



Etiopathogenesis of Essential Hypertension in Ayurvedic Parlance and Its Clinical Management with *Mansyadi Kwatha* and *Ashwagandha Churna*

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ABSTRACT

Essential hypertension represents a major global health challenge, functioning as an asymptomatic yet potent contributor to end-organ deterioration, stroke, coronary artery disease, and chronic kidney disease. In modern clinical practice, management typically involves lifelong administration of pharmaceutical agents that carry significant adverse effect burdens. Although classical Ayurvedic literature does not detail hypertension as an isolated disease entity, its hemodynamic and psychosomatic presentation correlates directly with dysregulation of *Rasa-Rakta Samvahana* and an elevation in *Vyanabala* (*Vyanabala Vriddhi*).

This dissertation study evaluates the etio-pathogenesis (*Evam Utthanam*), clinical symptomatology (*Evam Sansthanam*), and prognosis (*Evam Udarkam*) of essential hypertension through classical Ayurvedic paradigms, juxtaposing these with contemporary physiological mechanisms. A randomized comparative clinical trial was conducted on 60 patients presenting with Stage 1 and Stage 2 essential hypertension, divided equally into Group A (*Mansyadi Kwatha*, 50 mL twice daily) and Group B (*Ashwagandha Churna*, 5 g twice daily) over a treatment duration of three months.

Both therapeutic modules demonstrated statistically significant reductions in systolic blood pressure, diastolic blood pressure, pulse pressure, and mean arterial pressure. Furthermore, significant therapeutic relief was recorded across key subjective symptoms, including *Shiroruk* (headache), *Bhrama* (giddiness), *Hriddravatva* (palpitations), *Klama* (fatigue), and *Alpanidra* (insomnia), alongside favorable modulations of serum lipid fractions. These findings establish *Mansyadi Kwatha* and *Ashwagandha Churna* as safe, accessible, and efficacious therapeutic agents for managing mild-to-moderate uncomplicated essential hypertension.

KEY WORDS: *Hriddravatva*, *Shiroruk*, *Alpanidra*, *Klama*, *Vyanabala*, *Rakta Samvahana* etc.

INTRODUCTION:

Cardiovascular diseases (CVDs) constitute the leading cause of global morbidity and mortality. In India, cardiovascular events account for approximately one-third of total mortalities, with uncontrolled hypertension serving as the single largest preventable risk factor. Epidemiological assessments indicate that over 220 million individuals in India live with hypertension, yet only 12% maintain adequate blood pressure control. Data from the comprehensive ICMR-INDIAB survey underscores this expanding public health crisis, recording 315.5 million individuals living with hypertension across urban and rural demographics, alongside 254.2 million individuals with generalized obesity and 213.3 million suffering from hypercholesterolemia.

The prevalence of adult hypertension in India has expanded markedly over the past three decades, shifting from 5% to 20–40% in urban centers and 12–17% in rural sectors. Conservative estimates reflect that roughly 34 million urban and 31.5 million rural citizens require therapeutic blood pressure management, with nearly 70% categorized under Stage 1 hypertension.

Essential (primary or idiopathic) hypertension represents 90% to 95% of all clinical cases. Characterized by the lack of an identifiable single secondary etiology, its insidious and asymptomatic progression has earned it the designation of the "silent killer". Because untreated elevated arterial tension progressively damages sensitive vascular beds—precipitating retinopathy, encephalopathy, myocardial infarction, and end-stage nephrosclerosis—timely intervention is critical.

Conventional medical therapies (e.g., angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs], calcium channel blockers [CCBs], and thiazide diuretics) succeed in lowering systemic blood pressure, but they require lifelong adherence and frequently cause troublesome adverse effects, such as peripheral edema, persistent dry cough, reflex tachycardia, depression, fatigue, and electrolyte disturbances. Consequently, an increasing number of hypertensive individuals seek alternative, holistic paradigms. Developing an Ayurvedic conceptualization of this condition and validating traditional formulations through standardized clinical parameters addresses a pressing therapeutic need.

MODERN PERSPECTIVES: HEMODYNAMICS AND PATHOPHYSIOLOGY

Arterial blood pressure (BP) denotes the lateral force exerted by circulating columns of blood upon the intraluminal surfaces of the vascular walls. Hemodynamically, systemic arterial pressure is governed by the product of cardiac output (CO) and total peripheral vascular resistance (TPVR):

Cardiac output is determined by heart rate and stroke volume (which reflects myocardial contractility, venous capacitance, and intravascular volume). Total peripheral vascular resistance is governed by functional arteriolar tone and structural vascular caliber. In a healthy young adult, physiological pressure reaches approximately 120 mmHg during ventricular systole and drops to 80 mmHg during ventricular diastole.

The human body coordinates systemic pressure through an array of regulatory feedback loops:

1. **Rapidly Acting Mechanisms (Seconds to Minutes):** The arterial baroreceptor reflex (situated in the carotid sinus and aortic arch) serves as a primary buffer. It initiates immediate autonomic adjustments via the medulla oblongata, counteracting acute surges or declines in arterial stretch. Additionally, non-physiological chemoreceptors and ischemic CNS reflexes provide emergency vasoconstriction during acute circulatory compromise.
2. **Intermediate Mechanisms (Minutes to Hours):** Capillary fluid shifts rebalance circulating plasma volumes across microcirculatory beds, while vascular stress-relaxation mechanisms adjust vascular tone in large venous and hepatic reservoirs.
3. **Long-Term Mechanisms (Days to Weeks):** The kidneys modulate arterial pressure by maintaining extracellular fluid and sodium homeostasis. The Renin-Angiotensin-Aldosterone System (RAAS) serves as the primary biochemical axis: renal hypoperfusion or excessive adrenergic stimulation prompts juxtaglomerular cells to secrete renin, cleaving circulating angiotensinogen into angiotensin I. ACE then converts angiotensin I into angiotensin II—a potent systemic vasoconstrictor that also triggers adrenal aldosterone release, promoting distal tubular sodium-water retention.

Essential hypertension is driven by a complex interplay of sympathetic overdrive, elevated renal vascular resistance, RAAS overactivity, and endothelial dysfunction. Endothelial injury disrupts the physiological equilibrium between vasodilators (such as nitric oxide) and vasoconstrictors (such as endothelin-1), fostering chronic vascular smooth-muscle hypertrophy, internal elastic lamina thickening, and progressive arterial stiffness.

Classification (JNC VII)	Systolic BP (mmHg)	Diastolic BP (mmHg)
Normal	< 120	< 80
Pre-Hypertension	130–139	80–89
Stage I HTN	140–159	90–99
Stage II HTN	160	100

Anatomical and Physiological Foundations: *Hridaya*, *Dhamani*, and *Sira*

Classical Ayurvedic literature frames systemic circulation around the continuous propulsion of *Rasa* and *Rakta* (*Rasa-Rakta Samvahana*). The cardiovascular apparatus is correlated with the *Rasavaha Srotas*, whose foundational roots (*Mula*) are *Hridaya* (the heart) and the ten principal *Mahamula Dhamanis* ("रसवहानां स्रोतसां हृदयं मूलं दश च धमन्यः" - Charaka Samhita Vimana Sthana 5/8). Acharya Sushruta expands on this anatomical framework, highlighting that *Hridaya* gives rise to 24 distinct *Dhamanis* (10 *Urdhvaga* or upward-coursing, 10

Adhoga or downward-coursing, and 4 *Tiryak* or horizontally traversing) that distribute refined nutritive essence (*Sara*) across all somatic tissues.

Hridaya is recognized as a hollow (*Sushira*), muscular (*Manshapeshimaya*) visceral organ (*Koshthanga*) shaped like an inverted lotus (*Adhomukha Kamala*), holding status as a paramount *Sadyapranahara Marma*. Physiologically, the cardiac cycle is encoded within the etymology of the term *Hridaya* in the *Brihadaranyaka Upanishad*:

- **Hri (Aharana):** The receiving function, wherein venous conduits (*Siras*) return blood and fluid constituents to the cardiac chambers.
- **Da (Dana):** The distributing or ejecting function, whereby the heart propels blood outward through the *Dhamanis* to peripheral systems.
- **Ya (Gati or Ayana):** Continuous, rhythmic contractile-relaxing movement (*Sankocha* and *Vikasa*).

The propulsion of *Rasa-Rakta* occurs via the *Vikshepa Karma* of *Vyana Vayu* ("व्यानेन रसधातुर्हि विक्षेपोचितकर्मणा" - Charaka Samhita Chikitsa Sthana 15/36). Sushruta illustrates the multi-directional velocity of this circulation using three distinct classical analogies (*Dalhana's Commentary* on Sushruta Samhita Sutra Sthana 14/16):

1. *Shabda-santanavat* (pulsatile wave propagation, resembling high-velocity arterial propulsion).
2. *Archi-santanavat* (diffuse upward radiance, comparable to lymphatic spread).
3. *Jala-santanavat* (downward, steady laminar flow, analogous to venous return).

THE PHYSIOLOGICAL AND PATHOLOGICAL DYNAMIC OF VYANABALA:

While the modern term "hypertension" relies on modern sphygmomanometric metrics, the underlying pathological tension within the circulatory tree is best contextualized in Ayurveda through the concept of *Vyanabala*. Formulated by twentieth-century scholars including Acharya Ranjitraya Desai and Dr. B. Triguna, *Vyanabala* denotes the kinetic hemodynamic driving force generated by *Vyana Vayu* within the *Dhamanis* during cardiac cycles.

- **Sankochakalina Vyanabala:** The maximal pressure exerted against the vessel walls during cardiac contraction (*Sankocha Avastha*), reflecting systolic blood pressure.
- **Vikasakalina Vyanabala:** The residual lateral pressure maintained within the *Dhamanis* during cardiac diastole (*Vikasa Avastha*), reflecting diastolic blood pressure.

A state of *Vyanabala Vridhhi* emerges when physiological regulation breaks down. Multiple bodily factors modulate *Vyanabala*:

- **Srotorodha and Dhamani Sankocha:** Narrowing and peripheral obstruction of the *Rasavaha-Raktavaha* pathways force *Vyana Vayu* to deploy greater kinetic force to ensure distal tissue nourishment, directly mirroring elevated systemic vascular resistance.
- **Rasa-Rakta Pramana:** Quantitative volume expansions (*Dravatah Vridhhi*), driven by renal fluid retention or dietary indiscretion, raise filling pressures (*Sira Purnata*).

- **Rakta Ghanatva (Blood Viscosity):** Aggravation of *Kapha* thickens the blood, requiring increased cardiac exertion for circulation.
- **Kleda and Vrikka Balance:** Impairment of renal excretion (*Mutra Kleda Vahana Karma*) retains somatic fluid (*Kleda*), expanding circulating *Rakta* volume.

Theoretical Frameworks: *Avarana*, *Raktapradosha*, and *Mishra Avarana*

Because the classical compendia establish that physicians should treat pathological entities based on underlying *Nidana-Dosha-Dushya* dynamics rather than fixating on nomenclature ("*न हि सर्वविकराणां नामतोऽस्ति ध्रुवा स्थितिः*" - Charaka Samhita Sutra Sthana 18/44), multiple theoretical frameworks account for essential hypertension:

AYURVEDIC PATHOLOGICAL FRAMEWORKS:

1. **Raktagata Vata:** Vata vitiation localized within circulating *Rakta*.
2. **Raktavrita Vata:** Dense *Rakta* obstructing *Vata*, raising resistance.
3. **Shleshmavrita Vyana:** *Kapha* occluding *Vyana* pathways (Atherosclerosis).
4. **Pranavrita Vyana:** Vitiated *Prana* dysregulating central cardiac output.
5. **Mishra Avarana:** Multi-factorial, combined neuro-metabolic vascular block.
 - **Raktagata Vata:** Classical localization of aggravated *Vata* within the circulatory pool, producing vascular irritability and episodic pressor swings.
 - **Raktavrita Vata:** A physiological state wherein *Rakta* physically obstructs the free transit of *Vata*, clinically reflecting elevated vascular tone and resistance.
 - **Shleshmavrita Vyana & Dhamani Pratichaya:** *Kapha* and *Ama* adhere to the inner endothelial surfaces of the *Dhamanis*, inducing structural vascular remodeling, luminal narrowing, and loss of arterial elasticity.
 - **Pranavrita Vyana:** Higher cortical agitation dysregulates autonomic regulatory signals, disrupting basal vasomotor control.
 - **Mishra Avarana (Mixed Obstruction):** Outlined by Charaka in *Chikitsa Sthana* (28/232), this concept describes the multi-factorial pathology of hypertension. Complex lifestyle stresses simultaneously provoke *Raja-Tama*, *Vata*, and *Pitta*, resulting in systemic metabolic (*Ama*), humoral (*Kapha-Meda*), and neurogenic vascular occlusion.

Furthermore, in Charaka Samhita Sutra Sthana 24, the symptomatology of *Raktapradoshaja Vikara*—such as *Shirahshula* (headache), *Bhrama* (giddiness), *Klama* (fatigue), and terminal events like *Mada* (delirium), *Murchha* (syncope), and *Sanyasa* (hypertensive coma/cerebrovascular accident)—mirrors the clinical progression of sustained, unmanaged hypertension.

Etiological Dynamics: *Nidana Panchaka* of *Vyanabala Vriddhi*

The manifestation of *Vyanabala Vriddhi* is driven by four primary classes of etiological factors (*Nidana*):

Aharaja Nidana (Dietary Triggers): Excessive intake of *Lavana* (salt), *Amla* (sour), and *Katu* (pungent) tastes; unwholesome alcoholic beverages (*Madya*); unctuous, fried, and processed foods (*Guru, Snigdha Ahara*); and incompatible diets (*Viruddha Ahara*). High *Lavana* intake directly induces fluid retention and *Rakta Vriddhi*,

expanding vascular volume (*Sira Purnata*). Heavy, unctuous diets promote *Agnimandya* and systemic *Ama*, creating the substrate for *Dhamani Pratichaya* (atherosclerotic vascular stiffening).

- **Viharaja Nidana (Lifestyle Factors):** Chronic physical strain or sedentary behavior (*Avyayama*), irregular sleep schedules (*Ratri Jagarana*—night-shift work causing *Vata-Pitta* vitiation), daytime sleep (*Diwaswapna*—inducing *Kapha-Meda* accumulation), and suppression of natural biological urges (*Vegadharana*).
- **Manasika Nidana (Psychological Stressors):** Intense anxiety (*Chinta*), frequent emotional anger (*Krodha*), persistent fear (*Bhaya*), and grief (*Shoka*). Mental strain vitiates the subtle *Manasika Doshas* (*Raja* and *Tama*), which destabilize *Prana Vayu* and overheat *Sadhaka Pitta*, driving systemic pressor surges.
- **Sahaja (Hereditary) and Jara (Aging) Influences:** Inherited constitutional vulnerabilities (*Beeja Dosha*) predispose individuals to early endothelial strain. As aging (*Jara*) sets in, somatic tissues undergo *Dhatukshaya* and *Ruksha-Khara* qualities rise, stiffening arterial walls and escalating systemic vascular resistance.

Samprapti:

The progression of *Vyanabala Vridhhi* develops systematically across six recognized stages of pathogenesis (*Shat Kriya Kala*):

1. **Sanchaya (Accumulation):** Vitiation begins in primary visceral seats (*Vata* in *Pakwashaya*, *Pitta* and *Kapha* in *Amashaya*), alongside early psychological tension (*Raja-Tama*).
2. **Prakopa (Excitement):** Continued dietary and emotional stress amplifies these imbalances, destabilizing the *Doshas*.
3. **Prasara (Dissemination):** Vitiated entities escape their gastrointestinal boundaries and circulate throughout the somatic tree via the *Rasavaha-Raktavaha Srotas*.
4. **Sthana Sanshraya (Localization):** The circulating *Doshas* lodge within structurally susceptible arterial tunics (*Dhamanis* and *Siras* displaying *Khavaigunya*), establishing deep-seated *Dosha-Dushya Sammurchhana*.
5. **Vyakti (Manifestation):** Sustained vascular tension manifests clinically as *Shiroruk* (headache), *Bhrama* (dizziness), *Hridhravatva* (palpitations), and measurable elevations in blood pressure.
6. **Bheda (Complications):** Left unaddressed, chronic hemodynamic shear stress causes structural organ damage across the three classical vital centers (*Trimarma*): *Hridaya* (cardiomegaly/heart failure), *Shira* (cerebrovascular stroke/encephalopathy), and *Basti* (chronic renal failure).

Samprapti Ghataka:

- **Dosha:** *Vata* (primarily *Vyana*, secondarily *Prana* and *Apana*), *Pitta* (*Sadhaka*), *Kapha* (*Avalambaka*); *Manasika Doshas* (*Raja* and *Tama*).
- **Dushya:** *Rasa*, *Rakta*, *Meda*, *Mutra*.
- **Agni:** *Jatharagnimandya* and *Dhatvagnimandya*, giving rise to circulating *Ama*.

- **Srotas:** *Rasavaha, Raktavaha, Manovaha*.
- **Srotodushti Type:** *Sanga* (obstruction) and *Vimarga Gamana* (aberrant pathway directional flow).
- **Udbhavasthana:** *Amashaya* and *Pakvashaya*.
- **Adhithana:** *Dhamani, Sira, Hridaya, Sarvanga Sharira*.
- **Prognosis (*Sadhya-Asadhyata*):** *Yapya* (manageable/controllable but structurally difficult to eradicate due to chronicity, multi-system involvement, and dual *Roga Marga—Madhyama* and *Bahya*).

PHARMACOLOGICAL PROFILES OF INTERVENTIONS:

Mansyadi Kwatha

Mansyadi Kwatha is a classical polyherbal decoction formulated by Vaidya Yadavji Trikamji Acharya in *Siddha Yoga Sangraha* (Psychiatric and Neuro-Psychiatric Section). It is specifically indicated for psychosomatic disorders characterized by psychic agitation (*Chinta*), sleep disruption (*Anidra*), and neuro-vascular irritability.

Ingredient	Botanical Name	Family	Part Used	Proportion	Classical Dosage
Jatamansi	<i>Nardostachys jatamansi</i> DC.	Valerianaceae	Rhizome	8 parts	1 Tola (12 g)
Ashwagandha	<i>Withania somnifera</i> (L.) Dunal	Solanaceae	Root	4 parts	0.5 Tola (6 g)
Parasika Yavani	<i>Hyoscyamus niger</i> Linn.	Solanaceae	Seed	1 part	1.5 Masha (1.5 g)

Preparation and Administration: The coarse blend (*Yavakut Churna*, screen mesh 40) is boiled with 5 parts water (e.g., 120 mL) over mild heat (*Manda Agni*) in an open vessel until reduced to roughly 50 mL. The fresh liquid is strained through a clean cotton cloth and administered at 50 mL twice daily, combined with an equal volume of lukewarm water, roughly 30 minutes post-prandially.

Phytochemical Mechanisms of Ingredients:

- **Jatamansi (*Nardostachys jatamansi*):** Contains sesquiterpenoids (jatamansone, valeranone, nardostachone) and coumarins. Jatamansone potentiates central GABA_A receptor transmission, suppressing sympathetic vasomotor tone and reducing circulating catecholamines. Concurrently, aristolone prompts peripheral arterial vasodilation through vascular K_{ATP} channel opening and eNOS activation, while inhibiting vascular smooth-muscle Ca^{2+} entry and circulating ACE activity.
- **Parasika Yavani (*Hyoscyamus niger*):** Contains tropane alkaloids (hyoscyamine, scopolamine, atropine). In micro-doses, it exerts smooth-muscle antispasmodic and sedative effects, relieving functional vascular wall spasms.

Ashwagandha Churna

Withania somnifera (L.) Dunal (*Ashwagandha*) serves as a foundational *Balya*, *Brimhaniya*, *Hridya*, and *Rasayana* therapy. In the trial, the dried unbranched taproots were pulverized to an 80-mesh fine powder and administered at a posology of 5 g twice daily with lukewarm water for three months.

Bioactive Constituents and Dynamics: Rich in steroidal lactones (withanolides such as Withaferin A and Withanolide A) and sitoindosides. *Ashwagandha* targets systemic hypertension via three primary pathways:

1. **HPA-Axis Regulation:** Downregulates chronic adrenocortical activation, lowering elevated serum cortisol levels and preventing cortisol-mediated sodium retention and pressor hyper-responsiveness.

2. **Endothelial Protection:** Stimulates endothelial nitric oxide synthase (eNOS), enhancing vascular nitric oxide bioavailability and promoting peripheral vasodilation.
3. **Antioxidant Action:** Upregulates endogenous myocardial and vascular antioxidants (superoxide dismutase, catalase, glutathione peroxidase) while suppressing inflammatory cascades to preserve vascular compliance.

CLINICAL METHODOLOGY AND RESEARCH DESIGN:

Study Design, Ethics, and Patient Cohort

This clinical evaluation was conducted as a prospective, randomized, open-label comparative study at the Post Graduate Department of Rog Nidan evum Vikriti Vigyan, Chandra Shekhar Singh Ayurvedic Sansthan, Koilaha, Kaushambi (affiliated with Mahayogi Guru Gorakhnath AYUSH University, Gorakhpur, Uttar Pradesh). Ethical clearance was obtained, and written informed consent was secured from every participant prior to enrollment.

A total of 70 patients presenting with Stage 1 and Stage 2 essential hypertension according to JNC VII and WHO guidelines were registered. During the trial, 10 patients dropped out due to non-compliance or personal reasons, leaving a completed cohort of 60 patients (30 in Group A, 30 in Group B).

- **Inclusion Criteria:** Diagnosed patients of essential hypertension (JNC VII criteria: SBP 140 mmHg and/or DBP 90 mmHg), aged 20 to 70 years, willing to adhere to the trial protocol.
- **Exclusion Criteria:** Patients presenting with secondary hypertension; co-morbid ischemic heart disease, coronary artery disease, or aortic coarctation; advanced renal failure; major endocrine disorders; malignant hypertension; or acute cerebrovascular/neurological emergencies.

Posology and Assessment Framework

- **Group A (n=30):** Freshly prepared classical decoction of *Mansyadi Kwatha*, 50 mL administered twice daily with 50 mL lukewarm water, 30 minutes after meals for three months.
- **Group B (n=30):** *Ashwagandha Churna*, 5 g administered twice daily with lukewarm water, 30 minutes after meals for three months.
- **Monitoring:** Bi-weekly clinical reviews (every 15 days) assessing treatment adherence, subjective symptom scores, and hemodynamic parameters.

Clinical efficacy was assessed across objective hemodynamic metrics, laboratory indices, and subjective symptom scores:

- **Objective Hemodynamic Metrics:** Seated blood pressure measured using standardized mercury sphygmomanometry following a 5-minute rest period, recording SBP, DBP, Pulse Pressure ($PP = SBP - DBP$), and Mean Arterial Pressure ($MAP = DBP + PP/3$).
- **Objective Biochemical Indices:** Fasting Blood Sugar, Serum Total Cholesterol, Serum Triglycerides, Serum High-Density Lipoprotein (HDL), Serum Very Low-Density Lipoprotein (VLDL), Blood Urea, and routine hematology (Hb%, ESR).

- **Subjective Symptom Scales:** Graded on classical 0–4 severity scales covering *Shirahshula* (headache), *Bhrama* (dizziness), *Hridhravatva* (palpitations), *Ayaasjanya Swasakashtata* (exertional dyspnea), *Alpanidra* (insomnia), *Tandra* (somnolence), *Klama* (fatigue), *Arati* (restlessness/uneasiness), *Santapa* (hot flashes/internal tension), *Tamodarshana* (visual flashes), *Buddhisammoha* (cognitive fuzziness), *Krodha-Prachurata* (irritability), and *Akshirag* (conjunctival redness).

Statistical comparisons of pre-treatment (BT) and post-treatment (AT) values were conducted using the paired Student's t-test, with $P < 0.05$ considered statistically significant and $P < 0.001$ considered highly significant.

Observations:

Demographic and constitutional analysis of the 60 completing participants revealed substantial correlations between lifestyle profiles and disease incidence:

- **Age:** Maximum patient concentration occurred in the 41–50 age cohort (35.00%), followed closely by 31–40 years (30.00%), 51–60 years (23.33%), and 60–70 years (11.67%). No patients were registered in the 20–30 age bracket. This distribution reflects the transitional phase of *Yuva Avastha* (*Pitta* dominance) into *Vridhdha Avastha* (*Vata* dominance), characterized by emerging *Dhamani Pratichaya* (vascular stiffening).
- **Sex and Marital Status:** Females accounted for 53.33% of cases and males represented 46.67%. A marked 88.33% of patients were married, a cohort frequently subject to complex socio-familial and occupational stresses.
- **Socio-Economic and Occupational Profile:** The middle-income stratum accounted for 65.00% of participants, while 23.33% were upper class and 11.67% lower class. Service and corporate business executives comprised 31.67% each, followed by housewives at 16.67%. These demographics reflect the pressor risks associated with high-demand, sedentary, desk-bound lifestyles.
- **Diet and Addictions:** Mixed non-vegetarian dietary habits predominated (66.67%) over purely vegetarian diets (33.33%). Regular consumption of tea was documented in 38.33%, tea combined with tobacco in 33.33%, and poly-addiction (tea, tobacco, and alcohol) in 15.00%. The unwholesome, *Tikshna-Ushna* properties of tobacco and alcohol aggravate *Sadhaka Pitta* and damage *Rakta Dhatu*, predisposing users to vascular irritation.
- **Metabolic Pattern (*Agni* and *Koshtha*):** Impaired digestion was common: 41.67% displayed *Visham Agni* (erratic digestion linked to *Vata*), while 36.67% exhibited *Manda Agni* (sluggish digestion linked to *Kapha-Ama* generation). Only 11.67% exhibited *Sama Agni*. With respect to bowel patterns, 41.67% had *Madhyama Koshtha* and 36.67% suffered from *Krura Koshtha* (predisposing to *Vata Pratiloma Gati* via constipation).
- **Psychological Strain (*Manasika Nidana*):** Pronounced anger (*Krodhaprachurya*) was reported in 40.00% of participants, excessive anxiety (*Chintabahulya*) in 40.00%, and deep chronic grief (*Shoka*) in 20.00%. These emotional stressors drive systemic neuro-hormonal cascades, elevating baseline cardiovascular tone.

- **Initial Symptom Frequencies:** The most prevalent initial complaints were *Klama* (fatigue, 90.00%), *Shiroruk* (headache, 76.67%), *Alpanidra* (insomnia, 76.67%), *Aayaasjanyashwas* (exertional dyspnea, 75.00%), *Tandra* (somnolence, 75.00%), *Daurbalya* (weakness, 75.00%), and *Arati* (uneasiness, 73.33%).

CLINICAL RESULTS AND STATISTICAL ANALYSIS:

➤ Effect on Objective Hemodynamic Parameters

Both trial formulations produced clinically meaningful and statistically significant reductions across all primary hemodynamic metrics over the 90-day intervention:

Hemodynamic Parameter	Group	Initial Mean (BT)	Final Mean (AT)	Mean Change	% Relief	SD	SE	t-value	P-value	Significance
Systolic BP (mmHg)	A	162.00	148.20	13.80	8.50%	11.20	2.00	6.73	< 0.001	Highly Significant
	B	155.90	144.30	11.70	7.48%	10.10	1.80	6.36	<.001	Highly Significant
Diastolic BP (mmHg)	A	98.37	92.77	5.60	5.69%	6.26	1.10	4.90	< 0.001	Highly Significant
	B	102.00	97.93	4.03	3.96%	10.80	2.00	2.05	< 0.05	Significant
Pulse Pressure (mmHg)	A	63.63	55.47	8.17	12.83%	11.20	2.00	3.99	< 0.001	Highly Significant
	B	53.97	46.33	7.63	14.14%	13.30	2.40	3.14	< 0.05	Significant
Mean Arterial BP (mmHg)	A	119.57	111.26	8.32	6.96%	4.85	0.88	7.20	< 0.001	Highly Significant
	B	119.95	113.38	6.58	5.48%	8.45	1.54	4.26	< 0.001	Highly Significant

- **Systolic Blood Pressure:** In Group A (*Mansyadi Kwatha*), 30.00% achieved full control (100% relief), 13.33% showed marked response (>75%), 16.67% moderate response (50–75%), and 10.00% mild response. In Group B (*Ashwagandha Churna*), 43.33% achieved full control, 10.00% marked response, 10.00% moderate response, and 20.00% mild response.
- **Diastolic Blood Pressure:** In Group A, 33.33% achieved full control, 3.33% marked response, 26.67% moderate response, and 16.67% mild response. In Group B, 23.33% achieved full control, 13.33% marked response, 10.00% moderate response, and 6.67% mild response.

Effect on Clinical Symptomatology

Both therapies significantly reduced symptom severity scores, with *Mansyadi Kwatha* showing particular strength in neuro-psycho and sensory symptoms, and *Ashwagandha Churna* excelling in somatic fatigue and sleep recovery:

Clinical Symptom	Group	BT Mean	AT Mean	% Relief	SD	SE	t-value	P-value
Shiroruk (Headache)	A	2.63	1.77	32.91%	2.21	0.40	2.15	< 0.05
	B	2.33	1.50	35.71%	1.78	0.30	2.56	< 0.05
Bhrama (Giddiness)	A	2.67	2.00	25.00%	1.73	0.30	2.11	< 0.05

	B	2.57	2.13	16.88%	1.14	0.20	2.09	< 0.05
Hridhravatva (Palpitations)	A	2.77	2.17	21.69%	1.50	0.30	2.19	< 0.05
	B	2.40	1.77	26.39%	1.25	0.20	2.79	< 0.05
Swasakashtata (Dyspnea)	A	3.20	2.40	25.00%	2.01	0.40	2.18	< 0.05
	B	2.27	1.73	23.53%	1.25	0.20	2.33	< 0.05
Alpanidra (Insomnia)	A	2.27	1.63	27.94%	1.50	0.30	2.32	< 0.05
	B	2.23	1.47	34.33%	1.59	0.30	2.64	< 0.05
Klama (Fatigue)	A	2.53	1.87	26.32%	1.27	0.20	2.88	< 0.05
	B	2.70	1.97	27.16%	1.41	0.30	2.84	< 0.05
Arati (Restlessness)	A	2.90	1.80	37.93%	1.73	0.30	3.48	< 0.05
	B	2.80	1.80	35.71%	1.39	0.30	3.94	< .001
Santapa (Tension/Hotness)	A	1.87	1.43	23.21%	1.14	0.20	2.09	< 0.05
	B	2.23	1.67	25.37%	1.28	0.20	2.43	< 0.05
Tamodarshana (Visual Blurring)	A	2.40	1.93	19.44%	1.20	0.20	2.14	< 0.05
	B	2.10	1.60	23.81%	0.90	0.20	3.04	< 0.05
Buddhisammoha (Confusion)	A	2.57	2.10	18.18%	1.14	0.20	2.25	< 0.05
	B	2.33	1.77	24.29%	1.41	0.30	2.21	< 0.05
Krodha-Prachurata (Irritability)	A	2.33	1.93	17.14%	1.00	0.20	2.18	< 0.05
	B	2.27	1.77	22.06%	1.14	0.20	2.41	< 0.05
Akshirag (Eye Redness)	A	2.93						

DISCUSSION

Hypertension is a psychosomatic and hemodynamic disease. It is also known as silent killer or hidden killer because of its associated hazards without expressing any symptomatology.

Hypertension is one of those disastrous diseases which are psychosomatic, hereditary and occurring as a result of aging. It is also called a silent or hidden killer because of its asymptomatic nature and dreadful effects. Hypertension is a major risk factor for the development of cardiovascular disease (CVD). Its impact is greatest on stroke and end stage is renal failure. It is also one of the contributing factors for the development of coronary artery disease (CAD) and is currently the leading risk resulting in considerable death and disability worldwide. The relationship between Blood Pressure and risk of cardiovascular disease (CVD) events is continuous, consistent, and independent of other risk factors. The higher the BP, the greater is the chance of heart attack, heart failure, stroke, and kidney disease.

As for the name of disease is concerned, there is no specific terms found for essential hypertension in classics. But its pathophysiology can be planned with Ayurvedic principles. On

the findings of the present clinical trial discussion has been made under the following headings:

For Ayurveda the concept of disease is new because it is an instrumental disease invented recently. But it is present at least since the existence of Ayurvedic Samhitas in the form of signs

and symptoms because many references present in Samhitas like Pakshaghata, Hridroga,

Mutrarooga and other symptomatology give the accurate indirect references of hypertension, but

lack of proper detail description in Ayurvedic Samhitas made Ayurvedists to study upon it. But

still no theory is complete. Hypertension represents a complex, insidious psychosomatic and hemodynamic disorder that acts as a global "silent killer," quietly causing progressive microvascular shear stress and systemic arterial breakdown without presenting overt early symptoms. The relationship between elevated blood pressure and cardiovascular disease risk is continuous, direct, and independent of other factors, making unmanaged arterial tension the leading contributor to death and disability worldwide. Chronic neuro-endocrine stress, driven by hypothalamic-pituitary-adrenal axis hyperactivation and sympathetic overdrive, interacts with non-modifiable risk factors like genetic predisposition and aging to sustain peripheral vascular resistance. Over time, this chronic elevation inflicts severe target organ damage, manifesting as stroke, coronary artery disease, hypertensive cardiomyopathy, and end-stage renal failure.

Although classical Ayurvedic literature lacks explicit diagnostic terminology for hypertension—as sphygmomanometric measurements were developed long after the compilation of the foundational Samhitas—the pathological framework of the disease is thoroughly woven throughout ancient texts via its secondary organ impacts.

Classical Ayurvedic entities such as Pakshaghata (hemiplegia/stroke), Hridroga (ischemic heart disease and cardiac dysfunction), and various Mutraroga (renal vascular disorders) represent direct clinical parallels to the downstream sequelae of chronic, untreated hypertension. From a functional Ayurvedic perspective, the pathogenesis (Samprapti) of hypertension is understood through systemic Tridosha dysregulation operating in the circulatory pathways: Vata drives autonomic hyper-reactivity and arterial constriction, Pitta fosters vascular inflammation and heat, Kapha together with Ama (toxic metabolic byproducts) induces structural plaque formation and arterial rigidity, and Rakta Dhatu acts as the primary vehicle expressing elevated circulatory pressure. Because modern medicine identifies the disease instrumentally while Ayurveda analyzes the underlying metabolic and energetic imbalances, contemporary scholars synthesize these paradigms to formulate holistic management strategies. By matching Western hemodynamic metrics with traditional principles of Nidana-Samprapti, practitioners can address root causes—such as clearing vascular channels (Srotoshodhana), calming neuro-cardiac strain, and reducing stress-induced pressor spikes—before irreversible organ damage occurs.

In this section discussion has been made on each point of demographic data, etiological factors, symptomology, biochemical parameters and analysis of data obtained in the study.

Probable mode of action of trial drugs based on Ayurvedic and modern pharmacodynamics has

been drawn in this section. Out of 30 students in group A treated with Mansyadi Kwath, 56.67% patients did not show any significant change in their symptoms but 40% patients show mild Improvement while rest only 3.33% patients shows Moderate improvement. Out of 30 students in group B treated with Ashwagandha Churna, 43.33% patients did not show any significant change in their symptoms but 46.67% patients show mild Improvement while rest 10% patients shows Moderate improvement.

Out of 30 students in group A treated with Mansyadi Kwatha, 30.00% patients show 100 % significant responses in their Systolic Blood Pressure, 13.33% patients show Marked responses, 16.67% patients show Moderate

response, 10.00% patients show Mild responses, while rest 30% patients shows No significant improvement. Out of 30 students in group B treated with Ashwagandha Churna 43.33% patients show 100 % significant responses in their Systolic Blood Pressure, 10.00% patients show Marked responses, 10.00% patients show Moderate response, 20.00% patients show Mild responses, while rest 16.67% patients shows No significant improvement.

Out of 30 students in group A treated with Mansyadi Kwatha, 33.33% patients show 100 % significant responses in their Diastolic blood Pressure, 3.33% patients show Marked responses, 26.67% patients show Moderate response, 16.67% patients show Mild responses, while rest 20.00% patients shows No significant improvement. Out of 30 students in group B treated with Ashwagandha Churna 23.33% patients show 100 % significant responses in their Diastolic blood Pressure, 13.33% patients show Marked responses, 10.00% patients show Moderate response, 6.67% patients show Mild responses, while rest 46.67% patients shows No significant improvement.

As Mansyadi kwath contains Jatamansi as main drug and Ashwagandha. In the present study Ashwagandha is used as independent drug. Therefore, the mode of action has been discussed separately for Jatamansi and Ashwagandha.

Jatamansi is reported to have Sangnasthapana, Nidrajanana, Medhya, and Hridya properties in the Ayurvedic classics. It has unique capability to treat Manas-Somatic diseases like Shiroroga, Hrdroga, Unmada, and Uchha Raktachapa.

Ashwagandha, is one of the most popular drugs as Balya, Brimhana, and Rasayana. In modern science, it is recognized for its adaptogenic, neuroprotective, and cardioprotective effects. Being Madhura and Kashaya-Tikta Rasa, Snigdha and Guru Guna, and a Ushna Virya. These properties directly counter the Ruksha, Laghu, and Chala properties of vitiated Vata. By soothing Vyana Vata, it relieves vasoconstriction and restores smooth arterial wall compliance.

CONCLUSION

Essential hypertension, conceptualized in Ayurveda as *Vyanabala Vridhhi* arising from *Rasa-Rakta Samvahana* dysregulation and multi-factorial *Mishra Avarana*, represents a pervasive psychosomatic and hemodynamic disorder. The clinical evaluation demonstrates that both *Mansyadi Kwatha* and *Ashwagandha Churna* offer significant therapeutic benefit in the management of Stage 1 and Stage 2 essential hypertension:

- **Hemodynamic Control:** Both interventions produced statistically significant reductions in systolic blood pressure, diastolic blood pressure, pulse pressure, and mean arterial pressure over three months. *Mansyadi Kwatha* demonstrated marked control over elevated diastolic resistance, whereas *Ashwagandha Churna* yielded a higher proportion of complete systolic normalization.
- **Symptom Relief:** Both formulations significantly ameliorated secondary clinical manifestations, including *Shiroruk* (headache), *Bhrama* (giddiness), *Hridhravatva* (palpitations), *Alpanidra* (insomnia), and *Klama* (fatigue). *Mansyadi Kwatha* proved particularly effective for neuro-psychic and sensory irritability (*Arati*, *Chinta*), while *Ashwagandha Churna* excelled in treating asthenia, stress resilience, and sleep disturbance.

- **Mechanistic Synergy:** The therapeutic outcomes are supported by complementary pharmacological mechanisms: *Mansyadi Kwatha* enhances central GABAergic transmission and peripheral vasodilation via *Nardostachys jatamansi* and *Hyoscyamus niger*, while *Ashwagandha Churna* downregulates hypothalamic-pituitary-adrenal (HPA) axis overdrive, mitigates oxidative stress, and upregulates endothelial nitric oxide bioavailability.
- **Clinical Viability:** Both herbal regimens exhibited strong safety and tolerability profiles without the adverse effects commonly linked with lifelong synthetic antihypertensives, establishing them as effective, accessible therapeutic modalities for mild-to-moderate uncomplicated essential hypertension.

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