and function,⁸⁵ recovery in AN patients has focused on total body weight, BMI, and fat mass. Therefore, muscle mass repletion after weight recovery seems to be incomplete,⁸⁶ which leads to diminished functional capacity in the form of muscle strength.⁸⁷ Furthermore, low-intensity exercise by AN patients has been shown to have a harmless effect on body composition and to improve muscular strength and agility.^{72,88} Chantler et al.⁷⁷ have shown that adding low-intensity strength training to the standard care of patients with AN resulted in an increase in the peak torque of their knee extensors ($P < 0.001$), knee and elbow flexors ($P < 0.0001$), and elbow flexors ($P < 0.01$) compared with standard care alone.⁷⁷ Fernandez-del-Valle et al.⁸⁹ have also studied the effects of a high-intensity resistance training program, designed for adolescents to improve strength and agility, in AN patients. Performance on leg-press, bench-press, and lateral row tests improved significantly ($P < 0.001$) in test patients after 8 weeks of training compared with controls. Beneficial effects on agility were also observed in the participating AN patients; however, further studies are needed to determine long-term effects of such training programs ($>2 \text{ mo}$) and to better understand muscle function recovery in AN patients.

Physical activity and bone health

Low bone mineral density (BMD) is a frequent complication in AN. In adult women with AN, more than 90% have T-scores lower than -1 , and close to 40% have T-scores lower than -2.5 at one or more sites.⁹⁰ Adolescent girls with AN are also at high risk for low bone density. Indeed, more than 50% had BMD Z-scores lower than -1 at one or more sites.⁹¹ Furthermore, low BMD often persists even after recovery from the disease, with serious implications for future skeletal health.⁹² Impaired bone metabolism in AN is multifactorial and includes hormonal changes, lack of estrogen, calcium and vitamin D deficiencies, and hypercortisolemia.^{90,93} Moreover, reduction of anabolic effect of muscle contraction on the bone, which is directly related to lean body mass, has also been implicated.⁹⁴ Conventional bed rest imposed on patients with AN is probably detrimental to BMD because immobilization is a well-established risk factor for bone loss.⁹⁵ Conventional treatment of low BMD during AN

relies mainly on compensation of calcium–vitamin D depletion, but has a limited efficacy in the absence of efficient refeeding.⁹⁶ The benefit of bisphosphonates for the treatment of BMD during AN has not been established.⁹⁷

A few studies have suggested that moderate exercise may be protective against osteoporosis in women with AN, whereas pathological hyperactivity may be harmful.^{98,99} It is generally accepted that exercise helps in maintaining BMD in postmenopausal women and increases BMD of the spine and hip in women with osteopenia and osteoporosis.¹⁰⁰ Therefore, some authors have hypothesized that, through increasing lean body mass, physical activity may improve BMD during AN management to compensate, in part, for the hypogonadism. Furthermore, as shown by Bratland-Sanda et al.,¹⁰¹ the type of physical activity has to be adapted to goals (e.g., gain of mineral content or maintenance of BMD) and to the patient's progress and needs. Finally, supplementation with estrogens during AN may be considered, but benefits have not been documented.¹⁰² Thus, prospective and controlled studies are needed to better understand how PPA may influence BMD evolution during the treatment of AN and may be integrated in a multimodal preventive or curative treatment of low BMD.

Impact of programmed physical activity on psychological status and quality of life in anorexia nervosa

The effect of physical activity as a treatment option for mental disorders is best documented for depression and anxiety,^{103,104} which are common comorbid conditions in individuals with eating disorders. As discussed above, anxiety is frequent during AN, and hyperactivity may be used by the patients as an anxiolytic addictive behavior. It has been previously shown that regular and moderate physical activity can reduce anxiety symptoms in healthy adults.¹⁰⁵ In a previously mentioned study, increased gain of lean body mass by resistance training was associated with better psychological well-being, as assessed by a significant decrease in the Beck Depression Inventory score from 21.9 ± 17.4 to 10.1 ± 9.9 .⁷⁸ In another study, AN patients who attended a supervised physical activity program during treatment exhibited a reduction in exercise compulsivity and dependence.¹⁰⁶ A meta-analysis of randomized controlled trials in different group of psychiatric patients with anxiety disorders or symptoms reported a marked, significant reduction of the level of anxiety.¹⁰³ This beneficial effect of physical activity on anxiety may be explained by an increased norepinephrine neurotransmission in the central nervous system, serotonin synthesis, and secretion of atrial natriuretic peptide.¹⁰⁷

Positive effects of PPA on depression have also been reported.¹⁰⁸ Whether PPA may have a beneficial effect on anxiety and depression in AN patients, and may, therefore, contribute to a better outcome, needs further investigation.

Thus, several studies have reported that physical therapy intervention in AN patients may reduce scores of eating pathology (restrictive symptoms, weight preoccupation, body dissatisfaction, drive to thinness) compared with standard care alone.⁷² Brewerton et al.¹⁰⁹ reported that AN patients with higher activity had a lower frequency of binge eating, vomiting, and laxative use than those who did not exercise. Apart from PPA, some other body-oriented strategies might be of interest. The addition of a massage program resulted in significantly lower overall Eating Disorder Inventory scores in patients with AN ($P < .05$) after 5 weeks.^{110,111} Adding basic body awareness exercises also resulted in lower overall Eating Disorder Inventory scores ($P < .05$).¹¹² Carei et al.¹¹³ have demonstrated that 8 weeks of yoga resulted in significantly lower Eating Disorder Inventory scores ($P < .05$). All of these adjunct therapies may contribute to the reduction of stress and related anxiety and hyperactivity.

Other benefits of PPA might be expected in AN patients, such as favoring social integration to counteract the negative effects of the disease on self-esteem and educational or professional activities.¹¹⁴ Indeed, it is well-known that physical activity enhances quality of life through social interactions resulting from group participation and time spent outdoors,¹¹⁵ whereas quality of life is markedly impaired in AN patients, both in the physical and psychological dimensions.¹¹⁶ Indeed, PPA had been reported to have positive effects on quality of life.⁶⁶

Limitations of studies on programmed physical activity

A previous review⁷² demonstrated that the existing literature on PPA in AN is still difficult to interpret due to significant methodological shortcomings. Indeed, exercise training programs (resistance training, stretching, yoga, aerobic exercise, and anaerobic exercise) are heterogeneous, and only a few of the referred studies have reported details about physical activity assessment. In addition, several studies have used self-reported physical activity only, which does not exclude either persistent hyperactivity or poor compliance with the PPA program. Moreover, heterogeneity among the studies, particularly with regard to the duration, frequency, and intensity of the experimental intervention, the supervised or nonsupervised programs, and the diversity of eating disorder subtypes studied together, makes it impossible to define any dose–response relationship. Also,

only a few of the referred articles have measured the impact of PPA in AN on muscle-related variables.^{77,89} These shortcomings limit overall conclusions and highlight the need for further rigorous research.

CLINICAL IMPLICATIONS FOR MANAGEMENT OF PHYSICAL ACTIVITY IN PATIENTS WITH ANOREXIA NERVOSA

Hyperactivity is a frequent and early symptom in the course of AN, and its persistence is predictive of a poorer outcome; thus, this compulsive and obsessive behavior

should be considered as a therapeutic target. The available literature supports the therapeutic potential of PPA in AN management (Table 2).^{75–78,89,98,106,111–113} There is some rationale for expecting PPA to benefit AN patients with regard to body composition, BMD, symptoms of anxiety/depression, and quality of life (Figure 1), but robust clinical data are still awaited.

Physical activity assessment

Various instruments are available to evaluate exercise behavior in individuals with eating disorders. Actimetry

Table 2 Potential benefits of programmed physical activity during anorexia nervosa

| Reference | Sample size | Age (years) | Protocol, duration | Effects on physical fitness |
|---|----------------|-------------|--|--|
| Thien et al. (2000) ⁷⁵ | <i>n</i> = 16 | 29 ± 4.4 | 3-mo pilot study of a graded exercise program ranging from levels 1 to 7 performed 3 times/wk. Level 1 included stretching exercises while sitting and lying; level 7 included stretching exercises, resistive strengthening, and low-impact cardiovascular exercise | No significant differences in weight gain in exercisers vs nonexercisers |
| Tokumura et al. (2003) ⁷⁶ | <i>n</i> = 9 | 14 ± 3 | 30-min stationary bicycle exercise at anaerobic threshold, 5 times/wk | Positive effects of physical activity on exercise capacity (endurance time, oxygen uptake, VO ₂ , peak VO ₂ , and heart rate) |
| Chantler et al. (2006) ⁷⁷ | <i>n</i> = 14 | 25 ± 10 | 8 wk of strength training; 60-min sessions, 2 times/wk | Significantly increased BMI and FM percentages; significantly increased peak torque of knee extensors, knee flexors, and elbow flexors in exercising group |
| Szabo et al. (2002) ⁷⁸ | <i>n</i> = 21 | 20.5 ± 0.5 | 8-wk program; 2.5-kg dumbbells for upper-body exercises; therapeutic elastic bands and body weight for lower-body exercises | Significantly increased BM, BMI, and FM; significantly decreased Beck Depression Inventory score |
| del Valle et al. (2010) ⁸⁸ | <i>n</i> = 22 | 14 ± 2 | 3 mo of strength training; 60–70 min, 2 times/wk | Significant improve of muscle strength only on upper extremity in exercising group |
| Fernandez-del-Valle et al. (2014) ⁸⁹ | <i>n</i> = 36 | 12.6 ± 0.69 | 2 mo of strength training; 3 times/wk | Significantly improved performance on leg-press, bench-press, and lateral row tests |
| Joyce et al. (1990) ⁹⁸ | <i>n</i> = 8 | 29 ± 9 | Bone densitometry; exercise graded as <1 h/wk (0), 1–6 h/wk (1+), and >6 h/wk (2+) | Protective effect of moderate physical activity (1+) on BMD |
| Calogero et al. (2004) ¹⁰⁶ | <i>n</i> = 127 | 22 ± 8 | Warm up, exercise activities (stretching, posture, yoga, Pilates, partner exercises, strength training, balance, exercise balls, aerobic activity, and recreational games), and cool down; 60-min, 2–4 times/wk | Decreased exercise compulsivity and dependence |
| Hart et al. (2001) ¹¹¹ | <i>n</i> = 19 | 25.9 ± 0 | 5 wk of massage therapy; 15 min in supine position, 15 min in prone position, 2 times/wk | Lower Eating Disorder Inventory scores in exercising group |
| Catalan Matamoros et al. (2011) ¹¹² | <i>n</i> = 11 | 28 ± 0 | 7 wk of basic body awareness therapy | Lower Eating Disorder Inventory scores in exercising group |
| Carei et al. (2010) ¹¹³ | <i>n</i> = 29 | 16.5 ± 2.3 | 8 wk of yoga; 2 times/wk | Lower Eating Disorder Examination scores, State and Trait Anxiety Inventory scores, and Beck Depression Index in exercising group |

Abbreviations: BMD, bone mineral density; BMI, body mass index; FM, fat mass; VO₂, oxygen consumption.

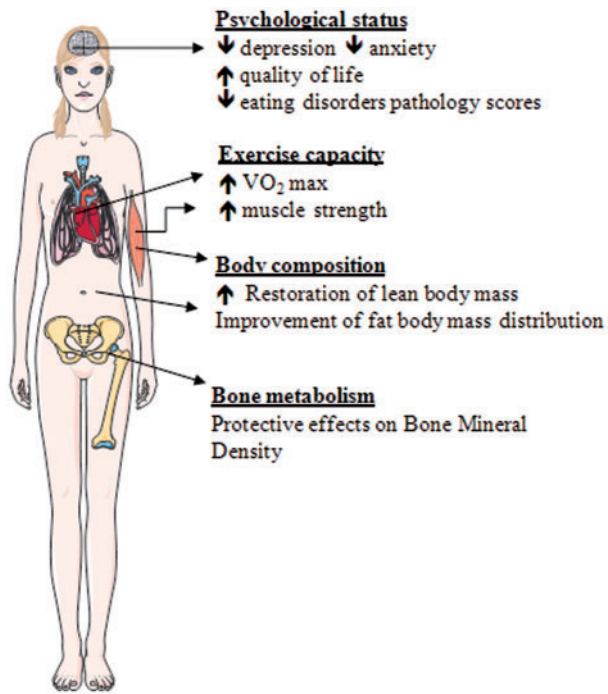


Figure 1 Potential benefits of programmed physical activity in anorexia nervosa. Preserving some kind of physical activity during refeeding of patients with anorexia nervosa should be safe and beneficial for the restoration of body composition and muscle function, preservation of bone mineral density, and management of mood and anxiety. *Abbreviation:* VO₂, oxygen consumption.

has been reported to be an accurate measure of physical activity¹¹⁷ but might not accurately reflect different types and/or intensity of physical activity and may not capture fine movements. Most of the studies to date used questionnaires to assess physical activity in patients with AN. The revised version of the Obligatory Exercise Questionnaire measures emotional elements, behavioral elements, and exercise preoccupation.¹¹⁸ The Reasons for Exercise Inventory also assesses management of fitness and health, attractiveness and weight loss, emotional regulation, and social interaction.¹¹⁹ Other questionnaires focus on exercise addiction and dependency. For example, the Exercise Dependence Scale–Revised includes subscales to evaluate dependence on exercise behavior¹²⁰; the Exercise Addiction Inventory is a short version related to addiction models for understanding exercise.¹²¹ Nevertheless, self-reports of physical activity, as well as general staff member reports, could result in over- or underreporting.¹²² Thus, assessments by personnel specially educated in physical activity (type, motives, intensity, duration, and frequency) may produce more reliable results.

It is worth noting that none of the questionnaires discussed above have been developed in clinical eating disorder units. Recently, Danielsen et al.¹²³ tested the

Exercise and Eating Disorder (EED) self-report questionnaire in 224 patients with eating disorders who were recruited from an eating disorder unit, including 79 AN patients, in comparison with a healthy control group.¹²³ The EED is the first clinically derived, self-report questionnaire to assess compulsive exercise among patients with eating disorders. Results confirmed it is a valid and reliable measure of compulsive exercise in this patient group, including AN patients; however, the EED has not yet been compared with another exercise questionnaire.

Clinical management of physical activity in specialized units for eating disorders

Conventional management of AN involves treatment of physical, cognitive, and behavioral aspects of the disorder,¹²⁴ including nutritional rehabilitation, psychosocial interventions (cognitive behavioral therapy), and medications (anxiolytics, antidepressants).¹²⁵ There is no consensus or recommendation on how physical activity should be handled during the treatment of AN, and practice may differ markedly among specialized units. Only few teams have established internal protocols. In a survey performed in 49 units specialized in eating disorders in Scandinavia and the United Kingdom, it appears that physical activity was often integrated into the treatment of AN.¹²⁶ In most of these units, hyperactivity was considered to be a harmful symptom in AN patients, and internal guidelines were available to monitor hyperactivity at entrance and to handle it during the hospital stay. In addition, PPA was considered to be potentially beneficial. The authors also reported a more positive attitude toward physical activity in the treatment of bulimia nervosa and binge eating disorder as compared with AN. Some national differences appeared in this study¹²⁶: Swedish units agreed to limit physical activity only in low-weight patients (BMI <15 kg/m²), whereas this type of restriction was more common in the other countries. Another survey among units in the United Kingdom reported a lack of consensus among units, with most of them reporting some form of physical activity restriction.¹²⁷ Thus, some units allowed physical activity from a BMI of 15 kg/m², whereas other units prohibited physical activity until a BMI of 18.5 kg/m² was reached. These observations support the need for some expert-consensus statement while waiting for clinical studies to allow the definition of evidence-based guidelines.

CONCLUSION

In the absence of a consensus statement, it seems reasonable to limit activity in patients with low BMI

(under 14 kg/m²) and to allow some level of spontaneous exercise to limit anxiety during weight gain when no specific program is available. If a therapeutic program including PPA can be planned, it must be managed by multidisciplinary professionals who are specially trained in PPA and are able to assess the spontaneous physical activity (type, motives, intensity, duration, frequency) and to establish a personalized program. These personalized programs should aim to limit the harmful consequences of hyperactivity and optimize the expected benefits of PPA. In all circumstances, staff should pay attention to the concerns of the patient about body shape and increased abdominal circumference, and provide reassurance and symptomatic treatment of digestive symptoms as needed.

Acknowledgments

Author contributions. N.A. performed the literature search and wrote the manuscript; M.C. contributed to the draft of the manuscript; P.D. contributed to the draft of the manuscript and gave final approval of the manuscript.

Funding. N.A. was supported by the Conseil Régional de Haute Normandie and the French Health Care Ministry. No other funding was received.

Declaration of interest. The authors have no relevant interests to declare.

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