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Gender Affirming Hormone Therapy and Potential Effects on Myocardium, Ventricular Morphology, and Cardiac Function: A Literature Review

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Abstract

Introduction: Approximately 1.4 million adults in the United States identify as transgender, about half of whom are treated with gender-affirming hormone therapy (GAHT). Transgender individuals experience increased cardiovascular risk, which may be partly related to GAHT. However, GAHT's effects on cardiac morphology and function are not well understood. The role of echocardiography in assessing these potential effects remains unclear.

Methods: A literature review was conducted using four major databases with keywords relevant to transgender and cardiovascular health. Articles were screened for relevance based on titles, and selected abstracts were reviewed for the initial inclusion criterion: (1) GAHT and potential effects on cardiovascular health. Remaining articles were read in full and eliminated or stratified based on research themes: (a) GAHT's effect on cardiac myocardium, (b) effects on ventricular morphology or function, or (c) the role of echocardiography in evaluating GAHT-related cardiac changes.

Results: 960 articles were initially identified, of which 214 distinct abstracts were reviewed, and 85 publications were read in full. 9 articles contained information regarding at least one of the three research themes. Reviewed data suggest GAHT may affect myocardial mass, end diastolic volume, and diastolic function in transgender men. Transgender women may have increased LVEF during exercise. Few studies used echocardiography to evaluate these changes.

Conclusion: GAHT may increase cardiovascular risk in transgender individuals, though underlying mechanisms remain unclear. Few articles evaluate GAHT's effect on cardiac structure and function, though current data suggest possible cardiac effects. The use of echocardiography to assess GAHT-related cardiac changes remains underexplored.

Introduction

The World Professional Association of Transgender Healthcare (WPATH) defines transgender as a broad term encompassing individuals with gender identity that differs to varying degrees from the sex they were assigned at birth[1]. The terms trans feminine, transgender women or male-to-female refers to those assigned male at birth who identify as female, while the term trans masculine, transgender men, or female-to-male describes those assigned female at birth who identify as male. Non-binary gender identities include transgender people who do not identify as strictly feminine or masculine[1]. Approximately 1.4 million adults in the United States identify as transgender, with nearly 55% undergoing gender-affirming hormone therapy (GAHT) for the treatment of gender incongruence[2,3].

The principles of GAHT are to reduce endogenous sex

hormone levels thereby reducing natal secondary sexual characteristics, and to substitute sex hormones that are consistent with one's gender identity[4]. Transgender women are often prescribed topical or oral estrogen plus anti-androgens, such as spironolactone, which can promote breast development, decrease facial and body hair growth, decrease testicular volume, and alter body mass distribution. In transgender men, the primary hormonal treatment is testosterone, usually prescribed as intramuscular injections, or topical dermal gel formulations[1,4]. While full masculinizing or feminizing effects of GAHT can take up to 5 years to complete, the impact of GAHT on the heart remains understudied, and available literature highlights lack of standardization limiting generalizability of findings[1,5].

Sex Hormones and the Heart

Cardiac morphology and function differ between the sexes. Left ventricular mass is similar for both sexes in infancy, however it increases by 25-38% in cisgender males after puberty[6]. The adult cisgender female heart weighs on average 26% less than its male counterpart, but is richer in cardiomyocytes[7,8]. Importantly, the cisgender female heart is characterized by higher ejection fraction, higher resting heart rate, higher contractility, but lower left ventricular end-diastolic volume, lower stroke volume, and lower overall cardiac output[7,9]. These sex-differences persist even after scaling by lean body mass, proving that these observations reflect functional variation attributed to sex rather than simple anthropometric scaling[7]. Sex differences in myocardial aging have also been documented. Cisgender females experience increased wall thickening in response to risk exposure, concentric remodeling, and diastolic dysfunction. On the other hand, cisgender males experience eccentric remodeling and systolic dysfunction[10]. Indeed, echocardiographers use sex-based differences as measuring parameters for echography, with guidelines directing normal left ventricular diastolic dimension 2-SD range 42.0-58.4 for males and 37.8-52.2 for females respectively[11]. There are additional sex-based differences in normal values of left ventricular ejection fractions with females having a slightly higher normal range (54-74%) compared to males (52-72%)[11].

The extent to which sex hormones, in particular estrogen and testosterone, drive these differences remains unclear. Estrogens have been shown to exert anti-hypertrophic effects, improve mitochondrial efficiency, lower contractile function, and have anti-apoptosis effects on cardiac myocytes[12]. Meanwhile, testosterone has pro-hypertrophic effects, enhances contractility, and may impact cardiac metabolism and turnover[12,13]. Data has also suggested sex hormones impact the cardiac rhythm, with sex based differences in cardiac electrical activity noted in electrocardiography as well as circulating levels of cardiac biomarkers including natriuretic peptide and high sensitivity troponin[14–16]. These findings suggest that sex hormones may have direct and indirect effects on cardiac myocytes.

Gender Affirming Hormone Therapy and the Heart

The effects of GAHT on cardiac morphology and function are poorly characterized, though previous reviews attempted to extrapolate results from rodent models and cisgender populations with altered hormonal states[13,17]. For instance, Santos et al proposed potential harmful cardiovascular effects of testosterone therapy including increasing pro-contractile mechanisms on the heart[13].

Table 1: Summary of terms used

Term	Definition
Sex	Attributes that characterize biological maleness or femaleness, which may include sex determining genes, sex chromosomes, H-Y antigen, sex hormones, internal and external genitalia, and secondary sex characteristics
Sex assigned at birth	A person's sex that was assigned at birth, usually based on male or female genital anatomy. Acronyms such as AFAB (assigned female at birth) or AMAB (assigned male at birth) may be used
Gender	Social and cultural characteristics pertaining to and differentiating between perceived sex characteristics, typically described as masculinity and femininity
Gender Identity	A person's internal sense of gender, which may or may not align with their sex assigned at birth
Cisgender	A person who is not transgender; a person whose gender identity aligns with the sex they were assigned at birth
Transgender	An umbrella term for a person whose gender identity differs from the sex they were assigned at birth; also known as trans
Transgender Female (TGW)	A woman whose sex assigned at birth was male, but who identifies as female; also known as trans woman, male-to-female transgender, transgender woman
Transgender Male (TGM)	A man whose sex assigned at birth was female, but who identifies as male; also known as trans man, female-to-male transgender, transgender man
Gender Affirming Hormone Treatment	Varying exogenous hormone treatments aimed to reduce natal secondary sexual characteristics and substitute hormones that are consistent with one's gender identity

These findings come from experimental rodent models showing an increase in adrenergic receptors, L-type calcium channels, and sodium/calcium exchange channels in male and female rats treated with testosterone. Left ventricular hypertrophy and contractility also increased in female rats treated with testosterone[13,18,19]. Another rodent model showed supratherapeutic doses of testosterone in male rats were related to pathologic cardiac hypertrophy[20]. Beyond these structural and electrophysiologic alterations, testosterone has been shown to produce atherogenic lipid patterns via increase in LDL-C and reduction in HDL-C[21]. Testosterone increases hematocrit via erythrocytosis, which is associated with a modest increase in blood pressure and variably influence insulin sensitivity[22].

Additionally, extrapolated data from individuals of altered hormonal states, including hyper- or hypogonadism has been used to propose potential effects of GAHT. Cisgendered females with hyperandrogenemia from polycystic ovarian syndrome have been found to have cardiac hypertrophy[23]. Supraphysiologic androgen levels on the athlete heart have been shown to include increased myocardial thickness, reduction in left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS) pattern[24]. Another systematic review of 8 clinical trials looking at testosterone replacement therapy in hypogonadal cisgender men with congestive heart failure did not show significant changes to ejection fraction, however the majority of trials examined had a short follow up period of 12 weeks[25]. Data from prostate cancer cohorts undergoing androgen deprivation therapy demonstrate a reduction in left ventricular GLS pattern between 2.5 and 6.5% after six months of androgen deprivation[26,27].

Feminizing hormone therapy may increase insulin resistance, produce variable effects on lipids, and exert a pro-coagulant effect via complex interactions with clotting factors and endothelium[28,29]. Conjugated estrogen-based therapy has been associated with favorable diastolic changes in post-menopausal cisgender women[30,31]. Duzenli and colleagues observed that in 37 postmenopausal cisgender women, conjugated estrogen plus medroxyprogesterone acetate increased E/A ratio modestly from 0.96 to 1.01 (p<0.05) after six months of treatment[30]. No significant changes were noted in other echocardiographic parameters including LVEF or left ventricular mass. Similarly, Duygu and colleagues reported that in a cohort of 30 surgically menopausal cisgender women, conjugated estrogen increased E/A ratio from 1.15 to 1.42 (p=0.0001) over 12 months[31].

Since blood tests cannot monitor conjugated estrogen levels, their use is discouraged in transgender women; instead, The Endocrine Society recommends preferential use of transdermal or oral 17β-estradiol[4]. In a randomized controlled trial including 152 surgically menopausal cisgender women, 33 received transdermal 17β-estradiol and 37 received oral 17β-estradiol, no significant changes were noted in E/A ratio or LVEF by 12 months[32].

Comparing transgender individuals to cis-gender population may be problematic as these groups likely differ in age and comorbidities. Although testosterone and estrogen may exert direct and indirect effects on the heart, studies specifically examining the effect of GAHT on cardiac function and morphology in transgender individuals remain limited.

Methods

The authors conducted an extensive, date-specific search, including from January 1997 - February 2025 to identify relevant literature. Databases utilized include Access Medicine, Cardiosource Plus, Embase, and PubMed. Keyword search terms included combinations of words relating cardiovascular health and transgender populations including “transgender”, “gender affirming hormone therapy”, “GAHT”, and “heart”, “cardiovascular”, “cardiac”, “myocardium”, and “echocardiography”. The databases were accessed during the two search periods of December 2024-March 2025 and July 2025. Within these databases, articles, and book chapters were reviewed; editorial comments were excluded.

Literature Selection

The search included a three-stage screening process. In the first stage, publications were screened for relevance based on titles. Documents were excluded if they did not pertain to both cardiovascular health and transgender patients. Any publication that was deemed relevant was advanced to abstract review. During the second stage, abstracts were screened based on the initial inclusion criterion relating to GAHT and potential effects on cardiovascular health. Abstracts that met this initial criterion were then advanced to the third and final stage, during which their full text was accessed and further assessed. Publications were then stratified and eliminated based on three research themes.

Inclusion Criteria

Publications were included if they were articles or book chapters published between January 2005-March 2025 and met initial inclusion criteria of (1) focused on topics of GAHT and cardiovascular health. Peer-reviewed articles and book chapters meeting this primary inclusion criterion were read in full, without restrictions on study design or location. The remaining documents then underwent full-text review and were stratified based on three research themes: (a) cardiovascular effects of GAHT at the level of the myocardium, excluding articles pertaining to QTc, (b) the potential effect of GAHT on cardiac ventricular morphology or function, or (c) the role of echocardiography

in evaluating GAHT related cardiac changes. Articles that did not focus on these three themes were eliminated.

Exclusion Criteria

Documents were initially excluded if they were deemed irrelevant based on their titles in that they did not focus on both transgender and cardiovascular health. Additionally, publications were not included if they pertained to cis-gendered individuals receiving hormone therapy or if they relayed information based on overall sexual orientation or sexual orientation minority groups as such LGBTQ+ communities. Items pertaining specifically to vascular health, such as characteristics of large arteries or venothrombotic risk were excluded. Publications that focused on cardiometabolic risk factors, such as lipid profiles, metabolic disorders, psychosocial stress, and blood pressure were also excluded at either the first or second stages of screening. Additionally, articles relaying changes in EKG patterns were excluded from review as such topic was beyond the scope of this literature review. Duplicate records, non-English language publications, non-peer-reviewed works, abstracts, and editorial comments were additionally excluded from review.

Results

The search of the four databases identified 4363 records. Of these, 960 publications were identified based on title relevance. After removal of duplicates, 214 distinct abstracts were screened in secondary review. 95 abstracts met secondary inclusion criteria, of which, 85 publications were accessed and read in full. Of these reviewed articles, 9 pertained to the study research themes.

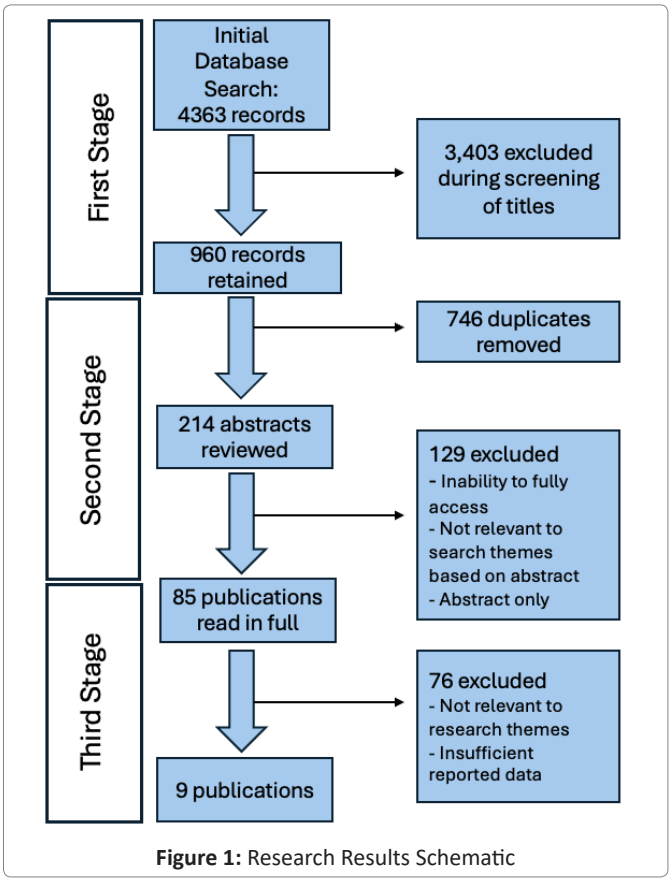

Figure 1: Research Results Schematic

Few studies evaluated echocardiographic changes in transgender populations, although they were limited by small sample size, short follow up duration, and variable GAHT regimens[33,34]. In a single center case-control study, authors compared stress echocardiography profiles of transgender women (n = 43) with those age-matched cisgender men and women (n = 84 and 86, respectively)[33]. At baseline, transgender and cisgender women had similar left ventricular end-diastolic diameter (LVEDD) (median [IQR], 48 [44-51] vs 47[43-50] mm, p=0.2) and similar left ventricular end-systolic diameter (LVESD) (30 [28-33] vs 30 [28-33] mm, p=0.92). In contrast, transgender women had a slightly smaller LVEDD and LVESD compared to cisgender men (48 [44-51] vs 49 [47-53] mm, p=0.23, and 30 [28-33] vs 32.5 [30-36] mm, p=0.018, respectively). The left ventricular mass index and left atrial volume index did not differ in transgender women when compared to both cisgender groups. With exercise, transgender women had a greater rise in LVEF compared to cisgender women (70% [70%-75%] vs 70% [65%-70%], p=0.007), but similar to cisgender men (70% [70%-75%] vs 70% [65%-75%], p = 0.08). Exercise mitral valve E/e' ratio was lower in transgender women compared to cisgender women (8.6 [7.5-10] vs 10 [8-12], p=0.029], but similar to cisgender men 8.6 [7.5-10] vs 10 [7.7-11.3], p= 0.53). The results suggest that transgender women's echocardiographic traits lie on a spectrum between cisgender men and women but may be more closely aligned with their affirmed gender.

Similarly, a study on transgender men included 32 individuals on long-term GAHT and 15 individuals newly initiating GAHT had found that new initiators showed a mild decline in diastolic function within 1 year[34]. The mild reduction in diastolic function was reflected by reduced septal and lateral early LV relaxation (from 14 to 11 cm/s, p = 0.04; and 18 to 15 cm/s, p = 0.02, respectively). In the same study, baseline measurements indicated that participants on GAHT for a median duration of 4 years had larger LVEDD compared with new initiators (4.6 cm vs. 4.4 cm; p < 0.05). Both groups demonstrated trends toward increased stroke volume, cardiac output, LV mass index, and relative wall thickness by the end of the study, though changes were modest and not statistically significant. The authors speculated that changes in LVEDD and diastolic function may represent an early adaptive response to volume overload mediated by testosterone rather than a pathologic response.

Limited studies evaluated cardiac MRI changes in transgender individuals[35,36]. In one study, transgender men demonstrated a statistically significant increase

Figure 2: Chart summarizing relevant studies

Study (Author, Year)	Population (N)	Objective	Key Cardiac Findings
Completed Studies			
Adel et al[33], 2025	213 total; 43 TGW, 84 CGM, 86 CGW	Compare stress echocardiography of TGW with age-matched CGM and CGW	TGW and CGW had non-significant difference LVEDD and LVESD; TGW had smaller LVEDD vs CGM (p=0.018); With exercise, TGW had greater rise in LVEF compared to CGW (p=0.007)
Buhl et al[34], 2025	47 TGM (15 initiated; 32 previously treated)	Evaluate changes in cardiac morphology, systolic and diastolic function after masculinizing GAHT	TGM on GAHT median 4 years had larger LVEDD vs new initiators; After 1 year, TGM had mild reduction in diastolic function; Trends toward increased stroke volume, cardiac output, LV mass index
Deischinger et al[35], 2025	35 total; 20 AFAB and 15 AMAB	Evaluate MRI for changes in myocardial mass after 6 months GAHT	AFAB patients had significant increase in myocardial mass (p=0.011) and end-diastolic volume index (p=0.02); No significant change in LVEF, stroke volume, cardiac output in either group
Slukova et al[36], 2024	35 total; 15 TGW, 20 TGM	MRI-spectroscopy to examine intramyocardial lipid content at initiation and after 6 months GAHT	Myocardial lipid content statistically unchanged in both TGW and TGM at 6 months compared to baseline
Proposed Studies			
Wiik et al[38], 2018	n/a	GETS study: cardiovascular function, metabolism, body composition	n/a
Christensen et al[39], 2020	n/a	BIC study: effects of testosterone on coronary disease, cardiac and respiratory function in TGM	n/a
Deischinger et al[37], 2024	n/a	Investigate effects of GAHT on cardiovascular risk profile, insulin, body composition (10-year extension)	n/a
Toribio et al[40], 2019	n/a	Cardiometabolic effects of initiating GAHT in TGW with/without HIV; cardiac MRI/MRS	n/a
Alvares et al[41], 2023	n/a	Aerobic capacity, strength, performance of TGW volleyball players undergoing hormone therapy	n/a

TGW = transgender women, TGM = transgender men, CGW = cisgender women, CGM = cisgender men, AFAB = assigned female at birth, AMAB = assigned male at birth, GAHT = gender affirming hormone therapy, LVEDD = left ventricular end-diastolic diameter, LVESD = left ventricular end-systolic diameter, n/a = not applicable

in mean myocardial mass by 6 g/m^2 (p=0.011) and an increase in end-diastolic volume index from 72 to 75 mL/m^2 (p=0.02) only six months after starting GAHT[35]. In the same study, transgender women had a nonsignificant decrease in myocardial mass by 4g/m^2 and decrease in end-diastolic volume index from 83 to 80 mL/m^2 during the same period. No significant changes were observed in LVEF, stroke volume, cardiac index, end-systolic volume index, peak filling rate, or diastolic function in either group. In another study by the same authors, MRI-spectroscopy imaging showed a non-significant reduction in median myocardial lipid content in transgender women (0.45% vs 0.4%, p=0.754) and transgender men (0.62% vs 0.29%, p=0.084) over a period of 6 months[36]. An extension of this study is planned for a follow up duration of up to 10 years, and may help shed light on long-term cardiac changes[37].

Many of the found abstracts were proposed study protocols. The Gender Dysphoria Treatment in Sweden (GETS) study will be looking at cardiovascular function as one of the secondary outcomes[38]. The Body Identity Clinic (BIC) study in Denmark will look at effects of masculinizing hormone therapy by utilizing coronary CT before and after 10 years of treatment[39]. A prospective study at MGH plans to evaluate the cardiometabolic effects of initiating GAHT among transgender women with and without HIV[40]. A group in Brazil will evaluate transgender women volleyball athletes longitudinally compared to age-matched cisgender men and women over a period of 6 to 12 months[41].

Conclusion

This paper is a literature review examining how GAHT affects cardiac structure and function. Existing studies are limited in number, generally small, and heterogeneous in design, with most prospective cohort studies conducted at single centers in Europe. Despite these limitations, available data suggest that GAHT therapy may have direct or indirect effects on the myocardium and may be associated

with changes in ventricular morphology and function, even in the shorter term. Testosterone masculinization therapy in transgender men may increase myocardial mass size, reduce diastolic function, and increase LVEDD over time. However, data regarding testosterone effects on LVEF, stroke volume, cardiac output remain inconclusive. Data is even less conclusive for transgender women receiving estrogen-based GAHT, with findings suggesting nonsignificant reductions in LVEDD compared to cisgender men, and no significant differences in LVEDD or LVESD compared to cisgender women. Additionally, GAHT does not appear to affect myocardial lipid content over short durations. Though there is increasing movement for longer longitudinal clinical trials, no current data exists on the long-term effects of GAHT on myocardial structure or ventricular function.

The cardiovascular mechanisms underlying GAHT are complex and not fully understood. Testosterone has been associated with an atherogenic lipid pattern, variable effects on insulin resistance, and may enhance adrenergic signaling and upregulate L-type calcium channels, potentially leading to myocardial dysfunction. Estrogen may influence cardiovascular risk through effects on insulin sensitivity, coagulation pathways, and endothelial function, including a pro-thrombotic tendency. Notably, both excess and deficiency of sex hormones have been linked to adverse cardiac performance.

The clinical significance of these findings is unclear due to methodological limitations across studies examined. All studies were conducted on small cohorts, limiting statistical power. Follow up periods were of short duration, ranging from 3 to 12 months. As transgender men and women may need up to 5 years for secondary sexual characteristics to fully take effect and are often on GAHT indefinitely, longer-term studies are essential for a more complete assessment of cardiac adaptations. GAHT regimens across studies were not standardized. Comorbidities such as tobacco or alcohol use, obesity, and hypertension, were not controlled for and may have confounded results. Finally, as most of the available studies originated from European centers, we are unable to generalize results to more diverse populations.

This study highlights the need for larger, long-term, multicenter or registry based prospective studies to evaluate the cardiac effects of GAHT in both transgender men and women. It also illustrates the role of echocardiography as a potentially important modality for healthcare maintenance of transgender individuals. Future investigations should incorporate advanced imaging modalities such as speckle-tracking echocardiography and cardiac magnetic resonance imaging to detect subclinical myocardial dysfunction. Standardized reporting of hormone type, dose, duration, and achieved serum levels should enable meaningful comparisons across studies.

Conflicts of Interest

The authors report no conflicts of interest.

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