International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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ARTICLE TITLE	PHYSICAL ACTIVITY LEVELS IN PATIENTS WITH ANOREXIA NERVOSA AND CARDIOVASCULAR COMPLICATIONS. INFORMATION OVERVIEW
ARTICLE INFO	Magdalena Mendak, Anna Hanslik, Agata Białek, Magdalena Domisiewicz, Monika Klimczak, Aleksandra Woskowska. (2025) Physical Activity Levels in Patients with Anorexia Nervosa and Cardiovascular Complications. Information Overview. *International Journal of Innovative Technologies in Social Science*. 3(47). doi: 10.31435/ijitss.3(47).2025.3476
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3476
RECEIVED	11 June 2025
ACCEPTED	23 July 2025
PUBLISHED	26 July 2025
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PHYSICAL ACTIVITY LEVELS IN PATIENTS WITH ANOREXIA NERVOSA AND CARDIOVASCULAR COMPLICATIONS. INFORMATION OVERVIEW

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ABSTRACT

Introduction and aim of the study: The main purpose of this article is to show the impact of eating disorders on the level of physical activity and the occurrence of cardiovascular complications. The development of anorexia- or bulimia-type disorders leads to prospective negative outcomes in physical performance and long-term cardiological issues and leads to decrease of quality of life.. Therefore, it is crucial to identify the causes and intervene quickly to prevent severe side effects. This article reviews issues about: physical and mental health risks in individuals with eating disorders, cardiovascular complications and quality of life among the patients with eating disorders.
Materials and methods: This article was based on literature gathered from various databases, including Google Scholar, ResearchGate, PubMed, ClinicalTrails, Cochrane database. The keywords anorexia nervosa, eating disorders, risk factors and cardiological complications were the basis of the review.
Results: Eating disorders represent a significant public health issue and require an interdisciplinary approach. These disorders develop insidiously, pose a therapeutic challenge, and leave long-lasting side effects including cardiovascular complications. Anorexia nervosa is associated with numerous cardiovascular complications, some of which can be life-threatening. Most of these abnormalities tend to resolve once a healthy body weight is restored. Therefore, treatment should primarily focus on addressing the underlying eating disorder, with symptomatic management provided as needed.

KEYWORDS

Anorexia Nervosa, Eating Disorders, Risk Factors, Cardiological Complications

CITATION

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Introduction.

Anorexia nervosa is a complex psychiatric condition marked by self-imposed dietary restriction, distorted body image, and an intense fear of gaining weight. Its clinical presentation often includes severe undernutrition, which can trigger multisystem complications. Though the disorder predominantly manifests during adolescence, it carries significant long-term health consequences. Among the most life-threatening are cardiovascular abnormalities, which account for a considerable proportion of anorexia-related deaths. This article aims to review the cardiovascular implications of anorexia nervosa, highlighting diagnostic challenges, clinical observations, and evidence-based approaches to management. Epidemiological data suggest that anorexia affects up to 4.3% of women and 0.3% of men, with onset typically occurring during adolescence or early adulthood. Psychological anorexia impacts nearly every organ system. Among the most common complications are growth retardation, osteoporosis, amenorrhea, delayed sexual maturation, and various neurological and cardiovascular disorders. While many of these issues improve with nutritional rehabilitation and recovery, some may result in lasting damage. Notably, anorexia nervosa has the highest mortality rate among all psychiatric disorders, with approximately one-third of deaths attributed to cardiovascular complications. Cardiovascular disorders arising in the course of anorexia have a complex etiology. The interplay of multiple factors distorts the clinical picture of patients and results in the disease manifesting differently in each individual. Dehydration and electrolyte imbalances in anorexia nervosa are largely the result of self-induced vomiting and chronic diarrhea. Protein deficiency combined with significant weight loss leads to a reduction in cardiac muscle mass, hypercholesterolemia, and accumulation of fluid in the pericardial sac. Persistent hypercholesterolemia can contribute to the development of atherosclerotic plaques in the vascular walls, increasing the risk of arteriosclerosis later in life. Although large pericardial effusions requiring pericardiocentesis are rarely reported in the literature and remain mostly anecdotal, they must be taken seriously. Accumulating pericardial fluid can compress the heart externally, limiting diastolic filling and potentially leading to congestive heart failure. In addition, the loss of cardiac muscle mass and the resulting decline in cardiomyocyte metabolic activity reduce myocardial contractility. The combination of these pathophysiological changes ultimately impairs the heart's function as a pump. Electrolyte deficiencies, dehydration, and the use of various pharmacologic agents (such as laxatives or diuretics) also contribute to hypotension and disruptions in ionic balance. These disturbances may lead not only to cardiac arrhythmias but also to complications beyond the cardiovascular system. Commonly observed effects include an increased risk of osteoporosis due to hypocalcemia, paralytic ileus caused by hypokalemia, and seizures resulting from hyponatremia.

Physical activity in individuals suffering from anorexia.

An increased level of physical activity in patients with anorexia nervosa is a frequently observed phenomenon and, according to various data, is reported in approximately 40-80% of patients. An intriguing phenomenon described in the literature is that patients are full of energy and show a greater willingness to participate in various activities. Their level of physical activity differs significantly from the fatigue and sluggishness typically observed in other individuals experiencing starvation and emaciation. This phenomenon has fascinated researchers for over 100 years and has been described in various ways in the literature. The most commonly used terms include: hyperactivity, overactivity, activation and arousal, paradoxical vitality, psychomotor agitation, diffuse motor restlessness, excessive need for physical activity, excessive exercise, excessive physical energy, compulsive exercise, and excessive vitality. A detailed review of concepts regarding the causes of this phenomenon is provided in the work of Casper (2006), in which the author describes the tendency toward hyperactivity ("the drive for activity") as a complex result of physiological and endocrine processes occurring in severely emaciated patients with anorexia nervosa (AN). Among the diagnostic criteria for anorexia in the ICD-10, there is no criterion explicitly mentioning hyperactivity [32]. However, exhaustive physical exercise (as part of a self-imposed regimen leading to weight loss) is considered a supporting feature for diagnosis (ICD-10 Classification, 2000). Similarly, such a criterion is also absent in

the American classification DSM-IV. Additionally, in patients with anorexia nervosa, there is a phenomenon of increased drive, which is not always consciously recognized. Remaining in constant motion gives a sense of being in a negative energy balance, and consequently, helps maintain a low body weight. In the common opinion of clinicians, excessive physical activity in patients with anorexia nervosa is associated with a poor prognosis [32]. It is important to determine the frequency of the symptom, its severity, the relationships between it and the course of the illness, the co-occurrence of disorders such as depression, the presence or absence of somatic complications, as well as the prognostic significance of the symptom. There are many methods for measuring physical activity, which can be divided into objective and subjective methods [32]. The most common include: in vivo observation, questionnaire surveys, actigraphy, photographic monitoring, motion analysis, videography, assessment of movement artifacts in EEG, motion and pressure-sensitive mattresses, bed and body transducers, pedometers, stabilimeters, ultrasonic motion detectors, and infrared detectors. in most studies discussing hyperactivity in anorexia, subjective measures are used, including retrospective analysis of medical information obtained from patient histories, prospective assessments of activity based on structured questionnaires for patients, semi-structured medical interviews, self-assessment scales or therapist assessments, and activity diaries. An example of an objective method that allows precise measurement of physical activity is actigraphy. An actimeter is an electronic device the size of a wristwatch that measures body movements, which the patient can wear, for example, on their wrist [32]. Actigraphy is mainly used in psychiatry for sleep disorders, ADHD, affective disorders, as well as in chronobiology and psychopharmacology. To date, only a few researchers have used actigraphy for the objective assessment of physical activity in patients with anorexia. An original method, an advanced version of the activity diary, called Experience Sampling Methodology (ESM), was applied by Vansteelandt et al. (2004). The study lasted for a week and involved patients answering questions multiple times a day at randomly selected times regarding their current inclination for physical activity, their compulsion drive, and their attitude towards hyperactivity [32].

The role of neuropeptides and their relationship to activity levels in individuals with anorexia nervosa.

Neurophysiological research on neuropeptides that inhibit and stimulate appetite sheds new light on their role in the development of eating disorders. Due to their pleiotropic effects, it is not possible to definitively determine their influence on the development of anorexia nervosa. An example is neuropeptide Y (NPY), which, in addition to regulating appetite (especially cravings for sweets), has anxiolytic and anticonvulsant properties, participates in the regulation of hormone synthesis and release, plays a role in the central regulation of the cardiovascular system, improves memory functions, and controls circadian rhythms [32]. It turns out that the concentrations of most discovered appetite stimulators (including neuropeptide Y, β-endorphin, and galanin, which influences cravings for fats) are similar in both healthy individuals and those with anorexia. The only exception is plasma ghrelin levels in anorexic patients, which correspond to post-meal levels in healthy individuals. In patients with anorexia nervosa (AN), ghrelin levels do not change even two hours after a meal, which may be an adaptation to prolonged food restriction [32]. Weight gain in anorexic patients and the correction of abnormal eating patterns lead to a decrease in plasma ghrelin levels and the restoration of a normal immediate ghrelin response to a single meal. According to reports from experiments on a rat model, it turns out that NPY, which has traditionally been considered an orexigenic substance, may actually increase hyperactivity and weight loss in individuals experiencing weight reduction [32]. This aligns with observations of anorexic patients exhibiting hyperactivity. Another group of substances influencing appetite are orexins, which are produced by the lateral and posterior hypothalamus. They are activated by hypoglycemia, but their role in the pathogenesis of anorexia nervosa is not yet fully understood [32]. However, it is known that one of the effects of their action, apart from regulating appetite, is their influence on spontaneous, involuntary physical activity by acting on similar areas of the lateral hypothalamus (Kotz 2006). Another function of orexins is the synchronization of appetite with arousal state, which is essential for an appropriate food-seeking response at the right time of day when hunger occurs. Under physiological conditions, orexins coordinate movement activity related to the anticipation of a meal. Among the neuropeptides closely associated with the development of AN, as well as the phenomenon of activity, leptin holds a special place. This hormone reduces appetite and stimulates the sympathetic nervous system. Disorders in its production or receptor insensitivity often lead to overweight. Leptin receptors are primarily present in the hypothalamus [32]. When leptin binds to receptors in the hypothalamus, neurons stop producing the neurotransmitter neuropeptide Y, which is an appetite stimulator. The gene expression site for leptin is the adipocytes of white adipose tissue. Leptin influences the levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin (PRL), and growth hormone (GH). It also regulates the process of steroidogenesis in the gonads. The main function of

leptin is to regulate the processes of hunger and satiety, as well as the size of fat tissue reserves. Leptin informs the brain about the body's energy resources, and its concentration in the blood is proportional to the amount of fat tissue [32]. It serves as a signal, whose disappearance triggers the body's survival response to starvation. A low plasma leptin concentration is an endocrine hallmark of the acute phase of AN. It is hypothesized that the subthreshold secretion of leptin by adipocytes (as a consequence of reduced energy intake) may play a significant role in the body's adaptation to self-starvation. Studies on leptin suggest an inverse correlation between plasma leptin levels and food intake restrictions in humans as well. (Holtkamp et al., 2004). In their search for clinical variables correlating with low leptin levels in anorexia nervosa (AN), they also found a relationship between leptin levels and excessive physical exercise (lower leptin levels in more physically active patients). In a subsequent study, the authors examined increased physical activity in anorexic patients during the acute phase of the illness (after hospital admission) by assessing it objectively using an actometer (Holtkamp et al., 2006). At present, it is recognized that the use of leptin may have a beneficial effect on the treatment and reduction of mortality in anorexia. Many findings regarding the pathophysiology of AN point to a connection between this disorder and the dopaminergic system [32]. Dopamine has a modulatory effect on the motor system, body weight, and eating-related behaviors, as well as on the reward and reinforcement system. Several studies have shown the therapeutic benefits of both typical and atypical antipsychotics, including olanzapine, in treating anorexia nervosa (Hillebrand et al., 2005). In the latter study, it was demonstrated that olanzapine administered to patients with anorexia reduced their activity levels compared to untreated patients, without affecting body weight or leptin levels. In rats, the use of olanzapine was associated with reduced physical activity, decreased starvation-induced hypothermia, and reduced hyperactivity of the hypothalamic-pituitary-adrenal axis. In women recovering from anorexia nervosa, studies have shown lower levels of the dopamine metabolite homovanillic acid (HVA) in cerebrospinal fluid compared to healthy controls (Kaye et al., 1999). Research by Frank et al. (2005), using positron emission tomography (PET), suggests that individuals with anorexia may have either reduced intrasynaptic dopamine levels or increased D2/D3 receptor density or binding affinity, especially in the ventral striatal region [32]. This alteration could be linked to the heightened physical activity typically seen in these patients. Conversely, decreased D2/D3 receptor binding has been associated with obesity (Wang et al., 2004). In the context of another mental health condition—Attention Deficit Hyperactivity Disorder (ADHD)—which is primarily characterized by symptoms such as impulsivity, inattention, and hyperactivity, research has indicated an increased affinity or sensitivity of dopamine D2/D3 receptors. Although ADHD is not inherently linked to appetite suppression or disordered eating behaviors, a notable side effect commonly observed in patients undergoing pharmacological treatment with psychostimulants—particularly methylphenidate, which selectively targets dopamine transmission—is a significant reduction in appetite. This appetite loss, although unintended, is frequently reported and highlights the broader impact of dopaminergic modulation not only on behavioral regulation but also on fundamental physiological processes such as hunger and satiety [32]. This connection emphasizes the role dopamine may play in mediating both activity levels and feeding behavior, a relationship that is also relevant in understanding conditions like anorexia nervosa. The above findings may suggest that excessive physical activity observed in anorexia nervosa could be associated with heightened activity of dopaminergic receptors [32]. It is hypothesized that the binding strength to D2/D3 receptors might be inversely proportional to body weight and food intake, positioning anorexia nervosa and obesity at opposite ends of this regulatory spectrum.

Physical Activity and the Treatment of Anorexia Nervosa

In treating anorexia nervosa, therapists often opt to limit patients' physical activity, particularly in the early stages of recovery. This approach is especially emphasized for individuals who are excessively active, as reducing movement can help facilitate weight gain and minimize health risks [32]. During the initial refeeding phase, when the body is still highly vulnerable—especially in severely underweight patients—limiting exercise becomes crucial. It serves not only to support the metabolic demands of restoring weight but also to prevent serious complications, including potentially fatal cardiovascular events. However, in the later stages of treatment, after serious somatic disturbances have been addressed, further restricting physical activity may hinder recovery [32]. This is because such limitations can be stressful for patients, increasing their levels of anxiety and negatively affecting their cooperation with the treatment. There is a lack of sufficient research on this matter. The impact of physical activity on the onset of osteoporosis has not been definitively clarified. Research results are inconclusive, and it remains unclear whether additional exercise protects against osteoporosis in anorexia nervosa, or conversely, contributes to an exacerbation of bone demineralization (Jagielska et al. 1999; Castro et al. 2000; Lichtenbelt et al. 1997) [32]. The effect of physical activity on the

development of osteoporosis in anorexia nervosa is unclear, in contrast to findings in the general population, where the effect is beneficial. High-intensity physical exercises (e.g., running) and weight loss are typically stimuli that promote bone mineral density increase [32]. However, in anorectic patients, the effect of physical activity can have twofold consequences – both protective and harmful. A higher level of physical activity is associated with higher average bone density compared to less active patients (Rigotti et al. 1984). This correlation was not dependent on variables such as age, body weight, duration of amenorrhea, or disease duration [32]. The bone density of more active patients was comparable to that of the control group. It has been shown that physical activity can be a protective factor against bone demineralization. A significant relationship with the onset of osteoporosis is associated with disrupted estrogen metabolism. It has been proven that the duration of low estrogen levels (and the related absence of menstruation), rather than the absolute concentration of this hormone, is linked to bone density (Salisbury et al. 1991). When it comes to the relationship with physical activity, there is some similarity between girls with anorexia and those who train in athletics, where, due to intense exercise, menstruation often stops, leading to a decrease in bone density and more frequent fractures [32]. These complications are more severe the longer the secondary absence of menstruation lasts or the earlier it occurs. Estrogens play an indirect but important role in regulating bone density. Intense physical exercise contributes to further weight loss, which, in turn, leads to a reduction in bone density. The interconnections between these phenomena are explained by Lanyon's theory of the minimum effective strain, which acts as a stimulus for osteogenesis. The rate of bone remodeling (bone turnover) in response to estrogen deficiency increases (with osteoblastic processes outweighing osteoclastic ones), ultimately leading to a decrease in bone mass [32]. In the case of estrogen deficiency, physical activity, as a stimulus, primarily reduces the rate of bone turnover, resulting in an increase in bone mass. However, endurance exercises, which provide insufficient stimulation, cannot balance these processes, which may explain why female athletes often have low bone mineral density. Diamanti et al. (2007) conducted a comprehensive study on a group of 57 patients with early-onset anorexia nervosa (AN), focusing on the relationship between the severity of osteoporosis, dietary habits, and the level of physical activity. The study found that adequate protein intake in the diet and moderate physical activity were associated with maintaining healthy bone density in these patients. This highlights the importance of balanced nutrition and exercise in managing the physical complications of AN, particularly osteoporosis, which is a common and serious concern for individuals suffering from this disorder [32]. The results of this study suggest that moderate physical activity is most beneficial in preventing osteoporosis in patients with AN. Moderate levels of exercise help protect bone density by promoting bone remodeling and improving circulation, which in turn supports the overall health of the skeletal system. On the other hand, too much intense physical activity can have the opposite effect, exacerbating bone loss and increasing the risk of fractures and osteoporosis. Additionally, the complete absence of physical activity, often due to the restrictive behaviors associated with AN, can lead to further bone demineralization [32]. Therefore, a careful balance of physical activity is necessary to prevent both the negative impacts of excessive exercise and the detrimental effects of inactivity. Building on this, the link between excessive physical activity and leptin levels also plays a critical role in the management of AN. As discussed in previous sections, leptin is a hormone closely associated with appetite regulation and energy balance. In patients with AN, low leptin levels are often indicative of starvation and malnutrition. Excessive physical activity, in turn, can exacerbate this condition by further reducing leptin levels and increasing the patient’s metabolic demand, which can complicate treatment. In this regard, researchers such as Sodresten et al. (2003) have suggested that limiting excessive physical activity should be a key goal in the treatment of AN, as it can help facilitate nutritional intake and encourage weight gain [32]. An effective strategy to support this process, according to Sodresten and colleagues, could be ensuring patients with AN are exposed to higher ambient temperatures. Many patients with AN experience hypothermia, which further complicates their physical and psychological condition. By providing a warmer environment, therapists may reduce some of the stress and discomfort caused by low body temperature, making it easier for patients to focus on eating and adhering to the treatment plan [32]. This adjustment in the environment may also help decrease anxiety, allowing patients to regain a sense of safety and comfort, which is crucial for their recovery. In summary, the management of physical activity and environmental conditions plays a significant role in the treatment of anorexia nervosa. While moderate exercise is protective against bone loss, excessive physical activity must be carefully controlled to prevent worsening of osteoporosis [32]. Coupled with strategies aimed at restoring leptin levels and providing a supportive environment, this approach may contribute significantly to the recovery process for individuals with AN. Gutierrez and Vazquez (2001) successfully applied a rehabilitation program for patients with anorexia nervosa (AN) that involved the use of saunas. This program was based on

the knowledge that reducing physical activity, combined with an increase in body temperature, could have a positive effect [32]. They found that not only did the patients' physical activity decrease, but it also did not lead to an increase in anxiety or depression levels. This finding suggests that excessive physical activity in AN may be driven by factors beyond the psychological urge to burn calories or cope with anxiety [32]. Instead, the lack of restraint in physical activity could be viewed as a physiological consequence of weight loss, which results in hypothermia. Another potential treatment approach could involve the administration of exogenous leptin. This approach would aim to address the low leptin levels often seen in individuals with AN, which are linked to starvation and metabolic disturbances. By restoring leptin levels, it may be possible to regulate appetite, energy balance, and even reduce some of the physiological symptoms, such as excessive physical activity, that accompany the disorder [32]. An increasing understanding of the complex interactions between neurohormonal factors regulating appetite may lead to the development of entirely new drugs for the treatment of eating disorders, including anorexia nervosa (AN), where no medications are currently recommended as the primary treatment [32]. Some neuropeptide hormones are already in the early stages of clinical trials as potential treatments for eating disorders.

Cardiovascular complications in patients with anorexia nervosa

Anorexia nervosa affects all systems of the human body. Among the most common complications are growth disorders, osteoporosis, amenorrhea and delayed sexual development, as well as neurological and cardiovascular problems [10]. While most of these issues improve with rehabilitation and recovery, some appear to be irreversible. Anorexia has the highest mortality rate of all psychiatric disorders, with approximately one-third of deaths resulting from cardiovascular complications. According to available publications, sudden cardiac death occurs in roughly 1 in 10 individuals with anorexia. Cardiovascular disorders associated with anorexia have a complex etiology [15]. The interaction of multiple factors complicates the clinical picture and causes the disease to manifest differently in each patient. The main contributing causes include: reduced intake of protein and electrolytes, weight loss, dehydration, and the use of laxatives. Electrolyte and cardiovascular disturbances in anorexia nervosa are further exacerbated by self-induced vomiting and the use of emetics, which contribute to dehydration and significant electrolyte imbalances [25]. A deficiency of protein, combined with severe weight loss, leads to atrophy of the cardiac muscle, hypercholesterolemia, and fluid accumulation in the pericardial sac (pericardial effusion) [10]. Chronic elevation of cholesterol levels may promote atherosclerotic plaque formation, increasing the long-term risk of arteriosclerosis. Although large pericardial effusions requiring pericardiocentesis are rarely reported and mainly described as case reports, they are clinically significant. Fluid buildup in the pericardial space exerts pressure on the heart, restricting its ability to relax during diastole, which can lead to congestive heart failure (CHF). Additionally, loss of cardiac muscle mass and a slowed metabolic rate of cardiomyocytes reduce their contractility [10]. The combination of these factors significantly impairs the heart's function as an effective pump. Low blood pressure, including orthostatic hypotension, is a frequent issue among patients hospitalized with anorexia nervosa. Studies, including those by Jagielska et al., report that up to 68% of these individuals may be affected. This condition is primarily due to a decrease in blood volume and weakened heart function [25]. Moreover, individuals with AN often experience muscle wasting in the limbs, which reduces the efficiency of the peripheral muscle pump—a mechanism that helps blood return to the heart. As a result, these patients are more prone to experiencing drops in blood pressure when standing up [25]. Patients with anorexia nervosa often experience drowsiness, difficulties with concentration, lowered body temperature, and a tendency to faint or lose consciousness. Blood pressure typically begins to normalize once the patient reaches approximately 80% of their ideal body weight. There is a noted correlation between blood pressure values and the severity of the illness, which has led researchers, including Shamim et al., to recommend regular blood pressure monitoring as a useful tool for assessing the clinical status of patients with anorexia nervosa.

Sinus bradycardia in patients with anorexia nervosa

According to various sources, sinus bradycardia affects 35-95% of patients with anorexia nervosa, with most publications citing the upper end of this range. Studies by Jagielska et al. show that sinus bradycardia is more common in adolescent patients than in adults [2]. The cause of this disorder is excessive activation of the parasympathetic nervous system, combined with increased sensitivity of baroreceptors to signals. An additional contributing factor is the slower metabolism resulting from the body's adaptation to limited energy substrate intake. Bradycardia becomes dangerous when it occurs alongside other abnormalities visible on an ECG, such as arrhythmias or a prolonged QT interval [2]. This significantly increases the likelihood of

ventricular arrhythmias, which require immediate treatment. A connection between bradycardia and an increased risk of sudden cardiac death has also been suggested [2]. Given how common bradycardia is among patients with anorexia nervosa, it is important to be vigilant for the development of resting tachycardia in these individuals. Any such case requires further diagnostic investigation, as an accelerated heart rate is often caused by an ongoing inflammatory process. It can also indicate the development of heart failure, or be a result of the use of medications or psychoactive substances by the patient. Heart rate normalization typically occurs when the patient reaches about 80% of their ideal body weight (IBW), usually within 3-4 weeks of treatment [2]. However, there are reports where bradycardia persists even after BMI normalization. Łętek et al. reported a 2-year observation of an 18-year-old patient with anorexia, in whom bradycardia did not completely resolve, despite achieving a BMI of 21.35 kg/m² [2]. The sick sinus syndrome was ruled out in this case. At the end of the study, the patient was in good overall health, not taking any medications. The average heart rateremained around 60 bpm, with intermittent episodes of bradycardia dropping to 37 bpm [2].. Cardiac arrhythmias in the form of bradycardia are relatively common in the course of anorexia nervosa and are generally considered a reversible condition, although they may be accompanied by other cardiovascular complications. In most cases, normalization of heart rate is observed within a few weeks after body weight has returned to normal conditions [3]. However, there are instances where bradycardia persists despite successful treatment of the underlying condition. A team from the 2nd Department of Cardiology at the Świętokrzyskie Cardiology Center of the Provincial Integrated Hospital in Kielce published a report describing a case of bradycardia with a somewhat atypical course [3]. The publication presented the case of a patient with a history of eating disorders of the anorexia-bulimia type, who continued to experience asymptomatic bradycardia for many months despite normalization of body weight and no evidence of sick sinus syndrome [3]. Over a two-year observation period, the bradycardia gradually subsided. An 18-year-old patient with a history of anorexia-bulimia type eating disorder was admitted to the cardiology department for the implantation of a pacemaker due to persistent sinus bradycardia observed on resting ECG and Holter monitoring, with heart rates ranging from 36–44 bpm, and nocturnal decreases to as low as 23 bpm [3]. The patient reported generalized weakness, but denied episodes of fainting or loss of consciousness. The standard ECG showed no QT interval prolongation, and biochemical tests revealed no significant abnormalities. Physical examination revealed a blood pressure of 110/80 mmHg, the presence of gynecomastia, and a BMI of 20.06 kg/m². In the atropine test, heart rate increased from 38 to 78 bpm [3]. The exercise stress test following the Bruce protocol lasted 14 minutes and 57 seconds, demonstrating good exercise tolerance (17.5 METs) with a satisfactory chronotropic response. Echocardiography showed no abnormalities, with a very good ejection fraction, normal global contractility, and a small amount of pericardial fluid. It was concluded that the bradycardia was secondary to the patient's eating disorder. Theophylline therapy was initiated in the patient, resulting in normalization of heart rate during both wakefulness and nighttime [3]. A follow-up Holter monitoring showed no episodes of bradycardia, with an average 24-hour heart rate of 47 bpm. During the two-year follow-up, a slight weight gain to 66 kg was observed, along with a gradual increase in heart rate. No episodes of bradycardia below 30 bpm were recorded. Currently, the patient is not taking any medications, and his BMI is 21.35 kg/m². A follow-up echocardiographic examination showed no pericardial effusion. Cardiovascular complications occur in 80% of patients with anorexia nervosa. The most common are bradycardia, hypotension, arrhythmias, and sudden cardiac death, which is reported in 10% of cases [3]. Patients with anorexia also demonstrate reduced exercise tolerance, although this is not always the case, as observed in the above case. Research shows that bradycardia occurs more frequently in adolescents with anorexia than in adults. The underlying causes of bradycardia in anorexia nervosa are believed to be increased vagal nerve activity and heightened baroreceptor sensitivity. In most cases, the abnormal heart rate resolves after approximately three weeks of treatment, once patients reach about 80% of their ideal body weight. However, as demonstrated in the case described above, bradycardia can persist despite normalization of BMI, gradually and progressively returning to normal over the course of several weeks. Bradycardia is a physiological response of the body to significantly reduced body weight; therefore, it is resting tachycardia that should raise particular concern. Cases have been reported in which symptomatic tachycardia was an early indicator of ongoing inflammatory processes in patients. In patients suffering from anorexia nervosa, the most appropriate approach appears to be close monitoring, exclusion of other potential causes of bradycardia and syncope, pharmacological management of bradycardia, and, most importantly, treatment of the underlying disorder [3]. Additionally, it is recommended that such patients undergo an atropine challenge, which can help determine the extent to which the observed arrhythmia is related to increased vagal tone versus an intrinsic dysfunction of the sinus node [3]. In turn, an exercise stress test

allows for the evaluation of exercise tolerance and the chronotropic response to physical exertion. After body weight normalization, a reassessment of the arrhythmia is necessary to guide further clinical decisions.

Prolonged QT interval in patients with anorexia.

In recent years, particular attention has been drawn to the phenomenon of QT interval prolongation in patients with anorexia. The QT interval reflects the depolarization and repolarization of both the left and right ventricles [2]. A prolonged QT interval is a known risk factor for the development of ventricular arrhythmias and sudden cardiac death. It is believed that the electrolyte disturbances commonly observed in untreated anorexic individuals—such as hypokalemia, hypocalcemia, and hypomagnesemia—are the direct cause of the altered repolarization period. A study comparing QT intervals in anorexic patients who had already begun treatment revealed no significant differences compared to a healthy control group [2]. This suggests that after correcting electrolyte imbalances and initiating appropriate nutritional therapy, repolarization abnormalities tend to normalize [2]. Therefore, regular monitoring of the QT interval is recommended in patients with anorexia, as it serves as a predictive factor for the development of ventricular arrhythmias and sudden cardiac death.

Orthostatic hypotension in patients with anorexia nervosa

Another health issue faced by approximately 68% of patients with anorexia is orthostatic hypotension. A drop in blood pressure in individuals with anorexia is primarily caused by dehydration and reduced cardiac output. Hypotension also increases the likelihood of fainting episodes [2]. This is attributed to the effects of starvation, which leads to weight loss and muscle atrophy, thereby reducing venous return to the heart. Studies have shown that blood pressure tends to normalize approximately three weeks after the initiation of nutritional rehabilitation, typically when patients regain about 80% of their ideal body weight. Therefore, blood pressure monitoring can serve as a useful clinical indicator of physiological stabilization in patients with anorexia nervosa [3]. Electrolyte imbalances in patients with anorexia nervosa are often secondary to the misuse of laxatives, diuretics, or self-induced vomiting. These behaviors not only contribute to weight loss but also lead to decreased exercise tolerance, cardiac arrhythmias, and, in severe cases, sudden cardiac death [3]. Lipid profiles in patients with anorexia nervosa are often abnormal [2]. However, as treatment progresses and body weight increases, these abnormalities tend to improve. Additionally, newly adopted dietary habits after recovery play an important role in normalizing lipid levels.

Echocardiography in patients with anorexia nervosa

The results of echocardiographic examinations in patients with anorexia nervosa often differ from those observed in healthy individuals. One of the indicators showing these differences is the measurement of heart muscle dimensions. Echocardiographic studies in patients with anorexia have revealed a reduced thickness of the interventricular septum (in 52% of patients), reduced thickness of the free wall of the left ventricle (in 61%), reduced size of the left atrium (in 31%), and decreased left ventricular mass (in 61%) [2].

However, other researchers did not find significant differences in the thickness of the interventricular septum or the posterior wall of the left ventricle compared to control groups. Instead, they highlighted differences in the end-systolic and end-diastolic dimensions of the left ventricle, which were significantly smaller in patients with anorexia. An increased frequency of mitral valve prolapse was also observed in this population. Additionally, approximately 35% of patients are found to have fluid in the pericardial sac, which should be considered an early sign of cardiovascular complications [2]. Although patients with pericardial effusion typically do not exhibit significant hemodynamic consequences, the literature does report cases where pericardiocentesis was necessary due to a life-threatening cardiac tamponade. Follow-up echocardiographic studies in patients with anorexia after hyperalimentation therapy show improvement in left ventricular parameters and myocardial muscle mass. Similarly, ejection fraction (EF) and heart rate tend to normalize. The amount of pericardial fluid also decreases as body weight increases. Studies show that patients with anorexia nervosa (AN) frequently exhibit abnormalities of the atrioventricular valves. Increased leaflet mobility is observed despite the normal anatomical structure of the valvular apparatus [2]. This may explain the higher incidence of mitral and tricuspid valve prolapse and regurgitation in this patient group compared to the general population [2]. The proportional relationship between valve size and myocardial thickness becomes disrupted, primarily due to the reduction in ventricular chamber dimensions while the leaflet size remains unchanged. As previously mentioned, hypoproteinemia and increased endothelial permeability in patients with AN also contribute to the accumulation of pericardial fluid [2].

The risk of congestive heart failure in patients with anorexia nervosa

Congestive heart failure represents a serious, although underrecognized, complication in individuals suffering from anorexia nervosa. Its pathophysiology is complex and multifactorial, involving both structural and metabolic alterations of the myocardium. Among the key contributing factors are severe malnutrition, reduction in myocardial mass, and functional impairment of systolic and diastolic performance. The atrophy of cardiac muscle fibers stems from a profound deficiency in protein, micronutrients, and essential vitamins, which are critical for cellular metabolism and contractile function [2]. Cardiomyocytes, due to their inherently high metabolic demands, are particularly vulnerable to these deficits. As a result, impaired contractility develops in a portion of the myocardial cells. In response, adjacent cardiomyocytes attempt to compensate by enhancing their workload, leading to progressive overstretching, cellular stress, and eventually degeneration and lysis of cardiac muscle tissue. A case study by Turillazzi et al. described the death of a young female patient with AN shortly after the onset of CHF symptoms. [2]. Post-mortem histopathological analysis revealed colliquative myocytolysis, a rare form of myocardial necrosis. With no evidence of other underlying causes, the authors concluded that profound malnutrition and starvation alone were sufficient to induce fatal cardiac failure. Another contributing factor is pericardial effusion, frequently observed in patients with AN due to hypoproteinemia and increased capillary permeability. Accumulated fluid within the pericardial sac can mechanically compress the heart, limiting diastolic filling and thus promoting or exacerbating heart failure [2]. Although often asymptomatic, in rare cases this can evolve into cardiac tamponade, a life-threatening condition requiring urgent intervention. Clinical manifestations of CHF in anorexia may include tachycardia, exertional dyspnea, peripheral edema, hepatomegaly, fatigue, dizziness, and poor exercise tolerance. These symptoms reflect the reduced efficiency of the heart as a pump in the context of myocardial atrophy and impaired preload. Early diagnosis of CHF in AN patients is essential, as nutritional rehabilitation often leads to functional improvement or even reversal of cardiac abnormalities. A multidisciplinary approach involving cardiologic monitoring, psychonutritional therapy, and close electrolyte management is critical for reducing mortality in this population.

Electrolyte disturbances in patients with anorexia

Electrolyte imbalances are commonly observed in the course of anorexia nervosa (AN) and may involve both macro- and micronutrients. In a study conducted by Miller et al. among women with AN, hyponatremia was found in 7% of participants. One patient even experienced a seizure attributed to this sodium deficiency. Notably, no cases of hypernatremia were reported. As for potassium, hypokalemia was detected in 20% of the women, even among those taking potassium supplements. Nearly half (48%) of those with low potassium levels admitted to regular use of laxatives. Another study found no hypokalemia in patients who did not use laxatives. Hypocalcemia was identified in 6% of participants. Due to the broad spectrum of possible electrolyte disturbances, the clinical presentation in patients with AN can vary significantly, making it difficult to identify a set of typical symptoms. However, some researchers have attempted to associate electrolyte abnormalities with QTc interval prolongation. Conditions such as hypokalemia, hypocalcemia, and hypomagnesemia are believed to contribute to delayed cardiac depolarization and repolarization, which manifests on an ECG as a prolonged QT interval. It has been clearly demonstrated that such electrolyte imbalances increase the risk of arrhythmias, which in turn can lead to sudden cardiac death.

Refeeding syndrome

The development of refeeding syndrome is a serious and potentially life-threatening complication in patients suffering from anorexia nervosa. It can occur regardless of the route of nutritional support—whether oral, enteral, or parenteral. This syndrome arises due to an excessively rapid and unbalanced intake of solid food and fluids, particularly when the diet contains a high amount of carbohydrates. [2]. Refeeding syndrome is associated with a range of symptoms, including cardiac, neurological, and hematological complications. This discussion focuses primarily on cardiac complications, which significantly increase the risk of sudden death. The primary cause of this condition is the onset of hypophosphatemia. On one hand, it may result from insufficient phosphorus content in the diet; on the other hand, large amounts of phosphorus are utilized in metabolic pathways to process the increased intake of energy substrates [2]. Due to the drop in phosphate levels, the production of energy substrates is impaired, which affects cardiomyocytes—cells particularly sensitive to energy deficits. As a result, their contractility becomes compromised, ultimately leading to the development of heart failure [2]. The literature highlights a significant risk of developing arrhythmias, bradycardia, hypotension, and even myocardial infarction during refeeding treatment. To minimize these complications, it is recommended that the size and nutritional content of meals introduced during nutritional rehabilitation be carefully planned. Early phosphorus supplementation is emphasized, along with the gradual increase in caloric intake. Additionally, regular monitoring of electrolyte levels and cardiac function is essential throughout the refeeding process [2].

Conclusions

Anorexia nervosa is associated with numerous cardiovascular complications, some of which can be life-threatening. Most of these abnormalities tend to resolve once a healthy body weight is restored. Therefore, treatment should primarily focus on addressing the underlying eating disorder, with symptomatic management provided as needed. In patients with anorexia who present with sinus node dysfunction, there are no indications for pacemaker implantation unless there are severe conduction disorders or congenital abnormalities of cardiac automatism. Such arrhythmias in anorexic patients—like other cardiovascular disturbances—respond best to hyperalimentation treatment, restoration of normal body weight, and appropriate psychological care. Typically, normalization of heart rate is observed within the first few weeks of treating the underlying eating disorder. In these cases, the temporary use of medications that inhibit vagal tone or stimulate beta-adrenergic receptors in the heart may be considered, particularly for symptomatic patients. The decision to implant a pacemaker should be carefully considered, even in cases of prolonged bradycardia. Additionally, all patients with anorexia require regular follow-up examinations, including tests to assess electrolyte levels, ECG with evaluation of the QTc interval, and, if necessary, echocardiographic assessment.

Disclousers

Author's contribution

Conceptualization: Magdalena Mendak; Methodology: Agata Białek; Software: Magdalena Domisiewicz; Analysis: Monika Klimczak; Investigation: Aleksandra Woskowska; Resources: Agata Białek; Data curation: Magdalena Mendak; Writing: Anna Hanslik; Preparation: Magdalena Domisiewicz; Visualization: Agata Białek; Supervison: Monika Klimczak; Project administration: Aleksandra Woskowska.

All authors have read and agreed with the published version of the manuscript.

Receiving funding: Not applicable.
Funding Statement: This Research received no external funding.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Acknowledgments: Not applicable.
Data Availability Statement: The authors confirm that the data supporting the findings of this study are available within the article's bibliography.
Conflicts of Interests: Authors declare no conflict of interests.

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