EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES

http://www.ejbps.com

ISSN 2349-8870
Volume: 12
Issue: 12
Year: 2025

"FROM ROOTS TO RHYTHM": 'SYNERGYSTIC ROLE OF ARJUNA TERMINALIA AND WITHANIA SOMNIFERA IN FORM OF POLYHERBAL CARDIOPROTECTIVE SYRUP'

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How to cite this Article: *1Poonam Ankush Jadhav (Asst. Proff.), 2Taher Unus Shaikh (Student), 3Dheeraj Jivan Sonkamble (Student), (2025). "From Roots To Rhythm": 'Synergystic Role Of Arjuna Terminalia And Withania Somnifera In Form Of Polyherbal Cardioprotective Syrup'. European Journal of Biomedical and Pharmaceutical Sciences, 12(12), 19–29.

Article Received on 28/10/2025 **Article Revised on 17/11/2025** **Article Accepted on 01/12/2025**

ABSTRACT

In 2022, the World Health Organization (WHO) reported that cardiovascular diseases (CVDs) caused about 19.8 million deaths worldwide. These diseases affect the heart and blood vessels, including heart disease, stroke, blocked arteries, heart defects, and blood clots. Common risk factors for CVDs include diabetes, high blood pressure, and high cholesterol, lack of exercise, obesity, and tobacco and alcohol use. WHO estimated that India spent $237 billion on treating CVDs between 2005 and 2015. High blood pressure is strongly linked to heart disease, as it accelerates the build-up of plaque in arteries due to inflammation. Treatments often focus on lowering blood pressure to reduce this risk. Medications like β-blockers relax blood vessels and improve blood flow. High cholesterol also raises risk, and doctors treat it with drugs such as atorvastatin, though long-term use may increase the risk of type 2 diabetes. The bark of the Arjuna tree (Terminalia arjuna), found throughout India, contains natural compounds such as tannins, flavonoids, and minerals, which offer anti-inflammatory and anticancer benefits. Traditionally, Arjuna has been valued for heart health support, where modern therapies may rely on drugs with side effects. It has shown potential to lower blood sugar, prevent blood clots, reduce blood pressure, and fight infections. Its triterpenoids benefit the heart, while flavonoids and tannins target cancer, demonstrating a broader therapeutic profile than many standard medications.

KEYWORDS: WHO estimated that India spent $237 billion on treating CVDs between 2005 and 2015.

INTRODUCTION

Cardiovascular diseases (CVDs) are a group of disorders that affect the heart and blood vessels, and they remain the leading cause of death globally. These conditions include coronary artery disease (CAD), heart failure, arrhythmias, congenital heart defects, rheumatic heart disease, and cerebrovascular diseases such as stroke.[26]

The primary causes of CVDs are lifestyle-related and include hypertension, diabetes, hyperlipidaemia, smoking, physical inactivity, obesity, unhealthy diet, and chronic stress. Atherosclerosis—the build-up of fatty deposits inside arteries—is a common underlying mechanism in many CVDs, leading to reduced blood flow and oxygen supply to vital organs.[26]

Symptoms can range from chest pain (angina), palpitations, and shortness of breath to more severe outcomes like heart attacks and strokes. Early detection, lifestyle modifications, pharmacological interventions, and in some cases, surgical procedures, are key to prevention and management.[28]

Due to the growing prevalence of CVDs, particularly in developing nations, there is increasing interest in complementary therapies, including polyhedral formulations, which may offer cardio protective, antihypertensive, lipid lowering, and antioxidant benefits.[11]

1) HYPERTENSION

Definition: Hypertension (high blood pressure) generally is when systolic BP ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg, or when a person is on antihypertensive medication.[27]

Why it matters: It is a major risk factor for cardiovascular diseases (heart attacks, stroke), kidney disease, and other complications. Often called a "silent killer" because many people are unaware they have it.[28]

Key Global Statistics

Table No. 1: Global Statics of Hypertension Disease.

Metric	Value / Estimate	Source / Notes
Number of adults aged 30–79 years with hypertension globally	~1.4 billion	2024 WHO estimate. World Health Organization
Proportion of adults 30–79 with hypertension worldwide	About 33%	WHO fact sheet. World Health Organization
Proportion undiagnosed among those with hypertension	~44%	WHO. World Health Organization
Year-2001 data: deaths attributed to high blood pressure	~7.6 million premature deaths (~13.5% of global total deaths)	The Lancet study on global burden in 2001. PubMed+1
Year-2001 data: DALYs (disability-adjusted life years) attributed to high blood pressure	~92 million DALYs (~6% of global total)	Same study. The Lancet+1

Mortality / Impact in Relation to Age[12]

Table No. 2: Mortality of Hypertension in Relation of Age.

Age Group	Mortality or Risk from Hypertension	Notes
Middle age (≈ 45–69 yrs)	Significant burden: many deaths attributable in these age groups	The global burden study indicated over half of the high blood pressure related disease burden occurred in people aged 45–69. ScienceDirect
Older age (≥ 70 yrs)	Even higher risk, though absolute numbers include large background mortality; control becomes more challenging	Studies show that very old age sees high prevalence-mortality, though risks must be interpreted in context. BioMed Central+1

PATHOPHYSIOLOGY OF HYPERTENSION

Primary Mechanisms

Diagram outlining the determinants of mean arterial pressure, showing dependencies on cardiac output (stroke volume, heart rate) and peripheral resistance (vascular tone, vascular structure).[30]

- Most hypertension cases (about 90–95%) are essential (primary), meaning they lack an identifiable secondary cause and arise from multifactorial origins.
- Hypertension results from increased cardiac output, increased peripheral vascular resistance, or both.
- Key determinants include:
- Over activation of the sympathetic nervous system, leading to increased heart rate and vasoconstriction.
- Renal dysregulation, causing impaired sodium excretion and subsequent fluid retention, contributing to increased blood volume and pressure.
- Stimulation of the renin–angiotensin–aldosterone system (RAAS), increasing vasoconstriction and sodium retention.
- Structural and functional changes in small arteries (arteriolar remodelling and rarefaction), raising vascular resistance.[15]

Additional Contributing Factors

- Chronic immune and inflammatory responses lead to vascular inflammation, endothelial dysfunction, and increased oxidative stress, all of which elevate resistance and arterial stiffness.
- Metabolic disturbances, especially obesity and insulin resistance, activate neurohormonal systems (RAAS, sympathetic drive) and promote vascular changes leading to hypertension.
- Genetic predispositions also affect sodium handling, vascular responsiveness, and hormone levels, influencing individual vulnerability.

Clinical Impact

- Persistent high blood pressure exerts excessive force against artery walls, potentially causing organ damage over time, including atherosclerosis, heart failure, kidney disease, and stroke.
- Untreated hypertension leads to further arterial remodelling and organ dysfunction, creating a cycle of worsening pressure and disease.

In summary, the pathophysiology of hypertension is a multifaceted process involving dysregulation in vascular tone, renal sodium management, neurohormonal systems, and inflammatory pathways, often acting together to maintain elevated blood pressure.

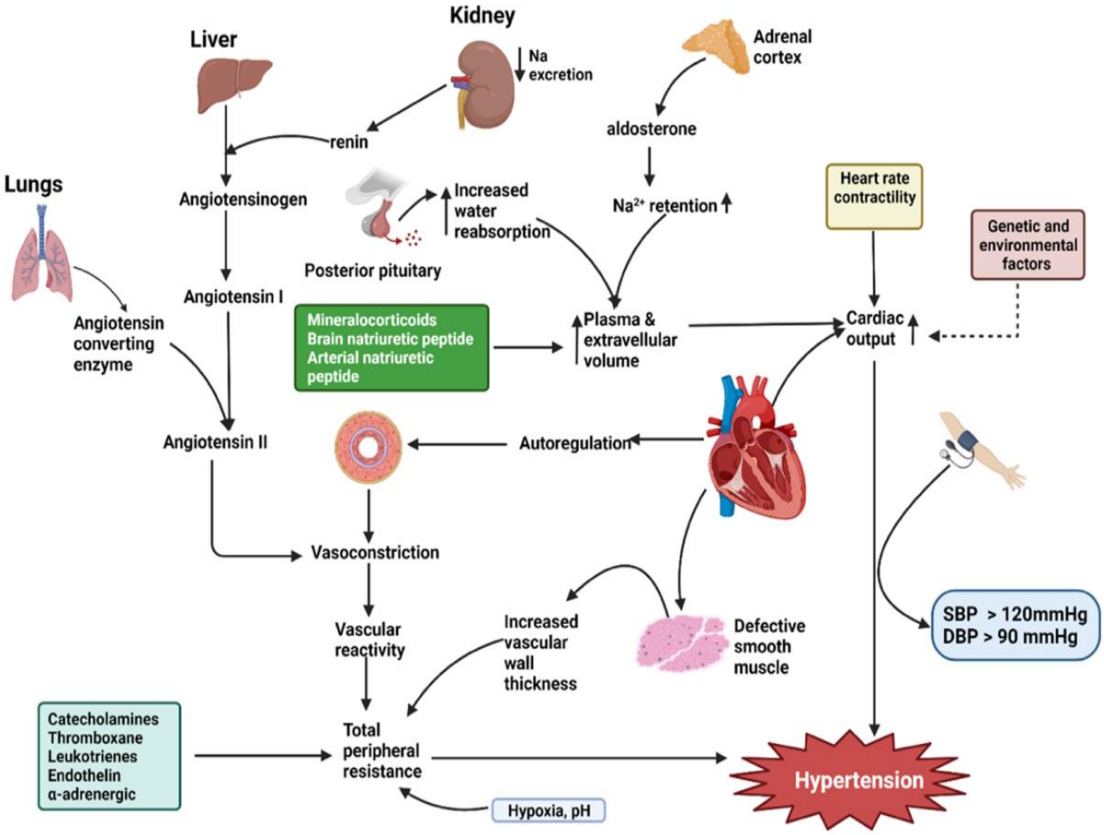

Figure No. 1: Pathophysiology Of Hypertension.

MODERN (allopathic) TREATMENT

The allopathic (modern medicine) treatment for hypertension (HTN) includes several classes of antihypertensive drugs, each with specific side effects. The main drug classes and their typical side effects are provided below.

Main Drug Classes and Their Side Effects

Diuretics ("water pills"): These help remove excess salt and water from the body, lowering blood pressure. Common side effects include excessive urination, dizziness, headaches, muscle cramps, weakness, insomnia, and, less commonly, sudden vision changes or severe rashes.

Beta-blockers: These slow down the heart rate and lower blood pressure. Typical side effects are cold hands/feet, depression, erectile dysfunction, insomnia, asthma symptoms, and fatigue. More serious effects, such as breathing difficulties or swelling in the extremities, can occur.[28]

ACE inhibitors: These prevent the formation of a hormone that constricts blood vessels. The commonest side effect is a persistent dry cough. Others include dizziness, abdominal pain, diarrhoea, hyperkalaemia, reversible kidney impairment, and, rarely, serious allergic reactions such as angioedema (swelling of lips, tongue, or throat).

Angiotensin II Receptor Blockers (ARBs): These have fewer cough and allergy issues compared to ACE inhibitors. Dizziness and sometimes hypotension (low blood pressure) are their main side effects.

Calcium Channel Blockers: These relax blood vessel walls. Side effects may include constipation, dizziness, headaches, irregular or rapid heartbeat (palpitations), and swollen ankles. Rare but serious reactions include rash, fainting, or significant facial swelling.

Alpha-blockers: These relax blood vessels by reducing nerve impulses. Dizziness, especially when standing suddenly and fast heartbeat are notable effects. Others may include fatigue or mild leg dryness.[28]

Centrally acting agents: These act on the central nervous system to lower blood pressure. Their side effects can include drowsiness, dry mouth, headaches, weakness, dizziness, and decreased sexual ability.

Important points

- Severe reactions (e.g., angioedema with ACE inhibitors, severe rashes, pronounced dizziness, chest pain, or breathing issues) require immediate medical attention.
- Side effects can vary and depend on a person's health, age, medication dose, and other individual factors.

- Do not change or stop antihypertensive medications without consulting a doctor, as abrupt changes can be dangerous.

This summarizes the primary antihypertensive classes and their most important side effects in allopathic (modern Western) medicine.

2) Angina Pectoris

Angina is a type of chest pain or discomfort that occurs when your heart muscle does not receive enough oxygen-rich blood, usually due to narrowed or blocked coronary arteries. It is a major symptom of coronary artery disease, the most common form of heart disease globally.[12]

Definition of Angina

Angina, also called angina pectoris, is chest pain or pressure caused by insufficient blood flow to the heart muscle (myocardium). This pain can feel like squeezing, heaviness, tightness, or pressure in the chest, and may extend to the arms, shoulders, neck, jaw, or back. There are three main types.

- Stable angina: Predictable, occurs with activity or stress, and is relieved by rest or medications.
- Unstable angina: Unexpected, can occur at rest, does not go away with rest or medication, and is a medical emergency.
- Variant (Prinzmetal) angina: Rare, usually occurs at rest due to coronary artery spasm.

Why Angina Matters

Angina is significant because it signals that the heart muscle is not getting enough blood and oxygen, frequently due to underlying coronary artery disease. This condition greatly increases the risk of major cardiovascular events—including heart attack (myocardial infarction) and sudden cardiac death. Angina is an important warning sign, offering an opportunity to intervene and prevent life-threatening complications through lifestyle modifications, medications, or surgical procedures. Furthermore, angina affects quality of life, causing pain, reduced physical ability, and increased risk of depression, disability, and higher health care usage.

Global Statistics

- Around 112 million people worldwide are affected by angina, making it a major public health concern.[12]
- In the United States alone, approximately 11 million adults live with angina, according to the CDC.
- The annual incidence for Western males aged 45–65 is about 1%, with a slightly higher rate in women, and the condition is more common in people over 65 years old.
- Angina and coronary artery disease are responsible for roughly 4.1 million deaths annually in Europe; overall, cardiovascular disease remains the leading global cause of death.
- Age is a major risk factor for mortality from angina and related coronary artery disease, with the mortality risk markedly increasing after age 65. Approximately 82% of coronary artery disease deaths (with angina as a primary symptom) in Europe occur in people older than 65 years.[11]

Here is a global overview of angina-related mortality and prevalence by age group.

Table No. 3: Mortality ratio of Angina pectoris in relation of age group.

Age Group	Angina Prevalence (%)	Mortality/Impact	Notes
18–44 years	0.3–0.5	Very low direct mortality	Prevalence stable, mortality rare
45–64 years	1.5–1.8	Mortality rises with age, still lower than older	Prevalence increases with age
65–74 years	3.2–4.3	Sharp rise in mortality, 27.7% IHD prevalence	Significant risk for events, deaths rise
75+ years	4.5–5.7	Highest rate: 82% of CAD/angina deaths occur >65	Prevalence and mortality highest

PATHOPHYSIOLOGY OF ANGINA

Mechanism of Angina

- **Oxygen Imbalance:** Angina occurs when the myocardial oxygen demand exceeds the oxygen supply delivered by the coronary arteries.
- **Coronary Artery Stenosis:** This imbalance often results from atherosclerotic plaques causing narrowing (stenosis) of the epicardial (surface) coronary arteries, which becomes especially problematic during increased demand (exercise, stress) or reduced supply (spasm, embolism).[14]
- **Contributing Factors:** Oxygen demand increases with a higher heart rate, elevated blood pressure, increased myocardial wall tension, or greater contractility. Oxygen supply depends on arterial diameter, vascular tone, perfusion pressure, collateral flow, and the heart's own rate.
- **Endothelial Dysfunction:** The lining of the coronary arteries regulates tone and prevents clotting; damage (due to hypertension, cholesterol, or inflammation) leads to impaired dilatation and increases risk of blockage and thrombosis.

- **Ischemia and Pain:** When blood flow is restricted, the heart muscle shifts to anaerobic metabolism, accumulating metabolites like adenosine, which triggers nerve endings. The pain is relayed through sympathetic afferent pathways, explaining why angina can radiate to the neck, arm, or back.[14]
- **Special Types:** Vasospastic (Prinzmetal) angina and microvascular angina often occur without significant atherosclerosis but with coronary spasm or microvascular dysfunction as the key mechanisms.

Molecular and Cellular Factors

- **Atherosclerosis Progression:** Chronic inflammation leads to plaque formation with foam cells, fibrous caps, and eventual calcification. Plaque rupture can cause acute coronary events or unstable angina.
- **Chemical Mediators:** Nitric oxide (NO) from healthy endothelium helps regulate vasodilation, while reduced NO and increased endothelin contribute to vasoconstriction and reduced blood flow.
- **Thrombosis:** In unstable angina, rupture or erosion of plaques induces platelet aggregation and thrombus (clot) formation, abruptly worsening blood flow and potentially leading to myocardial infarction.

TYPES OF ANGINA

- **Stable Angina:** Predictable chest pain usually triggered by physical exertion or stress. It follows a regular pattern, lasts a few minutes, and is relieved by rest or medication. It indicates the presence of coronary artery disease.
- **Unstable Angina:** Unpredictable and more severe chest pain that can occur at rest or with minimal exertion. It lasts longer, may worsen over time, and does not improve with rest or usual medications. This is a medical emergency as it signals a high risk of heart attack.
- **Variant (Prinzmetal) Angina:** Caused by a spasm in the coronary arteries, often occurring at rest and typically at night or early morning. It is not due to artery blockage but arterial spasm. Symptoms can be relieved by medication
- **Microvascular Angina:** Results from dysfunction in the smallest coronary arteries (microvascular disease). The chest pain may be more persistent and lasts longer, accompanied by symptoms like fatigue and shortness of breath.
- **Refractory Angina:** Chronic angina symptoms that persist despite medical treatment and lifestyle changes.

MODERN (allopathic) TREATMENT

1. **Nitrates (e.g., Nitroglycerin)**
- Used for acute relief and prevention by dilating coronary arteries.
- Side effects: Headache, dizziness, hypotension, flushing.

2. **Beta-blockers (e.g., Metoprolol, Atenolol)**
- Reduce heart rate and myocardial oxygen demand.
- Side effects: Fatigue, bradycardia, hypotension, bronchospasm (use cautiously in asthma).

3. **Calcium Channel Blockers (e.g., Amlodipine, Diltiazem)**
- Dilate coronary vessels and reduce workload on the heart.
- Side effects: Edema, dizziness, headache, constipation.

4. **Ranolazine**
- Used particularly in refractory angina; improves myocardial metabolism.
- Side effects: Dizziness, constipation, nausea.

5. **Ivabradine**
- Lowers heart rate by acting on sinus node If channels, used in some cases.
- Side effects: Bradycardia, visual disturbances (phosphenes).

6. **Antiplatelet agents (e.g., Aspirin)**
- Prevent blood clot formation, reducing the risk of heart attacks.
- Side effects: Gastric irritation, bleeding risk.

7. **Statins**
- Lower cholesterol and stabilize plaques.
- Side effects: Muscle aches, liver enzyme elevation.

These drugs are combined depending on the type and severity of angina and patient tolerance. Lifestyle modification and management of risk factors (like hypertension, diabetes) are essential adjuncts. In some cases, invasive therapies are considered if medications fail.

3) CONGESTIVE HEART FAILURE

Congestive Heart Failure (CHF) is a chronic and progressive condition in which the heart's ability to pump blood becomes inadequate to meet the body's metabolic demands. It does not mean that the heart stops working completely, but rather that it cannot circulate blood efficiently, leading to the buildup of fluid (congestion) in the lungs, legs, and other tissues.

Definition

According to the American College of Cardiology and the American Heart Association, heart failure is a complex clinical syndrome resulting from structural or functional impairment of ventricular filling or ejection of blood. Functionally, the heart may either fail to contract effectively (systolic failure) or fail to relax fully and fill with enough blood (diastolic failure). This leads to inadequate cardiac output, tissue hypoxia, and compensatory mechanisms such as ventricular enlargement, increased heart rate, and fluid retention.

Why CHF Matters

Congestive Heart Failure (CHF) matters because it is one of the most serious and widespread cardiovascular conditions, carrying a high burden on patients, families, and health systems worldwide. Understanding its impact helps in promoting early intervention, improving survival, and reducing healthcare costs.[2]

Major Health Burden

More than **64 million people** globally live with heart failure, making it the leading cause of hospitalization for adults over 65 years of age. The condition impairs the heart's ability to pump blood effectively, causing fatigue, breathlessness, reduced exercise capacity, and a serious decline in quality of life. Half of those diagnosed with CHF die within five years if not properly treated, underlining its severe prognosis.

Economic and Social Impact

CHF places a huge strain on healthcare systems due to frequent hospitalizations and long-term care requirements. In the United States alone, it accounts for about 2% of the total health budget, with billions spent annually on management and rehospitalisation costs. Approximately one in four patients is hospitalized within 30 days of discharge, emphasizing the diseases chronic and recurring nature.[2]

Public Health and Aging Population

CHF prevalence continues to rise largely due to aging populations and better survival rates following other heart diseases, such as coronary artery disease or myocardial infarction. It is especially common among older adults, with more than **10% prevalence in those over 75 years old**. This trend makes CHF a key focus in public health strategies for managing chronic illnesses and preventing cardiovascular complications.

Broader Medical Significance

Heart failure is not only a condition itself but also both a risk factor and a consequence of many cardiovascular diseases such as atrial fibrillation, stroke, and coronary heart disease. This interconnection means that addressing CHF can have wide-ranging benefits in preventing and managing other serious health conditions. Additionally, awareness and education about CHF can empower patients to adhere better to treatment and make lifestyle changes that enhance their quality of life.

Global Statics

- Approximately 64 million people worldwide are living with heart failure today.
- CHF contributes to nearly 9% of all deaths attributed to cardiovascular diseases globally.
- The global mortality from cardiovascular diseases (including CHF) was around 19.8 million deaths in 2022, with around 5–6 million deaths estimated as directly or indirectly linked to heart failure.
- Models predict a 73% increase in global cardiovascular mortality between 2025 and 2050, primarily due to aging populations.[12]

Global Heart Failure Mortality by Age (2025)

Table No. 4: Mortality ratio of congestive heart failure in relation of age group.

Age Group	Estimated Global Prevalence (%)	Annual Mortality Rate (%)	Major Risk Factors / Notes
18–44 years	~1.2%	~2–3%	Often secondary to congenital or untreated hypertension
45–64 years	~4.7%	~6–8%	Rising obesity, diabetes, and early coronary heart disease
65–74 years	~7.5%	~12%	Age-related vascular stiffening and multi-organ disease
75–84 years	~10.8%	~15–18%	High recurrence of hospitalizations and comorbidities
85+ years	~14.5%	~25–30%	Very high frailty, reduced physiological reserve, poor prognosis

Overview

- Mortality rises sharply with age**,** with those over 85 experiencing a mortality rate 10 times higher than those under 65 do.
- Men have a slightly higher prevalence (3–4%) than women do in younger groups, but after age 75**,** mortality is greater among women, due to longevity and diastolic heart failure dominance.[12]
- Heart failure hospital readmission and mortality remain highest within the first six months post-diagnosis.
- Aging and chronic diseases (hypertension, diabetes, obesity) are the key demographic drivers increasing CHF burden globally.

Types of CHF

Congestive Heart Failure (CHF) can be classified based on mechanism, anatomical involvement, and clinical staging. The main types reflect how the heart is pumping or filling function is impaired, providing guidance for diagnosis and management.

1) Based on Pumping Function (Ejection Fraction)

Table No. 5: Types of Congestive Heart Failure on basis of pumping function.

Type	Description	Ejection Fraction (EF)	Key Features
Heart Failure with Reduced EF (HFrEF)	Also called *systolic heart failure*; occurs when the heart muscle is weakened and cannot contract effectively.	EF ≤ 40%	Reduced cardiac output, ventricular dilation, usually due to ischemic heart disease.
Heart Failure with Mildly Reduced EF (HFmrEF)	A mid-range category reflecting partial reduction in heart pumping ability.	EF 41–49%	Transitional stage; patient's exhibit overlapping features of systolic and diastolic dysfunction.
Heart Failure with Preserved EF (HFpEF)	Also called *diastolic heart failure*; the heart contracts normally but is too stiff to fill adequately.	EF ≥ 50%	Common in older adults, hypertensive and diabetic individuals; causes pulmonary congestion without reduced EF.
Heart Failure with Recovered EF (HFrecEF)	EF improves to normal (>50%) after treatment, but ongoing care is required.	Previously ≤40%, now ≥50%	Seen after medical therapy or device support; relapse possible without maintenance care.

2) Based on Anatomical Location

Table No. 6: Types of Congestive Heart Failure on basis of Anatomical Location.

Type	Description	Key Symptoms
Left-Sided Heart Failure	The left ventricle fails to pump effectively, leading to fluid build-up in the lungs.	Shortness of breath, fatigue, cough, pulmonary congestion.
Right-Sided Heart Failure	The right ventricle cannot pump blood efficiently into the lungs, causing systemic fluid build-up. Often a consequence of left-sided heart failure.	Swelling of legs, ankles, and abdomen; weight gain from fluid retention.
Biventricular (Combined) Failure	Both ventricles are affected, leading to systemic and pulmonary congestion.	Severe breathlessness, generalized edema, low exercise tolerance.

3. Based on Duration and Onset

Table No. 7: Types of Congestive Heart Failure on basis of Duration of Action.

Type	Description	Onset Pattern
Acute Heart Failure	Sudden and severe impairment of cardiac function; often triggered by heart attack, arrhythmia, or hypertensive crisis.	Rapid onset requiring emergency management.
Chronic Heart Failure	Long-term progressive weakening of the heart muscle, often due to untreated hypertension or coronary artery disease.	Gradual symptom development and repeated hospitalizations.

4. Based on Clinical Stage (ACC/AHA)

Table No. 8: Types of Congestive Heart Failure on basis of Clinical Stage.

Stage	Description	Risk / Symptom Profile
Stage A	At risk due to factors like hypertension, diabetes, or obesity; no structural heart disease yet.	No symptoms.
Stage B	Structural heart changes but no symptoms yet (e.g., reduced EF, valve disease).	Asymptomatic.
Stage C	Evident structural damage with current or past symptoms of heart failure.	Fatigue, breathlessness, fluid retention.
Stage D	Advanced, treatment-resistant heart failure requiring hospitalization or transplantation.	Refractory symptoms despite therapy

Allopathic Treatment of Congestive Heart Failure (CHF)

Allopathic treatment for CHF focuses on relieving symptoms, preventing disease progression, reducing hospitalizations, and improving survival and quality of life. Treatment combines lifestyle modifications, pharmacologic therapies, and advanced interventions depending on the stage and severity of heart failure.[16]

Goals of Treatment

- Improve cardiac function and reduce the heart's workload.
- Manage fluid overload and congestion.
- Prevent complications like arrhythmias and sudden cardiac death.
- Slow or reverse cardiac remodelling.[23]

Drug Classification

1) **Angiotensin-Converting Enzyme (ACE) Inhibitors**
   a. Examples: Enalapril, Lisinopril
   b. Action: Dilate blood vessels, decrease blood pressure, reduce heart workload, and improve survival.
2) **Angiotensin II Receptor Blockers (ARBs)**
   a. Examples: Losartan, Valsartan
   b. Alternative to ACE inhibitors for those intolerant to ACEI; similar effects.
3) **Angiotensin Receptor-Neprilysin Inhibitors (ARNIs)**
   a. Example: Sacubitril-Valsartan
   b. Combines ARB with neprilysin inhibition to improve outcomes in heart failure with reduced ejection fraction (HFrEF).
4) **Beta Blockers**
   a. Examples: Carvedilol, Metoprolol, Bisoprolol
   b. Slow heart rate, reduce blood pressure, and improve heart function and survival.
5) **Diuretics**
   a. Loop Diuretics: Furosemide, Bumetanide
   b. Thiazide Diuretics: Metolazone
   c. Reduce fluid overload and relieve symptoms of congestion.
6) **Mineralocorticoid Receptor Antagonists (MRAs)**
   a. Examples: Spironolactone, Eplerenone
   b. Block aldosterone effects; reduce hospitalization and mortality in select patients.[25]

Pathophysiology of Congestive Heart Failure (CHF)

Congestive Heart Failure develops when the heart cannot pump enough blood to meet the body's needs. This failure can stem from damage or stress to the heart muscle due to conditions such as coronary artery disease, high blood pressure, or infections.

Initially, when the heart's pumping ability drops, the body activates several compensatory mechanisms to maintain adequate blood flow. These include.

Neurohormonal activation: The sympathetic nervous system releases hormones like norepinephrine, which increase heart rate and contractility. Simultaneously, the renin-angiotensin-aldosterone system (RAAS) promotes fluid retention and blood vessel constriction to maintain blood pressure.

Myocardial remodelling: Chronic stress stimulates changes in heart muscle cells, causing hypertrophy (enlargement) and fibrosis (scarring). While initially adaptive, these alterations ultimately impair contraction and relaxation.

Cellular calcium handling abnormalities: Proper heart muscle contraction depends on calcium cycling inside cells. In heart failure, these calcium dynamics become disrupted, reducing contractile strength and worsening relaxation.

MAIN FOCUSED HERBS

1) TERMINALIA ARJUNA

Terminalia arjuna, also known as arjuna, is a tree belonging to the Combretaceae family. In traditional Indian medicine, especially Ayurveda, its bark decoction has long been used to manage heart-related conditions such as angina, high blood pressure, heart failure, and imbalanced lipid levels.[3]

Figure No. 2: Terminalia arjuna.

ROLE

- Arjuna is a well-known herb traditionally used as a cardiac tonic in Ayurvedic medicine.
- It strengthens the heart muscle, helping to improve the heart's pumping ability.
- The bark of Arjuna has been shown to increase the force of cardiac contractions (positive inotropic effect).[3]
- It also helps regulate heart rhythm by stabilizing the electrical activity of the heart.[30]
- Arjuna exhibits antioxidant properties that protect the heart tissue from oxidative damage caused by stress or injury.
- The herb promotes improved blood flow through coronary arteries, aiding the heart muscle's oxygen supply.[1]

2) WITHANIA SOMNIFERA

Withania somnifera, commonly known as Ashwagandha, has been studied for its beneficial effects on heart health and cardiac treatment. It exhibits cardioprotective properties primarily due to its antioxidant and anti-apoptotic effects. In experimental models, such as rats with induced heart damage, Withania somnifera has been shown to reduce heart tissue injury caused by ischemia (restricted blood supply) and oxidative stress.[24]

Figure No. 3 – Withania somnifera.

ROLE

- Ashwagandha exhibits cardioprotective properties by helping to reduce oxidative stress in heart tissue through its antioxidant action.[1]
- It supports heart health by preventing damage to cardiac cells caused by ischemia (restricted blood supply) and reperfusion injury.
- The herb activates cellular pathways that inhibit apoptosis (programmed cell death) in heart muscle cells, promoting their survival.[7]
- It enhances energy regulation in cardiac cells by stimulating AMP-activated protein kinase (AMPK), which aids in maintaining energy homeostasis.
- Ashwagandha may improve cardiovascular endurance and reduce symptoms related to heart stress.[1]

DIFFERENT TYPES OF CARDIOTONIC HERBS

Table No. 9: Different types of Cardio Protective herbs.

Plant Name	Uses	Phytochemicals	Actions
Terminalia arjuna	Heart failure, angina	Arjunolic acid, flavonoids	Antioxidant, cardiotonic
Allium sativum	Hypertension, cholesterol	Allicin	Hypotensive, antiplatelet
Commiphora mukul	Hyperlipidaemia	Guggulsterone	Lipid-lowering
Curcuma longa	Inflammation	Curcumin	Anti-inflammatory, antioxidant
Hibiscus sabdariffa	Hypertension	Anthocyanin	Vasodilation, diuretic
Crataegus monogyna	Heart tonic	Flavonoids, oligomeric proanthocyanidins	Cardio tonic, vasodilatory

ADVANTAGES OF CARDIOPROTECTIVE FORMULATIONS

1) Herbal cardio protective drugs often work through multiple mechanisms such as antioxidant, anti-inflammatory, vasodilatory, and anti-platelet effects, providing comprehensive cardiovascular support.
2) They tend to have fewer side effects compared to some synthetic cardiovascular drugs, making them appealing for long-term use.
3) Many herbal medicines are cost-effective and accessible, especially in resource-limited settings.[13]
4) They may improve risk factors like high cholesterol, hypertension, and oxidative stress without the harsh impacts of conventional drugs.
5) Herbal drugs can complement conventional treatments by targeting additional molecular pathways involved in cardiovascular health.
6) Their natural origin often leads to better patient acceptance and adherence.[19]

DISADVANTAGES OF CARDIOPROTECTIVE FORMULATIONS

1) The potency and efficacy of herbal cardio protective drugs can vary widely due to differences in plant source, preparation method, and dosage.
2) Scientific evidence supporting clinical effectiveness is often limited or inconsistent, with few rigorous clinical trials.
3) Potential drug-herb interactions pose risks, especially when combined with conventional cardiovascular medicines, possibly leading to adverse effects like bleeding or altered drug metabolism.
4) Quality control and standardization of herbal products are often lacking, raising concerns about contamination, adulteration, or incorrect dosage.
5) Some herbal compounds may have toxic effects or contraindications that are not well documented.[13]
6) Regulatory oversight for herbal cardio protective drugs is less stringent than for conventional drugs, affecting safety and reliability.

TYPES OF CARDIOPROTECTIVE FORMULATIONS[13]

Table no. 10: Different types of Cardio protective formulations.

Dosage Form	Best Herbs for Formulation
Capsules/Tablets	Arjuna, Garlic, Guggul
Syrup/Tonics	Arjunarishta, Ashwagandha, Brahmi
Granules	Triphala, Methi
Decoction	Arjuna bark, Punarnava root

EVALUATION PARAMETERS[30]

Table no. 11: Evaluation parameters of Cardio Protective Formulations.

Evaluation Parameter	Description	Purpose
Organoleptic Evaluation	Assessment of colour, taste, odour, texture, and appearance	Ensures acceptability and consistency in formulation
Phytochemical Analysis	Identification and quantification of bioactive compounds like alkaloids, flavonoids, glycosides	Confirms presence of active ingredients
Physicochemical Parameters	Measurement of pH, moisture content, ash values, extractive values	Assesses quality, stability, and purity
Toxicological Evaluation	Tests for cell toxicity, acute and chronic toxicity in animal models	Ensures safety profile of the formulation
In vitro Antioxidant Activity	Evaluation of free radical scavenging and oxidative stress reduction	Indicates potential cardio protective efficacy
Pharmacological Testing	Studies on animal models to determine cardio tonic effect such as heart rate and contractility	Confirms therapeutic effectiveness
Microbial Load Testing	Detection of microbial contamination	Ensures microbiological safety and hygiene
Stability Studies	Assessment of formulation stability under different environmental conditions	Guarantees shelf life and efficacy over time
Standardization	Use of chromatographic or spectroscopic methods to ensure consistent concentration of actives	Maintains batch-to-batch uniformity

CONCLUSION

Herbal cardio protective drugs hold significant promise as complementary and alternative options in managing cardiovascular diseases. These natural products exert their beneficial effects through multiple mechanisms including antioxidant activity, anti-inflammatory effects, improvement in lipid profiles, enhancement of myocardial contractility, and protection against ischemic injury. Herbs like Terminalia arjuna, Ashwagandha, and garlic have demonstrated encouraging outcomes in both experimental and clinical studies, showing potential to reduce heart-related complications with fewer side effects than conventional drugs.

Despite these advantages, challenges remain in standardizing herbal formulations, ensuring consistent bioactive compound levels, and conducting large-scale, high-quality clinical trials to validate efficacy and safety. The integration of herbal medicines into mainstream cardiovascular care requires rigorous scientific evaluation and regulatory oversight. Overall, herbal cardio protective agents represent a valuable resource for holistic cardiovascular health management, especially when combined with lifestyle modifications. Further research is essential to fully harness their therapeutic potential and improve cardiovascular outcomes worldwide.

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