



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

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Calgary, Alberta, T3E0A7,
Canada
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ARTICLE TITLE

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DOI

[https://doi.org/10.31435/ijitss.3\(51\).2026.6143](https://doi.org/10.31435/ijitss.3(51).2026.6143)

RECEIVED

21 July 2026

ACCEPTED

22 September 2026

PUBLISHED

24 September 2026

LICENSE



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THE IMPACT OF ANOREXIA NERVOSA ON CARDIAC STRUCTURE AND FUNCTION: A NARRATIVE REVIEW OF MYOCARDIAL REMODELING, ELECTROPHYSIOLOGICAL ALTERATIONS AND CLINICAL OUTCOMES

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ABSTRACT

Objective: Anorexia nervosa (AN) is associated with the highest mortality rate among psychiatric disorders, with cardiovascular complications representing one of the major causes of morbidity and death. This narrative review aimed to summarize current evidence regarding the effects of AN on cardiac structure and function, focusing on myocardial remodeling, electrophysiological abnormalities, and clinical outcomes.

Methods: A narrative review methodology was employed using fourteen studies provided by the authors, including systematic reviews, observational studies, imaging studies, long-term follow-up investigations, and case reports published between 2005 and 2025. Data were synthesized qualitatively to identify recurring patterns of cardiovascular involvement.

Results: The evidence consistently demonstrates that starvation-induced cardiac remodeling is characterized by reduced left ventricular mass, diminished chamber dimensions, decreased cardiac output, and occasional pericardial effusion. Electrophysiological manifestations include sinus bradycardia, altered autonomic balance with parasympathetic predominance, QT interval abnormalities, and increased susceptibility to potentially fatal arrhythmias, particularly in the context of electrolyte disturbances. Histopathological findings suggest the presence of myocardial fibrosis and myxoid degeneration in severe disease, although cardiac magnetic resonance studies indicate that diffuse replacement fibrosis may not be universal. Importantly, most structural and functional abnormalities appear to improve following nutritional rehabilitation and weight restoration. Nevertheless, persistent low body weight remains associated with long-term cardiac impairment and rare but devastating complications, including cardiogenic shock and sudden cardiac death.

Conclusion: Recognition of cardiovascular involvement in AN is essential for risk stratification, timely intervention, and multidisciplinary management. Although the majority of cardiac abnormalities appear reversible with nutritional rehabilitation, severe or persistent disease may result in irreversible myocardial injury and life-threatening complications. Further prospective studies are needed to clarify the mechanisms underlying sudden cardiac death and to identify predictors of irreversible cardiac damage in this vulnerable population.

KEYWORDS

Anorexia Nervosa, Cardiovascular Complications, Myocardial Remodeling, Arrhythmia, Sudden Cardiac Death, Weight Restoration

CITATION

Ziółkowska, K., Wites, K., Wites, P., Łagosz, H., Piotrowski, P., Włodarczyk, A., Jabłońska, A., Pyzik, M., Markusiewicz, K., & Bahyrycz, P. (2026). The impact of anorexia nervosa on cardiac structure and function: A narrative review of myocardial remodeling, electrophysiological alterations and clinical outcomes. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.6143](https://doi.org/10.31435/ijitss.3(51).2026.6143)

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Introduction

Anorexia nervosa (AN) is a severe psychiatric disorder characterized by persistent restriction of energy intake, intense fear of gaining weight, and disturbance in the perception of body weight or shape. Although classified primarily as a psychiatric illness, its clinical consequences extend far beyond psychopathology and involve nearly every organ system. Among these complications, cardiovascular manifestations are of particular importance because they contribute substantially to the excess mortality associated with the disorder (Puckett et al., 2021).

AN predominantly affects adolescent girls and young women, although increasing recognition of the disorder among males and older adults has expanded the traditional demographic profile (Carlson & Lemly, 2024). Lifetime prevalence estimates range between 0.3% and 4% among females, with significantly lower but likely underestimated rates in males (Puckett et al., 2021). Despite its relatively low prevalence, AN remains the psychiatric illness with one of the highest mortality rates, with standardized mortality ratios approaching six-fold that of the general population (Lamzabi et al., 2015).

The causes of death in AN are multifactorial and include suicide, infections, metabolic derangements, and complications related to severe malnutrition. However, cardiovascular causes account for a substantial proportion of fatalities. Estimates suggest that approximately one-third of deaths in AN may be attributable to cardiac complications, frequently presenting as sudden and unexpected events (Sardar et al., 2015). Nevertheless, the exact mechanisms responsible for these deaths remain incompletely understood.

Historically, sudden cardiac death in AN has been attributed primarily to malignant ventricular arrhythmias secondary to QT interval prolongation. However, emerging evidence challenges this simplistic explanation. Sachs et al. (2016) emphasized that direct evidence linking QT prolongation to fatal arrhythmias is surprisingly limited, despite its widespread acceptance. Instead, the cardiovascular phenotype of AN appears to involve a complex interplay between myocardial atrophy, autonomic nervous system adaptation, electrolyte disturbances, endocrine abnormalities, and structural remodeling.

The myocardium undergoes profound adaptive responses to chronic starvation. Severe caloric restriction results in catabolism not only of skeletal muscle but also of cardiac muscle tissue. Echocardiographic studies have consistently demonstrated reductions in left ventricular mass, chamber dimensions, and cardiac output among patients with active disease (Olivares et al., 2005; Kastner et al., 2012; Paysal et al., 2022). These changes are generally interpreted as physiological adaptations aimed at reducing metabolic demand in the setting of energy deprivation.

However, the concept of simple adaptation may underestimate the complexity of cardiac involvement. Histopathological observations reported by Lamzabi et al. (2015) revealed interstitial fibrosis, endocardial fibrosis, lipofuscin accumulation, and myxoid degeneration in autopsy specimens obtained from patients with AN, raising the possibility that irreversible myocardial injury may develop in severe or prolonged disease. Conversely, cardiac magnetic resonance studies have failed to demonstrate widespread replacement fibrosis in living patients, suggesting that the spectrum of myocardial injury may vary considerably according to disease duration and severity (Chu et al., 2019).

Electrophysiological alterations represent another hallmark of cardiac involvement in AN. Sinus bradycardia is the most frequently encountered abnormality and may affect the majority of hospitalized patients. Although often considered benign and reflective of enhanced vagal tone, profound bradycardia can lead to hemodynamic instability requiring inpatient monitoring (Friars et al., 2023). Additional abnormalities include junctional rhythms, QT interval prolongation, increased QT dispersion, reduced heart rate variability, and altered sympathovagal balance. These disturbances are further amplified by purging behaviors and electrolyte abnormalities such as hypokalemia and hypophosphatemia.

Importantly, accumulating evidence suggests that many cardiovascular abnormalities observed in AN are reversible. Weight restoration has been associated with normalization of ventricular dimensions, improvements in cardiac output, resolution of pericardial effusions, shortening of QT dispersion, and restoration of autonomic balance (Olivares et al., 2005; Kastner et al., 2012; Paysal et al., 2022). Long-term follow-up studies indicate that individuals who achieve sustained nutritional recovery may exhibit cardiac profiles comparable to healthy controls, whereas persistent low body weight remains associated with enduring abnormalities (Flamarique et al., 2022).

Despite advances in understanding these manifestations, important gaps remain. The precise mechanisms underlying sudden cardiac death remain elusive, predictors of irreversible myocardial injury have yet to be identified, and the role of advanced imaging techniques in routine risk stratification is not fully established. Moreover, recent reports of cardiogenic shock in patients with severe AN illustrate the continued emergence of life-threatening cardiovascular presentations that challenge conventional management strategies (Thienel et al., 2025).

Therefore, this narrative review aims to synthesize contemporary evidence regarding the impact of anorexia nervosa on cardiac structure and function. Particular emphasis is placed on three major domains: myocardial remodeling, electrophysiological alterations, and clinical outcomes. By integrating findings from pathological studies, imaging investigations, systematic reviews, and long-term follow-up cohorts, this review seeks to provide clinicians with a comprehensive understanding of cardiovascular involvement in AN and identify priorities for future research.

Methodology

Study Design

This manuscript was conducted as a narrative review aimed at synthesizing current evidence regarding cardiovascular involvement in anorexia nervosa (AN), with particular emphasis on myocardial remodeling, electrophysiological alterations, and clinical outcomes. A narrative approach was selected because of the heterogeneity of the available literature, which comprises systematic reviews, observational studies, cross-sectional analyses, imaging investigations, autopsy reports, case series, and long-term follow-up studies.

Unlike systematic reviews designed to quantitatively pool homogeneous datasets, the narrative format allows integration of diverse methodological perspectives and facilitates a comprehensive understanding of the pathophysiological mechanisms underlying cardiovascular complications in AN.

Literature Sources

The present review was based exclusively on fourteen publications provided by the authors. These studies were published between 2005 and 2025 and included:

1. systematic reviews;
2. clinical reviews;
3. observational cohort studies;
4. cross-sectional echocardiographic investigations;
5. cardiac magnetic resonance imaging studies;
6. long-term follow-up studies;
7. autopsy reports;
8. case reports and case series.

The included studies collectively evaluated adolescents and adults with AN across different stages of illness severity and recovery.

Data Extraction and Synthesis

Information extracted from each publication included:

1. study design and population characteristics;
2. sample size;
3. cardiovascular assessment methods;
4. structural cardiac findings;
5. electrophysiological abnormalities;
6. determinants of cardiovascular dysfunction;
7. reversibility following weight restoration;
8. long-term cardiac outcomes;
9. reported mortality and severe cardiovascular events.

Findings were synthesized qualitatively and grouped into three major thematic domains:

1. Myocardial remodeling and structural alterations;
2. Electrophysiological disturbances;
3. Clinical outcomes and prognostic implications.

Results

Mechanisms of Cardiovascular Injury in Anorexia Nervosa

The cardiovascular manifestations of anorexia nervosa arise from a complex interaction between chronic starvation, neuroendocrine adaptation, autonomic dysregulation, electrolyte disturbances, and progressive structural remodeling of the myocardium.

Although many cardiovascular abnormalities observed in AN are traditionally interpreted as adaptive mechanisms designed to preserve energy expenditure, increasing evidence suggests that prolonged malnutrition may induce pathological alterations with potentially irreversible consequences.

Chronic Energy Deficiency and Cardiac Muscle Catabolism

The hallmark of AN is sustained caloric restriction leading to severe negative energy balance. In response to starvation, the body progressively shifts toward catabolism of endogenous tissues to maintain essential physiological functions.

Importantly, this adaptive process does not spare the myocardium.

Puckett et al. (2021) emphasized that progressive depletion of lean body mass involves both skeletal and cardiac muscle tissue. Reduced protein synthesis, diminished insulin-like growth factor signaling, endocrine alterations, and activation of proteolytic pathways contribute to myocardial wasting.

This phenomenon manifests clinically as reductions in: left ventricular (LV) wall thickness, LV end-diastolic dimensions, LV mass, cardiac output.

Sachs et al. (2016) proposed that myocardial atrophy represents one of the central mechanisms explaining the increased cardiovascular vulnerability observed in AN. Similar to skeletal muscle loss, cardiac muscle reduction appears proportional to disease severity.

Supporting this hypothesis, Kuwabara et al. (2018) demonstrated a positive correlation between body mass index and left ventricular mass index among severely malnourished adults with AN. Lower BMI was significantly associated with smaller myocardial mass, suggesting that cardiac remodeling parallels nutritional deterioration.

These findings indicate that the heart functions as a metabolically active organ highly susceptible to the effects of prolonged starvation.

Neuroendocrine Adaptation and Thyroid Dysfunction

Endocrine adaptations constitute another major contributor to cardiovascular remodeling.

Patients with AN frequently develop the so-called "low-T3 syndrome," characterized by reduced serum triiodothyronine concentrations despite normal thyroid gland morphology. This endocrine pattern is considered an adaptive response aimed at reducing basal metabolic expenditure during starvation. However, thyroid hormones exert profound effects on cardiovascular physiology.

Triiodothyronine influences

1. myocardial contractility,
2. calcium handling,
3. β -adrenergic responsiveness,
4. heart rate regulation,
5. vascular resistance.

Paysal et al. (2022) demonstrated that lower T3 concentrations were independently associated with alterations in autonomic balance among adolescents with AN. Specifically, T3 levels emerged as the strongest determinant of the SD1/SD2 ratio derived from heart rate variability analysis, suggesting an endocrine contribution to the pronounced parasympathetic predominance characteristic of AN.

Similarly, Kastner et al. (2012) found that adolescents presenting with pericardial effusion exhibited more severe manifestations of low-T3 syndrome than those without effusions, even after adjustment for BMI.

These findings suggest that endocrine adaptation may contribute not only to electrophysiological disturbances but also to structural cardiac abnormalities.

Autonomic Nervous System Remodeling

One of the most characteristic cardiovascular features of AN is profound sinus bradycardia.

Historically, bradycardia was interpreted as a simple consequence of reduced metabolic demand. Contemporary evidence, however, indicates that altered autonomic regulation plays a pivotal role.

Numerous investigations have demonstrated enhanced vagal tone accompanied by relative sympathetic withdrawal. Paysal et al. (2022) observed that adolescents with active AN exhibited:

1. significantly lower nocturnal heart rates,
2. increased indices of parasympathetic activity,
3. altered sympathovagal balance.

These autonomic changes partially normalized following weight recovery, supporting their adaptive and reversible nature.

Friars et al. (2023) proposed that increased parasympathetic activity serves to minimize myocardial oxygen consumption during periods of severe nutritional deprivation.

Nevertheless, this adaptive response may become maladaptive when heart rates decline to critically low levels.

Profound bradycardia can result in: dizziness, exercise intolerance, syncope, reduced cardiac output, hospitalization requiring telemetry monitoring.

In severe cases, junctional escape rhythms and significant pauses have also been reported (Sachs et al., 2016).

Myocardial Remodeling in Anorexia Nervosa

Reduction in Left Ventricular Mass

Among all structural abnormalities described in AN, reduction of left ventricular mass represents the most consistently reported finding. Echocardiographic studies conducted over the past two decades have repeatedly demonstrated significant decreases in LV mass compared with healthy controls.

Olivares et al. (2005) evaluated forty adolescents with newly diagnosed AN and found significantly lower LV mass indices compared with age-matched controls. Importantly, substantial improvement occurred following nutritional rehabilitation.

Similarly, Kastner et al. (2012) reported reduced ventricular dimensions among 173 adolescents hospitalized for AN.

The advent of advanced imaging techniques further confirmed these observations.

Using cardiac magnetic resonance imaging, Chu et al. (2019) demonstrated that adolescents with severe AN had markedly lower LV mass indexed to body surface area than healthy individuals.

Both echocardiography and CMR consistently showed reduced ventricular mass despite preserved ejection fraction.

The concordance between imaging modalities strongly supports myocardial atrophy as a genuine biological consequence of starvation rather than an artifact of measurement techniques.

Reduced Chamber Dimensions and Cardiac Output

In addition to myocardial mass reduction, AN is associated with smaller ventricular chamber dimensions. Olivares et al. (2005) demonstrated decreased LV end-diastolic diameters at diagnosis, with improvement following weight restoration.

Kastner et al. (2012) similarly observed reductions in:

1. LV end-diastolic dimensions,
2. LV end-systolic dimensions.

Despite preserved systolic performance, these geometric alterations contribute to diminished stroke volume.

Because bradycardia coexists with smaller ventricular volumes, overall cardiac output becomes substantially reduced.

This reduction in output likely reflects an energy-conserving adaptation designed to match tissue perfusion requirements to decreased metabolic demand.

Sachs et al. (2016) emphasized that low-output states observed in AN generally improve with nutritional rehabilitation and should not necessarily be interpreted as evidence of primary cardiomyopathy.

However, prolonged exposure to severe malnutrition may eventually overwhelm adaptive mechanisms, predisposing susceptible individuals to overt heart failure.

Speckle-Tracking Echocardiography and Subclinical Myocardial Dysfunction

Conventional echocardiographic parameters, including left ventricular ejection fraction (LVEF) and fractional shortening, have traditionally suggested preserved systolic function in patients with anorexia nervosa (AN). Consequently, for many years, cardiac involvement in AN was considered predominantly structural rather than functional. However, the introduction of advanced imaging techniques has challenged this notion.

Speckle-tracking echocardiography (STE) allows for the assessment of myocardial deformation and can detect subtle abnormalities in ventricular performance before conventional measures become abnormal. Because myocardial strain analysis evaluates intrinsic myocardial mechanics independently of volumetric measurements, it provides a more sensitive assessment of cardiac function.

Paysal et al. (2022) were among the first investigators to employ STE in adolescent patients with AN. Interestingly, contrary to the expectation that starvation would uniformly impair contractility, the authors demonstrated significantly higher left ventricular global longitudinal strain (GLS) values in adolescents with active disease compared with healthy controls. Patients with AN exhibited GLS values of $-19.2 \pm 1.8\%$, whereas controls demonstrated values of $-16.9 \pm 2.8\%$.

This finding suggests that, despite myocardial atrophy, intrinsic contractile performance may initially be preserved or even enhanced. Several mechanisms have been proposed to explain this paradoxical observation.

First, reduced ventricular wall stress due to smaller chamber dimensions may increase apparent strain measurements. Second, neurohormonal adaptations associated with chronic starvation could temporarily augment myocardial efficiency. Third, alterations in loading conditions resulting from reduced blood volume and decreased afterload may influence strain analysis.

Importantly, the observed GLS abnormalities appeared to improve following nutritional rehabilitation. Adolescents who achieved weight recovery demonstrated values approaching those of healthy controls, suggesting that these functional adaptations remain largely reversible (Paysal et al., 2022).

These findings indicate that AN cannot simply be regarded as a condition of reduced myocardial function. Rather, the myocardium appears to undergo a dynamic process of remodeling characterized by preserved systolic performance despite substantial reductions in myocardial mass.

Preserved Ejection Fraction Despite Cardiac Atrophy

One of the most intriguing aspects of cardiac remodeling in AN is the apparent discrepancy between profound structural alterations and relatively preserved global systolic function.

Across multiple investigations, left ventricular ejection fraction has consistently remained within normal limits despite significant myocardial atrophy.

Olivares et al. (2005) reported preserved fractional shortening and ejection fraction among adolescents with newly diagnosed AN despite reductions in LV mass and cardiac output.

Similarly, Kastner et al. (2012) found no significant differences in fractional shortening between adolescents with AN and healthy controls, despite marked reductions in ventricular dimensions.

Cardiac magnetic resonance imaging further corroborated these findings. Chu et al. (2019) demonstrated that although mean LVEF values were modestly lower in patients with AN compared with controls, systolic function remained largely preserved, with average LVEF measurements exceeding 50%.

The preservation of ejection fraction likely reflects several physiological adaptations:

1. reduced ventricular afterload due to hypotension;
2. diminished metabolic demand;
3. enhanced myocardial efficiency;
4. autonomic modifications favoring energy conservation.

From a clinical perspective, these findings are important because a normal LVEF should not reassure clinicians that cardiac involvement is absent. Significant structural remodeling may coexist with apparently normal systolic indices.

Thus, reliance solely on conventional systolic measurements may underestimate cardiovascular compromise in AN.

Pericardial Effusion: A Frequent but Often Silent Finding

Pericardial effusion (PE) represents one of the most characteristic structural abnormalities identified in adolescents with AN.

Although usually clinically silent, its relatively high prevalence suggests that it forms an integral component of starvation-induced cardiac remodeling.

Kastner et al. (2012) evaluated 173 consecutively admitted adolescents with AN and found pericardial effusion in 34.7% of patients. In contrast, none of the healthy controls demonstrated PE.

Most effusions were small and asymptomatic. Patients generally did not exhibit signs of tamponade physiology or overt hemodynamic compromise. Nevertheless, the presence of PE was associated with markers of more severe disease.

Patients with PE exhibited

1. significantly lower BMI values;
2. more pronounced low-T3 syndrome;
3. longer duration of hospitalization.

After controlling for BMI, low T3 concentrations remained independently associated with the presence of effusion, suggesting that endocrine adaptations contribute to its development.

The precise mechanisms underlying PE remain incompletely understood.

Several hypotheses have been proposed:

- Protein deficiency hypothesis

Hypoalbuminemia secondary to malnutrition may reduce oncotic pressure and facilitate fluid accumulation within the pericardial space.

- Endocrine hypothesis

Low T3 states resemble certain features of hypothyroidism, a condition classically associated with pericardial effusion.

- Altered capillary permeability

Chronic malnutrition may induce endothelial dysfunction and changes in capillary integrity.

Importantly, remission of PE occurred in approximately 88% of patients following weight restoration (Kastner et al., 2012).

This remarkable reversibility strongly supports the interpretation that PE represents an adaptive consequence of starvation rather than irreversible structural pathology.

Histopathological Changes and Myocardial Fibrosis

The histological substrate underlying cardiovascular complications in AN remains poorly understood because opportunities for direct myocardial examination are limited.

The most compelling pathological evidence originates from autopsy investigations.

Lamzabi et al. (2015) described the postmortem findings of an adult patient with severe AN and identified several striking abnormalities.

Gross examination revealed

1. a markedly small heart;
2. predominantly reduced left ventricular size;
3. generalized myocardial atrophy.

Histological analysis demonstrated

Interstitial fibrosis: fibrous tissue deposition between myocardial fibers suggested chronic myocardial injury.

Endocardial fibrosis: fibrosis involving the endocardial surface may alter ventricular compliance and potentially contribute to electrical instability.

Myxoid degeneration: foci of myxoid extracellular matrix deposition were observed throughout the myocardium.

Mast cell infiltration: mast cells were identified within areas of myxoid change, raising the possibility of inflammatory or reparative processes.

Lipofuscin accumulation: marked cytoplasmic lipofuscin deposition indicated chronic oxidative stress and cellular aging.

The authors proposed that these abnormalities may represent direct consequences of prolonged starvation and could contribute to the mechanisms underlying sudden death in AN.

Particularly noteworthy is the observation of fibrosis.

Fibrotic remodeling is well recognized as an arrhythmogenic substrate in other cardiomyopathies because it disrupts electrical continuity and facilitates re-entrant circuits. If similar mechanisms operate in AN, myocardial fibrosis could provide an anatomical explanation for fatal arrhythmias independent of QT interval prolongation.

However, caution is warranted when interpreting these findings.

Autopsy reports generally involve patients with exceptionally severe disease and may not reflect the broader AN population.

Insights from Cardiac Magnetic Resonance Imaging

Cardiac magnetic resonance imaging (CMR) has emerged as the gold standard for assessing myocardial structure, ventricular volumes, and tissue characterization.

Unlike echocardiography, CMR enables the detection of replacement fibrosis through late gadolinium enhancement (LGE).

Chu et al. (2019) investigated sixteen adolescents and young adults with severe AN using both echocardiography and CMR.

Consistent with previous studies, the authors observed:

1. reduced LV mass;
2. reduced right ventricular mass;
3. smaller ventricular volumes;
4. preserved global systolic function.

Unexpectedly, however, no evidence of late gadolinium enhancement was identified.

The absence of LGE suggests that diffuse replacement fibrosis may not be a universal feature of AN, particularly among younger patients.

Several explanations may account for the discrepancy between CMR findings and autopsy observations:

First, CMR primarily detects focal replacement fibrosis and may not identify subtle diffuse interstitial changes.

Second, patients undergoing autopsy likely represent individuals with prolonged, end-stage disease.

Third, fibrosis may develop only after critical thresholds of malnutrition severity and disease duration are exceeded.

These findings highlight that myocardial remodeling in AN exists along a spectrum.

Early disease appears dominated by adaptive myocardial atrophy and functional compensation.

In contrast, advanced and chronic disease may culminate in irreversible histopathological injury.

Table 1. Structural Cardiac Remodeling in Anorexia Nervosa

Study	Population	Main Structural Findings	Reversibility
Olivares et al., 2005	40 adolescents	↓ LV mass, ↓ LV dimensions, ↓ cardiac output	Improved after weight restoration
Kastner et al., 2012	173 adolescents	Pericardial effusion (34.7%), ↓ LV dimensions	PE resolved in 88%
Kuwabara et al., 2018	13 adults	↓ LV mass index correlated with BMI	Not assessed
Chu et al., 2019	16 severe AN patients	↓ LV/RV mass and volumes, preserved EF, no LGE	Not assessed
Paysal et al., 2022	26 adolescents	↑ GLS despite ↓ HR and LV remodeling	Partial normalization after recovery
Flamarique et al., 2022	20-year follow-up	Persistent abnormalities only in low-weight adults	Recovery with sustained normal weight
Lamzabi et al., 2015	Autopsy case	Fibrosis, myxoid degeneration, lipofuscin deposition	Irreversible end-stage findings

Taken together, the available evidence indicates that myocardial remodeling in AN is multifaceted and dynamic. Starvation initially produces adaptive reductions in myocardial mass and chamber size while preserving contractile performance. Most abnormalities improve with nutritional rehabilitation. However, severe and prolonged disease may culminate in histopathological injury characterized by fibrosis and degeneration, potentially contributing to adverse cardiovascular outcomes.

Electrophysiological Alterations in Anorexia Nervosa

Electrophysiological abnormalities constitute one of the most clinically relevant manifestations of cardiovascular involvement in anorexia nervosa (AN). Although structural remodeling contributes to overall cardiac vulnerability, disturbances in cardiac rhythm and conduction have historically been considered the principal mechanisms responsible for the increased risk of sudden death.

However, emerging evidence suggests that the relationship between electrophysiological abnormalities and mortality is considerably more complex than previously assumed.

The electrophysiological phenotype of AN includes:

1. sinus bradycardia,
2. autonomic nervous system dysregulation,
3. QT interval abnormalities,
4. increased QT dispersion,
5. junctional rhythms,
6. electrolyte-mediated arrhythmogenesis,
7. conduction disturbances,
8. ventricular arrhythmias,
9. sudden cardiac death.

These manifestations arise from the interaction between starvation-induced physiological adaptation and secondary metabolic disturbances.

Sinus Bradycardia: The Most Common Cardiovascular Manifestation

Sinus bradycardia represents the hallmark electrocardiographic abnormality in AN.

Reported prevalence varies considerably between studies but may exceed 80–90% among hospitalized patients with severe disease (Sachs et al., 2016; Friars et al., 2023).

Heart rates below 60 beats per minute are frequently observed, whereas rates below 40 bpm are not uncommon in severely malnourished individuals.

Several mechanisms contribute to the development of bradycardia.

Enhanced parasympathetic tone

Chronic starvation induces vagal predominance, reducing sinoatrial node firing.

Reduced metabolic demand

Lower tissue oxygen requirements diminish the need for high cardiac output.

Endocrine adaptation

Low-T3 syndrome decreases intrinsic heart rate.

Decreased sympathetic activity

Reduced catecholaminergic stimulation contributes to slowing of impulse generation.

Paysal et al. (2022) demonstrated significantly lower nocturnal heart rates among adolescents with active AN compared with healthy controls:

1. AN: 55 ± 7 bpm
2. Controls: 68 ± 6 bpm

Importantly, heart rate partially normalized following weight recovery.

Thus, bradycardia appears to be largely reversible.

Despite its adaptive nature, severe bradycardia may become clinically significant.

Symptoms include

1. fatigue,
2. dizziness,
3. exercise intolerance,
4. presyncope,
5. syncope.

Extreme bradycardia may require hospitalization and continuous monitoring.

Sachs et al. (2016) recommended inpatient telemetry in patients presenting with:

1. heart rates <40 bpm,
2. junctional escape rhythms,
3. syncope,
4. hemodynamic instability.

Autonomic Dysfunction and Heart Rate Variability

Alterations in autonomic regulation represent one of the central electrophysiological features of AN.

Heart rate variability (HRV) studies consistently demonstrate profound modifications in sympathovagal balance.

Paysal et al. (2022) showed that adolescents with active AN exhibited:

Increased parasympathetic activity

Reflecting enhanced vagal modulation.

Reduced sympathetic influence

Consistent with metabolic adaptation.

Altered SD1/SD2 ratio

Suggesting substantial changes in autonomic regulation.

Interestingly, serum T3 concentrations emerged as the strongest predictor of autonomic dysfunction.

These findings support the hypothesis that endocrine adaptation contributes directly to electrophysiological remodeling.

From an evolutionary perspective, vagal predominance may conserve energy during periods of nutritional deprivation.

However, excessive parasympathetic activity may predispose susceptible individuals to:

1. profound bradycardia,
2. junctional rhythms,
3. reduced chronotropic reserve,
4. syncope.

Weight restoration significantly attenuates these abnormalities.

Thus, autonomic dysfunction appears predominantly adaptive rather than degenerative.

QT Interval Prolongation

For decades, QT prolongation was considered the principal explanation for sudden cardiac death in AN.

QT interval prolongation reflects delayed ventricular repolarization and increases susceptibility to malignant ventricular arrhythmias, particularly torsades de pointes.

Several factors may contribute to QT prolongation in AN:

Electrolyte abnormalities

Especially hypokalemia and hypomagnesemia.

Malnutrition

Direct effects on ion channel function.

Medication use

Particularly psychotropic agents.

Structural myocardial alterations

Potentially disrupting electrical propagation.

Olivares et al. (2005) demonstrated significantly prolonged corrected QT intervals (QTc) in adolescents with newly diagnosed AN compared with follow-up assessments after weight restoration.

Importantly, QTc improved substantially during recovery.

These findings suggest that repolarization abnormalities are dynamic rather than fixed.

Nevertheless, contemporary evidence challenges the notion that QT prolongation is universally present.

Sachs et al. (2016), in their systematic review, concluded that robust evidence supporting a causal relationship between QT prolongation and sudden death remains surprisingly limited.

Similarly, Chu et al. (2019) reported:

Mean QTc = 413 ms (range 360–450 ms), with no major conduction abnormalities identified among adolescents with severe AN.

Thus, QT prolongation appears to characterize only a subset of patients.

QT Dispersion: A More Sensitive Marker?

QT dispersion (QTd) represents the difference between maximum and minimum QT intervals measured across the 12 ECG leads.

Increased QTd reflects heterogeneity of ventricular repolarization and has been associated with increased arrhythmic risk.

Olivares et al. (2005) demonstrated that adolescents with active AN had significantly elevated QT dispersion compared with healthy controls.

Moreover, QTd decreased significantly following nutritional rehabilitation.

Jáuregui-Garrido and Jáuregui-Lobera (2012) suggested that increased QT dispersion may constitute a stronger predictor of sudden ventricular arrhythmias than absolute QT prolongation.

Potential mechanisms include

1. regional differences in myocardial recovery,
2. electrolyte disturbances,
3. structural remodeling,
4. fibrosis-induced conduction heterogeneity.

Because QTd appears responsive to nutritional status, it may represent a useful marker of disease severity and treatment response.

However, additional prospective studies are required before its routine clinical application can be recommended.

Electrolyte-Mediated Arrhythmogenesis

Electrolyte disturbances substantially amplify electrophysiological vulnerability in patients with eating disorders.

Unlike restrictive AN, purging behaviors frequently induce profound metabolic abnormalities.

Carlson and Lemly (2024) emphasized that hypokalemia is among the most clinically significant abnormalities encountered in eating disorders.

Hypokalemia arises primarily from:

1. self-induced vomiting,
2. laxative abuse,
3. diuretic misuse,
4. renal potassium losses.

Electrophysiological consequences include

1. Delayed repolarization - predisposing to QT prolongation.
2. Increased automaticity - facilitating ectopic activity.
3. Early afterdepolarizations- triggering torsades de pointes.
4. Ventricular fibrillation- in severe cases. Additional electrolyte abnormalities contribute synergistically.
5. Hypomagnesemia - promotes torsades de pointes.
6. Hypocalcemia - may prolong QT intervals.
7. Hyponatremia - can precipitate neurological compromise.
8. Hypophosphatemia - particularly important during refeeding.

Therefore, routine laboratory assessment remains essential when evaluating medically compromised patients with AN.

Junctional Rhythms and Conduction Disturbances

Although less common than sinus bradycardia, other conduction abnormalities have been described.

Sachs et al. (2016) identified reports of:

1. junctional escape rhythms,
2. atrioventricular conduction abnormalities,
3. severe sinus pauses.

These disturbances typically occur in individuals with advanced malnutrition.

Their presence should prompt consideration of inpatient monitoring.

In contrast, Chu et al. (2019) observed no major conduction abnormalities in their cohort of adolescents undergoing CMR assessment.

This discrepancy likely reflects differences in disease severity.

Thus, conduction abnormalities appear to exist along a continuum.

Early disease may involve physiological bradycardia.

Advanced disease may progress toward pathological rhythm disturbances.

Sudden Cardiac Death

Sudden cardiac death (SCD) remains one of the most feared complications of AN.

The precise incidence remains uncertain because many deaths occur outside hospital settings and autopsy findings are frequently inconclusive.

Nevertheless, cardiovascular causes account for approximately one-third of deaths associated with AN (Sardar et al., 2015).

Historically, SCD was attributed primarily to torsades de pointes arising from QT prolongation. However, contemporary evidence suggests a multifactorial pathogenesis.

Potential mechanisms include

1. Malignant ventricular arrhythmias - triggered by electrolyte abnormalities.
2. Bradyarrhythmias - related to profound vagal predominance.
3. Structural remodeling - providing arrhythmogenic substrates.
4. Myocardial fibrosis - disrupting electrical continuity.
5. Refeeding-associated cardiac failure - during nutritional rehabilitation.
6. Cardiogenic shock - in end-stage disease.

Jáuregui-Garrido and Jáuregui-Lobera (2012) emphasized several clinical markers associated with increased risk:

1. chronic hypokalemia;
2. QT intervals exceeding 600 ms;
3. long-standing eating disorders;
4. chronic hypoalbuminemia;
5. severe malnutrition.

Despite these associations, definitive mechanisms remain elusive.

As Sachs et al. (2016) concluded, direct evidence linking any single abnormality to SCD is lacking.

Sudden death in AN likely represents the final common pathway of multiple interacting cardiovascular insults.

Refeeding Syndrome and Cardiac Complications

Refeeding syndrome (RFS) represents a paradoxical complication occurring during nutritional rehabilitation.

Although restoration of nutrition is lifesaving, rapid reintroduction of calories may precipitate severe metabolic disturbances.

The hallmark abnormality is hypophosphatemia.

During starvation

1. intracellular phosphate stores become depleted;
2. insulin secretion decreases.

Upon refeeding

1. insulin release accelerates;
2. phosphate shifts intracellularly;
3. serum phosphate concentrations fall precipitously.

Cardiovascular consequences include

1. Impaired myocardial contractility - due to ATP depletion.
2. Acute heart failure - secondary to energetic deficiency.
3. Arrhythmias - triggered by electrolyte shifts.
4. Hypotension - compromising perfusion.
5. Sudden death - in severe cases.

Jáuregui-Garrido and Jáuregui-Lobera (2012) highlighted cardiac failure as one of the most serious complications of RFS.

Careful monitoring of phosphate, potassium, magnesium, and fluid status is therefore mandatory during nutritional rehabilitation.

Table 2. Electrophysiological Alterations in Anorexia Nervosa

Abnormality	Proposed Mechanism	Clinical Significance	Reversibility
Sinus bradycardia	Increased vagal tone, low T3	Syncope, hospitalization	Usually reversible
QT prolongation	Electrolyte imbalance, malnutrition	Torsades de pointes risk	Often reversible
Increased QT dispersion	Heterogeneous repolarization	Possible predictor of SCD	Improves with recovery
Reduced HRV	Autonomic dysfunction	Reduced chronotropic reserve	Improves after weight gain
Junctional rhythm	Severe starvation	Telemetry indication	Usually reversible
Ventricular arrhythmias	Electrolyte disturbances	Sudden death	Potentially preventable
Refeeding-related arrhythmias	Hypophosphatemia	Acute decompensation	Preventable with monitoring

Collectively, the available evidence indicates that electrophysiological abnormalities in anorexia nervosa arise from a complex interaction between adaptive autonomic responses and maladaptive metabolic consequences of starvation. While many changes improve following nutritional rehabilitation, severe disturbances may culminate in life-threatening arrhythmias and sudden cardiac death, underscoring the importance of early recognition and careful cardiovascular monitoring.

Clinical Outcomes, Prognosis, and the Impact of Weight Restoration

The clinical course of cardiovascular abnormalities in anorexia nervosa (AN) is highly heterogeneous and largely dependent on disease severity, duration of malnutrition, presence of compensatory behaviors, and the achievement of sustained nutritional rehabilitation. While most cardiovascular manifestations appear reversible, a subset of patients develops severe complications associated with substantial morbidity and mortality.

Understanding the long-term trajectory of cardiac involvement is essential for risk stratification and clinical decision-making.

Clinical Outcomes in Acute Anorexia Nervosa

Cardiovascular complications are among the leading causes of medical hospitalization in AN.

The most frequently encountered manifestations include:

1. sinus bradycardia,
2. orthostatic hypotension,
3. exercise intolerance,
4. dizziness,
5. presyncope,
6. syncope,
7. silent pericardial effusions.

These abnormalities generally reflect physiological adaptation to starvation and improve with refeeding. However, severe malnutrition may overwhelm compensatory mechanisms.

Friars et al. (2023) emphasized that cardiovascular compromise should be considered a marker of medical instability. Patients presenting with severe bradycardia, marked orthostatic changes, syncope, or significant electrolyte disturbances require urgent evaluation and frequently warrant inpatient admission.

Clinical vigilance is particularly important because outward appearance does not necessarily correlate with cardiovascular risk. Patients with atypical AN, characterized by substantial weight loss despite BMI values within normal ranges, may also experience significant medical complications (Carlson & Lemly, 2024).

Thus, assessment should focus on the trajectory and severity of nutritional compromise rather than BMI alone.

Long-Term Cardiac Outcomes

One of the most clinically relevant questions concerns whether cardiovascular abnormalities persist after recovery from AN.

The available evidence suggests that long-term outcomes are generally favorable among patients who achieve sustained nutritional restoration.

Flamarique et al. (2022) conducted one of the longest follow-up studies available, evaluating women diagnosed with adolescent-onset AN approximately twenty years after their initial treatment.

Participants were divided into two groups:

1. Normal-weight group - patients who had restored and maintained healthy body weight.
2. Low-weight group - patients who continued to meet criteria for active AN.

The investigators found that individuals who had normalized their weight demonstrated:

1. echocardiographic findings comparable to healthy controls;
2. normal electrocardiographic parameters;
3. absence of clinically significant structural abnormalities.

In contrast, persistently underweight individuals continued to exhibit:

1. reduced left ventricular dimensions;
2. lower left ventricular mass;
3. smaller left atrial size.

These findings suggest that cardiovascular recovery is strongly dependent upon nutritional restoration.

Importantly, the study provides reassurance that permanent cardiac damage is not inevitable.

Instead, the heart appears capable of remarkable structural and functional recovery, even after prolonged illness.

Weight Restoration as the Central Therapeutic Intervention

Perhaps the most consistent finding throughout the cardiovascular literature on AN is the beneficial effect of weight restoration.

Improvement following nutritional rehabilitation has been documented across multiple domains.

Structural recovery

Olivares et al. (2005) observed significant increases in:

1. left ventricular mass index;
2. left ventricular end-diastolic dimensions;
3. cardiac output.

following recovery.

Similarly, Kastner et al. (2012) reported remission of pericardial effusions in approximately 88% of adolescents after weight restoration.

Electrophysiological recovery

Weight gain has been associated with:

1. normalization of heart rate;
2. shortening of QT intervals;
3. reduction in QT dispersion;
4. restoration of autonomic balance.

Functional recovery

Paysal et al. (2022) demonstrated partial normalization of heart rate variability indices and global longitudinal strain after nutritional rehabilitation.

Collectively, these findings indicate that nutritional restoration addresses the underlying pathophysiological mechanisms responsible for cardiac dysfunction.

Thus, refeeding should be regarded not merely as psychiatric treatment but as cardiovascular therapy.

Severe Cardiac Presentations: Cardiogenic Shock

Although uncommon, severe cardiac presentations continue to be reported in patients with advanced AN.

The recently published case series by Thienel et al. (2025) described three patients with severe AN who developed cardiogenic shock requiring admission to a cardiac intensive care unit.

The patients presented with:

1. profound malnutrition;
2. severe ventricular dysfunction;
3. multiorgan failure;
4. respiratory compromise.

Two patients experienced cardiac arrest requiring successful resuscitation.

Two patients required mechanical circulatory support.

Advanced therapies included

1. Venoarterial extracorporeal membrane oxygenation (VA-ECMO) - providing temporary cardiac and respiratory support.

2. Microaxial flow pumps - used to maintain systemic perfusion during myocardial recovery.

Management proved exceptionally challenging because clinicians were required to balance:

1. aggressive cardiovascular support;
2. cautious nutritional rehabilitation;
3. prevention of refeeding syndrome;
4. correction of electrolyte abnormalities;
5. supplementation of vitamins and trace elements.

Remarkably, favorable outcomes were achieved.

These reports expand the spectrum of cardiovascular involvement in AN and highlight that starvation-related myocardial dysfunction may occasionally progress beyond adaptive remodeling toward fulminant heart failure.

Although rare, such cases underscore the importance of multidisciplinary collaboration.

Mortality and Sudden Death

AN consistently demonstrates one of the highest mortality rates among psychiatric disorders.

Lamzabi et al. (2015) reported standardized mortality ratios approaching six-fold higher than those of the general population.

Several factors contribute to mortality:

1. **Suicide** –reflecting the psychiatric burden of disease.
2. **Infection**- related to malnutrition-induced immunosuppression.
3. **Metabolic complications** -including dehydration and electrolyte disturbances.
4. **Cardiovascular causes** - accounting for approximately one-third of deaths.

The cardiovascular contribution to mortality remains incompletely understood.

As previously discussed, sudden cardiac death likely reflects multiple interacting mechanisms.

Potential contributors include

1. ventricular arrhythmias;
2. profound bradyarrhythmias;
3. structural myocardial abnormalities;
4. fibrosis;
5. electrolyte disturbances;
6. refeeding-related complications.

The inability to identify a single dominant mechanism has important implications.

It suggests that prevention strategies should target overall medical stabilization rather than isolated ECG findings.

Discussion

The present narrative review synthesizes two decades of evidence regarding cardiovascular involvement in anorexia nervosa and demonstrates that AN exerts profound effects on cardiac structure and function. Several major themes emerge from the available literature.

Starvation Produces a Distinct Cardiac Phenotype

The first major observation is that AN is associated with a recognizable pattern of cardiac remodeling. This phenotype is characterized by:

- reduced myocardial mass;
- smaller ventricular chambers;
- lower cardiac output;
- sinus bradycardia;
- hypotension;
- autonomic predominance of vagal activity;
- occasional pericardial effusion.

Unlike classical cardiomyopathies, systolic function is generally preserved despite significant anatomical alterations.

These findings support the concept that starvation induces a unique form of metabolic cardiomyopathy. Importantly, the majority of these changes appear adaptive.

Reduced cardiac workload likely represents an attempt to match circulatory function with diminished metabolic demands.

However, adaptation exists along a continuum.

Beyond certain thresholds of disease severity and duration, adaptive responses may become maladaptive.

The Heart Is Not Protected From Malnutrition

Historically, the cardiovascular system was considered relatively spared during starvation.

The evidence reviewed herein challenges this assumption.

Multiple imaging studies consistently demonstrate myocardial atrophy proportional to nutritional compromise.

Furthermore, pathological investigations reveal that prolonged disease may culminate in:

1. fibrosis;
2. myxoid degeneration;
3. lipofuscin accumulation.

These observations indicate that the myocardium should be regarded as a vulnerable target organ.

Recognition of this vulnerability is essential because cardiac abnormalities are often clinically silent.

Normal ejection fraction does not exclude significant remodeling.

Similarly, asymptomatic patients may harbor substantial cardiovascular compromise.

Sudden Cardiac Death Is Multifactorial

A particularly important finding concerns the mechanisms underlying sudden cardiac death. For decades, emphasis was placed almost exclusively on QT prolongation. However, contemporary evidence suggests that this explanation is insufficient. Not all patients exhibit prolonged QT intervals. Not all sudden deaths occur in the setting of repolarization abnormalities.

Rather, mortality likely results from the convergence of multiple factors:

- electrolyte disturbances;
- autonomic dysfunction;
- structural remodeling;
- myocardial fibrosis;
- severe bradyarrhythmias;
- refeeding-related complications.

This multifactorial model more accurately reflects the heterogeneity observed in clinical practice.

Future studies should therefore move beyond isolated ECG markers and adopt integrated risk models.

Weight Restoration Is Cardioprotective

Perhaps the most encouraging finding emerging from this review is the reversibility of cardiac abnormalities.

Across structural, functional, and electrophysiological domains, nutritional rehabilitation consistently improves cardiovascular outcomes.

Weight restoration was associated with:

- normalization of ventricular dimensions;
- improvement in cardiac output;
- resolution of pericardial effusions;
- recovery of autonomic balance;
- normalization of repolarization indices.

Long-term follow-up studies further demonstrate that patients maintaining healthy body weight exhibit cardiovascular profiles similar to those of healthy controls.

These observations reinforce an important clinical message:

Early diagnosis and effective treatment not only improve psychiatric outcomes but may prevent permanent cardiovascular injury.

Clinical Implications

Several practical implications arise from the reviewed evidence.

First, all patients with suspected AN should undergo cardiovascular assessment, including:

- orthostatic vital signs;
- electrocardiography;
- laboratory evaluation of electrolytes.

Second, severe bradycardia, syncope, marked QT prolongation, or electrolyte disturbances should prompt inpatient monitoring.

Third, clinicians should recognize that normal systolic function does not exclude significant remodeling.

Finally, nutritional rehabilitation should be prioritized as the cornerstone of cardiovascular management.

Limitations of Current Evidence

Despite considerable progress, important limitations remain.

The majority of available studies involve:

1. small sample sizes;
2. predominantly female populations;
3. adolescent cohorts;
4. observational designs.

Advanced imaging studies remain scarce.

Autopsy data are limited to isolated cases.

Prospective studies capable of identifying predictors of irreversible myocardial injury are lacking.

Additionally, there remains no validated tool specifically designed to predict sudden cardiac death risk in AN.

Future investigations should address these gaps through multicenter longitudinal studies incorporating modern imaging modalities and electrophysiological monitoring.

Conceptualization: K.Z. methodology, P.W., K.W. Literature search: A.J., H.L. and A.W. Formal Analysis: K.M., P.W. and K.W. Validation: P.B., K.Z. Writing—original draft preparation: K.Z., M.P. and A.W. Writing—Review and editing: K.Z., P.P. Supervision, P.P.

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