MANAGEMENT OF PULMONARY HYPERSENSITIVITIES IN AYURVEDIC PERSPECTIVE – A CLINICAL STUDY.

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Declaration

I, **Dr. SAMEER N. NAIK**, PhD scholar declare that my research work in the thesis entitled **"MANAGEMENT OF PULMONARY HYPERSENSITIVITIES IN AYURVEDIC PERSPECTIVE A CLINICAL STUDY."** has been carried out by me under the guidance of Dr. B K. BHAGWAT, Professor, Dept of Kayachikitsa, College of Ayurveda, Pune, for the degree of AYURVEDA VARIDHI, DOCTOR OF PHILOSOPHY in Kayachikitsa. This work is original and has not been submitted for any degree or diploma or associate ship of this or any other University.

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CERTIFICATE

This is to certify that, this thesis embodies the out come of original observation made by Dr.Sameer.N.Naik on the **"Management Of Pulmonary Hypersensitivities In Ayurvedic Perspective A Clinical Study."** This *work has been carried out under my immediate direction and guidance in the Department of Kayachikitsa, Bharati Vidyapeeth Deemed University, College of Ayurveda and Hospital, Pune during 2005-2012.*

This thesis bears ample evidence of original thought and devoted work. It makes a distinct advance on scientific lines in this subject. I strongly recommend and forward this thesis for acceptance of the degree of 'Ayurveda Varidhi' Doctor of Philosophy (PhD) of the Bharati Vidyapeeth Deemed University, Pune.

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CERTIFICATE

This is to certify that Dr Sameer N. Naik a PhD Scholar in the speciality of Kayachikitsa has successfully completed his thesis entitled **"Management Of Pulmonary Hypersensitivities In Ayurvedic Perspective A Clinical Study."** *for the degree of Ayurveda Varidhi (Doctor of Philosophy) under the direct supervision and guidance of Prof. Dr. B.K.Bhagwat*

The scholar has presented the research work before experts committee of Institutional Research and Ethical Committee (IREC) and Pre PhD Seminar on 20-4-2009 and 10-6-2010 respectively. The suggestions given by experts are all incorporated in this thesis.

The scholar has put hard work in bringing about this thesis after making an intensive study on the subject from Ayurvedic and Conventional view as well as clinical study. This work makes a distinct advance on scientific lines in this scientific.

This is further certified that he has completed all the formalities laid down in the regulation governing the award of 'Ayurveda Varidhi' PhD degree of Bharati Vidyapeeth Deemed University, Pune.

Hence, his thesis is recommended for being submitted to adjudication for the award of PhD degree in Kayachikitsa.

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Dr. Sameer N. Naik.

Date:

Place: Pune

Abbreviations

- **C.S.**Carakasaṁhitā
- **S.S.**SuṣṛtaSaṁhitā
- **A.S.**AṣṭaṅgaSangraga
- **A.H**.AṣṭaṅgaHrudaya
- **B.S.**BhelaSaṁhitā
- **K.S.**KashyĀpa Saṁhitā
- **M.**N.MadhavaNidāna
- **Sha.S.**SharaṅgadharaSaṁhitā
- **B.P.**BhāvaPrakash
- **Y.R.** Yoga Ratnākara
- **R.R.S.**Rasa RatnaSamuchaya
- **B.R**.BhaishajyaRatnāvali
- **Basava.R.**BasavaRājeeyam
- **CH.D.**Chakra Datta
- **Ch.S.S.**Chikitsā Sara Sangraha
- **R.Y.S.**Rasa Yoga Sarasangraha
- **CP.**ChakarĀpani
- **JJ.**Jejjata
- **Dal.**Dalhaṇa
- **Su.**SūtraSthāna
- **Ni.**NidānaSthāna
- **Sh.**ŚarīraSthāna
- **Vi.**VimanaSthāna
- **Chi.**ChikitsāSthāna
- **Si.** Siddhi Sthāna
- **Ka.**KalpaSthāna
- **Pu.**PurvaKhanda
- **Ma.**MadhyāmaKhanda
- **Ut.**UttaraKhanda
- **w.**s.r.With Special Reference

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ABSTRACT

"MANAGEMENT OF PULMONARY HYPERSENSITIVITIES IN AYURVEDIC PERSPECTIVE

A CLINICAL STUDY

1. ***Scholar – Dr. Sameer N.Naik.***
2. ***Guide – DR. Bhalachandra K.Bhagwat.***

Introduction The respiratory tract while performing its physiological function is exposed to a wide variety of air-borne environmental antigens. The lungs are working as filter for the entire circulating blood volume. Thus, these are constantly exposed to various blood & air-borne agents that possess potential to accelerate inflammation, infection or immune processes. Pulmonary hypersenssitivitie can be named as Atopic disease. Atopy is defined as familial tendency to sensitization to environmental allergens. Atopic allergy is a type 1 hypersensitivity reaction that produces IgE antibodies to allergens viz. pollen, dust, etc. pulmonary hypersensitivities has always been proved to be a problematic ailment to the doctors. The magnitude of the condition can be understood by the fact that, though it is known from the ancient era & inspite of worldwide efforts to combat this impediment, still there is no definite solution for the problem. *The objective is to find out the efficacy of shodhana,and shamana in the management of* Pulmonary hypersenssitivities.

Hypothesis – *The Śodhana,and Śamana therapies do have a significant role in the management of* Pulmonary hypersenssitivities

Materials and Methods – *According to Sahasrayoga and Bhavaprakash Shireesharishta and Bhriguharitaki are best in the management of* Pulmonary hypersenssitivities *respectively.150 patients selected as per criteria anddevided them in to three groups. Examined by means of Roga and Rogi Parīkṣā methods.* After Virechana, **For Group A patient – Shirisharishta** was given in the dosage of 20ml three times in a day with ushnajala as anupan after food. **For Group B patient – Bhriguharitaki Leha** was given in the dosage of 10ml three times in a day with Sukhoshna dugdha as anupan before food.**For Group C patient- Shirisharishta** was given in the dosage of 20ml three times in a day with ushnajala as anupan after food. **Bhriguharitaki Leha** was given in the dosage of 10ml three times in a day with Sukhoshna dugdha as anupan before food. Subjective *and Objective parameters were assessed before and after treatments.*

Results - *Paired't' test and Anova were adopted for statistical significance. Results revealed that subjective and objective assessment by score method shows that majority cases got Significant improvement.*

Discussion – *All the clinical observations in respect of incidences of Nidāna, Lakṣaṇa, and percentage of relief are illustrated by tables, charts, diagrams and discussed with comparing Āyurveda.*

Conclusion – *In the present research work undertaken, Ayurveda formulations of Shodhana, and shamana is clinically provedvery effective.*

Key words – *Respiratory hypersensitivities, Shireeshrishta, Bhriguharitaki, astmyaja pranavaha sroto vikara.*

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1.3 INTRODUCTION

For most of us, the process of breathing in and out is effortless, thus hardly noticeable, and therefore, often taken for granted. Through a life span, consider the newborn's first gasp for air outside mother's womb, signifying the wonderful act of entry into this world, the infant's first vocal sounds and lisps that express emotions enabling communication with the world, and finally, the inevitable act of dying, or expiration, marked by giving the spirit away with the last breath - all tied to the respiratory tract.

Respiratory hypersensitivity is a frightening condition which can seriously impede one's ability to breathe, and suddenly rob the individual of the most important nutrient of all - oxygen. People who are having an Asthma attack have real trouble taking a breath. Many people with

Stuffy noses from hay fever or colds say, "I can't breathe," but they retain the option of breathing through the mouth. Asthmatics, however, know what "I can't breathe" really means. Instead of their nasal passages, it is the bronchial tubes in their lungs that become swollen and clogged. Breathing can become frighteningly difficult. Asthma involves two conditions:

(1) Contraction of the small muscles surrounding the bronchial tubes and
(2) Inflammation of the lining of those tubes.

The GINA Workshop report 2005 says, "The rate of asthma increases as communities adopt western lifestyles and become urbanized. With the projected increase in the proportion of the world's population that is urban from 45% to 59% in 2025, there is likely to be a marked increase in the number of asthmatics worldwide over the next two decades. It is estimated that there may be an additional 100 million persons with asthma by 2025."

Allergy accounts for a substantial number of human diseases with significant morbidity. The respiratory tract is in constant contact with external environment & is therefore a major site of antigenic challenge.

The term allergy is used to mean an altered or acquired state of sensitivity, abnormal reaction of the body to substances normally harmless. Such as, Pollen allergens, Animal allergens, Food allergens, Arthropod allergens, Fungal allergens, Drug & Chemical allergens, Occupational allergens. Allergic respiratory disorders are on the rise due to the changed life style of the 21^{st} century man which incorporates extraordinary thrust on cosmetics, preservatives & drugs & increasing population. Every 7^{th} person in the world is suffering from these problems.

Respiratory Allergies / hypersensitivities constitute the greatest stress on the people in most of the developed and developing countries. Problem of allergy particularly, respiratory allergies are increasing throughout the world at an alarming rate. In India, prevalence of Respiratory Allergic Disorders (RADs) in school going children has been reported between 5-20% in different geographic regions. Respiratory allergy is one of the

commonest illnesses during childhood and in the population who indulge in mining, coir industry, paint industry etc. Typical complaints include intermittent nasal congestion, itching, and sneezing, clear rhino rhea, conjunctival irritation, loss of sense of smell and taste, headache, wheezing, coughing, and dyspnoea. Nasal congestion is often more severe at night causing mouth breathing and snoring interfering with sleep and inducing irritability. It mainly includes Allergic rhinitis and allergic asthma. People with Respiratory Allergic Disorders experience frustration, anxiety and physical, social and emotional disturbances that affect their learning and ability to integrate with peers. The disorder contributes to headaches, fatigue, limits daily activities, interferes with sleep and leads to school absenteeism [1] in children and disturbance in the routine work in other population.On comparing the symptoms of RADs with different diseases in Ayurveda, it shows close resemblance with *Sadyah Pratishyaya*, *Vatic Pratishyaya* and *Tamak shwasa.*

Despite an alarming growth of incidence & prevalence, prevention of these ailments is a neglected area.

Alarmingly one in every 6 persons diagnosed for chest problem is found to be suffering from Asthma. The prevalence of Asthma is increasing worldwide despite to improved treatment options. Asthma is a complex genetic disorder. Globally over 1, 80,000 people die from Asthma each year. Till date there is no total cure for these hypersensitive allergens, reactions due to their complicated aetio pathogenesis.

Even though the modern science is well equipped with advanced technology it has failed in giving satisfactory results (Complete cure) for most of the pulmonary hypersensitivities. Now the Western people are showing lot of interest towards the other faculties of medicine for this current burning problem.

According to modern medical science, its management includes Antihistamines, Bronchodilators, Mast cell stabilizers and corticosteroids apart from avoidance of allergens. But most of the time, these are associated with many adverse effects like, tachycardia, tremors, headache, hypokalemia, sedation, weight gain, oral thrush, reflex coughing etc.[1] According to Ayurvedic concept, both the *Ama* and the *Vyadhikshamatva,* are involved in the pathology of RADs. A large number of drugs are mentioned in the classics which are capable of breaking the pathology of RADs at various levels, and giving prompt symptomatic relief to the patient. Also, the drugs possessing immunomodulatory action are supposed to gear up the immune system (*Vyadhikshamatva*) of the body and prepare the body to combat allergens. Thus, it is supposed that these drugs can prove beneficial and provide effective and long term solution to allergic disorders and thereby may improve the quality of life and school performances in children.

Classics of Ayurveda offer a very detailed illustration of respiratory disorders. It includes allergic as well as non allergic disorders. It is our interest & duty to find out allergic types so as to offer the best remedy.

Hence the present study is formulated to find out different types of respiratory allergies & to see the effect of Virechana **& Shirisharishta , Bhrigu haritaki rasayana** when used in combination & when used singly.

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AIMS & OBJECTIVES

1. To develop a concept for pulmonary hypersensitivity in Ayurveda.

2. To identify the different types of respiratory allergens which are prevalent in this area.

3. To evaluate the efficacy of **virechana, Bhrigu haritaki Rasayana & Shirisharishta** in the management of different types of **pulmonary** hypersensitivities.

CONCEPTUAL STUDY

DISEASE REVEIW

PREFACE

Ayurveda has the better understanding of hypersensitivity with relevant examples. Ayurveda believes in the maintenance of health through different measures. Few of them are in the form of daily regimen (*Dinacharya)*, *Rutucharya* (seasonal regimen) etc. The measures to identify healthy status is on the lines of *Dosha, Dhatu ,Agni,* Evacuation of excreta(*mala*) at right or appropriate time, Tranquil mind, fit sensory and motor capacity and of course Souls tranquility.

All these measures will help in maintaining swasthya of swastha. Ultimately bala/vyadhikshamatva of an individual plays an important role in maintenance of health, preventing disease manifestation and in getting rid of diseases. If bala /vyadhikshamatva is good even though the person get exposed to nidanas, disease will not manifest because of absence of vikara utpattikara bhavas and if the bala is avara then the person is more likely to suffer from respective disease.

There are many instances in the text books of Ayurveda, where we may identify the Features, Treatment, and Causes about Allergy. Only thing is that they are described in different parts of the Samhitas.

The concept of *Satmya* and *Asatmya* is clearly an indication of immunity and allergy. Here *Satmya* means Compatibility. The S*atmya* may be towards the Medicine or to the Food. When *Satmya* is quoted for food, it indicates that the food which is practiced for long duration helps the person to develop his Immune system. In the same way there is one more concept called "*Oka Satmya*". That is compatibility attained through slow adaptation mechanism. That is a drug/ food is given in daily basis in smaller quantity for prolonged period, so that the person is gradually developing adaptability towards it, even though it is incompatible to him previously.

The matter of *Asatmya* means Non compatible to body. I.e. if a medicine or food is not acceptable to the body, body tries to throw it away in different fashions. It may be Diarrhea, Vomiting, and Skin manifestations and so on. Hence the treatment runs according to the presenting complaint. Whenever the combination of two drugs or food is going to take place it acts as either *Samyoga* or *Viruddha.* The *Viruddha* is the causative factor for *Atma/Shareera asathmya* in many instances.

Eg; (1) continuous consumption of Fish and milk

(2) Consumption of Milk and Salt.

(3) Consumption of Honey and Ghee in equal quantity.

The above are very few selected examples where in various skin ailments are the resultant. But these changes require longer duration for Manifestations. The prolonged administration of such things in due course ends up with the mechanism of manifestation of disease. The above discussion clearly indicates the allergen in the form of Milk\ Salt\ Fish\ Honey\ Ghee to the respective person. The whole mechanism may be considered as ALLERGY.

In the same way if there is some specific changes in food, nature of composition of food, nature of consumption of food, time of consumption of food then it will lead to Diarrhea. This is one of the acute manifestations. This is nothing but food allergy of acute origin. The immediate treatment if done in the form of stoppage of disease, it will lead to some skin reactions and some gastro intestinal tract pathologies. Hence the above allergy may be considered as food allergy and acute allergy.

While discussing the causative factors for diseases like Shwasa, Kasa, 'Raja", Dhooma, are identified. The "Raja" means nothing but dust particles .Dhooma is irritant fumes. If the person is having allergy towards them, then only he will develop pathologies like Shwasa, Kasa. Hence we may consider Raja and Dhooma as Allergens. Like wise there are other such identified Allergens in such scenario.

The complete description of allergy in Ayurveda can be traced under following contests.

- Viruddha ahara
- Visha (gara / dooshi visha)
- Satmyasatmya vikaras.
- Concept of hereditary diseases.

  Hence in the present context the conceptual part of respiratory hypersensitivity is discussed in two chapters,

  i) Concept of bala/vyadhikshamatva/immunity.

  ii) Concept of asatmya vikara/ hypersensitivity/allergy.

VYADHI KSAMATVA

The concept of immunity, *Vyadhi Ksamatva* or *Bala*, as it is known in Ayurveda, is a fascinating and vast subject. The body's resistance is of tremendous importance in the daily welfare of living beings; for prevention and rapid recovery from diseases, immunity plays a key role. It can be observed that among a group of people exposed to a given disease, only some will be afflicted, while others are left without any effect. This phenomenon itself illustrates two important points -- that the pathogenic factors require some essential favorable conditions to flourish, and that the individual is susceptible to the disease. In the absence of such conditions, an individual's immunity or resistance can eradicate the disease, preserving and maintaining a balanced condition. This concept is akin to the principle explained in ancient Vedic literature: Manusmruti, Mahabharata and Panchatantra all explained that the seed sown in non-fertile soil will be destroyed, just as fire thrown in a fuel less or air-less place subsides.

In Ayurveda, ojas is considered as vital in the defense mechanism of the body. In conditions like diabetes mellitus and malnutrition, where loss of ojas is a constant feature, people are known to be susceptible to various other interrelated and degenerative diseases or recurrent infections. As a general rule, those who indulge in an irregular routine and eat unwholesome food tend to suffer ill health. Conversely, those who maintain a regular, healthy routine and take wholesome food generally maintain good health. However, it can be observed that some people can tolerate and overcome disease even after indulging in irregular routine and unwholesome food, managing to live healthily and happily.

It can also be noted that though some people follow a regular routine and eat wholesome food, they are still susceptible to disease and suffer ill health. These are unanswered questions that are worthy of investigation and analysis. Ayurveda offers multi-faceted and profound explanations for this phenomenon.

Intake of wholesome food and a regular routine alone are not enough to prevent disease. Additional factors such as mistaken intellect, constitution, moral conduct, karma and unsuitable contact of sensory perceptions like sound, touch, vision, taste and smell are also responsible for the onset of disease. Due to these factors, diseases manifest as mild or severe, acute or chronic, easily curable or difficult to cure/incurable.

02.2: CONCEPT OF OJA AS IMMUNITY

Man is a creature composed of millions of cells. A microbe is composed of only one. Yet, through out the ages, the microbes have had the upper hand in their ceaseless conflict with man. The above sentence is narrated from Atharvaveda, which dates backs to 5000 years.

Ayurveda describes human body as, seat of Chetana (consciousness) and a product of Panchabhoutika Vikara, existing in equilibrium gets disturbed. That results in defective bodily tissues. This is the beginning of any disease [1]**.**

The normal healthy state of a body requires normalcy of several factors they are Dosha, Agni, and Dhatu, and Mala*,* Peaceful deposition of Atma, Indriya and Manas.[2] for their normal functioning, body requires strength, which is called as Bala. The same normal strength had synonym as Shleshma [3.] That is also known as Ojas. Ojakshaya is a broad understanding of immuno deficiency, depleted vigor and vitality. This Ojas is transformed from the parents to the pregnancy through Sukra and Shonita[4] (Beeja of Purusha and Stri) at the time of zygote formation. Ojas is the essence of all the Dhatus. The Beeja *i*s responsible for the formation of particular organ or tissue, if it is vitiated, that results in deformity of the respective organ. If it develops undisturbed there will not be any deformity of the respective organ.

There for it is clearly understood that every part of the healthy human body (Dhatu and Mala*)* develops according to the healthy state of the Beeja and Beejabhaga. There for the essence of Dhatus as represented by Ojas of Pumbeeja and Stribeeja plays the major role in this mechanism.

Ojas depends upon healthy state of Kapha. Physiologically Kapha represents a potential sour of strength and resistance to disease and decay. Those are Bala and Ojas. These terms reflect to the force and power which resists the factors responsible for decay and disease. Bala may be Sahaja (inherited), Kalaja (seasonal) and Yuktikruta (acquired)[5]. But these all are equally capable of resisting the diseases. As Vyadhikshamatva is a force antagonistic to virulence of diseases causative factors[6]. Susruta clarified further stating Balam is Ojas[7]**.** As long as Dhatus are strong and healthy and are conducting their normal functions which their essence i.e. Ojas being both qualitatively and quantitatively effective.

The body will be strong enough to resist and counter the decay and degeneration caused by either the natural processes or disease. So in this contest this is very essential that, to know about etymology and normal physiology of Ojas.

Nirukti (Etymology) of Ojas

'Ojas' the word has its root in "uj"or "vaj" Dhatu. That means confer or strong (Ugra) Ojas is the Subanta Pratyaya of word Ojas, which means Deepti, Prakasha and Balam[8.] Kalidasa in Raghu Vamsha Kavya writes 'Rudraoujasa' with reference to the potency of Shiva**.**

Paribhasha (Definition) of Ojas

Ojas is the essence of all Dhatus. Ojas is nothing but the Bala or Strength of the body, which is the ultimate end product of the seven Dhatus starting from Rasa and ending at Shukra[9]. Chakrapani contradicts this opinion and says Ojas sustains the body but does not nourish it.

The normal healthy state of a body requires normalcy of several facts. They are Dosha, Agni, Dhatu and Mala, along with peaceful deposition of Atma, Indriya and Manas. For this normal functioning, body requires strength which is called as Bala. The normal strength is called as Shlesma, which has synonym of Ojas, This Ojas is transformed from the parents to the pregnancy through Shukra and Shonita which is the essence of the Dhatus. If a part of Beeja, which is responsible for the formation of particular organ or tissue, is vitiated, that results in deformity of the respective organ. If it develops undisturbed there will not be any deformity of the respective organ. There for it is clearly understood that every part of the healthy human body (Dhatu and Mala) develop according to the healthy state of the Beeja and Beejabhaga, there for the essence of Dhatus as represented by Ojas of Pumbeeja and Steebeeja plays the major role in this mechanism.

Ojas depends upon healthy state of Kapha. Kapha physiologically represents potential source of strength and resistance to disease and decay of Bala and Ojas. These terms reflect to the force and power which resists the factors responsible for decay and disease. Bala may be Sahaja (inherited) Kalaja (seasonal) and Yuktikrita (acquired). But these all are equally capable of resisting the diseases.

As long as Dhatus are strong and healthy and conducting their normal functions with the essence i.e. Ojas being both qualitatively and quantitatively effective. The body will be strong enough to resist and counter the decay and degeneration caused by either the natural processes or disease. So in this contest this is very essential that, to know about etymology and normal physiology of Ojas.

Formation of Ojas in the body

Ojas is the 'Sara' i.e. essence of all Dhatus [10]. It is produced In the body as honey, Which is collected by bees from various flowers and fruits. Ojas is derived from all the Sapta Dhatus in other words all the Dhatus contributes to its making. Ojas is the product of the Prasada Paka of Dhatvagni Vyapara.That has the essence of all the Sapta Dhatus in it. Essentially Ojas depends on Ahara for its production and sustenance [11.]

Panchaboutika Sanghatana

Apara Ojas is also known as Shlaishmika Ojas and it is considered Somatmak denoting the predominance of Aap and Prithvi Mahabhootas [12.]

Physical properties of Ojas [13]

- ✓ Color: Whitish yellow or whitish red resembling the color of Ghee
- ✓ Taste: Sweet like honey
- ✓ Odour: Smell like fried paddy or Laja gelatin
- ✓ Oja Sthana: Ojas is present all over the body and in each cell of the body [14]

Oja Karya [15]

- ✓ Dosha Nigrahana
- ✓ Sthiropachita Mamsata
- ✓ Cheshtasu Apratighata
- ✓ Svara Varna prasadhana
- ✓ Karananam Atma Karya Pratipatti
- ✓ Preenitaha Sarvadehinaha
- ✓ Prana Yatra Pratishtita

Consistency

Snigdha(unctuous)[16], Guru(heavy)[17], Pichchila(gelatinous)[18], Mridu (smooth)[19],Sheeta, [20] or Somatmaka (mild to touch)[21], Sthira (stable)[22] Shukla varnam, [23] Saram [24] Viviktam[25] ,Guru [26] ,Bahalam[27], Madhu Rasam[28],Lajagandhi [29] Lohita Peetakam, [30]

Ojas Poshana [31]

Ojas is nourished mainly by Ahara, from the Ahara–Ahara rasa- Rasa Dhatu- Rakta-Mamsa-Medha-Asthi- Majjaa-Shukra dhatus

Classification of Ojas [32]

There are two classifications of Ojas, made by Charaka, Susruta, Chakrapani, and by all other acharyas. Those are

- ✓ Para Ojas
- ✓ Apara Ojas [33]

These two types of Ojas which have a direct bearing on body's defense against degeneration and infection

Para Ojas

Ojas marks the beginning of the formation of embryo. It is the essential nourishing fluid developed from the Rasa of the embryo. It enters the heart right at the stage of the letter's initial formation and is permanently locates there, sustaining the life of fetus. Loss of Ojas amounts to the loss of life itself. Chakrapani comments that the above function pertains to both the Ojas and further explains that Ojas plays a role in three different stages of the life of the fetus. It permeates to through Rasa in entire body and nourishes entire body [34] and Ojas is transported through the Ojovaha Dhamanis. [35]

- ✓ At the time of conception it is the essence of Shukra and Shonita.
- ✓ In the second stage it is the essence of the Rasa Sara, which provides nutrition to the embryo.
- ✓ The third stage, when there is formation of various organs, Ojas manifests with its own action.

Apara Ojas or Sleshmaka Ojas

It performs the Tarpana action in the entire body. It is source of strength to the Dhatus. Any loss in the quantity would cause sudden death. Commenting on function of Ojas Susruta has made a significant observation. Ojas permits entire body nourishes limbs and organs. In the absence or deficiency of Ojas in the body there will be wasting, decay, degeneration, and destruction of the body. This statement indicates the nutritive nature of the Apara Ojas in preventing the decay of the body. It is one of the ten seats of life. It gives firmness to physical structures and gives strength to motor activity. Ojas spreads all over the body. In the absence of it life does not exist. The seat of Apara Ojas is the ten dhamanis connected with Hrudaya [36.]

According to Vagbhata, the function of Apara Ojas is "Dehastitanibhandana". Which means it keeps the physical fitness of the body. Chandranandana [37] clarifies that it is the protection of the body in all the states. Hemadri also states that the changes in the Ojas are the root cause for all the changes in the body. Ojas is Bala, which is a potential source of resistance to disease and decay. Bala controls the Doshas that cause disease. This is called Vyadhi kshamatva.[38.]

Synonyms of Ojas

The term Ojas has been stated in Ayurvedic classics represents Kapha, Bala, Shleshma, Rasa and Rakta.[39]

Sleshma in normal state apart from other confers [40.]

- ✓ It gives Weight and bulk.
- ✓ It gives Strength to perform work
- ✓ It resists disease and decay.
- ✓ Promotes durability, (preserves the body from decay)
- ✓ Promotes healing process (Ropanam)
- ✓ It promotes tissue building.

Ojas Vriddhi Lakshanas

Increase in Ojas results in vriddi of Bala, Varna, Agni, Medha, Ayu and Sukha.Decrease results in kshaya. [41]

Nidana of Ojakshaya

The pathological state of Oja is called as Ojokshaya .[42.] Charaka and all other classics have described this Oja vikriti as Ojokshaya. Susruta has classified this condition in to three different stages [43] as 1) Ojo Vishrams, 2) Ojo Vyapat, 3) Ojokshaya, The Nidana which causes depletion of any Dhatu, can also causes depletion in the Ojas qualitatively and quantitatively. The factors influencing the Ojokshaya are as follows.

- ✓ Ahar karana.
- ✓ Vihar karana.
- ✓ Manashika karana,
- ✓ Aagantu karana.[44]

Ahar karanas

- ✓ Alpaasha (mal nutrition)
- ✓ Anashana (starvation)

Vihara karanas

- ✓ Vaata atapa shevana (expose to sun heat and winds)
- ✓ Ativyayama (excessive work beyond the capacity)
- ✓ Ativyavaya (excessive sex)
- ✓ Shonit ativartana (loss of blood) and
- ✓ Prajagara (keeping awake at night)

Manasika Karanas

- ✓ Kopa, (anger)
- ✓ Shoka (grief)
- ✓ Chinta (worry)
- ✓ Bhaya (Phobia).

Agantu Karana

"Sankramana" or "Upasarga" denote infection

- ✓ Krimi, Rakhsa, Rakshasa, yathudhana, Pishacha, Gandarva, Bhoota, Nishachara, presents different types of microbes, and
- ✓ Oja bhakshaka rajanichra. [45]

Many diseases like Rajayakshma, Abhishyanda, Kushta, Jwara, Upadamsha, Pooyameha, Apatantraka, Visarpa, Masoorika, Rohini, are some examples of infectious diseases coming to the mode infection, Bootopaghata, due to Bhuta, Pishacha, Rakshasa, etc. Charaka has mentioned that the Ojas is the Ahara for rakshas and if they consume the Ojas, which leads to depletion of Ojas. Here Rakshasa i.e. Rajanichara [46] can be correlated to infectious organism which spreads through Prasanga (sexual contact), Gatra samsparsha (physical touch), Nishwas (droplet infection), Saha bhojana (eating together) and Saha shayyasana, (sharing bed).

Nidanartkara Vyadhi for the Oja vikriti

Rajayakshma [47], Prameha [48] Pandu [49,] Raktarsha, Raktatisara, Kshayaja kasa, Kshataja kasa, Sannipata jwara [50], are the diseases which causes Ojakshaya in there later stage. [51] Susruta pointed such possibility while dealing Abhinyasa jwara where in he used the term Hataujas [52] indicating the Ojakshaya.

The clinical futures are low or even sub normal temperature, sub comatose state, loss of voice, cracked tongue, dry ness of throat, suppression of stools, perspiration, micturation, hardness of chest, aversion to food, dull complexion, difficulty in breathing, and delirium. Susruta observed that disturbance of Ojas to the various parts of the body is affected either due to leakage or loss or obstruction to the Ojas carrying tiny shrotases in Sannipataja jwara, such condition is called as Oja – Nirodhaja sannipata. Inertness of the limbs, chills, fits, loss of consciousness, delirium, etc.

The acute condition referred above illustrates how the pronounced loss of Ojas contributes to an extra –ordinary state of susceptibility to increased microbial / viral activity and to toxins produced by these agents. Other clinical conditions which are slow in progression, chronic in nature, and cause profound Dhatuksaya (wasting of body tissues) occur due to metabolic abnormalities leading to diminished production of Ojas. This will happen due to loss of structural integrity of Dhatu vaha srotas and obstruction in the supply system. Such other disease syndromes are Rajayaksma, Madhumeha, Ojomeha [53], Pandu, Sannipata jwara, and etc.

Charaka has enumerated the pathological sequences very clearly while explaining the Samprapti of Rajayaksma (both Anuloma and Pratiloma) in Charaka Chikitsa. After explaining the manner in which nutrient materials are normally metabolized and assimilated

by the Dhatus he elaborates it further, due to the obstruction of srotas. As a result of a deficiency of nutrients Raktadi Dhatus, lowered functioning of dhatwagni and catabolic events, the food ingested, which under goes Pachana in Koshta, is changed in to Malas. Charaka in the Samprapti of Madhumeha observed that Vata by its Ruksha Guna.

Charaka in the Samprapti of Madhumeha observed that Vata by its Ruksha Guna transforms the Ojas, sweet in taste to astringent and transports it to the Mutrashaya leading to the causation of the condition known as Madhumeha. It is another disease where Ojokshaya is evident. Several other conditions creep in long with the main disease. Here the Ojas produced in this person it self is vitiated [54.]

In case of Pandu Roga the Samprapti is dominated by Pitta. The aggravated Dosha vitiates the dhatus, which in turn loose their integrity and loss of normal colour, Bala (resistance) and Sneha, which are the Gunas of Ojas are depleted by the Dosha-Dhatu Sammurchana resulting the clinical features; impoverished Rakta, and Medha dhatus leading Shitilendriyata and Vaivarnyata.

Prameha, the urinary disorder is of two types. One caused due to endogenous factors like Vatadi doshas, another one caused by exogenous or Agantuja factors like indulgence in sex with the unfit and diseased partners. Agantuja Prameha is infectious and communicable disease, transmitted through Agamya, and Dushita yoni Samsarga (sexually transmitted). In these Pramehas Ojakshaya occurs as a consequence of the passage of Ojas – mixed with urine excessively. Here Ojakshaya takes place in two different ways [55.]

Dosha Dhatu Kshayajanya Ojakshaya. (That is depletion of the Ojas, due to the endogenous factors such as Dosha, Dhatu.etc.) Aupasargika / agantuja meha janya Ojokshaya (depletion due to exogenous factors like infections etc). But there is lot of difference in treatment between above said two entities.

In case of Madatyaya, that severely affects Ojas, Bala and Prana. Madya is having qualities exactly opposite to that of Ojas. Hence the Vyadhikshamatva is affected in it's totally. Krimi and Visha also lead to severe loss of Ojas.

Ojovisramsa

Ojas mixes with Rasa Dhatu in Hridaya, from there it circulates trough out the body via various Srotases. In this condition, the circulating Ojas leads leaks out or oozes out from

the tiny distributing channels as a result; this vital substance may not reach certain organs / parts of the body and leads to the following signs and symptoms.

Ojovyapat

It is a pathological condition of Ojas because of vitiation by the Dosha as a result, the Ojas looses its physiological or normal qualities and properties as described to it, this vikruta Ojas prod uses the following laxanas.

Ojakshaya This is the final stage of Ojo-vikriti represents the loss and wasting of Ojas.

According to Susruta [56]

Ojovisramsa

- ✓ Sandhi Vishleshana (loss of firmness of the joints)
- ✓ Gatrasada (inertness of the extremities)
- ✓ Doshachayana (disturbance) displacement of Doshas from their own places
- ✓ Kriyasannirodha (impairment of kaya Vak-Mano Vyapara)

Ojovyapat

- ✓ Sthabdagatrata. (Heaviness and stiffness of the body and extremities.)
- ✓ Vata –Shopha (edema of Vataja nature)
- ✓ Glani (malaise)
- ✓ Varna Bheda (impairment of normal color of the skin complexion)
- ✓ Tandra (drowsiness)
- ✓ Nidra

Ojakshaya

- ✓ Murcha (loss of conscious ness)
- ✓ Mamsa Kshaya (emaciation of mussels)
- ✓ Moha (stupor)
- ✓ Pralapa (delirium)
- ✓ Marana (death)

Kapha and Vyadhikshamatva

Health and longevity depends on the Bala as represented by Kapha. Charaka has explained the same in the words "Baladisthanam Arogyam". Bala denotes two vital aspects of life process namely Vyayama Shakti. Vyadhikshamatva is further classified into three types – Sahaja, Kalaja and Yuktikrita.

Sahaja Bala [57]The Sahaja Bala or resistance to the disease is stated to be Prakriti. I.e. Inherent genetic from of resistance existing in the individual body since birth and this also increases along with the growth of the body elements i.e. Sapta Dhatus .It comprehends both Sharira and Satva i.e. body and mind.

Kalaja Bala [58]

Kalaja Bala is influenced by the factors like seasonal variations and age of the individuals. Thus Kalaja Bala is supposed to be dissipated at its lowest leveling the Adana Kala comprising of Sishira.Vasanta and Greeshma Ritus. On other hand Bala is stated to be conserved and at its high peek level in the Visarga Kala, increasing over Varsha, Sharat and Hemanta Ritus. Those are known as Sheeta Kala or cooler period.

Yuktikrita Bala [59.]

Yuktikrita Bala refers to the body's resistance against disease, which can be enhanced by appropriate nutrition such as meat, Ghee, etc. Restorative and Rasayana therapy in keeping with the seasonal requirements, adaptation of Swastha Vritta principles of Ayurveda along with Achara Rasayana also contributes the growth of **Yuktikrita Bala.**

Dalhana in his commentary on Bala Lakshanas as explained by Susruta observes Ojas and Bala as synonyms, especially with Chikitsa point of view. However they are distinct in the sense the former is the essence of all the dhatus and it has physical properties like Roopa, Rasa, Veerya etc., the later has to be determined from the power to lift heavy weight and the capacity to bear heavy loads etc. it does not possess physical properties. [60]

The Vyadhikshamatva is not the same merit/order in all constitutions. In other words-This Shakti varies from individual. The same is explained in the Charaka Samhita as "Na cha Sarva Shareerani Vyadhikshamatva Samarthani Bhavantani." In the discussion on factors that influence Bala, held between Punarvasu Atreya and Agnivesha is recorded in the

chapter Vividaashitiyapeeya in the Charaka sutra sthana. This discussion throws considerable amount of light on the views held on resistance to disease.

Kapha is five types [61.] Those are Kledakakapha, Bodhakakapha, Tarpakakapha, Avalambakakapha and Shlesmakakapha. Each one is limited to some part or parts of body by their functions. They look after the functions of the Kapha locally and project the body collectively.

The function of Kapha [62]

The important functions, attributed to the Sleshma by the different Acharyas are,

1. Kapha is responsible for growth, weight, and bulk of the body. That is Brimhanam, and Gouravam.
2. It is Vrishya, a function relates to sexual stamina and productivity.
3. Sthairyam- it imparts stability and durability to the body and strength to the limbs.
4. It confers strength required to perform labors physical activity i.e. physical activity i.e. Vyayama Shakti.
5. It also provides the power to resist and overcome forces or factors, which bring about disease and decay popularly known as Vyadhikshamatva viz. Vyadhibala viroditva, Vyadhi utpadaka hetupratibhandakatvam.[63].
6. Ropana- promotes healing process.
7. Ambukarma- Kapha being a repository of water, Makes this important fluid function to sub serve its vital secretary activities.
8. It has a function responsible of cohesion of various units and structures of the body.

02.3: INTRODUCTION TO BALA

Bala is mentioned as the foundation on which individual's health is resting. On one hand Bala is the capacity of strength and on other hand controlling of vitiation of doshas. Bala that is fully and securely supports the body and is the fore most important in the human beings, which help a person to keep healthy and to get rid of the disease. Therefore, Bala is only one of the weapons in the basic struggle of life and denotes the resistance that an organism offers against aggression by a parasite. Bala is further defined as the capacity of the tissue to grow and the capacity to with stand physical hardship and stress.

A) INSTANCES OF BALA IN CHARAKA SAMHITA

In Charaka Samhita an exhaustive discussion has been made regarding the word Bala, it has mentioned that maintenance of health depends upon entirely on Bala. When Sleshma is in Prakruta avastha it is responsible for Bala, but when it is Vikruta avastha then it considered as mala. In other words, the normal kapha is said to be ojus while abnormal one is leads to causes of various disease.[64]

He has mainly concentrated on prakrut kapha, vyadhikshmatwa along with some factors like Bala pramana, Dashavidhapariksha, Adana and Visarga kala, and relation with Agni, Dehagni, Vayu and Tridoshas. While discussing these points bala was discussed either directly or indirectly. Bala is the skill to do any activity and this has been said that Bala is the matter of anumanapramana, which is guessed through the capacity of an individual to do exercise. So Chakrapani stated that Bala is also termed as Kriyasamarthya.[65]

1. BALA PRAMANA

In every human being, Bala is very important and is mandatory to assess the rogi Bala for the management of diseases.[66] Examination of the patient reveals the knowledge of the span of life or the degree of strength and morbidity. Hence the patient should be examined in the respect of 1) Prakrutitah 2) Vikrutitah 3) Saratah 4) Samhanatah 5) Pramanatah 6) Satmyatah 7) Sattwatah 8) Aharasaktitah 9) Vyayamasaktitah 10) Vayatah [67]

Strength is very important in every human being to lead the life. Charaka[68]has explained ten types of pramanas, are very important to assess the rogi bala for the better management of diseases. By dashavidha pareekshya, the immunity can be assessed & ascertained.These factors would reveal the relative strength of the patient to withstand or oppose the disease process. Hence prakruti, Vikruti, Sara, Samhanana, Pramana, Satmya, Satwa, Ahara, Vyayama, Vaya are considered as the factors helpful for assessing the Bala[69]

1) PRAKRITI

The foetus is determined by the nature of sperm and ovum in the uterus. Dosha one or more than one, which predominates in these factors get attached to the foetus. This is

known as Doshapakruti and it is emerged from the initial stage of foetus. Hence, some persons are constitutionally Sleshmala, Pittala, and Vatala prakruti.[70]

a) Slesma - It is snigdha, slakshana, mrudu, madhura, sara, sandras, manda, sthimita, guru, shita, picchila and swacha. Because of these qualities the sleshmala persons are strong, wealthy, learned, brave, calm and long-lived.

b) Pitta - It is ushana, tikshna, drava, vishra, amla, and katu. Because of presence of these qualities the persons having predominance of pitta are moderate in strength, life-span, knowledge, understanding, means and wealth.

c) Vata - It is ruksha, laghu, chala, bahu, sighra, shita, parusha, vishada. Those having predominance of vata have mostly low degree of strength, life-span, progeny, means and wealth.

2) VIKRUTI

In respect of vikrti as well vikrti is vikara (disorder). It should be examined in terms of the nidana, dosha, dushya, prakriti, desha, kala, and Balalakshanas, because the severity of disease can not be known without knowing the strength of nidana, dosha, dushya and etc. The disease having Bala similar to that of dusya, prakruti, desha, and kala along with great strength of nidhan and severity of lakshanas is taken as severe.[71]

3) SARA

There are eight types of Sara, which are described for the knowledge of the degree of strength such as the types of Sara relating to each of Twak, Rakta, Mamsa, Medas, Asthi, Majja, Sukra and Sattwa.[72]

4) SAMHANANA

Samhanana, Samhati and Samyojana are its synonyms. Evenly well-demarcated bones, well-bound joints, well-formed muscles are known as compact body. Who having well compact body they are strong and having good strength[73]

5) PRAMANA

The pramana of the body (anthropometrics) will be described by the pramana of individual fingers in terms of height, breadth and length respectively. The persons having normal measurement of the body are endowed with ayu, Bala, ojus, aishwarya and other desired qualities. Those having body with less or more measurement have qualities contrary to these. [74]

6) SATMYA

Satmya is that which being used constantly has wholesome effect; those used to gruta, kshera, taila, mamsa rasa, and to all rasas are strong, enduring and long-lived. On the contrary, those used to rough diet and single rasa are often weak, un-enduring, and short-lived with a little means. Those having mixed suitability have medium strength **.**[75]

7) SATTWA

Sattwa is known as mind. It controls the body by conjunction with the self. Sattwa is of three types according to strength uttama, madhyama and heena. Accordingly the persons are also (of three types) having superior, medium and inferior sattwa. Amongst them, those having superior sattwa are in fact sttwasara, and have been described in context of saras. Those having medium sattwa sustain themselves at the instance of other. But those possessing inferior sattwa can sustain neither by themselves nor by others. [76]

8) AHARASAKTI

This is examined by the power of ingestion as well as digestion. Strength and life depend on diet. [77]

9) VYAYAMASAKTI

The power of exercise should be examined by the capacity for work. The three types of strength are inferred from the capacity for work**.** [78]

10) VAYA

Age is defined as the state of body corresponding to the length of time. Age is broadly divided into three stages baala, madhya, and jirna. Childhood is determined up to sixteen

years when the dhatus are immature, sexual characters are not manifested; the body is delicate, unenduring with incomplete strength The middle age is characterized by strength, energy, virility, prowess, acquisition, retention, recollection, speech, understanding and qualities of all dhatus having reached the normal limit; with proper physical and mental strength, without degeneration in qualities of dhatus, The old age is characterized by strength, organs, energy, virility, prowess, acquisition, retention, recollection, speech and understanding gradually degenerate, one should determine the three division of age on the bases of strength of the factors like prakrati etc. (except vikruti) and also character of different periods of life span.[79]

2. IMPORTANCE OF BALA IN DASAVIDHA PARIKSHA

The Bala is classified on the bases of working capacity of an individual and is of three types. [80]

1**) Pravara Bala:** Pravara Bala in which the person is capable of performing all types of physical and mental activity and never feels tired very easily.

2) Madhyma Bala: The Bala that come in between the pravar and avara Bala is known as madhyma Bala.

3) Avara Bala: It is quite opposite to the pravara Bala; those persons who possess such type of Bala become tired just after a slightest physical and mental activity.

3. BALA IN ADANA AND VISARGA KALA :

In the beginning of Visarga kala and the end of Adana kala weakness prevails in human beings. In the middle of the both kalas strength becomes moderate, at the end of the Visarga kala and the beginning of the Adana kala human beings get considerable amount of strength.[81]

4. BALA IN RELATION WITH AGNI:

Bala, arogya, ayu, and prana depend upon the agni. When the agni is supplied with proper fuel in the form of food and drinks, it is sustained and bestows Bala to the body.[82] Hence Bala is dependent on agni and its proper function. Such type of agni helps in bringing about the Bala, varna, sukha and ayu.[83] Bala, varna, swasthya, utsaha, upachaya, tejas, ayu etc are

dependent on agni and its proper functions [84]. So as long as agni is normal the person can have a healthy and long life. Loss of agni leads to loss of life. If this agni becomes vikruta the person becomes rogi. The dosha, dhatu and mala are maintained normally by this agni. Among 13 types of agni, pachakagni is pradana and all the other agnis are dependent on this pachakagni. Agni is also attributed for the production of two kinds of strength, Strength to resist the occurence of disease Strength to perform the physical exercise It is stated in Ayurveda that, all the internal diseases are caused by the vitiation of agni. Hence, it is said that treating the vitiated agni it- self is the chikitsa. Thus, it is one of the ten factors that are required to be examined before initiating the treatment and itself is responsible for the increase of strength [85]

IMPORTANCE OF VAYU

In vatavyadhi chikitsadhyaya the vayu has been taken as Bala. Acharya Charaka[86] has described that; vata constitutes every life of the living beings. It has been stated that, the vata is the foremost dosha which provides the life to the living organisms so, it is termed as prana.

Apart from this, Sushruta has said that, since the vayu provides ayu by means of transportation, movement of the dosha, dhatu and malas and by means of ucchwasa and nishwas. The statement of Bhela that life exists as long as vata lasts in the body and movement in the body is indicative of the presence of life. In the same way, Vagbhatas statement regarding the function of vata dosha shows its importance that 1) It controls over the function of the body 2) Swift action 3) Strength 4) Capacity to vitiate other doshas 5) Independent movement and 6) Large number of diseases caused by vitiation.

5. BALA IN RELATION TO VYADHIKSHMATVA:

The Bala or strength of the person helps him to counter the disease by decreasing the strength of the disease. Rogotpatti in shareer and its Pratibhandhak shakti is called as Vyadhikshmatwa. When the etiological factors come in to contact with the body they try to produce the diseases. At the same time the body tries to resist against the diseases. The power of the body which prevents the development of diseases or resists developed diseases is called Vyadhikshamatwa.[87] The human body has the ability to resist almost all

types of organism or toxins that damage the tissues and organs (leading to the causation of disease) this capacity is called as immunity.

Charak states that not all prakritis are equally capable of Vyadhikshamatwa (capable to resist diseases). Since long it is a matter of observations that in a given mass of population under same etiological and environmental condition, one person remain healthy while the other becomes sick. All people are not same in their response to strain and stress and disease producing factors. This quality of the body is confined by Charak to "Kshmatwa" he had also classified eight kinds of personalities due to the reason they are considered possessing less kshmatwa and are always susceptible to various kinds of diseases.[88] Vyadhikshamatva according to our Acharyas has been considered as the power of resistance or tolerance towards the diseases. Hence the importance of psychosomatic health as well as good social religious and spiritual behavior which lead to the healthy and disease free society.

Vyadhikshamatva is made up of two words vyadhi and kshamatwa. Vyadhi is the disturbance of body elements and kshamatva is the ability or capability to resist the disease. There are so many etiological factors which try to produce the diseases but the body try to resist it. The power of the body, which prevents the development of disease or resists the development of disease, is called Vyadhikshamatva.Here two significant terms, vyadhibala virodhitwa and vyadhiutpada pratibandhakatwa have been used in a particular order The vyadhi balavirodhitwa which is antagonistic to the strength and virulence of diseases. It is considered with the action after the disease has manifested in the body.The vyadhiutpadana pratibandhakatwa is the capacity to inhibit certain or bind the cause or factors of disease. It is commonly observed that, certain disease do not develop even after coming into context with causative factors while others become victims of the diseases. The reason is that when the resistive power of the body is sufficiently strong it destroys the causes if it is weak then person become victim to the disease. Bala is the factor that controls the dosha and Bala is that whichresist the disease. Bala is the power to perform the work which depends on the musculature, conferring karma sadhanashakti on body opposition. Bala as potential source of resistance to disease and decay represented by the apara or shleshmika ojus. Then the commentary Chakradatta is describe that the ojus has the function of rakshana of the body i.e. the protection, preservation, guarding etc indicating it

acts against the degeneration and decay this function is known as Vyadhikshamatva. Dalhana in his commentary states that the body strength i.e. abhyantara prana or Bala is derived from ojus hence; Vyadhikshamatva is also dependent on ojusbecause ojus is seat of Vyadhikshamatva. So it is observed that in disease accompanied by profuse dhatukshaya there is also diminution of Bala, ojus and Vyadhikshamatva.

Generally it is said that ojus and Vyadhikshamatva are same or vice versa but it seems to be the partial statement because, ojas is dravyathamaka with its pramana, lakshana, guna, where as vyadhikshmatwa is karmatmaka, anumana and yuktijanya. By knowing the function of ojus it can be considered as one of the factors relating to Vyadhikshamatva. This statement can be justified by increasing the Bala. Still the doubt remains regarding the relation between ojus and Vyadhikshamatva that the lakshanas of oja kshaya had been explained by our Acharyas where as there is no direct reference regarding the Vyadhikshamatva kshaya laxana hence; it can be interpreted that the vikruti in the ojus may lead to kshaya of Vyadhikshamatva.

6. BALA IN RELATION TO SLESHMA (KAPHA)

Sleshma is the cause of strength in the body. Bala also represents a potential source of resistance to disease and decay. This particular quality is represented by ojus which is treated as an essence of all dhatus. But Sleshma indicates the apara ojus the second variety which is a direct product of kapha. Food provides supply nutrient materials from which the bodybuilds up its structural and functional elements. The nutrient substances which are homologue both inside and outside the body contribute to dhatupushti. The substances which nourish both kapha and dhatus contribute to the pushti of slashmika ojus. In view of the qualities of kapha and slashmika ojus beingmadhura, guru, snigdha, pichila, sthira, manda etc. the dominant bhuta is pruthvi and apa. The most important point to be noted here is that jivaparamanus which are different in different dhatus contributes to the genesis of slashmika ojus which has the function of kapha, which is present in these jivaparamanu.

Therefore, when kapha located in jivaparamanu the slashimika ojus should be proteinious in nature. Bala is balakrit i.e. it confirms strength to the body. Bala is one of the synonyms of somatic factor. Bala is interpreted in two aspects Karmasadhana shakti OjusIn this context, Bala and ojus are synonym of kapha but it is slaishmika ojus which bestows

Vyadhikshamatva that is the power to resist and over come the force which brings about the disease and decay. But according to Dalhana, bala interpreted as karmasadhna shakti i.e. energy to perform work and a sign represent the sign of growth and is devoid of rupa, rasa and virya but ojus is an essence of all dhatu which are moortimata with rupa, rasa, virya etc. Therefore it may be surprised that Bala is energy and ojus is a substance. Karma sadana shakti – Bala identified as kamasadhana shakti is dependent on well grown muscular performance of work without opposition. Therefore it can be assumed that sthiropachit mamsat bestow kamasadhan shakti.

CONCEPT OF SAHAJA BALA

WORD MEANING OF SAHAJA

According to Sanskrit dictionary [89]meaning of Sahaja : along with indicator, born or produce together, congenital, innate, hereditary, original, natural by birth and by nature, naturally with desha, Birthplace, always the same as from the beginning. Among the trividha Bala Sahaja Bala is the most important one because of the fact that it comes right from the birth.

CLASSIFICATION OF BALA

Bala has mainly classified in to three types Sahaja, Kalaja, and Yuktikruta.[90] Constitutional strength is the one, which exists in the mind and body from the very birth. Temporal is the one which is based on the division of seasons and the age of the person. The acquired strength is the one which is achieved by the combination of diet and other regimen.

Sahaja Bala:

This is natural inherent capacity to oppose & with stand disease. It exists since birth, promotes the growth of the dhathus, independent of other causes. The sahaja bala also depends upon Kula (recital traits, jaathi (genus) & pratyatma (individual / species). In this resistance to disease is stated to be Prakrita (natural, inborn, genetic) and it exists from birth. It is known as Sahaja bala and increases with the growth of dhatus and does not depend up on any other cause (Chakrapanidatta) it comprehends both sharira and satwa

Among these 3 types of balas, Sahajabala is very important because it comes right from the birth. It represents the resistance power inherited from the parents to their offspring which is basically depended on the character of the shukra and shonita. This cannot be adopted or acquired after birth but can be improved or enhanced by proper conduct such as sadvruthpalana, dinacharya, rutucharya, ahara vihara, rasayana and vajeekanana therapies by mata and pita. Hence it is better to follow the proper conduct to avoid such disease in their offspring. By adopting these conduct the coming offspring will develop the Sahaja bala.

The resistance that is possessed by an individual from his birth onwards is called Sahajabala. The perfect condition of shukra and shonita of the parents and proper intake of healthy food during pregnancy by the mother makes the child healthy and good resistance is known as Sahajabala.

This type of Bala or resistance to disease is stated to be prakrita (natural inborn genetic) and it exists right from the birth. It is inherent in every individual to lesser or greater degree. It is said to increase with the genuine growth of the dhatu and does not depend upon any other cause. Some individuals are observed as physically strong from their birth where as some are observed as physically weak from the very birth.

Kalaja Bala:

This type of Bala is stated to be influenced by seasonal traits and the age of the person. Thus Bala is stated to be dissipated and at its lowest Bala in adanakala, corresponding to shishira, vasanta, and grishama rutus. On the other hand the Bala should be highest optimum level in visargkala corresponding to varsha, sharad and hemanth. Strength increases the stage of development, in the human beings. Regarding the vaya sampath it is termed as the storehouse of strength. It is the more appropriate to state that in the youth all the dhatus attain the perfection. So it is natural that each individual at this stage acquires more strength. As regard to Bala the child hood and old age come in the last descending order.

Yuktikruta bala

In this shareera bala refers to the induction of body resistance against diseases by resorting appropriate nutrition such as meat, ghee, etc. physical exercise, rest, restorative and rasayana therapies keeping with seasonal needs, and artificially produced by use of patent drugs, gems and etc.According to Chakrapanidatha the strength begins to develop by regular diet, rest and exercises but rasayan administration plays an important part in the development of the body resistance. Strength of the human being according the seasons [91]

3. BALAVRUDHIKARA BHAVAS

Ayurveda suggested some factors which are quite capable of enhancing the Bala. Along with this adetailed, explainational regarding Balavrudhikara bhavas has been explained in shrira sthana of charaka samhita. The formation of three types of Bala fully depends on many factors. Here Acharya Charak has attempted in pointing out these factors through balavrudhikabhavas.[92] 1.Balavat purushe desh janma 2.Balavat purushe kala janma 3.Kalayoga sukhata 4.Bija kshetra sampth 5.Ahara sampath 6.Sharer sampath 7.Satmya sampath 8.Sattva sampath 9. Swabhava samsidhi 10.Yavana avastha 11.Karma sampat 12.Samharsha

Formation of these three types of Balas fully depends on these factors. Among 13 factors some are helpful towards the Sahajabala and some are helpful towards kalaja and yuktikrut Bala.

BALAVAT PURUSHE DESHE JANMA:

Acharya Charak has mentioned in the begining that an individual born in a desa where the strong persons are born would also be strong (balavat purushe desa janma.) By this statement the he wanted to stress on the fact that the desh has got an impact or influence over the physical strength of an individual. Acharya had mentioned three types of desa are Jangala, Anupa, and Sadharana. In kalpasthana of Charaka Samhita a detailed description of the characteristic of these deshas in terms of the condition of the soil, the status of water, the air, the persons, the dominating doshas and over the above in terms of the Bala of the inhabitant of that particular desha has been illustrated.[93] Commenting on this statement

Chakrapani says on account of specific characteristics of desha the inhabitants of that desha acquire a specific shareerbala.

Regarding jangala desha it's having the soil dominated by sikata, sharkara etc and very hard land and big and small hills are found. In such places the land for cultivation would be very less and yield also would be less. In this desha vata is more predominant so the person who born in this desha will be having comparatively less shareerabala.

Regarding the anupadesha, it contains a soft and fertile land the crops can be had at least twice a year due to sufficient water resources. Here the materials we get green vegetables which are high in proteins so kapha is more predominant in this desha hence, the person who born in this desha is having more shareerabala.

Regarding the sadharana desha it would have the land by mixed type. Somewhere the crops would be good where as some parts the eatables and water will be less, so the person who born in this desha is having mixed types of shareera bala i.e. uttama as well as heena bala.

BALAVAT PURUSHE KALA JANMA:

The second factor responsible for the Balavriddhi has been described as Balavat purush Kala janma that mean that a man should be born in such a time when only the strong men are borne. According to the second ideology the birth, time and the Bala have been said to be interrelated in Ayurveda the time factors has been described with different angles.This concept has been also explained based on adana and visarga kala. Based on this concept individuals born in the visarga kala would be strong, stout and sturdy, and the person born is the adana kala would be weak, lean, and thin.[94]

This statement can be explained in two ways

According to the first view, those persons would be strong enough who are born at a time when strong and stout persons are born. So clarity this concept that where the prehistoric persons like Rama, Bheemasena, Arjuna etc were born that time the kala is more important for getting super strength. This statement may be correct but it may not be an all time. The authors accepted that the time factor is a non stable entity and is every changing.

According to the changes in time and environment would also change the physical strength of an individual. So we cannot expect a person of 21st century to be physically strong just similar to Bheemasen who has the physical strength equal to the 60,000 Elephants.

According to the second ideology the birth time and Bala have been said to be interrelated. While commenting upon the statement of Charak the balavat purusha kalajanma Acharya Chakrapani has clarified that in this context kala has to be accepted in term of hemanth, shishira, etc season and a person born in these hemant and shishiva seasons would be sufficiently strong in comparison to the person born in other seasons. In this connection Chakrapani has stated that this strong ness of the body is related with the prashasta parinama of kala. Based on this concept individuals born in the visarga kala would be strong, stout and sturdy and the person born in the adnakala would be weak, lean and thin.

KALAYOGA SUKHATA:

Ayurveda considered kala to have an influence on the bala of an individual; there are mainly three bad association of kala that decrease the bala. They are, 1.Heena 2) Mithya and 3) Atiyoga of kala. If the seasons are absolutely normal they are considered as the saviors of the individual as well as the bala of human beings.

As already been stated that kala is divided into different rutus. The Bala gets increased in different states of kala. In each kala a specific type of atmosphere which is supposed to be natural. This naturality is disturbed then it is not called as sukhakala, this is termed as heena, mithya, and atiyoga of kala. With this ideology, it has been postulated that if the seasonal status is absolutely normal and person born during such kalayoga sukhata they are supposed to possess the normal status of strength. Otherwise condition would be lacking in the physical strength.

BEEJA KSHETRA GUNA SAMPAT:

The Beeja sampat and the Kshetra sampat have been also considered as the factors responsible for Bala vridhi of an individual.In connection with the concept of procreation almost all authorities have postulated that for conception rutu, ambu, kshetra, and beeja are the essential factors.66 rutu, kshetsa, ect are the technical terms used in Ayrveda, and

are analogue to the phenomenon of cultivation and its components. In the same way, if the shukra or pumbeeja is a purest, when it embedded in the healthiest garbhashaya at an appropriate time or in the proper rutu kala as well nourished by the finest ahara rasa of the mother. The conception takes place normally and the progeny taken place proper time and in healthy state.In almost all the Ayrurvedhic text the general properties and normal characteristics of shukra and arthva has been mentioned, at the same time 8 dosha of shukra and arthra along with their clinical features and treatment have been also described. So that if an individual whether the male partner or the female partner is suffering of any abnormality then the coming offspring will suffer from many diseases along with loss of strength. If it is treated before indulging in the phenomenon of conception then coming offspring have good strength.Kshetra is the Garbhashaya and to have healthy and strength progeny.

The Garbhashaya should be of normal size and free from all the types of diseases. The normalcy of the garbhashaya is termed as kshetra sampat.The superior quality of the ahara rasa of the mother which is utilized for the fetal nourishment may be considered as the ambu sampat. Regarding the beeja, Charak has particularly stated that for a healthy and strong progeny, the beeja must be free from the Beejadushti, Beejabhaga dushti, and Beejabhaga avayava dushti. In connection with the beeja sampat and the kshetra sampat Chakrapani has considerd only shukra from term beeja and kshetra as arthava and Garbhashaya. On the basis of above description it is deducted that if there is a beeja sampat and a kshetra sampat aided by the rutu sampat and ambu sampat the offspring would be healthy and strong. In al-most all texts, the general properties and normal characteristics of shukra and shonita are mentioned. If these are in normal state then coming offspring may be having good natural immunity. Regarding beeja sampat Charaka particularly stated that for a healthy and strong progeny the beeja must be free from the beejadushti, beejabhaga dushti and beejabhaga avayava dushti. Kshetra is the garbhashaya and to have healthy and strong progeny the garbha should be of normal size and free from all the types of diseases.

AHARA SAMPAT:

The next Balarvadhikara bhava informed by Charak is in term of Ahara sampat. Here the term Ahara refers to the substance ingested for the purpose of strength. Food sustains

the life of living beings. All living beings in the universe require food and it gives complexion, clarity, good voice, longevity, geniusness, happiness, satisfaction, nourishment, strength and intelligence. This ahara should be consumed for the purpose of shareer vruddi, but this shareervruddhi embodies various other factors also and one of them is the production of strength, vigor, vitality in the individuals. In the context of the evaluation, in taittireeyo Upanishad it is discussed that the process of the creation of universe after formation of earth the aushadhi was evolved. Form aushadhi the anna was evolved, and from anna the purusha or the living organism was evolved. Thus purusha has been considered as to be formed from anna and rasa. Food which is one among the Upasthambha has a prominent role in Bala Vrudhi. Charak has stated that the life of all the living things it food and it gives complexion, good voice, long life, happiness, satisfaction, growth, strength and intelligence. Therefore the Ahara sampat should always be maintained to maintain the bala vitality energy etc, by following diet rules. Eight aspect of dietetics, Dietetic regulation, measured diet, Diet at proper time, wholesome diet.

SHAREERA SAMPAT:

Shareera sampat is the next factor included under the Bala vrudhikara Bhavas by Charak. The term Shareer sampat refers to a condition is which, the sharer is observed as gifted by its virtues. While commenting upon this particular sutra, almost all the commentators have maintained a silence on this statement, but Kaviraj Gangadhar has explained the term shareer sampat as the sadgunya of the shareer gunas. 69 Charak has enumerated certain qualities that termed as sharer “sankhya samarthyakara gunas”. Which has been described as the gurvadayah and they are 20 in number, Charak has explained the phenomenon of shareer samkhy samarthya vriddhi by applying the theory of samanya and vishesha siddanth. According to this statement similar gunas are responsible for increases the shareera and dissimilar guns are decreasing the shareera.In this way charak has mentioned those in whom all the element are in perfect tone are very strong endured with very happy circumstances, able to trouble self confident in all enterprises, given to good pursuits of firm and well kit bodies, deep and big voice, and are passed of happiness, power strength, wealth, honor.They are slow in ageing and slow in being attacked by the diseases, and have offspring of similar qualities in great number and long lived.

SATMYA SAMPAT:

In this context, the term satmya stands for homologation that has become agreeable to a person by constant use. In connection with the concept of homologation Dalhan acharya has given some more elucidating account. He has mentioned the satmya is of various types such a dosha satmya, desha satmya, jagarana, vyayama, kala, rasa, etc all these types of satmya are very important for the enhancement of Bala. If parents are satmya to all these types of homologues then the offspring will have all satmya qualifies to resists the diseases and also have good and sufficient Bala.

FACTORS RESPONSIBLE FOR ENHANCING THE SAHAJA BALA

There are so many factors which are responsible for the enhancing the Sahaja bala. Among these foremost importances given to the ojus because, ojus is produced first in the body of the living beings and it helps to foetus for growth and stability. Without ojus the foetus will not stay and grow in the uterus. Suppose the foetus will grow without ojus definitely there is a lack of Sahaja bala in that child.

INTERPRETATION OF SLESHMA (KAPHA) AS SAHAJA BALA

This can be attempted in two ways – one on the grand of its functional (karma) ability, secondly, its dravyatmak (quantity). Considering latter one the total quantity of Kapha in a human being is 6 anjali, but part of it about ½ anjali is Sleshmika oja which is a purest part of Kapha which in the literature of Charak says sleshmika oja refers to apara ojas. By the above discussion ojus has been established by two factors for Sahaja bala i.e. dravyatmak and karmatmak. Besides which the function of kapha are also seen in the growing foetus such as kalala, alingana, vrudhi of different angas etc. are because of dravyatmak of kapha, there by the karmatmaka property attributed in month wise growing foetus can be establish as the function of Sahajabala pertaining to the kapha.

INTERPRETATION OF SHUKRA AS SAHAJA BALA

Through Ayurvedic, literature supports Shukra in the following headings As one among the sapth dhatus, Which help for the garbha dharana With regard to the garbhotpadaka bhavas both the Shukra and shonita unites and form the foetus. So formed

foetus will determine the next progeny for particular race. So formed each race will posses its own Bala to resists diseases having a drawback of repeating the disorders too which are formed as hereditable disorders because of beejabhag, beejabhaga avayaya and beejabhaga dushti. By the factors of gene transmigration from the parents in the form of haploid chromosomes to a diploid Zygote is brought up by the factor Shukra through its shukra beejabhaga is clearly explained in Sushruta Samhita. Hence the shukra can be taken for Sahajabala. This Sahajabala carries immunity but also hereditary transmitted disorders.

INTERPRETATION OF KALA AS A SAHAJA BALA

In classics, Kala is divided into adana kala and visarga kala and here stated that the person born in visarga kala will naturally bestowed with uttamabala and those born in adanakala have less bala. This can be substantiated that a part of matruja ahara is provided to the growing foetus for its nourishment; hence the food materials which is consumed in visargakala will naturally have madhura, lavana, apya dravyas so it enhancing the ojus like gunas resulting in uttamabala. Nevertheless, this point in the present era needs a survey base study to look over; however, literary evidence supports the concepts. The above factor that kala has a constant influence on Sahajabala which is seen in the time of birth only on the literary ground.

INTERPRETATION OF BALAVRUDHIKAR BHAVAS AS SAHAJ BALA

13 factors are explained in classics they can be classified under Sahajabala, Kalajabala, and Yuktikruta bala. In particular Sahajabala the balavat puresh desha janma, balavat purushe kalajanma, beejakshetra sampat, are influence for the Sahajabala in the child. All these factors are naturally in their healthy form, which is the cause for the natural strength.

INTERPRETATION OF DESHA AS SAHAJABALA

According to Charaka three desha are explained mainly anupa, sadharana, and jangaldesha possess sukumar, uttama, and sadharana bala respectively. This concept can be evenly applied to the Sahajabala on the basis of immune mechanism of foetal life. The immunoglobulin (IgG) which is present in the pregnant woman passes through placental barrier and protects the foetus throughout its intra uterine life.

IgG is active in early life and protect the new born baby up to stimulation of T and B lymphocytes with response to providing the environmental condition. Hence the environmental condition known as Desha.

CORRELATION OF SAHAJABALA AND INNATE IMMUNE SYSTEM

The word Sahajabala specifies the strength of naturally occurring mechanism in protecting the new born child. When in the innate immune system is defined as, to resist the infection is a natural gift and is seen in all forms of life. This natural non susceptibility to infection is called as innate/ natural immunity.

Having dealt the detail interpretation of Sahaja bala earlier under specific headings such as ojas, kapha, shukra, kala, etc appears that Acharyas indirectly refers to the inbuilt immune system that present naturally in our body protecting in the most sensitive age of the early life. So the correlation that exhibits the Sahaja bala for a modern innate immune system in the specific areas like immunoglobulin, cytoplasm, genetics etc which are part of protecting, nourishing and transmigrating of genacy to the new offspring. Here I made an attempt to correlate of Sahajabala and innate system.

SHUKRA AS INNATE IMMUNE SYSTEM

According to modern science, fertilization is the process of fusion of the spermatozoa with the matured ovum. It begins with sperm egg caelision and ends with production of a mononucleated single cell called as zygote. Its main objects are 1) To initiate the embryonic developed of egg and 2) To restore chromosome number of the species. The same explanation that is explained by Sushruta might be correlated with the modern theory that combination of shukra and sonata leads to a garbha and its objectives are 1) Garbha dharana 2) To garbha vrudhi

CONTACT AND FUSION OF THE GAMETES

Growth or regeneration is the characteristics of life, growth occur through division of cells and their subsequent enlargement. This division of cell is known as mitosis. In the human body the head and tail of the spermatozoa enter the cytoplasm of the occytes but plasma membrane is left behind on the occyte surface head and neck of the spermatozoa

become male pronucleus containing haploid number of chromosomes (23 X) or (23 Y). The male and female pronnelie units at the center with restoration of the diploid number of chromosomes (46) which is constant for the species. The zygote then formed, contains both paternal and maternal genetic materials. In some instances an antigen called fertilizen present on the cortex and its coat of the ovum, reacts with the antibody called antifertilizer liberated at the plasma membrane of the sperm head. Then the union between the two gametes may be affected by an immunological reaction. But according our Acharyas they foresighted on Beeja for getting the Sahajabala by some factors like rutu, ambu, kshetra, beeja along with the matrujadi shadbhavas are responsible for the Sahajabala in coming offspring. Among these factors Beeja plays an important role for natural immunity and this can be correlated with the paternal and maternal genetic materials (chromosomes) of modern science.

OJAS AS INNATE IMMUNE SYSTEM

According to Charak ojus is a first product in the shareer and it is responsible for the Sahajabala in coming offspring. Ojus plays a vital role in defense mechanism of the body or body resistance, which is originated from the birth. According to Chakrapani after the union of sperm and ovum, ojus enters in the body of the embryo and it provides nutrition, resistance to the organism and manifests the cardiac activity. Thus in three different stages of the foetus ojus play an important role, At the time of conception it is essence of shukra and shonita In the second stage it is the rasa sara which provides nutrition to the embryo In the third stage when there is formation of various organs ojus manifest its own action Hence ojus originates in the first month and helps for development and leading the life.

According to the modern science the ojus can be correlated with the cytoplasm which is present in the sperm and ovum which form the zygote. Its main function is embryonic development after the formation of zygote, a typical mitotic division of segmentation leads to produce two blastomeres. Each contains equal cytoplasmic volume and chromosomes number. The blastomeres continue to divide by binary division through 4,8,16 cells stage until a cluster of cell is formed and is called morula. The central cell of the morula is known as inner cell mass which forms the embryo in proper way and peripheral cells called outer cell mass which will form protective and nutritive membrane of the embryo.Ojus plays a

defensive mechanism in the foetus which can be correlated with the immunoglobulin because immune function in the foetus and newborn are physiologically competent at birth Various types of T cell are identified as early as 7-8 weeks of infantine life. Lymphocytes infiltrate into thymus by 8-9 weeks, thymus is matured by 12 weeks. B cell is detectable by 13 weeks and major classes of immunoglobulin can be formed by 20 weeks of gestation.

Above discussion or correlation of ojus and cytoplasm along with immunoglobulin are seems to same by its origin and function.

Asthira astame oja is noticed in 8th month of pregnancy. As similar reference is available in modern context of immune complex in that among five immunoglobulin the IgG immunoglobulin is only capable for transforming across the placental barrier reaching the fetus. Hence, it provides the positive immunity to the fetus and early neonate life.

SLESHMA (KAPHA) AS INNATE IMMUNE SYSTEM

Kapha is balakrit i.e. it confers strength to the body. Bala referred to the essence of all dhatus is ojus. With reference to the function of kapha this indicates the apara / slasihmika oja which is ½ anjali pramana. In this context, the slaishmik oja/ apara ojas that bestows Vyadhikshamatva shakti i.e. the power to resist and overcome the forces or factors which brings about disease and decay. All organs and parts of the body are made up of their ultimate units of jivaparamanu. These innumerable jivaparamanu are needed for developing the different structure and organs of the body. The kapha has some qualities i.e. snigdha, picchila, satva, sthira etc which are helpful for the development of the shareera and also maintain the shape of the organ/ structures. In addition, to avoid the sithilata of that organ the bandhan should be stable because of this kapha.

According to modern science the intracellular substance or protoplasm that can be correlate with the kapha because, the intracellular substance in the tissues performs the similar function. The main function of the protoplasm is through the intercellular substance to pass the nutrient material from the capillaries to cell and the metabolic waste passing in the reverse direction, therefore it is very important for the preservation of health. Interference with its function of transport leads to depression of cellular respiration and nourishment with result necrosis. It forms a medium or an obstacle to the passage of invading

microorganism.Protoplasm contributed in modern physiology including biochemistry as detailed below:

1) Apadhatu is dominant in both substances. The pruthvi bhuta or solid play only a secondary role, through signification of protoplasm and this fact agrees with the definition of kapha i.e “kena jalena phalati iti kapha”. The water content of different tissues (dhatus) is different, the maximum of 90 to 92 per cent in rasadhatu and the minimum of about 16 per cent in medodhatu

2) Both sleshma and protoplasm embrace or keep together the components of the entire body or of the jivaparamanu in keeping with the definition of the former slish alingane.

3) The description of the physical characteristics of kapha suklatva, madhura and lavana tastes, sita, snigdhatva, mrudu, slakshna, sandra, guru, sthira, picchila, manda likewise represent thephysical properties of the protoplasm.

a) Suklatva and acchatva refer to the whitishness and transparency respectively of kapha. The protoplasm is translucent and colourless, but to the presence of the colloid and the gel state, it may appear whitish.

b) Madhura and lavana tastes are also exhibited by the protoplasm.

c) Sitaguna is attributed due to the more dominant presence of apadhatu. This guna may be interpreted as the cool, touch and conservancy of energy. Even through a lot of heat is produced by the chemical reaction in the body by the action of pitta, The kapha maintains the sitaguna due to the higher latent heat of vaporization of the watery element.

d) The snigdhatva of kapha corresponds to the viscosity of protoplasm. The quality is due to the presence of fat protein complexes and lipids in a collodial solution.

e) Slakshnatva refers to the smoothness, a characteristic of both kapha and protoplasm. This quality is due to the colloidal nature of the soutes and the gel state of the protoplasm.

f) Sandratva refers to the density of kapha. This quality along with the gurutva and stiratava lakshanas are conferred by the gel state of kapha due to the preference of the constituents with high molecular weight like glycoprotein and fat protein complexes which reflect the

physical- chemical qualities/ properties of pruthvi and apabhutas. These constitutes resist the degeneration and decay.

g) Picchilatva has referred to the sliminess of the kapha, also a characteristic of protoplasm due to the dissolved protein complexes of high molecular weight

4) kapha, like protoplasm is the basic matter stuff of all dhatus or tissues or cells, even through there may be some difference in character, qualities and function due to specialized function of each respective dhatu. These differences are due to differentiation of cells during their development. In general, the physical and chemical characteristics of kapha appear to be similar to protoplasm. After going through a correlation of sahaja bala and innate immune system I come to know that some of the Ayurvedic concept like shukrashonita, shleshma, matrujadi shadbhavas can be correlated with some modern theory of chromosome, cytoplasm, immunoglobulin, protoplasm, in particular natural immunity system. So it appears that fore sights of our Acharyas in particular Sahajabala which was explained 5000 years ago are some what relating to the innate immune system at present era.

Thus it can be said that

1. Bala plays an important role in the physical activity.
2. Bala cannot be correlated to any single specific factor because; it can be interrelated with many of the factors such as Ojus, Prakrutha sleshma, Shukra, Agni, Vyadhikshamatwa, etc.
3. Apara ojus will be directly proportional to the Bala of the individual to combat against the diseases.
4. Variation in the environmental condition plays an influential role on the body resistance.
5. Bala can be assessed on the bases of Balapramana.
6. Normal characteristic of Ojus, Shukra, and Shleshma are responsible for Sahaja Bala.
7. The normal state of Garbhodpadaka bhavas and proper functioning of Garbhavrudhika bhavas are contributes for Sahaja bala.

8. By adopting proper Garbhiniparicharya is also an important factor for Sahaja bala.

9. Sahaja bala can be interpreted as Ojus, Prakruta shlesha, Shukra, etc on the bases of their dravya, guna, and karma.

10. The Sahaja bala can be correlated with innate immune system in the specific areas like, cytoplasm, immunoglobulin, chromosome and protoplasm.

02.4 CRITICAL ANALYSIS OF VYADHIKSHAMATWA

When etiological factors are exposed to the body, they try to produce disease. At the same time the body tries to resist the disease. This power of the body, which prevents the development of diseases, is called Vyadhikshamatwa.[95] It is commonly observed that certain persons do not develop a disease even after coming into contact with the relevant etiological factors, while others become victims of the disease. The reason is that, when the resistive power of the body is sufficiently strong it destroys the causes. A beautiful simile is used in manusmruti and mahabharata to illustrate this fact when a seed is sown in non – fertile soil, it is destroyed. The fire which fell in an environment devoid of grass would naturally get extinguished.

SYNONYMS

According to Sushruta, Praana & Bala, Ojus is the seat of Vyadhikshamatwa.

TYPES

It is classified with a two different perspectives as follows

1. Based on vyaayaam shakthi. (Physical strength)

2. Based on bala

TABLE NO 1: CHARAK EXPLAINS concept of ACTIVE & PASSIVE immunity,

TATHA VIDHA DRAVYA	**TAD VIRODHINA DRAVYA**
It is simulative therapy	It is restorative therapy
Treating with similar	Treating with contrast measures
It is like impletion therapy	It is inducing antibodies
It is an active process	It is a passive process
Ex. Antigens.	**Ex. Antibodies**

This give rise to the concept of ACTIVE & PASSIVE immunity. Charka narrates this in the context of treatment of diseases that produced by viruddha- ahara factors. He mentioned TAHAVIDHA DRAVYA & TAD VIRODHINA DRAVYA. [96]

MECHANISM OF VYADHIKSHAMATWA (IMMUNITY)

It deals with methods to improve the qualities & properties of vyadhikshamatwa & the mechanism which explains the interaction of body with the etiology. Charka says the diseases which are likely to be caused by viruddha-aahar can be encountered by SHODHAN & in the form of antidotes OR by using similar etiological causes as prophylactic basis so that it seldom affects the person but supports his immunity . The prophylaxis here refers to the habitual use of unwholesome foods/viruddha ahara in meager quantity, provided the person has strong agni, strong young body /taruna), an oleated body/snigdha & an acquired good physique, out of physical exercises.[97] So the person possessing these contributing factors would never be affected easily & be victimized to minute alteration & deviation from purity of food or such etiological factors. Chances of becoming ill are least.

CHARAKA postulates two more concepts, which find their application in aiding Explanation to the mechanism.

1 .Theory Of Natural Homeostsis (Swabhaavo Param vaada).[98]

The disturbed equilibrium of dhatus is the result of disturbed etiology. There is a self limiting factor which controls spontaneously. The principle appears to be operating automatically to fade away the diseases & restore the health soon after they are manifested. This is referred to as natural homeostasis (SWABHAAVA). The impairments perish by themselves as the time advances. The time factors (kaal) to accomplish natural homeostasis may be minimized by vyadhikshamatwa.

2, Theory Of Homologation (saathmikarana).[99]

The seasonal homologation related to diet & regime; even injurious diet & regime may become non-injurious to the body when they are habituated by regular use. They probably tend to develop & establish an intimacy with the body components by nature .This is referred to as Oak Saathmya. The vyadhikshamatwa can be recognized by the following facts.

1. The factor that bring back any deviation from normal hygiene, does not affect adversely, when mithya-aahaar vihaar are carried out.

2. Though there are visible etiological factors & abnormal interaction of dacha & dhatu (dosha-dooshya sammoorchana) appears to be operating to bring about diseases, the strong immunity may resist & may not permit the disease to manifest at all.

3. The nidaan & vyadhikshamatwa are the two relative deciding factors responsible for incidence or suppression, mildness or severity & acuteness or chronicity of diseases.

4. Sometimes patients get over the diseases without any remedy. This signifies the role of self limiting principle of the disease or the immunity of the person.

IMMUNE-MECHANISM.

Charka says –When agents injurious to the tissue elements/**deha dhatuhu pratyanika bhootani dravyani** enter the body, they are bound to face the opposing force/**virodham** from the tissue-elements / **deha dhatuhu** there; the offence & defence mechanism thus come into play . It means that the stronger would overcome the weaker/**alpam avajeeyate.**[100]Here prathyanika bhoota means – an armed enemy who is ready to fight (antibodies)[101] **Vaagbhata says** –when a person gets modified by virtue of opposing powers

(to fight against) to morbid doshas , poisons(toxic factors) or antigens ; he / she is enabled to resist those factors in his future life , if he / she were to come across them abruptly.[102]

Location of vyadhikshamavatva

Bhela- enumerates Ojus to have 12 entities. They are saptha saar (7), Thrimala (3) , pittha & jaleeyasraava; body secretions such as – saliva, lacrimal secretion , nasal secretion , sebaceous secretion, bronchial secretion , urethral secretion vaginal , seminal , intestinal secretions &sweat are said to contain LYSOZYME , which have some bacteriolytic properties physically , the Ojus has 4 forms [103]

1. Dhaatu tejo roopa (par-Ojus) --- It is present in tissue element . It imparts energy to fortify their strength.

2. Jeevitha shonitha roopa Ojus. --- It is the blood embodied with life, the living leucocytes (WBC). They help to phagocytose (cell eating) the living micro-organisms & hence form the immune mechanism.

3. Rasaathmaka Ojus – The defending property of the body fluids & secretions being drained in the Dashamoola siraa, throughout the body is included under this category.

Ablastine present in serum is said to be bacteriostatic. A& B- Lysine is said to contain bactericidal principle & sensitive against gram +ve & gram –ve organisms. Serum-lipids seemto possess & exhibit viricidal property.

4. Shleshma roopa Ojus -The physiological Kapha is the source of energy, whereas the pathological Kapha becomes the Mala. The albumin, globulin etc present in blood plasma are said to play an important role in body defence mechanism.

02.5 IMMUNE SYSTEM

DEFINITON

Immunity is the capacity of an individual to resist an infection that is invasion, multiplication and protection of a disease by an organism. In the broadest sense the protection of self is called as immunity. [104]

Early bacteriologists adopted it to signify the resistance to infection. Burnest (1957) described it in the broadest terms, the development of experience of past infections to deal more efficiently with subsequent one. Antibodies appear in the blood, but it is even more important that the antibody producing cells are so modified that they can rapidly produce fresh supply of antibodies on renewed contacts with the antigen.

But, immunity is not mere a defense against the infections. It is concerned with the structural and functional integrity of the body. It is indeed a reaction which protects the self and rejects the factors those are not a part of its own cellular structure. Basically immunology depends on the recognition of differences in the chemical structure of the substance.

It is not easy, therefore at present to give a concise definition of the immunity. The subject is expanding in an explosive fashion, which including subjects like tissue and organ transplantations, tagging of antigen and antibodies. Concepts of auto – immunity etc. in spite of a great deal of knowledge acquired during recent years about various organs cells and tissues comprising immunologic system, the exact manner in which their functions are linked together is still not clearly know. It is useful to classify immunity as follows.

INNATE IMMUNITY:

The resistance to certain infection is a natural gift and is seen in all forms of life. This natural non – susceptibility to infection is called innate immunity. The actual mechanisms if its development is not know. This is the type of immunity related to the species and heredity.

In spite of wide distribution of pathogens and their frequent as well as constant contact with the body nevertheless in many people they remain inoffensive. Among human beings some are subject to disease and others are constitution of the tissues and humors which oppose the penetration of pathogens is called **innate or natural immunity**. An individual enjoys the innate immunity by virtue of his constitutional and genetic make up. Innate immunity may be species – specific racial – specific or individual – specific.

SPECIES SPECIFIC IMMUNITY: Certain infections occur in particular race e.g. syphilis gonorrhea leprosy measles diphtheria cholera and many other diseases occur in man only but not in lower animals.

RACE SPECIFIC IMMUNITY: No races of some species are found to exhibit the same degree of susceptibility or resistance to infection e.g. Algerian race of sheep is immune to anthrax that is a common disease of other races of sheep

INDIVIDUAL SPECIFIC IMMUNITY: Some differences in individuals of some species in susceptibility to certain infection do occur. Intra uterine trans placental transfer of antibodies to infection in infants depending upon immunology status of the mothers. Genetically acquired immune deficiency will also result in increased susceptibility to infection in some individuals.

SURFACE DEFENCE: The pathogens are prevented from getting an access to the tissues by the surface covering of the skin. The skin forms the first site of defense. Apart from the skin there is mucous membrane distributed at certain parts of the body and various kinds of secretions and excretions that are directed towards the prevention of bacteria from penetrating into the interior.

TISSUES AND THEIR DEFENCES: In spite of the surface defense micro – organisms do gain access to tissues and blood. The defenses offered at tissue level, which act on or destroy them, are, Tissue fluid, which contains lysozyme, Natural antibodies, Properdin, Phagocytes the important biochemical changes that occur are: Glycolytic metabolism of leucocyte with the liberation of lactic acid.

Liberation of thrombokinase from damages tissues with precipitation of fibrin barrier. Tissue environment becomes anaerobic by the raised Co2 level. Liberation of antibacterial or bactericidal chemicals such as verdo – peroxidase lysozyme lipoids and basic peptoids by the dead tissues.

INNATE IMMUNE IN EMBRYO

FERTILIZATION

Fertilization is the process of fusion of the spermatozoa with the mature ovum. It begins with sperm egg collision end with production of a mononucleated single cell the zygote. It objectives are 1, to initiate the embryonic development of the egg 2, to restore the chromosome number of the species.

CONTECT AND FUSION OF THE GAMETES

Complete dissolution of the cell of the corona radiate occurs probably by the chemical action and liberates from the acrosomal cap of the hundred of sperms present at the site or by the action of the mucosal enzyme. The acrosomal cap probably facilitates penetration of the zona pelucida. More than one sperm may penetrate the zona pelucida. Out of many sperms, one touches the vitelline membrane. Soon, penetration of the other sperm is prevented and immobilization of the sperm present in side the zona pelucida occurs by zonal reaction and vitelline block. Completion of the second meiotic division of the oocyte immediately follows each containing haploid number of chromosomes (23 X) the bigger one is called the female pronucles and the smaller one is called second polar body which is pushed to the perivitelline space.

In the human, both the head end tails of the spermatozoa enter the cytoplasm of the oocyte but the plasma membrane is left behind on the oocyte surface. Head and neck of the spermatozoa becomes male pronucleus containing haploid number of chromosome (23, X) or (23, Y). The male female pronuclie unite at the centre with the restoration of the diploid number of chromosomes (46) which is constant for the sepses. The zygote thus form contain the both the parental and maternal genetic materials. In some instant an antigen called fertilizen present on the cortex and it coat of the ovum, reacts with the antibody called antifertilizen liberties at the plasma membrane of the sperm head. Thus the union between the two gametes may be affected by an immunological reaction.

After the zygote formation, typical mitotic division of the segmentation nucleus occurs producing two blastomeres. The two cell stage is reached approximately 30 hours after the fertilization. Each contains equal cytoplasamic volume and chromosome number. Blatomers continue to divide by binary division through 4, 8, 16 cell stage until a cluster of cell is formed and is called morula. The cell of the morula us known as inner cell mass which forms the embryo proper and the peripheral cell are called outer cell mass which will form protective and nutritive membrane of the embryo.

While the morula remains free in the uterine cavity on the 4th and 5th day it is covered by a film of mucus. The fluid posse through the canaliculi of the zona pellucida which separates the cell of the morula is termed as blastocyte. The zona pellicida became

stretch, thinned and rgatually disappears soon prior to implantation. The cell of the outer cell mass forms the wall of the blostocyte and is known as trophoblast or primitive o ectoderm which is concerted with embedding and attachment of the zygote. The inner call mass is concerned with development of the embryo.

IMMUNOLOGY IN OBSTETRICS

The antigen present over the periphery of the trophoblast cells, are in some way hidden from the maternal immune mechanism.

A sialomucopeptide coating is present over the trophoblastic cell which possibly impedes recognition of cell surface antigen by maternal lymphocytes. Trophoblast produce large amount of chorionic gonadotophin, a glycoprotein which is know to have some depressing effect on antigen recognition system of maternal lymphocytes. It is further known that local concentration of chorionic gonadotrophin far exceeds the peripheral level and is sufficient to prevent immune recognition. It is through that maternal immune response may remain depressed during pregnancy as evidence by depressed mixed lymphocyte reaction between maternal lymphocyte and lymphocyte from unrelated non pregnant individuals

Alpha fetoprotein is a major serum protein in the foetus which across the placental barrier in to the maternal circulation. Alpha fetoprotein abrogates maternal immune response against the foetus and this may act as a natural immunosuppressive.

Prolactine has been known to depress maternal immune reaction against the foetus. Raised level of prolactine characteristic of pregnancy, drops to normal within the first two week of parturition. The same holds good for the newborn whose prolactine level at birth is very high the fall of this level during the first week is exceptionally steep.

IMMUNE FUNCTION IN THE FEOTUS AND NEW BORN

The infants are physiologically competent at birth. Various types of T cells are identified as early as 7-8 weeks of intrauterine life. Lymphocytes infiltrate to thymus by 8-9 weeks. Thymus is matured by 12 weeks. B cells are detectable by 13 weeks. Major classes of immunoglobulin can be formed by 20 weeks of gestation.

LYMPHATIC AND IMMUNE SYSTEM

The lymphatic and immune system consists of fluid called lymph, vessels called lymphatic vessels to transport the fluid a number of structures and organs containing lymphatic tissue and red bone marrow. When stem cell develops into various types of blood cell including lymphocytes, it assists in circulating body fluids and helps defend the body against disease causing agents.

Whereas interstitial fluid is found between cells, lymph is located within lymphatic vessels and lymphatic tissue Lymphatic tissue is specialized from of reticular connective tissue that contains large numbers of lymphocytes those lymphocytes are a granular white blood cells. Two types of lymphocytes participate in immune responses: B cell and T cells. Draining excess interstitial fluid. Transporting dietary lipids. Carrying out immune responses. Lymphatic Vessels and Lymph Circulation- Tissues that lack lymphatic capillaries include avasular tissues (such as cartilage, the epidermis, and the cornea of the eye), the central nervous system, portion of the spleen, and bone marrow.

SPECIFIC RESISTANCE: IMMUNITY

The ability of the body to defend itself against specific invading agents such as bacteria, toxins, viruses and foreign tissues is called specific resistance or immunity. Substances that are recognized as foreign and provoke immune responses are called antigens (Ags) the immune system includes the cell and tissues that carry out immune responses.

MATURATION OF T CELL AND B CELL

The cells that develop immunocompetence, the ability to carry out immune responses if properly stimulated by lympocytes called B cell and T cell. Both develop in primary lymphatic organs (red bone marrow and the thymus) from pluripotent stem cells that originate in red bone marrow, B cell complete their development in bone marrow a process that continues throughout T cell develop from pre T cell that migrate from bone marrow into the thymus where they mature. Most T cell arises before puberty, but they continue to mature and leave the thymus through out life. Before T cells leave the thymus or B cell leave red bone marrow, they begin to make several distinctive proteins that are inserted into the plasma membrane.

TYPES OF IMMUNE RESPONSES:

Immunity consists of two kinds of closely allied responses, both triggered by antigens. In cell mediated immune responses, CD8+ T cell proliferate into cytotoxic T Cells that directly attack the invading antigen. In antibody- mediated immune responses, B cells transform into plasma cell, which synthesize and secrete specific proteins called antibodies immunoglobulin.

CELL MEDIATED IMMUNITY

(Activation, Proliferation, and Differentiation of T cell)

A cell mediated immune response with activation of a small number of T cells by a specific antigen. Once a T cell his been activated, it undergoes proliferation and differentiation into a clone of effectors cells, a population of identical cell that can recognize the same antigen and carry some aspect of the immune attack.

TYPES OF T CELL: The three main types of differentiated T cell are helper T cells, cytotoxic T cell, and memory T cells.

HELPER T CELLS: Most T cells that display CD4 develop into helper T cells, also known as CD4+ T cells. Helper T cell starts secreting verities of cytokines. Different subsets of T cells specialize in the production of particular cytokines.

CYTOTOXIC T CELLS: T cells that display CD8 develop in to cytotoxic T cells, also termed CD 8+ cells. However, to become cytotoxic T cells need

co stimulation by other cytokines produce by helper T cell. Thus maximal activation of T cells requires presentation of antigen associated with both MCH -I and MCH -II molecules.

MEMORY T CELLS: T cells that remain from a proliferated clone after a cell mediated immune response are termed memory T cells.

ANTIBODY MEDIATED IMMUNITY: (activation, proliferation, and differentiation of B cells) the body contains not only millions of different T cells but also millions of different B cells, each capable of responding to a specific antigen. Where as cytotoxic T cells leave lymphatic tissues to seek out and destroy a foreign antigen, B cells stay put. In the presence of a foreign antigen, specific B cells in lymph nodes, the spleen or mucous associated lymphatic

tissue become activated. They then differentiate in to plasma cells those secrets specific antibodies, which in turn circulate in the lymph and blood to reach the site of invasion.

ANTIBODIES An antibody can combine specifically with the epitope on the antigen that triggered its production. The plasma cell could secrete as many different antibodies as there are different B cell receptors because the same recombined gene segment code for both the antibodies which ids secreted by plasma cells.

ANTIBODY STRUCTURE Antibodies belong to a group of glycoprotines called globulins, and for this region, they are known as immunoglobulins (Igs). Most antibodies contain four polypeptide chains. Two of the chains are identical to each other and are called heavy chains, each consists about 450 amino acids. Short carbohydrates chains are attached to each heavy polypeptide chain. The other two-polypeptide chains also identical to each other are called light chains, and each consists about 220 amino acids. A disulfide bonds (s-s) holds each light chain to a heavy chain. Two disulfide bonds also link the two heavy chains, this part of the antibody displays considerable flexibility and the called the hinge region.

The remainder of each H and L chain, called the constant region, is nearly the same in all antibodies of the same class and is responsible for the type of antigen antibodies reaction that occur. However, the constant region of the H chain differ from one class of antibody to another, and its structure serves as a basic for distinguishing five different classes, designated IgG, IgA, IgM, IgD, and IgE. Each class has a distinct chemical structure and a specific biological role. Because they appears first and are relatively short lived.

MOLECULES OF THE IMMUNE SYSTEM

IMMUNOGLOBULINS

Immunoglobulin (Ig) molecules are the effectors products of B cell and although they all have a broadly similar structure, minor difference within the main immunological classes they are IgG, IgA, IgM, IgD, and IgE, are associated with a range of important

biological properties. Molecules almost identical; to secreted immunoglobulins are incorporated in the cell membranes of the B cell and there are many related molecules concerned with antigen recognition and cell-cell communication

STRUCTURE OF IMMUNOGLOBULIN MOLECULES

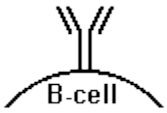
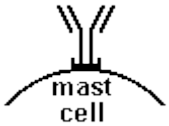
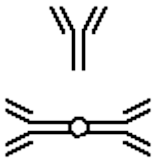
An Immunoglobulin (Ig) molecules is made up of distinct sub units held together by disulphide (s-s) bonds, which can be broken by reducing agents so that molecules falls apart in to pairs of polypeptide4 chains called light and heavy chains. Two types of light chains exist, kappa and lambda, of which individual immunoglobulins molecules have only one type and there are several different heavy chains, which confer on the Ig molecules its class specific properties.

A typical immunoglobulins molecule such as IgG has two antigen binding region and one Fe component that is the part of the molecules, which performs the class related function such as complement fixation. The section of the heavy chain contained in to the Fe component is responsible for the antigenic difference between the classes of the immunoglobulin. Which enable their laboratory measurement by the use of heavy chain specific antisera.

The molecular bases of diversity of antigen binding functions reside in the so called variable region of the Fab component. The antigen combining site of an immunoglobulins molecules can if self be recognized and reacted to by other immunocopetent cells, with the production of the anti idiotype antibodies which can influence the magnitude and duration of antibody production to a given antigen.

TABLE NO:2 CHARACTERSTIC FEATURES OF IMMUNOGLOBULINS

Isotype	Structure	Placental transfer	Binds mast cell surfaces	Binds phagocytic cell surfaces	Activates complement	Additional features

IgM		-	-	-	+	First Ab in development and response.
IgD		-	-	-	-	B-cell receptor.
IgG		+	-	+	+	Involved in opsonization and ADCC. Four subclasses; IgG1, IgG2, IgG3, IgG4.
IgE		-	+	-	-	Involved in allergic responses.
IgA		-	-	-	-	Two subclasses; IgA1, IgA2. Also found as dimer (sIgA) in secretions.

IMMUNOGLOBULIN G (IgG)

In healthy adults, IgG accounts for more than 70% of the immunoglobulins in normal serum and is distributed equally between the blood and extra cellular fluids. About a quarter of all the body's IgG passes out of the bloodstream each day and the same amount returns via the thoracic duct. In man, IgG is the only immunoglobulin that is transported across the placenta to reach the fetus and provide the newborn baby with passively acquired antibody during its early life. IgG antibodies are very important in anti bacterial immunity.

They readily neutralize soluble toxins such as those responsible for diphtheria or tetanus. IgG also has an opsonic effect on bacteria, coating them so that their ingestion by phagocytes is facilitated. IgG antibodies also can produce disease, e.g. by forming immune complexes, or as auto auto

IMMUNOGLOBULIN M (IGM)

The macro molecular IgM is predominantly intravascular. It is made up of five immunoglobulin units linked with disulphide bond to provide ten identical antigen combining – sites, together with a J (joining) chain. IgM is especially effective in activating complement to produce immune lysis of foreign cells. IgM antibodies are also much more efficient than IgG antibodies in linking and particulate antigens together for agglutination and phagocytosis, and would seem to be specially adapted for dealing with cell debris or bacteria in the bloodstream.

IMMUNAGLOHUIIN A (IGA)

IgA accounts for about 20% of the serum immunoglobulins. However its function, if any within the bloodstream and tissues is thought to be much less important than its role as a secretary antibody. The major site of IgA synthesis are the laminae propriety underlying the respiratory tract the gut and other mucosae. IgA is fount in three main molecular forms (Fig. 2.6). In blood it is 7S, monomeric, similar in size to IgG. IgA (also containing J chain) is produced by plasma cell in the mucosae and transported across epithelia into colostrums, saliva, intestinal juice, respiratory secretion. Tears and several other body fluids during trans- epithelial transport, another polypeptide, secretary components is incorporated to form secretary IgA, relatively resistant to digestive enzymes.

Secretary IgA confers immunity infection by enteric bacterial and viral pathogens, and may also be involved in the regulation of the commensal gut flora. Oral immunization is now being used to try to try to induce protective immunity to intestinal infection such as cholera and rotavirus.

IMMUNOGLOBULIN D (IGD) IgD is almost exclusively found on the surface of immature B lymphocytes and may be involved in their maturation and regulation.

IMMUNOGLOBULIN E (IGE)

IgE concentration in serum is very low. This is in part because it has a considerable affinity for cell surface and bind firmly to mast cells and basophile. IgE antibodies are necessary for immediate hypersensitivity reactions, such as occur in atopic individuals e.g. in hay fever.

The physiological function of IgE antibodies is obscure but appears to be important in defense against helminthes parasites (worms)

FACRTORS INFLUNCING IMMUNITY:

The resistance to infections is due to many factors some of which remain as the individual and his previous history. In the last century there was much controversy over the respective of the cellular and humoral factors in immunity. It is now recognized that both these factors are of importance in resistance to infection.

In our attempts to naturally increase the immunity we have to understand many divers' factors upon which it probably depends. They are as follows

HEREDITY: It is possible that a certain degree of capacity to resist against particular infections may be derived from the past experience of parents and immediate ancestors. This would be a trait additional to the circulating antibodies inherited at birth from mother. Capacity to respond to a particular antigenic group genetically determined.

TRADITION: These may provide increased opportunities for contact with an infectious agent resulting in the building up of high specific immunity among the survivors.

AGE: The new born is immune to many common infections because of antibodies passively received from the mother. However the bacteriostatic activity of serum is poorer in the premature than in the full term infant.

In old age there is a reduced immunity towards the streptococcal and pneumococcal infections but there are increased resistances to certain other bacterial and viral infections because of specific immunity build up during the course of life.

SEX: Female of any species responds better to antigenic stimuli as compared to the male. Immunoglobulin levels are usually higher in females.

HORMONES: It appears that the growth promoting hormone thyroid hormone and female sex hormones influence the immune response.

LYSOZYME : Lysozyme is law molecular weight substance found in various phagocyte cells and many tissue fluids such as tears saliva etc. which resists microbial multiplication by virtue if its enzymes.

NUTRITION: Good nutrition obviously helps to build up resistance against the common respiratory infections. However certain viruses are know to have predilection towards the attacking well nourished cells.

Adequate vitamins and other food factor especially the vitamins A.B. complex and C is probably of importance in the defense of the body against infections against. High degree of protein calorie malnutrition adversely affects the immune response.

LOCAL TISSUE RESISTANCE: The healthy intact skin or mucosa presents a formidable barrier to the entry of most pathogenic organisms. However the thin – layered vaginal membrane of the child is very muscles membranes of the young infants seem to be more resistant to the infection than those of older subjects and this may be one factor in the lower incidence of diphtheria in infants. Previous infection may cause certain changes in the target cells in the tissue. This may render them resistant to subsequent infection by the same agent. Recently a direct correlation of the resistance of the respiratory mucosa to infection by Para influenza type 1 and measles viruses has been established with the present of local secretary antibody IgA. The intestinal mucosa in know to contain a large number of plasma cells which are capable of providing immunoglobulin especially IgA. Some evidence is now available to associate the local antibody production with the protection at mucosal surface.

ACQUIRED IMMUNITY: Acquired immunity is that which is acquired by the individual during the course of his life and due to the presence of antibodies in his body. It is usually specific. As shown above these types of immunity may be actively or passively acquired and either of these may be acquired by natural or artificial means. This is of two types:

ACTIVE IMMUNITY: Active immunity the most ideal form takes some time to develop but is lost very slowly and is capable of rapid restoration by the slightest stimulations. It takes about 8 – 10 days to establish itself inside the body so as to produce antibodies entirely by the cells of the body itself. It persists for long periods if developed once. The cells of the body are so changed and so trained that when the specific antigen is re – introduced more

and more antibodies are produced afresh. This type of immunity is called as active immunity.

PASSIVE IMMUNITY: On the other hand the passive immunity although rapid in development is only of a short duration lasting for a few weeks at the most and once lost can not be revived. If the cells of the body do not take much part or active role in the production of antibodies in the course of exposure to antigen the immunity is called as passive. Antibodies are introduced with the serum of a highly immunized animal to blood of the person. In this process the body cells do not undergo any changes but the immunity is acquired immediately. The passive immunity can be produced by antibacterial as well as anti – toxic serum.

But the active and passive immunities are further grouped as naturally acquired and artificially acquired one.

02.6 ALLERGY & CONCEPT OF VIRUDHA AHARA

Food plays an important role in healthy diseased and convalescent states. It is more important than the medicine itself. The body can be nourished and maintained in good health, by adopting suitable diet. Ahara is the basic factor that sustains life. It supports the body like one of the pillars of building [105]. It is essential for the nutrition of dhatus. Proper maintenance of power of digestion also depends upon the intake of proper ahara [106].

Importance of Ahara

Ayurveda is the health science of prevention and cure of diseases. Ahara has given prime importance in both the aspects.

I Acharyas have given instructions for taking food daily and also according to personal constitution. The changes to be followed for different seasons have also given. By following these instructions one can sustain his healthy life. Thus ahara can be considered as one among the prime factor for longevity of life [107].

II. The Yukthivyapasraya chikitsa is performed by the administration of dravya either as diet or as medicine [108]. Thus ahara and oushadha are complimentary in the treatment of diseases for the management of every rogas ahara too has been prescribed as pathya or apathya. That is why Kasyapacharya [109] has called it as 'Maha bhaishajya'.

Ahara as an Etiology for Diseases

In take of right kind and amount of food can ensure good health. Ahara if not taken properly will lead to ill effects. Carakacharya has said that the body is made by ahara and the diseases are also caused by ahara [110]. Virudhahara is considered to be one of the important causes of diseases.

Etymology

The word virudha is derived from the root words Vitrudh ktha [111]. The meaning of the word 'virudh' is virodha visista. 'Virodha' means incompatability, inconsistency, opposition and 'Visista' means distinguished, distinct, particular, and peculiar. Thus the whole meaning is distinguished incompatability.

Synonyms

In Ayurvedic literature Ahita ahara, (un acceptable food) apathya (incompatable diet) and Midhya Aahara (Improper diet) words are used very frequently as synonyms for the term virudhahara.

'Hitahara' is that which maintain samatha [the equilibrium] of bodily dhatus and help in eliminating the disturbance of their equilibrium. Ahitha ahara is having quality opposite to it[112]. 'Pathya' is that which protects the swastha's health and helps the patient to be cured of disease[113.] Caraka has described eight factors for determining the utility of food called as 'Astavidha Aharavidhi visesayatanani'. The diet opposite to 'Astaaharavidhivisesayatanani' is designated [114] as 'Mithya Ahara'.

Definition

Dravya that which causes ulklesa [agilates, enrages, and move doshas from their normal places] of doshas and do not expels it out is termed as Virudha [115] It will also possess qualities opposite to dhatus. Virudhahara produce utklesa or agitation of doshas which is not sufficient to get expelled from the body. In short, all drugs and diet that dislodge the various doshas but do not expel them out of the body are to be regarded as unwholesome. [116]

Factors that cause Virudha

The drugs and diet that are virudha for the normal dhatus and doshas of the body are infact opposed to the proper growth of such dhatu and doshas[117.] Some act due to 'paraspara guna virudha' [Mutually contradictory qualities] some by samyoga virudha [combination] some by samskara virudha [method of preparation], some by virtue of desa virudha [Land and body], Kala virudha [time] Matra virudha [dose] and some others by swabhava virudha [inherent nature] virudha occurs due to visamata, samyata, visamata samyata, samskara and swabhava of anyonya gunas which has got immense strength [118].

Types of Virudha

Carakacharya has classified virudhas into eighteen different types [119]. Susruthacharya cited many examples under mana, samyoga, karma virudha. He has specially mentioned karma and Rasa – veerya – vipaka virudha [120] Vagbhatacharya has just

mentioned samskara, matra, desa, kala and samyoga virudhas[121]. The elaborate description of the eighteen types of virudhas is as follows.

I. Desha Virudha

The habitat (Desha) is divided as [122]

a. The country or habitat of the drug
b. The patient himself.

Here regarding this subject, the habitat is important further Caraka classifies Desha into three kinds.

(i) Anupa (Wet Land) (ii) Jangala (Arid Land) and (iii) Sadharana (Ordinary)

Anupa desa is predominant of kapha dosha and madhura rasa people resideing here will be of kapha vata predominant. Use of snigdha, sita and other food substances of similar qualities in anupa desa are considered as Anupa desa virudha. Jangala desa is predominant of Vata dosha and katurasa. People residing here will be of vata pitta predominant consuming ruksha, tikshna and other food substances of similar qualities in jangala desa is virudha. Anupa sadharana is predominant of kapha dosha and Lavana and Amla rasa. So use of Lavana & Amla and other kapha aggravating food substances is considered as virudha. Jangala sadharana is predominant of kapha dosha and Tikta & Kashaya rasa. So use of Tikta & Kashaya and other Vata aggravating food substances is considered as virudha.

Chakrapanidatta has given examples in his commentary for both Bhoo desa virudha and sharira desa virudha. According to him use of "Bhasmapamsu paridhwastham mamsam" is Bhoo desa virudha and the use of honey by 'ushnabhitapta sariram' is sarira desa virudha [123.]

2. Kala Virudha

Kala or time is divided into two types – related to the individual (Avastika kala) or to the environment (samvatsara kala). The samvatasara kala is very important in terms of food. The six seasons or ritu of the year have different levels of doshas. Regimen for each ritu has been advised and observing regimen contrary to the specific season is considered as virudha.

In Hemanta ritu, agni is in prabala state [124]. Therefore, intake of guru brimhana, snigdha, swadamla lavana rasa pradhana ahara is advised [125.] Consuming sita and ruksha ahara in sita kala is considered as virudha sisira ritu is similar to Hemanta ritu but predominant of ruksha and sita. In take of katu, tiktha, kashaya, sita and vatala ahara is considered as virudha.

In Vasanha ritu Jataragni is manda and kapha is in prakopa state. So intake of guru sita snigdha, madhura and lavana ahara is virudha. In Grishma ritu, Kapha is in prasama state and vata is in caya state [126]. Madhura, sita, drava, snigdha, annapana has to be taken [127.] In take of Lavana, amla, katu and ushana ahara in ushana kala is considered as virudha.

In Varsha ritu Agni and bala will be in a state of kshina. So laghu, snigdha, ushna, amla, lavana, ahara has to be taken [128]. Consumption of food against this quality is considered as virudha. In Sarat ritu, pitha is in prakopa state. Sita, laghu, kashaya, madhura and tiktha annapana has to be taken. In take of food against this quality is considered as virudha [129].

3. Agni Virudha

Agni in the body is differentiated into four types according to its intensity.

a. Mandagn - Mild intensity

b. Tikshnagni - Acute intensity

c. Vishamagni - Irregular intensity

d. Samagni - Regular intensity.

One should take diet (Ahara) after considering four types of agni respectively. If food has not been taken in accordance to respective digestive capacity (Jataragni bala) then it will become Agni virudha.

Due to kapha dosha, agni is manda and even minute quantity of ahara will be digested with difficulty. Intake of Guru, Snigdha and Madhura food stuff is Virudha. Due to pitha predominance, the agni is tikshna and can digest even heavy foods fastly. Intake of Laghu, tikshna, suksma, vidahi, ushna food stuff is virudha. Due to vata dosha the agni

become irregular and digestion too will be irregular. In take of Ruksha. Laghu, Suksma, Guru food substances is agni virudha. When vata pitta – kapha is in equilibrium samagni occurs. Excessive quantity of food and taking food substances irrespective to agni causes virudha[130].

4. Matra Virudha

Food taken in sufficient quantity is termed as Matravat Ahara. If one cannot take matravat ahara then it is called matravirudha ahara. Caraka has given example of intake same quantity of Madhu and Gruta for explanation of Matra virudha.

Hina matra and atimatra could be taken under matra virudha. Caraka has described matra as 'Rashi' in Ashtavidha Ahara visesayatana.

Ayurveda believes that every human being is unique and different from each other hence the ahara matra for each person is different. The hunger or the quantity of food needed also differs from one person to other. So it is not possible to decide a specific quantity of food, which is applicable to all. Considering these points Ayurveda contributes a special view in regard of matra of food. To decide appropriate quantity of food which is sufficient for person's good health Carakacharya had given some good lines. The stomach is imagining to be divided in three equal parts. One part for solid food one part for liquids and remaing one is for doshas.

In matra total quantity as well as quantity of different items is considered. The former is known as sarvagraha (consideration of the whole) and the latter as parigraha (consideration item wise).

This is very important from the view of the concept of "balance diet "because each items is considered with regarded to the requirment of the body.

Matra virudha may be of two types

1. Hina (deficient)

2. Adhika matra (excessive)

And these two types of virudha matra can be divided into further two types in context of saravagraha and parigraha

1. Saravagraha hina matra (deficiency in quantity of whole food)
2. Parigraha hina matra (deficiency in quantity of ingredients)
3. Saravagraha Adhika Matra (Excessive quantity of whole food)
4. Parigraha Adhika Matra (Excessive quantity of ingredients)

Hina mantra and Adhika mantra against the proper quantity normally required for a person is Matravirudhahara. When combining certain food substances, particular ratio has to be maintained in the quantity.

If this ratio is not maintained it will also lead to matravirudha. For eg; mixing madhu and sarpi in equal quantity.

TABLE NO: 03 EXAMPLES FOR MATRA VIRUDDHA

NO	**Example**	**C.S**	**S.S**	**A.H**	**A.S**
1.	Honey + Ghee in equal quantity	+	+	+	+
2	Honey + rain water in equal quantity	+			
3	Honey + seed of the east Indian lotus	+			
4	Honey + water in equal quantity		+		+
5	Honey + sneha in equal quantity		+	+	
6	Water + sneha in equal quantity		+		
7	Honey + senha + rain water		+		
8	Honey + ghee + vasa			+	
9	Honey + vasa			+	
10	Honey + Taila			+	
11	Honey + Taila + Ghee + Vasa			+	

5.Satmya virudha

Satmya is defined as substances which are accoustomed for a person[131.] Satmya is of three type (i) Avara satmya-homologation to a single taste ii) pravara satmya-homologation to all the six tastes iii) Madhya satmya- homologation between the avra and pravara satmya. Virudha occurs if one takes substancae to which he is not accoustomed for eg. Consuming swadu and sita substance by person accoustomed to katu and ushna substance.[132]

6.Dosha virudha

Every individual has unique prakrithi and there is some preponderance of particular dosha in particular habitus. One should take food substances which are opposite in quantity to that predominat dosha. Utilization of anna, ousahda and kriya having similar qualities with doshas of individual is called dosha virudha (humoral incompatibility) for eg; consuming ruksha sitadi dravyas in vata dosha. [133]

7. Samskara virudha.

Samskara is the method in which food is cooked (Processing method). It imparts acceptable taste and better qualities for the foodstuff [134.] The different agents like water, fire, untesils employed in samskara should be properly used with care. Other ise during the processing method food substance is converted into poison and is termed as samskara virudha for eg; meat of sikhi (Peacock) roasted on eranda (castor) stick and fire[135]. According to Dalhanacharya the term 'Karma' is used for samskara. So Susruthacharya's karma virudha can be classified into samskara virudha of Carakacharya. For eg; meat of kapinjala, mayura, lava, tithiri, godha cooked with eranda taila in Eranda/darvi fire [136].

8. Virya virudha

Virya is defined as the potency or efficacy of a dravya. Virya is the power by which an action takes place. Some Acharyas says it is of eight types, while some other says it is of two types – ushna and sheeta. These two have opposite function. Ushna veerya dravya helps in stambhana, jivana and pitha shamana [137].

When Dravyas are taken in combination, they should be of same virya. When a substances having opposite viryas are used in combination it is known as Virya virudha. For eg. Consuming substances having sita virya in combination with ushna virya substance [138].

9. Koshta virudha

Depending upon predominance of doshas, the koshta is of three types. Krura koshta occurs due to vata, madhyama koshta occurs due to kapha and mridu koshta occurs due to pitta. Understanding the nature of koshta is very important in treatment. Krura koshta needs a strong medicine for purgation. Administration of manda virya and alpa matra dravyas in krura koshta causes virudha. While mridu koshta requires only mild drugs for purgation. Administration of guru purgatives in heavy doses for mridu koshta results in koshta virudha[139.]

10. Avastha virudha

Avastha indicates the state of dosha in the body. Depending upon condition of body, dosha too deranges. For eg; vyayama, vyavaya and srama increases vata in the body. One should take snigdha, ushna ahara after it. Consuming vata aggravating food soon after physical exertion is considered as Avastha virudha. Similarly, intake of kapha aggravating food by a person soon after sleep is also virudha[140.] When one person takes food substance similar to his own prakrti dominant dosha, then it is called avastha virudha. For eg; childhood intake of kaphavardhaka ahara. If a person who is suffering from provoked dosha and he takes food substance having similar quality to provoked dosha, it is also called avastha virudha.

11. Krama virudha

Food is to be eaten only by observing certain order like do not take ushna food soon after sita food or snigdh food soon after ruksha food and vice versa. Taking food without attending the nature's call for defecation and micturition or when not having proper appetite or after his hunger has been aggravated is also considered as virudha [141.]

12. Parihara virudha

Parihara means the food substance which are to be avoided depending upon healthy or unhealthy condition. In diseased condition, in take of food substance, which are mentioned as apathya in that particular disese is called parihara virudha. Similarly, in healthy condition, after intake any type of meals, one should avoid those food substances, which are similar in quality with previous meal. Consuming of hot things after taking pork and cold things after taking ghee are examples for Parihara virudha [142.]

13.Upacara virudha

Upacara means chikitsa or treatment. Ahara is utilized as a part of treatment. Use of food incompatible to treatment is called Upacara virudha. For eg drinking cold, water after Snehapana .[143]

14. Paka virudha

Paka denotes the process of cooking which makes the food palatable and easy for digestion. The kind of fuel and limits of proper cooking is important. Over cooked and under cooked foods, burnt food all are cause for ajeerna and ama formation. Food looses its nutritious value by such injuditious cooking. A bad or rotten fuel also affects quality of cooked food. All these wrong practices constitute Paka virudha.

15. Samyoga virudha

Samyoga denotes combination of two or more substances. Usually combination of substances like milk and sugar will be beneficial for the ebody. Some substances on combination may become incompatible for the body. For example milk is jivana for the body and amla rasa is said to be tastemaker. Nevertheless, the combination of the two-i.e consuming sour substances with milk is samyoga virudha[144].

16. Hrut virudha

The term 'Hrdaya' meas 'Manas'. Food items which are pleasing to the mind will also be pleasant to the sense organs. Taste of the food is an important factor promoting likeness of food. Such food items will get assimilated and absorbed into the body quickly.

This likes and dislikes will be varying from one person to the other. Consuming food that is not pleasant in taste is Hrdaya virudha [145.]

17. Sampath virudha

Sampath refers to the richness of quality of food products. The substance should reach natural maturity before they are consumed. Only the naturally matured foods tuffs have good quality and will perform intended function in the body. Eating of substance that are not matured/over matured /putrified will lead to Sampath virudha[146.]

18. Vidhi virudha

Dietetic regulation and procedures have been advocated in Ayurveda like Upayoga Samstha (Ashta aharavidhi visheshayatana) [147.] These include eating food, which is hot, unctuous, non-antagonistic in potency and in due measure, after full digestion of the previous meals, in exclusive place, provide with all the accessories, neither too hurry, nor too leisurely, without talking or laughing with full concentration and having proper regard to oneself. Not obeying these rules for eating food is virudha. For eg. Taking meals in public is Vidhi virudha.

Incidence of virudhahara in Modern Generation

Ahara should promote growth, reproduction, maintainance and repair of the body. The present generation gives more importance for satisfying the sense of taste without judging whether the food is beneficial to the body or not. The elaborate example for virudhahara given by Acharyas is still relevant today. As a part of modern life style man is always in habit of taking virudha knowingly or unknowingly.

Drinking milk with salty food, milk with fruits, drink milk when body is hot are example of virudhahara. Taking ice cream, fruit salads, and cold drinks soon after taking hot foods or when the body is hot will lead to virudha. Taking curd/ honey/ alcohol with hot food and drinking tea/coffee/milk after taking fish will cause virudha. Biriyanis with curd, salad and smoked / grilled/ Tandoor/ Barbecued food are source of virudhahara. Preserved food and tinned food are kind of paka virudha observed in modern era.

Modern fast life style increases the chance of virudhahara. In many houses food cooked once in a week is storing in refrigerator for wholeweek's use. The busy daily schedule has forced the employees and students to take food in different time. Many people hold the natural urges for micturition and defaecation while taking food.

The increasing complexity of our modern industrial society and the wide ranging nature of the international food trade have increased the risk of contamination of food by chemical and biological agents.

General Symptoms

The symptoms appear at the stage of stanasamsraya when vitiated doshas get lodged in the deformed srotases General symptoms of vitiation of srotases have been described by carakacharya (i) Atipravruthi [eg. Atisara, prameha etc] (ii) Sanga [eg. Jwara, ama Vata etc] (iii) Siragranthi [arsas] and (iv) Vimarga gamana [Udara] [148]

Doshas are vitiated due to the imperfectly formed anna rasa which leads to deranged dhatus. Both these deranged doshas and dhatu leads to the vitiation of malas. These malas further derange malayanas or passages that facilitate their excretion [urdha dwa – Sapta sirasi] and result in diseases. [149]

When Virudhaharas are followed the dhatu parinama process will be deranged and affect the ojas. The ojokshaya causes the nasa of teja and bala.

Indriyas depend upon bhutagnis for nutrition and proper functioning. Each bhutagni depends upon jataragni for its ideal functioning. Thus faulty functioning of jataragni will not only affect the physical but also the psychological functioning of the body. This leads Indriya dourbalyam, Indriya nasam, Andhyam, Badhiryam, Mookatha and minmina.

Diseases caused by Virudhahara

Virudhahara causes pathological state in the srotas. This Srothodushti produces diseases of concerned srotas diseases caused by virudhahara with respect to concerned srotas can be tabulated as follows.

Table no: 04 Diseases caused by virudhahara

No	Srotas	Diseases according to	
		CarakAcharya	VagbhatAcharya
1	Annavaha	Grahani Adhmana Amlapitha Amavisha	+ + Gulma -
2	Udakavaha	Udara	+
3	Pranavaha	Peenasa	Yakshma
4	Raktha Vaha	Kustha Kilasa Visphota Visarpa Bagandara - +	+ - + - + Vidradi Asrapitha
5	Mamsa Vaha	Galamayam	Arsa
6	Medo vaha	-	Meha
7	Asthi Vaha	-	Vatavyadhi
8	Majja vaha	Murcha Mada	- +
9	Sukravaha	Shandya Santhana Dosha	-
10	Mutra Vaha	-	Asmari
11	Purisha Vaha	Atisara	+
12	Manovaha	Unmadam, Chithanasam	Mathi – smrithi - chithanasam

Why some people following Virudhahara are not affected by diseases? Atreya says that due to certain factors even ahitahara does not produce disease immediately in every individual. Because all apathya are not potential enough to vibrate tulya dosha; all doshas are not of tulya bala nor do all individuals have equal vyadhikshamatwa.

The apathya due to atiyoga of desa, kala, samyoga, veerya, pramana, will be more harmful. This exceeded doshas if combine with each other, if they require mutually contradictory therapies, if they are deep seated, if chronic, if vitiated in one of the ten resorts of life and if they afflict the vital organs will be difficult to cure and may be fatal to that individual.

Vagbhata says the mutually antagonising articles of food taken together will not harm those who habitually taken physical excersise, whose body is oily whose digestive

power is good, who are youthful and strong, who are habituated to such food, and who normally eat only little food. [150]

Factors that pacify ill effects of Virudhahara

Agni is the important factor that determines the well being of life. If agni is strong, the body will not be affected by virudhahara. Vyayama promotes agni and snigdha ahara fuels jataragni. Youth is the stage in which the jataragni is at its peak. Thus in young age, body is able to nullify the ill effects caused by virudhahara.

Vyadhikshamatwa is the power of the body that prevents the development of diseases or resists a developed disease [151]. Ojas is also called Balam which is related to the Vyadhikshamatwa of the body. Satmyam refers to the ahara and vihara to which the person is accustomed. Even virudhahara can be made to be familarised for the body. Due to satmyata equilibrium of doshas will not be tilted. Virudhahara will remain harmless due to satmyatwam only when the agni is strong enough to handle it.

The ill effect of virudhahara will be nullified as long as the person is in stage of youvana, performs vyayama and take snigdhahara. The jataragni will be able to deal with harmful effect to a certain extent if virudhahara is of less quantity. If virudhahara quantity is more then, it is able to produce devastating effect even if it is satmya.

Modern Concept

Various topics in modern science like food incompatibility, free radicals, Antagonism, Food processing is similar to the concept of virudhahara.

1. Food incompatibility[152]

The term food incompatibility was introduced by Dr. Howard Hay in 1930. He classified carbohydrates as alkaline forming foods and proteins as acid forming foods. He suggested that an ideal diet should contain 80% alkaline forming foods and 20% acid forming foods.

Concentrated protein foods should not be mixed with concentrated starch foods as they are digested differently in the body. Fruits which contain a large amount of glucose should not be combined with protein rich foods and it is better eaten alone either thirty

minutes before a meal or one or two hours after it. This will prevent fermentation of fruits in the acidic environment of stomach.

Milk when drunk with meals interferes with digestion process as it acts as a gastric insulator. When fat and proteins are digested together facts decrease the gastric juice secretion and hamper the digestion process of proteins.

The sugars and acids in the fruits slow the digestion of starches in the vegetables and may cause fermentation. So fruits and vegetables should not to be mixed. Acids fruits should be eaten separately from sweet foods. When sour substances are consumed along with starches, enzyme ptyalin cannot digest starches in the acidic medium resulting in fermentation. This concept is similar to samyoga virudha explained by Acharyas.

2. Free Radicals

The body generates energy by gradually oxidizing food in a controlled manner and storing it in the form of chemical potential energy called ATP. Some times free electrons may escape the transport system. These unpaired electrons readily form free radicals, which are unstable, short lived and highly reactive as they combine with other atoms new radicals are created and a chain reaction of free radical formation begins.

Cell membranes are made of unsaturated lipids. The unsaturated lipid molecules of cell membranes are particularly susceptible to this damaging free radicals process and readily contribute to the uncontrolled chain reaction. Oxidative damage, another name for the chemical reaction that free radicals cause can lead to a break down or even hardening of lipids which make up all cell walls. If the cell wall is hardened (Lipid peroxidation) then it becomes impossible for the cell to properly get its nutrients, get signals from other cells to perform an action (such as firing of a neuron) and many other cellular activities can be affected. In addition to the cell walls, other biological molecules are also susceptible to damage, including RNA, DNA and protein enzymes. The primary site of free radical damage is the DNA found in the mitochondria extensive DNA damage accumulates over time and shuts down mitochondria, causing the cells to die and the organism to age.

Free radicals naturally produced have some beneficial effects. Foreign invaders or damaged tissue is marked with free fadicals by immune system. This allows for determination of which tissue need to be removed from the body.

Free radicals play a key role in the pathogenesis of certain human diseases. The diseases caused by free radicals include Cancer, Atherosderosis, Heart diseases, RA, Diabetes, Senility and Mental disorders. They may also be involved in Parkinson's disease, senile and drug – induced Deafness, Schizophrenia, and Alzheimer's. The classic free radical syndrome, the iron storage disease hemochromatosis, is typically associated with a constellation of free radical related symptoms. Smoked and barbecued foods and processed foods contain high levels of lipid peroxides, which produce free radicals that damage the CVS, Virudhahara may lead to formation of free radicals.

Excersise and Oxidative damages

If free radical formation and attack are not controlled within the muscle during exercise a large quantity of muscle could easily be damaged. Damaged muscle could in turn inhibit performance by the induction of fatigue. Intense exercise in untrained individuals over whelms the defenses resulting in increased free radical damage. On the other hand regular physical exercise enhances the antioxidant defense system and protects against exercise induced free radical damage. This gives scientific basis for the fact that proper vyayama nullify the harmful effect of virudhahara.

3.Antagonism

In modern pharmacology antagonist is a drug that binds to a cellular receptor for a hormone, neurotransmitter, or another drug blocking the action of that substance without producing any physiological effect it self. Antagonism reduces the biological activity and increases the adverse effect of the drug. Virudha can be explained on the basis of antagonism. For eg. Combination of milk and sour fruits can be considered as antagonism.

4.Food processing

It is method of improving the palatability and increasing the shelf life of food. Heat treatment in the presence of reducing sugars such as lactose or glucose with a protein

causes the loss of available lysine (amino acid) this can even occur when the protein and sugar are stored together at lower temperature under moist conditions.

When proteins become resistant to digestion bioavailability of amino acids will be reduced. This occurs in severe heating conditions like burnt, overcooked in the presence of either sugars or oxidized lipids or even without either of these. When proteins are heated strongly, peptide bond formation occurs between the side chains of lysine and dicarboxylic acids leading to resistance in enzymatic digestion. These may be considered as example for 'Paka virudha' and samskara virudha.

5. Food allergy

Food allergy is defined as a complex of clinical syndromes resulting from the sensitization of the patient to one or more food. Immediate pattern of food allergy is mediated by IgE and delayed pattern by non - IgE mediated response. Allergens are usually proteins present in each food. The major allergens in milk are the caseins and the whey protein b-lactoglobulin the main allergens are the egg white, proteins ovomucoid, ovalbumin, and ovatransfferin. Severe anaphylactic reactions may occur due to fish flesh proteins called parvalbumins. Seed storage proteins (such as wheat gluten) and other protiens present in grain to protect it from attack by moulds and bacteria, have been found to be major allergens.

a. Symptoms of food allergies

Reaction to foods are some times rapid appearing within an hour (or sometimes even seconds) of consumption. IgE attaches to the allergens triggers the release of histamine causing symptoms like urticaria or wheezing, chronic skin reactions, such as atopic dermatitis may also occur. Other symptoms include nausea, cramping pains, bloating, vomiting and diarrhea. The delayed pattern of food allergy can be the cause of many chronic and disabling hypersensitivity diseases like asthma, allergy, rheumatic diseases, auto immune diseases, multiple sclerosis, diabetes, thyroiditis, psoriasis etc. Thus food allergy can be compared with many of the diseases caused by virudhahara.

b. Fatality due to Food allergy

Most reactions are short lived and relatively harmless. But an un common allergic reaction called anaphylaxis is a sudden severe potentially fatal systemic hyper sensitivity reaction that can involve various areas of the body such as skin, respiratory tract, gastro intestinal tract and cardiovascular system. The 'Sadyo marakatwa' of certain virudhahara can be explained through this phenomenon.

c. Exercise induced anaphylaxis

Eating allergic foods within 2 – 3 hrs of vigorous excercising may trigger anaphylactic reactions called as exercise – induced anaphylaxis. This condition may be life threatening. It may be the reason why Acharyas have advised not to take food after physical exertion (Avastha Virudha)

6. Food intolerance

It is an unpleasant and abnormal reaction to a chemical within a food. Some times the sufferer cannot identify the offending food and the condition is not psychological like neither food aversion, nor it is true immunological like food allergy. Most of the mechanisms of intolerences are unexplained, but some are due to inherited deficiencies of the enzymes needed for the efficient metabolism of specific chemicals for eg. Lactose (milk sugar) intolerance is due to deficiency of the enzyme lactase tyramine in chocolate can stimulate the production of histamines leads to pseudo allergic reactions in certain sensitive individuals. Dosha virudha may be explained through this phenomenon.

7. Food Borne Illness

Micro organisms of animal origin are frequently found in animal foods such as meat, poultry, fish and sea food. Bacteria break down the proteins into amino acids which are an important nutrient source for the bacteria. The onset of symptoms occurs two or more days after the contaminated food was taken. So many people fail to recognise the illness as food borne. The symptoms include nausea, vomiting, diarrhoea and fever. It may be the reason why Acharyas have said many virudhahara involving animal foods.

8. Toxins in food

Toxins in food are usually present in small concentrations. Raw foods like meat, fish, milk, vegetables grown on sewage are likely to be contaminated with harmful micro organisms. These are generally destroyed during cooking or processing of the food. However some toxins are heat stable. For example, staphylococcus can produce toxins that are not destroyed by high cooking temperatures. To prevent toxins from developing in food, don't leave food at room temperature for more than 2 hours. On a hot day (90^{o}F or higher), food should not be kept out for more than 1 hour. Paka Virudha, samskara virudha can be explained through this phenomenon.

9. Food adulteration

If the food stuff contains any other substances which affects or if the material is so processed as to affect injuriously the nature of subsance or quality there of is considered as adulteration. This is similar to samskara, paka and guna virudha.

If the foodstuff had been prepared, pack or kept under un sanitary conditions where by it has become contaminated or injurious to health. If the food stuff consists wholly or in part of any filthy, putrid disgusting rotten decomposed or diseased animal or vegetable substance or is insect infested or other wise unfit for human consumption.

10. Food additives

Food additives may be natural or artificial. Common natural additives include sugar, salt, corn syrup, baking soda and pepper. The most controversial additives are those which are completely synthetic and have no natural counter part. Food additives such as Meta bisulphate, Benzoic acid, Sorbic acid etc are added for preservation and to improve shelf life. If they are present above the prescribed safe limits it will be harmful for the body. This is similar to samskara virudha.

11. Toxicants naturally occuring in some foods.

Most toxins in foods are natural which may cause serious illness when consumed in large amounts. An important example is the legume Lathyrus sativus (Kesari dal), contain an active neurotoxic principle B(N) Oxalyl Amino Alanine [BOAA] which is present as free

amino acid in the seed cotyledons. BOAA has a predilection for the pyramidal tracts. When consumed in large amount it causes lathyrism (similar to matra virudha) some varieties of mushrooms contain toxic active principles like phalloidins, Amatoxins and Virotoxins which when consumed produce serious ill effects.

12. Toxic plant metabolites

Number plants undergo metabolic changes, which can result in the production and accumulation of abnormal and in some cases toxic, metabolites. This occurs when plants are subjected to a variety of stress conditions, most commonly due to fungal invasion.

Legumes contain compounds, which can inhibit the proteolytic activity of certain enzymes. For eg. Raw soyabeans contain trypsin inhibitor. The highest concentration of phytohemagglutinins are found in leguminosae and euphorbiaceae

Phytate (hexa phosphate of inositol) in the presence of calcium and magnesium forms insoluble complexes with iron and causing anemia. Unrefined cereals contain more phytate than refined or polished cereals which can be considered as samskara virudha.

Tannin is a condensed polyphenotoxic compound which is a potent inhibitor of iron absorption. They are also known to bind with proteins and reduce their availability. Removal of seed coat of legumes can reduce the tannin content.

13. Genetically engineered vegetables and fruits

Genetic engineering is inserting only specific characteristic (gene) to the living organism for further reproduction. Plants and animals are the most complex systems on earth, and no one gene is isolated from the others. Artificially changing just one gene could have adverse and random effects on the entire organism. The ability to splice genes between species brings up several ethical issued as well. The first issue concerns the use of human genes in transgenic plants and animals produced for human consumption. This leads to questions about cannibalism. Genetic engineering can actually result in unexpected allergens and toxins in food and reduced nutritional value.

The release of genetically engineered organisms into the environment may upset the ecology. The possible complications are extremely difficult to evaluate.

Allowing the release of GE products into nature is nothing less than blind experimentation with the environment with unknown and unpredictable consequences, especially as the released genes can not be recalled. These GE products can be considered as dosha virudha and samyoga virudha.

Protein hypersensitivities.

Theoretically any protein material may act as antigen and induce hypersensitivity. Most common allergies are reported by consuming following foods.

- Milk and its products e.g. curd, cheese
- Oil e.g. ground nut oil, cottonseed oil
- Cereals e.g. Rye, wheat
- Non vegetarian diet e.g. fishes, tuna varieties, beef, chicken
- Others e.g. Mushrooms, honey, sprouts
- Legumes e.g. beans, pea, ground nut
- Fruits e.g. pine apple, banana, citrus fruits, strawberries

Nutritional aspects of these materials reveal that presence of potent vaso-active amines like Histamine, phenyl thymine, serotonin and 5-hydroxyl tryptamine etc in substantial qualities may be responsible for allergic symptoms. Various combinations and adulterated food when consumed, will not produce potent and immediate effects. Prolonged consumption results in untoward effect. This may be due to following mechanisms

Synergistic effect-	A potent sudden effect, e.g. Ksheera + Cilicima mastya → *sadyamarana*, *Kapotamamsa* + *Sarsapa tailaa* → *Sadya Marana*
Additive effect-	A delayed and less harmful effect e.g. *Ksheera* + *lavana* → *Tvak vikaara*

Harmless in individual state- Harmful in combination e.g. Madhu, *ghritha, haritashaka, ksheera*

This may be due to

1) Conversion of partial heptanes to full antigenic substances, when combined with other or

2) Interference with digestive processes.

Now recent studies, dieticians say that Transfats are slow poisons that result in high incidence of diabetes and cardiovascular diseases among thirty plus individual. Tran fatty acids are unsaturated fatty acids with one double bond in transfiguration formed during partial hydrogenation of vegetable oil. The most common food items containing Transfats are Pizza, butter popcorn, French fries, cakes, cookies, pancake, chocolate bar and peanut butter. Besides traditional foods such as puri, paratha, bhatura, samosa and pakodas contain high level of TFA.Transfats are found in junk food items made with hydrogenated oil in an unregulated market are already a threat to youngsters. All cooking is done in Vanaspati which is a common adulterant used in edible oils. Amount of Transfats in vanaspati is extremely high which acts as slow poison.

Much importance is given in Ayurveda to avoid food combinations like

1) High protein and concentrated fat substances

2) Mixing of vegetable protein and animal protein together

3) Consuming sour fruits and sprouts e.g. germinated seeds

4) Combining milk along with high protein diet.

Based on chemistry of digestion and assimilation processes, following points can be interpreted –

When sour materials are consumed along with starches, enzyme ptyalin cannot digest starches in acidic medium resulting in fermentation. Citric, malic, oxalic acids found in fruits interfere with carbohydrate digestion in the stomach. Since the digestion process of

carbohydrates and proteins are very much different, rich quantities of them when consumed in different combinations together interfere with each another's digestion.

Fats decrease the gastric secretions and hamper digestion process of proteins when consumed together. Experiments have proved that heavy fat diet may reduce rate of digestion by more than 50%.

02.7 VISHA AS A CONCEPT OF HYPERSENSITIVITY

Visha an Overview

Derivation

he term 'Visha' has its origin from the Sanskrith dhatu 'Vishnu vyapthou' which denotes the spreading nature of visha. Another derivation is 'Visha viprayoge', which shows the capacity of visha to derange the body and mind.

Definition

Charaka has defined visha as 'that which has its origin from Ambu, and hence of dual nature, similar to Pavaka, having eight gunas and twenty four upakramas.[153]

In Sushruta Samhita another myth is given as originated from the wrath of Brahma.[154] Visha can be defined as a substance, by virtue of its ten gunas, can act against the Ojus, adversely affecting the body and mind.

Types of Visha

According to Susruthacharya [155] Carakacharya [156] there are two types of visha – sthavara and jangama Vagbhatacharya ,[157] also says about two types of visha (i) Akritrima visha – includes sthavara and jangama visha (ii) Kritrima visha – Gara visha is included in it. According to Sarngadharacharya, [158]visha is of three types sthavara, jangama and kritrima. Kritrima visha is again of two types – Gara and Dushi visha.

Visha is divided into two types – Krithrima (artificial) and Akrithrima (natural). Krithrima visha is Garavisha. Akrithrima is again divided into Sthavara (inanimate) and Jangama (animate). Sthavara visha are vegetable and mineral toxins. Jangama visha includes snakes, spiders, and rats' etc.[159]

Sthavara visha adhishtana

Among these, Sthavara (vegetable and mineral) has ten abodes such as root (moola), leaf (patra), flower (pushpa), fruit (phala), bark (tvak), hard wood or pith (sara) exudates or gum (niryasa), sap (ksheera), mineral ores (dhatu) rhizomes tubers (kandha).[160]

Jangama visha adhishtanas

Seats of Jangama visha are sixteen; viz, Sight (drushti), breathing (nisvasa), touch (sparsa), tusk, canine teeth (damshtra), mouth or teeth (mukha), nails, claws (nakha), bone (asthi), urine (mootra), excreta (pureesha), semen (sukra), menstrual blood (arthava), saliva (lala), hair (roma) bristle, stings (sooka) bile (pitha) and dead body (sava).[161]

TABLE-5 STHAVARA VISHA ADHISHTANA

Sthavara visha adhistanas	**Example**
Moola (Root)	*Kleetanaka, Ashwamaraka, Gunja, Sugandhaka, Gargaraka, Karkara, Karighataka etc.*
Patra (Leaf)	*Kalapatrika, Lamba, Varada, Karambha, Arka etc.*
phala (Fruit)	*Vallirenuaka, Karambha, Mahakarambha etc.*
Pushpa (Flower)	*Vallirenuaka, Karambha, Mahakarambha etc.*
Tvak (Bark)	*Karaka, Karaghata, Karambha, Mahakarambha, Naracha*
Ksheera (Sap)	*Kumudvati, Danti, Snuhi, Arka, Jalinee, Vyali etc.*
*Sara (*Pith)	*Kumudvati, Danti, Snuhi, Arka, Jalinee, Vyali etc.*
Niryasa (Exudates)	*Kumudvati, Danti, Snuhi, Arka, Jalinee, Vyali etc.*
Dhatu (Mineral ores)	*Haritala, Phenasma Bheda, and Rakta*
Kanda (Rhizome)	*Halahala, Kalakoota, Vatsanabha, Srungi, Sarshapa, Jalaka, Kardamaka, Vairataka, Mustaka, Mushkaka, Saktuka, Kraunchuka, Valaka, Mahavisha, Puṇdareeka, Galava, Moolaka, Marketaka, Karkataka, Karaveeraka, Indrayudha, Sankochaka, Langalaka, Tailapeya, Kusha Pushpaka, Pushpaka Rohisha, Anjanabhaka.*

TABLE 6 JANGAMA VISHA ADHISTANAS

Visha adhishtana	Example
Drushti (sight)	*Divya Sarpas*
Nisvasa (breathing)	*Divya Sarpas*
Sparsa(Touch)	*Lootha*
Damshtra (Canine teeth)	*Bhouma Sarpas , Marjara, Shvana, Vanara, Mandooka, lootha, paka matsya, etc.*
Mukha (mouth or teeth)	*Marjara, Shvana, Vanara, Mandooka*
Nakha (nails or claws)	*Marjara, Shvana, Vanara, Mandooka, paka matsya, snail, lootha, etc.*
Asthi (bone)	*Varati, fish*
Mootra (urine)	*Chipita, picchitaka, kasha, vasika, Sarshapika, lootha, Chitrasiras etc.*
Pureesha (excreta)	*Chipita, picchitaka, kasha, vasika, lootha, Sarshapika etc.*
Sukra (semen)	*Mouse, Lootha etc.*
Arthava (menstrual blood)	*Lootha*
Lala (saliva)	*Lootha*
Sooka (hair, bristle, sting)	*Vruschika, visabhara, varatika, ucchitinga, sea scorpion.*
Pitha (bile)	*Sakuli fish, rakta raji, varati fish.*
Roma (hair)	*Lootha*
Sava (Dead body)	*Sarpa, Keetas*

Visha gunas

Poisons, animate or inanimate possesses properties such as *teekshna* (penetrating), *ushna* (hot), *rooksha* (dry), viscid, *visada* (non unctuous), thin and entering into *sookshma* (minute pores) spreads all over the body quickly and then under goes *vyavayi* (processing), asukari (quick acting), *vikashi* (causes loosening of the joints and tissues), *laghu* (light), *avyakta rasa* (imperceptible in taste), With these properties, it remains *apaki* (indigestible).[162]

Visha karmas

By *teekshna* (sharp) and *ushna* (hot) properties poisons or toxins aggravate *pitha* and *rakta,* by *rooksha* (dry) property it aggravates *vata* (vayu), by *visada* (non-viscid) they do not adhere to any spot of the body due to their non-viscid nature. *Sookshma* (By fine) and *vyavayi* (diffusible) properties it enters into all the *doshas*, *dhatus* and *malas* and all the parts and the organs of the body; by *asukar*i (instantaneous) property it kills the patient quickly, by *vikashi* property it annihilate the doshas, *dhatus,* and *malas* due to their natural power of disintegration, by *laghu* (light) property it becomes difficult to remove it out of the body, by *avyakta rasa* (imperceptible to taste perception) property it aggravates *sleshma* (kapha) and follows (gets mixed up well) all of the taste goes away undetected of the foods. By *apaki* (indigestible) property, it does not undergo digestion and so surely kill the person who has consumed it. Though mitigated to some extent by the strength of mantras and *Oushadha,* obtaining an exciting cause it gets aggravated again.[163]

2.7.1 Definition of Gara Visha

Artificial poisons, also termed as Concocted poisons are prepared by the combination of Animate and Inanimate poisonous substances. They contain

- different body parts of insects
- blood, menstrual blood, urine, feces etc of human and animal origin
- medicines having diametrically opposing action on the same Physiological system
- incompatible foods
- ashes of metallic or mineral origin
- poisons of having low potency

According to *Charakacharya, Gara* is *Samyogaja visha* unlike that of *Sthavara* and *Jangama visha*. These artificial poisons are also named as *Gada* (disease producing).[164]

Sushrutacharya has mentioned while telling definition *Agadatantra.* He mentioned *Garavisha* as *Vividhavisha samyoga kritha*. It is a combination of different poisonous

substances.[165] while describing *Keeta kalpa* chapter*, Sushruta* mentions that *Keeta deha churnas* can be mixed with various *yogas*. External application of these *yogas* will produce symptoms similar to *Dooshivisha*.[166]

According to *Vaghbhata, Gara Visha* is a type of *Krithrima visha* prepared by various medicines.[167]

Commentators' View on Garavisha

Chakrapani commentary on *Charaka* explains that *Gara Visha* is combination of *savisha* and *nirvisha dravyas*. Always chirakaari rogajanaka does not prove fatal immediately.[168]

Vriddha Kashyapa opines the same view of *Chakrapani*.

Dalhana in his commentary on *Sushruta Samhita* tells the opinion of *Alambayana* in the context of *Garavisha* as follows - One should have knowledge about *Keetas* which are used for *Gara yogas* and their specific body parts which have more *visha veerya*. He further adds that *Keetas* not only produces symptoms of *visha* by bites alone but also in the form of symptoms of *dushi visha* when applied its body parts externally*[169]*

Indu in his commentary states that *Gara* is *Vishesha visha* (special type of visha) which is usually given along with food and it includes '*Sakalamapi vastujaatam*' i.e. combination of all substances.[170] Further *Indu* states that excretory products from different parts of the human body may also be included under preparation of *gara yogas*.

Madhukosha commentary on *Madhava Nidana* explains *Gara Visha* as the drugs with special properties like *Apaki* (nondigesting) and they will reside in *Jathara*.[171]

The opinion of *Bhavaprakasha* while explaining *Kritrima visha* is as follows: *Garavisha* are made out of non-poisonous substances. These substances will get *visha* symptoms by combination through its S*wabhavikatva*.[172]

Yogaratnakara opines as *Bhavaprakasha* in the following way that *gara* is a combination of *Savisha* and *Nirvisha dravyas.*[173]

In the text *Basavarajeeyam* by *Basavaraja, Gara Visha* is a type of *krithrima visha* which affects the person within a period of fortnight to a month.[174]

Mode of administration

According to *Vaghbhata,* the mode of administration of *Garavisha* is usually *Ahara sampruktha* (through food).[175] Commentator *Indu* gives clarification to this context saying *Gara* is special type of poison (*Vishesha Visha*); usually given with *Ahara*. Here the word *Ahara* comes first, but it does not mean only through food but includes all substances he uses daily *(Sakalamapi vastujaatam).*[176]

Sushruta has explained the modes of administration of *Gara yogas* through external application of powders prepared of different *yogas.*[177] In *Kalpasthana,* he has told the mode of administration of poisons, for which *Dalhana* comments as these are *Garavisha adhistanas.*[178] If the poison is going to be administered it should be easily administered and it should not be detectable easily. Sushruta while protecting the king from different modes of administration.[179] explains these things

The Garavisha Adhistanas are as follows:

- *Annapana (*foods that we take*)*
- *Dantakasta (*twigs used for brushing the teeth*)*
- *Abhyanga (*massage*)*
- *Avalekhana (Kesha prasadana* or the anointments *)*
- *Utsadana (*powders*)*
- *Kashaya (*decoctions*)*
- *Parisheka (*baths*)*
- *Anulepana (*after- pastes*)*
- *Srakshu(Pushpamaleshu* or garlands*)*
- *Vastra (*clothes*)*
- *Sayyeshu (*beds*)*
- *Kavachabharana (*armour, ornaments*)*
- *Padukapadapiteshu (*foot wears*)*
- *Pristeshu Gaja vajina (*back of elephants and horses*)*
- *Nasya (*snuff*)*
- *Dhooma (*smoking*)*
- *Anjana (*collyrium*)* etc.Symptoms of *Garavisha*

Symptoms of *Garavisha* explained in *Ayurveda* includes mainly

- Physical symptoms
- Psychological symptoms

Special symptoms of *Garavisha* in *Vridha vaghbhata*[180]

- *Garopahata pavaka*(Digestive fire affected by poison)
- *Garopahatatwacha* (Skin afflicted by poison)- This symptom told in *Yogaratnakara*[181]*Garenakshapitoujasam* (Loss of ojus due to artificial poison)

Psychological symptoms-

- *Swapnachintaparayana* (Drowsiness and worry)
- *Suskavanaspati jalasaya*(Dreams of dried river, withered trees etc)
- *Krishna* looks *Goura/Goura* looks *Krishna*(If he is of dark complexion, he dreams himself to be of white colour and vice versa)
- *Vikarnanasanayanam* (He dreams himself to be without ears and nose, to be with distorted sensory organs)
- *Vihatendriya* (Disturbance in functioning of sense organs)
- *Swapne gomaya marjara nakula vyala vanara* etc(Dreams of cats, jackals, wild animals, mangoose and monkeys)

Table-7 Symptoms Of Garavisha [182,183, 184.]

Somatic symptoms	Ashtanga hrudayam/sangaham	Yogaratna-karam/bhavaprakasam	Kriyakoumudi	Visha vaidya tharangini	Bhasha-vishavaidya kalika	Vishavaidya sam-graham/
Pandu	✓	✓	✓	✓	✓	✓
Krisatha	✓	✓	✓	✓	✓	✓
Dourbalyam	✓		✓	✓		✓
Alpagni	✓	✓	✓	✓		✓
Kasa swasa	✓		✓			✓
Jwara	✓	✓				✓

Chardi	✓				✓	✓
Arditha	✓					✓
Prathiloma vayu	✓		✓			
Swapnac-Hinthapa-rayanam	✓		✓			✓
Mahodaram	✓	✓	✓	✓	✓	✓
Yakritodaram& pleehodaram	✓		✓	✓	✓	✓
Deenavak	✓		✓	✓	✓	✓
Alasa	✓		✓	✓	✓	✓
Sopham	✓	✓	✓	✓	✓	✓
Satatadmatha	✓		✓	✓	✓	✓
Diarrhoea with colour change	✓	✓	✓	✓		✓
Romachuthi	✓		✓	✓	✓	✓
Romaharsha	✓		✓	✓	✓	✓
Rashes formation	✓		✓	✓	✓	✓
Excess Salivation	✓		✓	✓	✓	✓
Karacharana sosham	✓	✓	✓	✓	✓	✓
Mental symptoms						
Seeing animals like ongoo-se,snake & dried trees & rivers in dreams	✓		✓	✓ ✓	✓ ✓	✓ ✓
Feeling of having						

Irregularity of ears,nose etc.	✓		✓	✓	✓	✓
Pandu	✓	✓	✓	✓	✓	✓
Krisatha	✓	✓	✓	✓	✓	✓

Action of *Gara Visha*

Action of Garavisha in samhita is explained in two ways according to the administration of poison both externally and internal.

Action of Garavisha after intake or exposure to Garavisha mainly depends on combination of poisons, dose and root of administration. Action of Garavisha when it is applied through external skin by different churnas (Keeta deha Churnas) mixed with yogas, symptoms caused, are explained in Sushruta samhita in Keeta kalpa chapter.[80]. In this condition, action of Garavisha may be through transdermal absorption of Garayogas causing symptoms of Visha. Action of *Garavisha* after entering our body explained in the pathogenesis of *Udara* roga *samprapthi* of *Sannipatodar*a, *baddodara* and *chidrodhara* respectively. Here in *Udara roga* main *nidana* is intake of *Gara* (*stridatta rajo mala, pakshmavalai*) along with food.[185]

In samprapti chapter, it is discussed in detail.

Psychological actions of Garavisha

The definition of visha indicates that which produces Vishaada. Here, vishaada meaning manovikaara or chittaavasaada.[186, 187.] any visha dravyas having ten qualities specially vyavahi, vikaasi, suksma and tiksna etc these qualities swift in action affects the body and mind quickly. These visha gunas are opposite to ojus. Poisons like Gara Visha (artificial poisons) can influence human mind very intensely.

This can be broadly classified into two

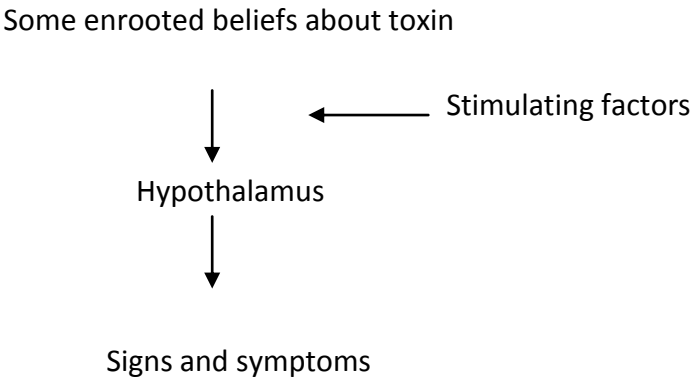
1. Direct toxic effects
2. Indirect toxic effect

Direct toxic effects

Generally, poisons are potent and they can be absorbed quickly. Upavishas, comparatively slow poisons in broader sense can be considered under Gara Visha have action on CNS which will affect the function of mind. Euphoria, confusional psychosis and non specific personality changes etc are some of the examples. In Ayurvedic, samhita there is explanation of treatment Hridayaavarana that is mainly for Chitte visha vyapthi prtisheda according to Dalhana.[188] this shows Samhitas give more importance to psychological aspect while treating poisons.

Indirect toxic effects

Some enrooted beliefs of people such as poison given to them by others etc. along with some stimulating factors, causes stimulation of hypothalamus. There are some specific centers in hypothalamus to regulate or to modulate different activities in our body. They have crucial role in body functions like circulation, fluid and electrolyte balance, temperature regulations, feeling of hunger and thirst. So, stimulation of nuclei may result in various somatic manifestations, as a general body mechanism. Some patients may have false belief that their disease is due to ingestion of toxic materials. They are obsessed that they were poisoned in deceit. In many cases, more than toxic effect, fear and anxiety causes much severe symptoms.



In Ayurveda it is clearly explained that psychological action of poison in the context of Shanka Visha (Dubious poison) is a clinical condition generated by fear alone when a person is pricked by something in darkness, as he assumes that he is bitten by a snake and exhibits signs and symptoms accordingly. The appearance of symptoms of dubious

poisoning may be psychological reaction. Same mechanism holds well in Gara Visha aspect also. In this condition treatment is psychological.

Cumulative period

Yogaratnakara mentioned Garavisha will affect the person after fifteen days or Basavarajeeyam tells month period.181 same opinion in 23rd chapter.

2.7.2 DUSHIVISHA

Etymology

The word *Dushivisha* comprises of two words "*Dushi"* and "*Visha"*. "*Dushi*" means altered, denatured, attenuated, latent, vitiated and "*Visha*" means poison [189]

It is derived form the word "*Dush*" which means polluting or defiling,

Definition

Any poison that is having low potency as compared to that of the natural ten properties of *Visha*, incapable of producing symptoms of poisoning is called Dushivisha.[190]

Chakrapandatta defines *Dushivisha* as *"Kalantara prakopi visham Dushivisham*" ie, the poison which manifests its effects after sometime.

Dushivisha is not a type of *Visha* like gara but it is only a transformational characterstic of *Visha.*[191]

Dushivisha is any kind of poison originating from inanimate or animate sources, any artificial poison (kritrima visha) retained in the body after partial expulsion, or which has provisionally undergone detoxification, by the antipoisonous drugs, forest fire, the wind or the sun.[192]

A constant exposure to that particular time (i.e. time is meant a cloudy and windy day, as well as rainy season), place (i.e. place is meant a *Anupa Desha*, extensive windy cold rainy place) and diet (i.e. diet is meant wine, sesame, *kulatha,* pulse) as well as constant and regular day sleep tends to vitiate the *dhatus* (fundamental root principal) of the body and this poison is consequently known as *dooshi visha*.

It can be concluded that along with *Sthavara*, *Jangama visha* and *Gara Visha* depending on the stages these could become *dooshi visha*. Though *Gara Visha* is slow acting in nature, it can affect like *dooshi visha*. But the severity of the *Gara Visha* will vary depending on the potency of the combination of the poisonous drugs. But *dooshi visha* will be always a slow acting in nature, as it is entangled by *kapha*, causing the discomfort to body by residing in the body years together.

Both *Gara* and *dooshi visha* are late in action both digest latter and remain in the body for a long time. *Dooshi visha* looses its potency in cases of sunlight, air , and heat . Acute symptoms occur only in *Gara Visha* because it is artificially prepared by combination of drugs.

Transformation of *Visha* into *Dushivisha*[193]

***Jeernam* (partially metabolised)**

"*Dehadshesam yat nirgirati tat jeernam*". Any type of *Visha (sthavaram*, *jangamam, kritriman*) which is not completely eliminated from the body or partially detoxified and being deposited and accumulated in the body at various *dhatus* producing cumulative effect. When *Visha* which is capable of producing acute ill effects is kept for a long time some of them may loose their original *gunas* and ultimately gets converted into low potency *Visha* leading to formation of *Dushivisha.* [194]

Vishaghnoshadibhir hatam

It means partial detoxification by antidotes or incomplete metabolism of poison that also retains some properties (Detoxification is the chemical process occurring in the body, which convert toxic substances to non-toxic substances).

Davagni

Exposure to intense heat and fire alters the property of *Visha* resulting in reduced potency of it and hence leading to the formation of *Dushivisha.*

Exposure to *vata*

Exposure to *sheetala vata* (cold wind) dries up the water content of *Visha* and reduces some of the natural properties like *teekshna, ushna* etc and makes it less potent.

Exposure to *Aatapa*

Exposure to sun rays (especially UV radiations– which disinfects air, water and even poisonous substances) causes the detoxification process to get accelerated and reduces the potency of the poison.

Swabhavato va swagunair na yuktham visham

Naturally all *Visha (sthavaram, jangamam, kritrimam*) have all the classical ten properties like *teekshna, ushna, rooksha* etc. Presence of these properties can cause acute or sub-acute poisoning. But if any one or more of these properties are absent in a particular poison, it can become *Dushivisha* that is of low potency by nature. Some substances are naturally less potent and produce symptoms of latent poison. *Charaka* has classified *keetas* into *Pranahara keeta* and *Dushivisha keeta*. Commentator *Chakrapani* has commended on *Dushivisha keeta* as *alpa visha keeta*.

Properties

Any poisons that are not having natural ten properties of *visha*, producing only chronic symptoms is known as *Dooshivisha*[195].

Dalhana, the commentator of *Susruta Samhita*, clarifies this statement as [196]

A poison, which is having fewer properties, which means less than ten classical properties that actually a poison should have, or either the poison, which is having lesser potency of all the ten properties, attains a latent or hidden stage in the body called Latent poison (*Dooshivisha*). Low potency of all the ten qualities is said to be responsible for the delayed action and cumulative toxicity on the body.

Acc to *Charaka* another main characteristic feature of *Dooshivisha* is *Bahudoshakaratvam* and is not going to take away the *Prana* .[197] Again, *Charaka* has said they are two types of *Keetas*

- *Dooshivisha Keeta*
- *Pranaharakeetas*

Even he told *Dooshivisha Loota* and *Dooshivisha Mushika* [198]

Susruta has said in the contest of Garavisha that external application of *Keeta deha churnas* along with *yogas* will produce symptoms similar to *Dooshivisha*.

Aetilogy (*Nidana*)

A poison either *Sthavara*, *Jangama* or *kritrima,* whenever not fully eliminated from body and attenuated by antipoisonous remedies or gets dried up by d*avagni*, *vata, atapa* (the fire, the wind and the sun) or when the foresaid natural ten qualities of poison becomes less potent is called as dooshi visha. Because of its mild potency does not prove fatal for an individual and as it get enveloped by the *kapha* it resides in the body for many years.

Aggrevating factors of *Dushivisha* [199]

The factors that aggravate Dushivisha can be divided into two groups as follows:

Exogenic factors Endogenic factors

As per *Ayurveda Sabdakosam "prakvata*" means *'poorvadi gatho vata'*. It is considered as a factor for *tridosha dushti*. During *Sarath ritu prakvata* is to be avoided where pitta is in *prakopa* state. Thus *prakvat*a is more *pitta pradushaka* and should be avoided in *Visha. Sushrutacharya*[200] says that it aggrevates *raktha* and *pitta* and aggrevates disease in a patient suffering from wound, ulcer, or any kind of poison.[201]

Ahitadesam – According to *Dalhanacharya* the *desa* which aggrevate *Dushivisha* is *anoopa desa*. In *anoopa desa kapha* and *vata* are more prominent and may promote the activity of *Dushivisha.*[202]

Ahitakalam – The *kala* which aggrevate *Dushivisha* are *varsha ritu*, *seeta kala* and *durdina* ie cloudy wind and cloudy days. In *varsha ritu* rain makes body klinna, digestive power is reduced, vata is in *prakopa* state*, pitta* also gets vitiated. All these lead to aggrevation of *Dushivisha.*

Ahita Annam*:* The *ahara* which aggrevates *Dushivisha* include alchohol, sesame oil, horse gram etc. T*eekshna* and *vidahi dravyas* will also enhance the potency of *visha.* Activities like day sleep, exercise, sexual act, suppression of natural urges, anger, grief etc will also provocate *Dushivisha*

***Dushivisha* – Different opinions**

According to ***Madhava Nidana Madhukosa* commentary** *Seetanila* (cold wind) *durdina* (cloudy days) are considered as aggravating factors of *Dushivisha* and causes skin lesions such as *visarpa, visphota* etc. It is said as "*Kshapayet cha shukram iti shandyam karoti*" ie, it causes sterility.[203]

According to *Bhavaprakasa*

Bhavaprakasa clarifies some of the points like *'kaphanvitam'* means "*kaphena mandeekritha ushnadi gunam*", which means that the potency of *gunas* like *ushna, sookshma, rooksha* etc are reduced by *kapha.* '*Varshagananubandi*' means *"kaphena agni mandiditwat apakat chirasthayi*".The *avarana* of *kapha* causes *agnimandhya* (indigestion) and *dhatwagni mandya* (defective metabolism) occurs which then causes *apakata of visha* and it stays for a longer time in the body without producing any signs and symptoms.[204]

According to *Yogarantnakara*

Kritima visha (artificial poison) is of two types, one is *Dushivisha* (latent poison) formed by mixing *savisha dravyas* (toxic components) and another is *Gara visha* formed by mixing *nirvisha dravyas* (non-toxic components). All the concepts of *Dushivisha* are exactly similar to that of *Sushruta.*

***Poorva roopa* of *Dushivisha*[205] (Prodromal symptoms)**

- ✓ *Nidra* (sleepiness)
- ✓ *Vijrimbanam (*yawning)
- ✓ *Harsham (*Horripilation)
- ✓ *Avipaka (Indigestion)*
- ✓ *Mandalam* (eruption of circular patches on skin)
- ✓ *Moham* (fainting)

- ✓ *Padakarasyasopham (Swelling of face and extremities)*
- ✓ *Dakodaram* (Ascites) *Chardi* (Vomiting) *Moorcha Vishama jwaram*
- ✓ *Prabalam trisha Unmadam Anaham (*Constipation of bowels)
- ✓ *Kshaypayati shukram (*Involuntary emission of semen*)*
- ✓ *Kushtam* (Skin diseases)
- ✓ *Gurutvam (*heaviness of body)
- ✓ *Vishlesham* (looseness in joints*)*
- ✓ *Angamardam (*aching of limbs)
- ✓ *Arochakam (*disrelish for food*)*
- ✓ *Kota(* urticaria)
- ✓ *Dhatu kshayam* (loss of vital *principles of organism)*

Pathogenesis of *Dushivisha*

Nidana sevanam (*Sthavara visham, jangama visham, kritrima visham)* (Failure of complete elimination) Acumulation of toxin at the *ashaya* or *dhatu* level (*chaya) (prakopa)* Toxins get *kapha avarana* Sub-clinical condition Exposure to secondary aggrevating factors Vitiation of *tridoshas* and *dhatus* (*dosha dooshya sammurchana) Poorvarapa* of *Dushivisha (sthana samsrayam)*

Roopa depending upon the *dhatu Rasa, Rakta, Mamsa ……Sukra (vyakthi) Kushta, Visarpa etc Kitibha Kushtam (bhedam) (prasara*)

Roopas of *Dushivisha*

Symptoms of *Dushivisha* according to the site of their location.[206]

Amasaya : Symptoms of deranged *kapha* and *vata doshas*, ie, unconsciousness, vomiting, diarrhoea, giddiness, burning sensation, tremors, altered sensorium.

Pakvasaya : Symptoms of deranged *vata* and *pitta doshas* ie, burning sensation all over the body, fainting diarrhoea, loss of hair, and strength

PROGNOSIS OF LATENT POISON (*DOOSHIVISHA*)

Dooshivisha in the prudent and in early cases of poisoning is curable. Cases of one year's standing become relievable, while even this type of poisoning in an enfeebled and

imprudent patient, who is taking unwholesome food, should be considered to be incurable. [207]

Author *Bhavamisra,* in his work *Bhavaprakasha* has also agreed to the same explanation as of Sage *Sushruta*. The commentator has clarified some of the points like '*kaphavrutam*' means *kaphena mandeekrta ushnadigunam*-, which means that the potency of hot (*ushna)*, minute (*sukshma)*, and dry (*rooksha*) etc gunas are reduced by *kapha*. *varshaganubandhi'* means *kaphena agnermandyaditwat apakat chirasthayi*'. It means because of *kapha dosha avarana* defective digestion (*agnimandya*) and defective metabolism (*dhatwagni mandya*) occurs in turn leads to *apakata* of latent poison (*Dooshivisha)* and stays for long time in the body with out producing any signs and symptoms. [204]

Yogaratnakara in *vishadikara* explained a different opinion. He considers *Dooshivisha* under *Garavisha* classification.

Artificial poison *(krtrima visha*) is of two types, one is latent poison (*Dooshivisha)* formed by mixing toxic components (*Savisha Dravyas*) another is *Garavisha* formed by non-toxic components (*Nirvisha Dravyas*). *Kriyakoumadi* author says that even external application of medications may transform into *Dooshivisha* after initial absorption. He adds that *mala, mootra* and *artav*a which are not properly discharged from the body may become *Dooshivisha.*

Diagnosis of *Dooshivisha*

Diagnosis of *Dooshivisha* and *Garavisha* is a difficult task. Identifying the difference between these two concepts is also difficult. But some of the traditional books in *Agadatantra* explained diagnosis of *Dooshivisha* (*Visha chikitsa* of *Vaidya vachaspati M.balakrishnan nair*),

Some of the specific *lakshana*s are helpful to understand the presence of *Dooshivisha in* the body. By careful examination of the *Nethra, vadana, dantha, roma,* and *charma*

1) *Nethralakshana*- In *dooshivisha rogis,* the *nethramoola* ie *kannenaka* will be blood red in colour. Other parts of *suklamandala* will be white in colour.

2) *Vadanalakshana*- On their faces, black bindus (Spots) or discolouration will appear.

3) *Dantalakshana*- By keen observation, instead of white colour a *syava varna* is seen.

4) *Romalakshana*-The normal color of *roma* is changed into white or coppery.

5) *Charma lakshana*- In *charma*, various *visharpa* or *pama* or *dadru kushta* are seen.

Among these, if any one or two *lakshanas* are seen, then, one can doubt the presence of *dooshivisha.* If all five *lakshanas* are seen, it is confirmed that *Dooshivisha* is present in the body. *Garavisha* is combination of poisonous and non poisonous substance.*Dooshivisha* is always a combination of poisonous substance. Even non poisonous substance can act as poison in the contest of Garavisha due to their combination. These substances before combination are *Avisha dravyas. A*fter combination they become potent poison. There is difference between these two concepts explained separately by *acharyas*

DUSHIVISHA – Modern concept

It is very difficult to define *Dushivisha* (latent poison) exactly and accurately by any modern terms. Hence an attempt is done here to put forward some of the modern terms which are close to the concept of *Dushivisha.*

ALLERGY / HYPERSENSITIVETY

Allergy refers to an exaggerated reaction by our immune system in response to bodily contact with certain foreign substance that is usually harmless. The substances that often cause reaction are pollen, dust, pet dander, food, insectsting, medicine etc. The terms allergy and hypersensitivity are synonymous. Allergy is one of the four forms of hypersensitivity and is called type I (Immediate) hypersensitivity. Allergies can cause a running nose, sneezing, itching, rashes, swelling or asthma. Allergies won't become fatal, but only create some sort of discomfort in the body. However a severe reaction called anaphylaxis is life threatening. Hypersensitivity refers to undesirable (damaging, discomfort producing and sometimes fatal) reactions produced by the normal immune system. There are four basic types of hypersensitivity

1. Type I (Immediate hypersensitivity) reaction

2. Type II (Cytotoxic hypersensitivity) reaction

3. Type III (Immuno complex) reaction

4. Type IV (Delayed hypersensitivity / Cell mediated) reaction

- ✓ **Cumulative toxicity**

Cumulative toxicity is associated with agents whose residence time in the body for outlast their duration of effective concentration.This category include agents with biological half lives measured in days, weeks or months instead of hours where concentration may fall to ineffective level long before the drugs disappear from the body. Such agents also have the expected propensity for slowly accumulating upon repeated exposure even to small quantities until the total amount in the body is sufficient to produce toxicity however, cumulative toxicity is associated with certain agents whose duration of action is relatively short. Agents in this category are highly lipid soluble and have their sites of action within tissues that are rapidly perfused with blood. Redistribution from *Dushivisha – Modern concept -31* these sites to the tissues receiving lower rates of blood flow leads to the termination of action, even though considerable quantities of drug still remains in the body. This is well exemplified by certain metals like lead. Individuals living in industrialized

countries are exposed to lead in air and food. 5-10% is absorbed from GIT. 30-50% inspired with air finds its way to blood stream. When rate of entry exceeds the rate of elimination excess lead is stored in various tissues such as kidney, hair and bone. It inhibits essential enzyme processes.

- ✓ **Delayed toxicity**

It is a predictable adverse drug reaction. Keratosis or raindrop pigmentation of palms following arsenic ingestion is examples.

- ✓ **Drug interaction**

It is an adverse drug reaction Interaction can take place when two or more drugs are given simultaneously. The skin is one of the most important targets for adverse drug reaction. The lesions may appear as lichenoid, pustular, bullous reactions. In case of patients taking NSAID, antibiotics and anticonvulsants 1-5% may develop a skin eruption.

- ✓ **Intolerance**

It is the appearance of characteristic toxic effect of a drug in an individual at therapeutic doses due to the low threshold to the drug.

- ✓ **Carcinogenicity and mutagenicity**

It refers to the capacity of drug to cause cancer and genetic defects due to the production of reaction intermediates. Chemical carcinogen takes several (10-40) years to develop.

- ✓ **Free radicals**

These are highly reactive unstable species that can interact with proteins, lipids and carbohydrates and are involved in cellular injury induced by a variety of chemical and biological effects. They produce serious damage in all physiological systems by causing both ageing and diseases like cancer, atherosclerosis, rheumatoid arthritis etc. Free radicals have two fold action-slow cumulative damage or disastrous crisis. *Dushivisha – Modern concept - 32*

- ✓ **Acute toxicity**

It occurs almost immediately after an exposure. An acute exposure is a single dose or a series of doses received within 24 hour period. Death is a major concern in cases of acute exposure. Many people die each year from inhaling carbon monoxide from faulty heaters.

- ✓ **Chronic toxicity**

It represents cumulative damage to specific organ systems and takes many months or years to become a recognizable clinical disease.

- ✓ **Auto immunity**

This is the failure of an organism to recognize its own constituent parts as self which results in an immune response against its own cells and tissues. Any disease that results from such an aberrant immune response is termed as autoimmune disease. It has been established that an essential pre-requisite for the development of autoimmune disease is the breakage of immunological tolerance. Certain individuals are genetically susceptible to the development of autoimmune disease. Certain chemical agents and drugs can also be assosciated with the genesis of autoimmune disease. Over exposure to pesticides and toxins may also induce autoimmunity.

- ✓ **De toxification**

This is the process by which toxic substances are rapidly made excretable through different biochemical changes. Liver is the major site of detoxification and done by the processess of oxidation, hydrolysis, reduction and conjugation. A large member of foreign substances are metabolized in the body by oxidation. Many primary aliphatic amines are detoxified in the liver with the formation of corresponding acid and nitrogen is converted into urea. The sulphur of many of the organic compounds are oxidized to sulphates of varying degrees. Some detoxification process is also taking place in the effector cells of kidney. In a cell the smooth endoplasmic reticulum (SSER) contains an enzyme mixed function oxidase system which plays a major part in the detoxification mechanism with or without the help of mitochondria. *Dushivisha – Modern concept -33* it is proposed that toxins could be bio transformed in two phases. Functionalization that uses oxygen to form a reactive site and conjugation that results in addition of a water-soluble group to the reactive site. These two steps are termed as phase 1 and phase 11 detoxification respectively. The result is the biotransformation of a lipophilic compound not able to be excreted in urine to a water soluble compound able to be excreted in urine. If any failure or partial functioning of this detoxification process occurs it will result in accumulation of metabolites and affect the normal physiological function of the body and result in various diseases. This accumulation first remains as latent (dormant, in active) and on suitable conditions it manifests disease symptoms. This is also known as denatured poison. In the process of detoxification either partially or completely cell undergoes degeneration mechanism which is either reversible or irreversible. Irreversible may cause necrosis of the cell. Reversible degeneration is of two varieties, late and early reversible degeneration. On implementing latent poison the kind of

degeneration manifested is of former type (reversible degeneration). *Dushivisha – Modern concept -34.*

02.8 SATMYASATMYA VIKARAS AS A CONCEPT OF HYPERSENSITIVITY

Allergy and intolerance are explained in Ayurvedic literature as the concept of asatmya. “Aushadha anna viharanam upayogam sukhavaham sahi satmyam” is an ancient Ayurvedic phrase that means when a person is exposed to medicines, diet, lifestyle, and environmental changes, the body can adjust and accept these things[208]. This is tolerance (satmya). Conversely, intolerance (asatmya) happens when the body can no longer accept the changes and an adverse reaction occurs. Intolerance and allergy are both conditions of hypersensitivity, a reaction of the body to factors that it can no longer deal with in a healthy way.

The concept of *Satmya* and *Asatmya* is clearly an indication of immunity and allergy. Here *Satmya* means Compatibility. The S*atmya* may be towards the Medicine or to the Food. When *Satmya* is quoted for food, it indicates that the food which is practiced for long duration helps the person to develop his Immune system. In the same way there is one more concept called “*Oka Satmya*”. That is compatibility attained through slow adaptation mechanism. That is a drug/ food is given in daily basis in smaller quantity for prolonged period, so that the person is gradually developing adaptability towards it, even though it is incompatible to him previously. The matter of *Asatmya* means Non compatible to body. I.e. if a medicine or food is not acceptable to the body, body tries to through it away in different fashions. It may be Diarrhea, Vomiting, and Skin manifestations and so on. Hence the treatment runs according to the presenting complaint.

Ritu Sandhi

• It is clearly described by Vagbhat[209], which, indicates that a specific “Dinacharya” is to be followed in that particular period.

• Duration – Ritu Sandhi is a period of 14 days between two consecutive seasons

• In Ayurveda, a specific diet regimen (Ahara & Vihara) is indicated for each & every season under the heading of 'Ritucharya.'[210]

• Indications & contraindications for each season are described which should be followed strictly in order to remain healthy.

• Our Acharayas have well planned the concept of Ritucharya by considering the proportions of Tridosha, Panchamahabhoota, Rasapanchaka, etc. in various seasons & its adverse effect on a human body.

• Similarly prior adopting Ritucharya of the coming season, there are certain rules regarding the abandonment of the Ritucharya of previous season. These rules are mentioned under 'Ritu sandhi'.

Adverse effect If "Do's & Don'ts" of Ritu Sandhi are not followed properly than it leads to:

• "Asatmyaja Roga[211]

• Tridosha Prakopa, which is the main nidana for almost all the diseases.[212]

• Vitiation of Dhatus, giving rise to Dhatu Pradoshaja Vikara[213]

In the same context, Acharya Sushruta has also mentioned that, due to toxic Vayu following disease are caused[214].

• Kasa

• Shwasa

• Pratishyaya

• Shiroroga

• Netraroga

All the above diseases are included under allergic disease in modern texts. The concept of 'Asatmjaja Vyadhi' is very scientific. All factors are favorable in the period of 'Ritu Sandhi' for a disease to manifest itself. Following environmental factors described in the Modern pathology for allergic diseases can be compared with 'Ritu Sandhi' period & hence it can be said that these diseases has direct relation with Ritu Sandhi.

In preventive & social medicine, it is mentioned that health status of an individual, a community or a nation is determined by the interplay & integration of two ecological universes. The internal environment of man in himself and the external environment, which surrounds him.

In the modern concept, disease is due to a disturbance in the delicate balance between man & his environment. Three ecological factors, Agent, Host & Environment are responsible for the disease. But the environment from which the patient comes is largely unknown. From this a positive support is obtained to scientific concept of 'Ritu Sandhi', further more. Mites of the genus Dermatophagoides have been shown to the commonest arthropods present in the dusts of foods, mattresses, carpets & upholstered furniture, etc. by several authors all over the world.

Three main factors, which influence the population of house dust mites, are –

The temperature,

Relative humidity of the air,

Food material,

They seem to be most abundant in those areas of the world where the climate is damp & temperate as compared to dry & cold climatic regions. Relative humidity >65% & <30% are unpleasant. Permanent exposure to such low humidity can cause drying of the nasal mucosa, which may predispose to infection (viz. – Sore throat, cough). Research on 'Seasonal periodicity of House dust mite population' published in "Aspects of Allergy & Applied Immunology." Showed following conclusions.

• Maximum positivity was found in the months of August & September & minimum in the months of March, April & May.

• Indicating that the mite population in house dusts show a periodic increase & decrease with the change of season.

• It appears that more rapid growth of these organisms occurs during a period when the temperature is moderate & the relative humidity is high & a slow growth or no growth when it is relatively dry plus either too hot or too cold.

When we make our selves to under go a task, we toil for days and nights to explore pros and cons of the problem, strive for solutions and at the end of the road look back for our achievements, failures; imposing a question mark for ourselves and striving with their answer for the people, this is called discussion.

02.9 HEREDITARY DISEASES AS A CONCEPT OF HYPERSENSITIVITY

- Acharya Sushruta has clearly defined hereditary diseases[215]
- Causative factor- Vitiation of sperm & ovum
- Chances of Allergic disorders increases in bilateral inheritance
- Unilateral inheritance decreases the chances of Allergic disorders.
- Acharya Charaka has described it as "Kulaja Roga"[216]
- Acharya Vagabhata has described it as "Kulaja" & "Kulodbhava"[217]

In the nutshell a healthy sperm & ovum gives rise to a healthy progeny.According to an Ayurvedic principle there is a balance of Vata, Pitta & Kapha within the body of each & every individual. Its proportion (Qualitatively & Quantitatively) is fixed at the time of birth i.e. its Tara & Tama bhava. On the bases of Dosha pradhanata, Prakriti (Sharira & Manasa) of an individual is formed. All these factors play an important role in forming ones immunity. Acharya Charaka[218] has described 6 varieties of Prakriti in individuals. From which the first 2 i.e. "Jatiprasakta" & "Kulaprasakta" may throw light on this view. "Jatiprasakta"- in some races there is tendency for hypersensitivity e.g. a "Bhanushali" race of Jamnagar is highly sensitive to certain drugs, which are commonly used. "Kulaprasakta"i.e. Family disposition. This can be related with chromosomal abnormalities. There is genetic inheritance, influencing the future generation. The above explanations may be enough to throw light on the role of heredity in the manifestation of allergic diseases.

Viruddhahara act as Garavisha - its possible mechanism

Most of the people depend on fat diet especially non-vegetarian diet and neglecting vegetarian diet rich in protein and carbohydrate. It is known that people eating more protein and carbohydrate rich diets get protection against toxicity,While fat diet make them victims of toxicity. Another reason may be intake of *viruddhahara* in many forms like junk food, adulterated food which are more acidic in property. It may cause disturbance of ph level in the body and finally decrease of ph level. Decrease in ph level increases the susceptibility to diseases. So, our food should maintain ph of the body which helps to increase immunity and thereby lessens the chance of disease.

Clinical application of *Viruddhahara* (Incompatible food) and *Garavisha*

It is interesting to know that some incompatibles foods and drugs act as a *Rasayana.* In *Kriyakoumad*i Sri V.M.Kuttikrishnan, menon mentions that the incompatible combination of buttermilk (*Takra*) with unripe plantain (*Kadali*-Musa paradisiacal) administered to babies, who instead of causing harm, develops the immune power. In *Astanga Hridaya* there is indirect reference of using incompatible drugs that is administration of herbal drugs along with Madhu, *Ghrita, Swarna, a combination* to newborn baby as an immune booster.

Astanga sangraha dedicated separate chapter for indication of different visha (poison) *prayoga* in various disorders[219] *Indu,* a commentator in this context explains that there is no rule like prativisha could be used in Visha condition only. However, *Visha* can be used successfully in many diseases which were not responding to other treatments. Those people who are in search of *Rasayan*a (Rejuvenation) can also use poison (*Visha*) regularly.

Common diseases which are indicated for *Visha Prayoga*- [220]

- Haritala Prayoga after shodhana with Gomutra – This poison can be used as Brihmana, Sannipataja jwara etc.
- Bhallatakaadi yoga along with Visha – Vicharchika
- Rasanjana along with Visha – *Dustavrina*,
- *Sannipatajwra*, *Raktapitta*, *Dustavrina*, *Udavarta*, *Kusta, Vicharachika* etc. are other diseases where visha can be used as Rasayana.

Gara Visha v/s Dooshi visha

In the Ayurveda, the visha is classified into three types that are *Sthavara, Jangama* and *Gara Visha*. There is no separate classification of the *dooshi visha* but Ayurveda has defined it. Sthavara, *Jangama visha* or *kritrima visha* after treatment, may become less potent and when their effects are not nullified radically because of which, they resides in the body. That particular less potent part of the above said poisons is called *dooshi visha*. Dooshi visha is a residual poison. Not an independent variety of visha. It is not included under any classification. But it is an *avasthabhed*a of *Sthavara*, *Jangam*a or *kritrima visha*. This *visha* gives trouble to patient frequently.

Difference between Dooshivisha and Garavisha

Dooshivisha	*Garavisha*
1. It is *Avasthabheda* or *avisista visha* of *Sthavara, Jangama* or *Krithrima visha*	1. It is an artificially prepared poison.
2. It is always late in action.(*Varshagananubhandhi)*	2. Acute or late in action according to its Combination
3. It is a residual poison and not Included under any classification.	3. It is an independent poison.
4. It is *Savisha dravyas samyoga* only.	4. It is combination of *Savisha* and *Nirvisha dravya*

Viruddhahara* V/S *Garavisha

Panchamahabhutas (Five elements) are very essential for the continuous existence of man. Therefore to man, these elements are his natural food. This is the real *Annam* (Food) of man. *Annath Bhavathi Bhootani (Thaitiriyopanishad)* which means all living beings are made up of food they eat. The scriptures say that this is 'Annam *Brahmeti Vyanjante*' which means, food is renowned as God. The world famous psychologist Sigmund Freud said, 'you are what you eat'. The food, which contains all these five elements, can be considered as divine food.

Incompability to various foods is common complaint now days. Main reason for this may be Indians are undergoing rapid nutrition transition, when they are moving from traditional food to western food. Foods which are prepared with innovative methods and combination neglecting traditional methods are also responsible. Today what we are taking as food, every item of it is posing a threat. Adulteration may also be a predisposing factor.

Food is responsible for stability of the body by maintaining *Antaragni* (*Jatharagni*) of a person. Because ….*Agnimoolam cha dehadaranamiti…*[221]. This *jatharagn*i is responsible for

human beings to gain *ayu, varna, bala, utsaha, prabha,oja* and *prana.*[222] To maintain *antaragni,* whatever the food we are taking should be ideal. According to Indu, our food acts as *Indhana* (Fuel). This food is running Jatharagni…*Karya karanabhavath*. If our food is not good, then it leads to all ailments.

02.10 HYPERSENSITIVITY / ALLERGY

The word allergy was applied originally by Von Pirquest to any alteration in the state of reactivity of an organism, due to contact with any organic substance. Among other terms used to describe this condition are pathergy, atopy, anaphylaxis, hypersensitivity and idiosyncrasy.

"Allergy" is an individual's sensitivity to a foreign substance that is usually harmless. This substance, called an allergen or antigen, is introduced the immune system by a number of different routes; either by ingestion, inhalation, injection, or simply by touch. Allergy is the most personalized of diseases and can occur at any point in an individuals; lifetime. Once an allergic individual's immune system has identified an antigen, it sets to work producing antibodies to defend itself. Normal individuals produce immunoglobin G to ward off invaders. It does not cause an allergic reaction. Allergic individuals also produce immunoglobin G. but – in addition –they produce immunoglobin E, an antibody with a "memory" for specific substance. Histamine, and related substances, cause allergic symptoms to occur. This is a vastly simplified expansion of allergic reaction. There are many chemicals that become part of the allergy chain. An allergic reaction can occur almost anywhere in body. The symptoms of the reaction often occur at the site of the reaction. Hence, the sneezing and stuffy nose of allergic rhinitis, the stomach cramps of food allergy and the itching rash of poison ivy. At other times, however, the symptoms may occur in a separate part of the body. Allergic reactions to insect stings can cause hives, dizziness and other symptoms. And any type of severe allergic reaction can cause systemic symptoms that can be life threatening.

Mechanism of the allergy

The mechanism of allergy has become widespread only since 1920. The two major discoveries, which led to recognition of allergic phenomena, were the discovery of anaphylaxis & the development of immune sera for the treatment of various diseases like allergic rhinitis.

Immunochemical aspect of allergy

In this subheading briefly reviewed the mechanism of the ordinary types of immunologic reactions, with special reference to similar phenomena in allergy. The nature of antigens,

An antigen is any chemical substance that, when introduced into the body, causes the body to produce specific antibodies, which can react with the antigen. The antigens thus have two important characteristics. - The first is immunogenecity or the ability to stimulate the formation of specific antibodies. - The second is reactivity or the ability of the antigen to react specifically with the produced antibodies. Chemically, the vast majority of antigens are proteins, nucleoproteins, lipoproteins (lipid + protein), glycoproteins (carbohydrate + protein), and certain large polysaccharides. In general, they have a molecular weight or 10,000 or greater. The antibodies do not form against the whole antigen. At specific chemical groups of the antigen combined with the antibody. This combination depends upon the size & shape, of the determinant site & the manner in which it corresponds to the chemical structures of the antibody.

Antibodies

An antibody, like an antigen, also has a valency, whereas most of the antigens are multivalent. The antibodies belong to a group of proteins called globulins & for this reason; they are also known as immunoglobulins or Ig. Five different classes of immunoglobulins are known to exist in humans. These are designated as IgG, IgA, IgM, IgD & IgE. Explanation regarding these immunoglobulins is available in the context of immunity.

Antigen – Antibody Reactions

The antigens & the antibodies, by definition, combine with each other specially & in observable manner. The reactions between the antigens & antibodies serve several purposes. In the body they form the basis of antibody-mediated immunity in infectious disease, or of tissue injury in some types of hypersensitivity & autoimmune diseases.

Hypersensitivity

Allergic hypersensitivity predisposes to tissue damage. The processes of allergic hypersensitivity are lymphatic reactions of two sorts; indirect action by T lymphocytes themselves in close contact with antigen. Types of tissue damaging allergic hypersensitivity reaction If the reaction is tissue damaging allergic hypersensitivity, then the two processes are respectively cell mediated hypersensitivity & antibody – mediated hypersensitivity have classified the tissue damaging allergic hypersensitivity reaction into four reaction types based on animal models; types I, II & III are antibody mediated; type IV is cell mediated hypersensitivity as mentioned above. There are four basic types of hypersensitivity

reactions; type I (anaphylaxis), type II (cytotoxic), type III (Immune complex); type IV (cell mediated). The first three involves antibodies; the last involves T cells.

Type I (anaphylaxis) reactions are the most common & occur within a few minute after a person sensitized to an allergen is reexposed to it. Anaphylaxis (an 'afi- LAK-sis) literally means "against protection" & results from the interaction of allergens with IgE antibodies on the surface of mast cells & basophils. Basophils circulate in blood; mast cells are especially numerous in connective tissue of the skin & respiratory system & endothelium of blood vessels. In response to certain allergens, some people produce IgE antibodies that bind to the surface of mast cells & basophils. The next time the same allergen enters the body, it attaches to the IgE antibodies already present on the surface of mast cells & basophils. In response, the cells release chemicals called mediators of anaphylaxis, among which are histamine, prostaglandins, leukotrienes & kinin. Collectively, the mediators cause vasodilation, increased blood capillary permeability, increased mucus secretion. As a result, a person may experience inflammatory response, difficulty in breathing through the constricted bronchial tubes, & a "runny" nose from excess mucus secretion.

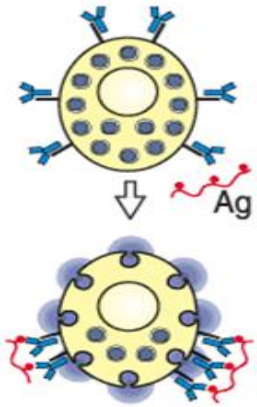
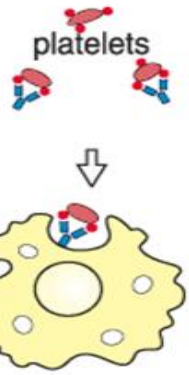
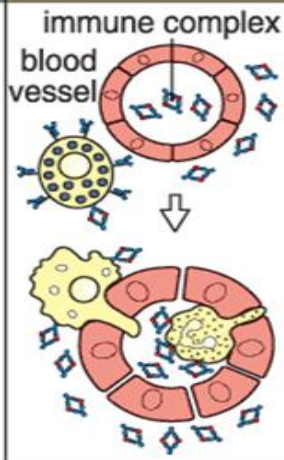
Type II (cytotoxic) reactions are caused by antibodies (IgG or IgM) directed against antigens on a person's blood cells (red blood cells, lymphocytes, or platelets) or tissue cells. The reaction of antibodies & antigens usually leads to activation of complement. Type II reactions, which may occur in incompatible transfusion reactions, damage cells by causing lysis.

Type III (immune complex) reaction involve antigens (not part of a host tissue cell), antibodies (IgA or IgM), & complement. When certain ratios of antigen to antibody occur, the complexes are small & escape phagocytosis. The complexes of blood vessels, activate complement, & cause an inflammation. Conditions that so arise include glomerulonephritis, systemic lupus erythematous (SLE), & rheumatoid arthritis (RA).

Type IV (cell-mediated) reactions or delayed type hypersensitivity (DTH) reactions are carried out by macrophages that have become activated by T cells. They usually appear 12-72 hours after exposure to an allergen. Type IV reactions occur when allergens are taken up

by antigen-presenting cells, such as Langerhans cells in the skin, which then migrate to lymph nodes & present the allergen to T cells. This result in sensitizations & proliferation of T cells, some of which migrate via the lymph& blood to the site of allergen entry into body. There they secrete cytokines, such as gamma-interferon, which activates macrophages, & tumor necrosis factor (TNF), which stimulates an inflammatory response. Intracellular bacteria, such as Listeria monocytogenes & Mycobacterium tuberculosis, trigger this type of cellmediated immunity, as do certain haptens, such as poison ivy toxin. The skin test for tuberculosis also is a delayed hypersensitivity type reaction.

TABLE NO: 181 CHARACTERSTICS OF IMMUNOGLOBULINS

	Type I	Type II	Type III
Immune reactant	IgE	IgG	IgG
Antigen	Soluble antigen	Cell- or matrix-associated antigen	Soluble antigen
Effector mechanism	Mast-cell activation	FcR^+ cells (phagocytes, NK cells)	FcR^+ cells Complement
			
Example of hypersensitivity reaction	Allergic rhinitis, asthma, systemic anaphylaxis	Some drug allergies (e.g., penicillin)	Serum sickness, Arthus reaction

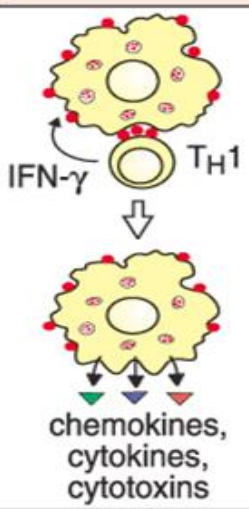
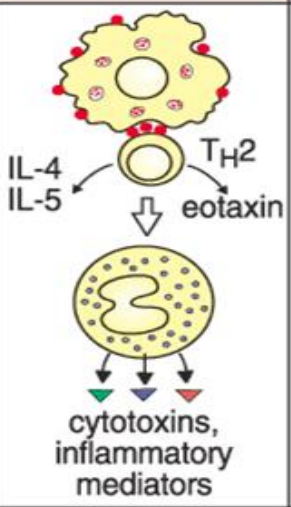
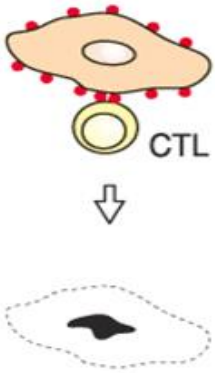
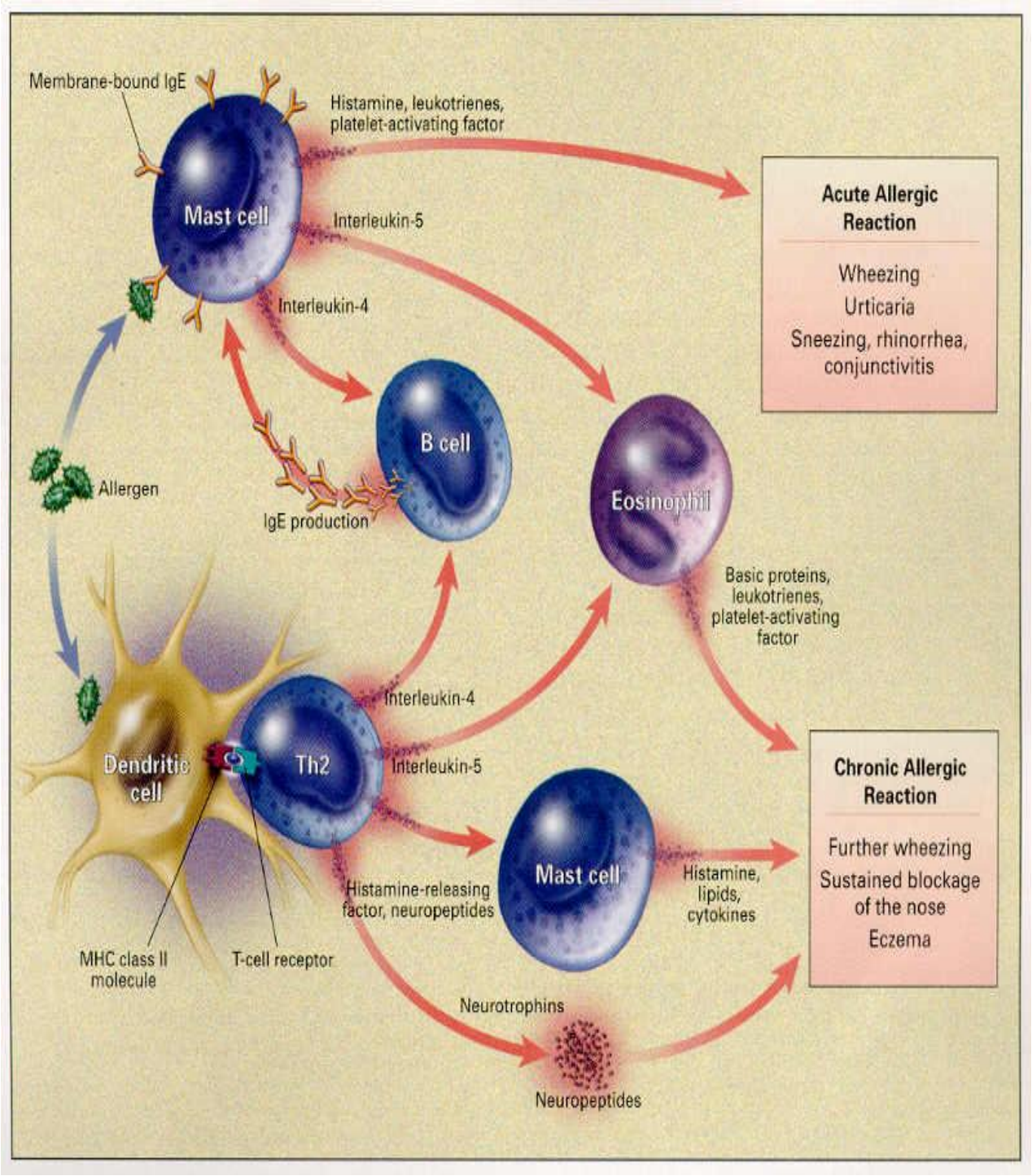
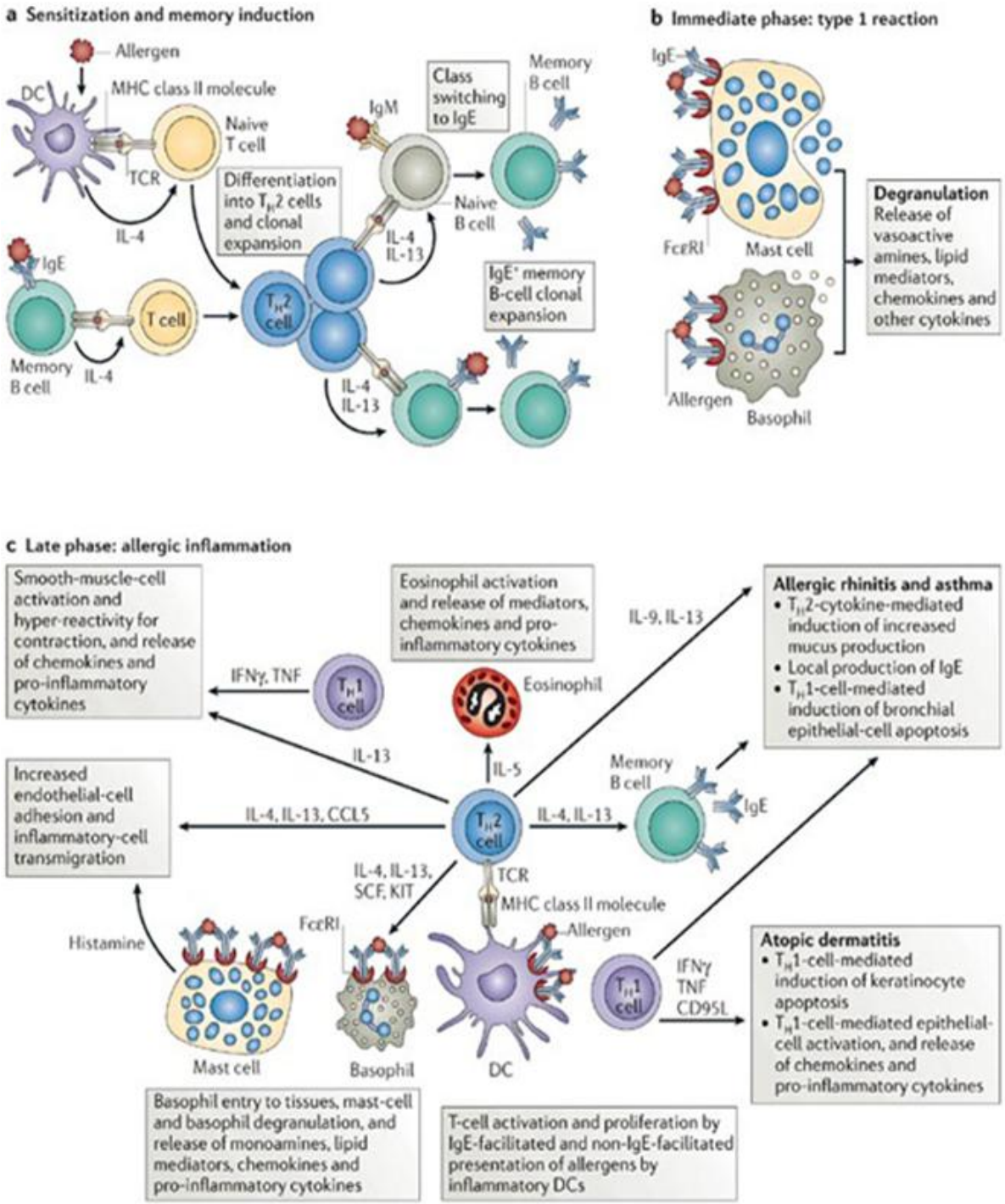
	Type IV		
Immune reactant	T_H1 cells	T_H2 cells	CTL
Antigen	Soluble antigen	Soluble antigen	Cell-associated antigen
Effector mechanism	Macrophage activation	Eosinophil activation	Cytotoxicity
			
Example of hypersensitivity reaction	Contact dermatitis, tuberculin reaction	Chronic asthma, chronic allergic rhinitis	Contact dermatitis

Diagram No 1


Membrane-bound IgE
Histamine, leukotrienes, platelet-activating factor
Mast cell
Interleukin-5
Interleukin-4
Acute Allergic Reaction
Wheezing
Urticaria
Sneezing, rhinorrhea, conjunctivitis
B cell
Allergen
IgE production
Eosinophil
Basic proteins, leukotrienes, platelet-activating factor
Interleukin-4
Dendritic cell
Th2
Interleukin-5
Chronic Allergic Reaction
Further wheezing
Sustained blockage of the nose
Eczema
Mast cell
Histamine-releasing factor, neuropeptides
Histamine, lipids, cytokines
MHC class II molecule
T-cell receptor
Neurotrophins
Neuropeptides



Kinins

The kinins represent a small group of polypeptides formed by enzymatic mechanisms and which have potent common pharmacologic activities in extremely minute dosages (nanograms). The principle pharamalogic actions include dilation of blood vessels, hypotension, white blood cell migration, increased vascular permeability, smooth muscle

contraction and pain. Proteolytic activation of kallikreinogen (prekallikrein),an active precursor to the enzyme kallikrein, occurs by a variety of enzymes, dilution, acidification, and activation of Hageman factor (factor XII of the coagulation system). Kallikrein, in turn, is responsible for the conversion of a normal serum alpha-2 globulin, kninogen, to the vasoactive peptide bradykinin, a non-apeptide. A naturally occurring kinin inhibiting enzyme knininase (carboxypeptidase B) serves to control the activity of tissue kinins. The kinin system proteins have been demonstrated in nasal secretions in allergic rhinitis and also in otitis media with effusion. An important aspect of the kinin system that should be considered in relation to its potential role in otitis media and allergic rhinitis is the effect of kinin on the mucosa. Injection of bradykinin has been shown to produce gaps in the venular side of the vascular bed in vessels with a caliber of greater than 60 µm in diameter. It has been further has shown that this initial change in permeability results in the accumulation of large molecular weight plasma proteins in the interstitial fluid. A colloid-osmotic passage of fluids through the whole capillary bed takes place resulting in tissue edema.

IgE

IgE normally occurs in adult human serum in a concentration of 10 to 70 g/100ml and thus represents 0.001% of the total serum protein. The molecular weight of IgE is slightly heavier than IgG and IgA and is about 200,000 daltons. IgE type immunoglobulins comprise the reaginic antibodies and do not cross the placental barrier nor fix complement or rheumatoid factor. These antibodies sensitize human mast cells and basophils and when coupled with specific antigen cause the release of inflammatory mediators. It is believed that some IgE antibodies are formed locally in the respiratory and gastrointestinal tracts and play a major role in the pathogenesis of acute allergic disorders. On the other hand, IgE may play a protective role at mucosal surfaces and may function in the organism’s defense against parasitic infection.

Histamine

Instillation of histamine into the nose produces immediate itching followed by sneezing, nasal discharge & blocking. Thus the symptoms are very similar to those produced by allergens in the sensitized subject. It should be noted that allergen provocation, unlike histamine provocation, results in infiltration of inflammatory cells, particularly eosinophils, & heightened nasal reactivity. Small amounts of histamine can be identified in nasal washings after allergen challenge. Selective H1 antagonists such as astemizole (Hismanal)

inhibit sneezing & watery discharge but not nasal blockage, but only a short lasting & insignificant challenge of contra lateral nasal patency indicating that a direct histamine effect on blood vessels may be important for any persistent nasal blockage in allergic rhinitis. Histamine affects the vascular tube by both H1 & H2 receptors resulting in dilatation of some & constriction of other blood vessels & edema formation. Histamine has a fairly weak H2 mediator effect on mucous glands & appears to increase mucous glycoproteins without significantly affecting the total volume of nasal discharge. Thus the inability of ordinary antihistamines & the new selective H1 antagonists to deal with nasal blockage in allergic rhinitis could be due to the presence of H2 receptors in nasal vasculature. Combined use of H1 & H2 antagonists in the nose only partially prevents histamine provoked vascular changes. Thus the effects of histamine are complex. Although there is slight bilateral blockage, there is considerable bilateral hyper secretion after histamine instillation, which is stimulated through H1 receptors. Thus sneezing & a large part of mucous secretion appear to be reflex mediated.

Inflammatory Reaction and Allergic Diseases Effectors Mechanisms

(Philippe Gosset & Michel Joseph)

The development of the inflammatory reaction is based on three crucial cell partners: "regulating" lymphocytes, "filtrating" endothelial cells, and "effector" cells (eosinophils, monocytes, macrophages, platelets). Our investigations focused on human IgE receptors, more particularly on the regulation of their expression and on the analysis of their functions in mononuclear phagocytes (monocytes, dendritic cells and alveolar macrophages). The duality of IgE receptors is now a generally accepted concept, with a high affinity receptor (FceRI) originally identified on basophils and mast cells only, and a low affinity receptor (FceRII or CD23) recognized initially on monocytes, eosinophils, B-lymphocytes and platelets. The opposition between FceRIpositive cells and CD23-positive cells was recently reconsidered. FceRI is also expressed Langerhans cells, dendritic cells, monocytes and eosinophils. Through monoclonal antibodies against each receptor type and IgE-anti-IgE complexes, we have evaluated their capacity to induce production of cytokines and the implication of these mediators in allergic diseases. Cell activation with IgE and anti-IgE significanthly increased the production of pro-inflammatory cytokines (such as TNFalpha, IL-1béta), of chemokines (IL-8, MCP-1, MIP-1alpha), but also, to a lesser extent, of anti-inflammatory cytokines (IL-10 and IL-1Ra). Pro-inflammatory cytokines were synthesized

earlier (4h) than anti-inflammatory cytokines. It appears therefore that the IgE-dependent activation *via* CD23 exerted globally proinflammatory effects on AM. Through this mechanism AM could participate to the development of the inflammatory reaction following the exposure of asthmatic patients to allergen). In contrast, anti FceRI antibody had no activity on the generation of these cytokines. However the percentage of monocytes and macrophages positive for FceRI was very low compared to dendritic cells derived from monocytes. For this reason, the effect of cytokines has been evaluated on FceRI expression in monocytes. IL-4 and IL-13 enhanced after 24h incubation FceRI alpha chain expression at the mRNA and protein level in monocytes. The response of IL-4 was significantly higher in monocytes from allergic patients compared to healthy subjects. In contrast, a glucocorticoid such as methylprednisolone greatly inhibited IL-4-induced FceRI alpha chain expression in monocytes. In addition, *in vivo* treatment of allergic patients with glucocorticoids also decreased the FceRI expression. The *in vitro* activity of both IL-4 and glucocorticoids can explain in an opposite manner the modulation of FceRI alpha chain expression in monocytes from allergic patients. Moreover the effect of IL-4 is sustained by GM-CSF (granulocyte macrophage-colony stimulating factor), a combination of cytokines, which allows the differentiation of monocytes in dendritic cells. This mechanism is responsible for the high level of FceRI and CD23 expression in dendritic cells.

The technics of generation of dendritic cells from monocytes, which expressed high levels of FceRI, has been developed in the laboratory and allows measuring the effect of IgE-dependent activation to define the respective function of FceRI and CD23 in these types of cells. Preliminary results showed that IgE-dependent stimulation enhanced the membrane expression of CD86 and CD40 as well as the production of TNF and of the chemokine TARC. The second step is now to evaluate the respective role of FceRI and CD23 in this process and to compare the response of dendritic cells from allergic patients and from healthy subjects. These data should allow to define the function of IgE receptors in the monocyte-derived dendritic cells and to identify potential dysregulation in their behaviour in allergic patients compared to healthy subjects.

TABLE No 11 CATEGORIES OF DISEASES RELATED TO ALLERGY

		Common	Main	Other key

Disorder	Symptoms	allergens or other causes	disease mechanism	features
Allergic rhinitis:	Blocked, runny nose, sneezing, itching and streaming eyes		IgEmediatd	Mild winters and warmer springs mean that pollination in the United Kingdom now starts earlier than it did 50 years ago. Therefore symptoms can be well established by the first week in May and peak around mid-June to early July. When pollen counts are very high, some wheeziness can also coexist with rhinitis, in a condition known as seasonal allergic asthma
Seasonal allergic rhinitis(hayfever or rhino conjunctivitis)	Worst symptoms occur at the height of summer when vast clouds of grass pollens become airborne	Pollen (commonly grass, but also tree and weed pollen)		
Perennial allergic rhinitis	Chronic symptoms occur all year round	Housedust mite, allergens derived from cats, dogs, horses and pet rodents. In some patients, perennial rhinitis is due to non-allergic causes such as infection or structural abnormalities of the airway. A small minority of patients also have underlying		

		immunodeficiency problems		
Asthma	Characterised by episodes of wheezy breathlessness, but may also present as an isolated cough, particularly in children. "Non-atopic" asthma often starts later in life and can be more severe	The cause is still uncertain, and it is often difficult to determine the role of allergy. Allergy to house dust mite, pollen, moulds and pets can trigger an attack in a significant proportion of patients; food allergens and additives may rarely trigger symptoms. A significant proportion of patients are not sensitised to allergens so are "non-atopic" or "intrinsic" asthmatics	IgE-mediated	Pathology involves inflammation and muscular contraction of the large and small airways (bronchi and bronchioles—see Figure 2). The consequence is an irritable, easily constricted airway in which a variety of non-specific irritants causes airflow obstruction (bronchial hyper-responsiveness). Triggers include viral infection, exercise, certain drugs, and exposure to fumes or tobacco smoke
Anaphylaxis:	Anaphylaxis describes a group of symptoms affecting several parts of the body, caused by a hypersensitivity reaction to an allergen in a previously sensitised individual. "Anaphylactic shock" is an extreme hypersensitive reaction characterised by an overwhelming sense of impending doom, a dramatic fall in blood pressure, swelling in the throat and mouth, chest tightness, breathlessness from severe asthma and unconsciousness. In a small number of cases,		IgE-mediated	A rash may herald that a more severe reaction will occur in the future, but in some cases anaphylactic shock occurs without any previous warning

	anaphylactic shock results in death			
Sensitivity to insect venom	Some reactions are life-threatening but most result in a temporary irritation or swelling around the site of the sting	Can be caused by wasp or bee stings		
Sensitivity to drugs	Rash anywhere in the body	Almost any drug, but the most common causes are penicillin and other betalactam antibiotics		Only a small proportion of adverse drug reactions have an allergic background, and an even smaller proportion are IgE-mediated
Sensitivity to foods	Rash anywhere in the body, especially around the mouth and throat	Peanuts, tree nuts (such as almonds, hazelnuts, walnuts and brazil nuts), milk, eggs, fish and shellfish		
Oral allergy syndrome	Swelling in the lips, mouth, tongue or throat	Occurs in tree, grass, weed and latex allergy sufferers immediately after contact with certain foods. A significant proportion of people who are allergic to birch trees, suffer oral allergy syndrome after eating raw apples	IgE-mediated	The reaction is caused by a cross-reaction between the allergen to which the patient is sensitised, and the food protein
Urticaria and Angioedema:	Itching and swollen, red welts known as "hives" or "wheals" on the surface of the skin (urticaria) or deeper in the skin, particularly around the mouth and eyes (angioedema)			
Acute	Rash suddenly occurs and usually disappears	Food allergy, especially to	IgE-mediated	

	within 24-48 hours	peanuts, tree nuts or shellfish. Viral infection is more commonly the cause than food allergy		
Chronic	Symptoms last intermittently or continuously for more than three months, but often clear up without treatment	Underlying cause is rarely found	Non-IgE-mediated	
Atopic dermatitis (Atopiceczema)	Chronic, recurrent inflammation of the skin, characterised by intense itching which particularly affects the flexures (creases of skin) at joints such as the wrists, elbows, ankles and knees	Egg or cow's milk allergy sometimes triggers symptoms in children, but this is rarely the case in adults. A number of external influences may trigger or exacerbate symptoms, including emotional stress, irritation of the skin by wool or nylon, infections and vaccinations	IgE-mediated	Patients often also suffer from other atopic disorders such as allergic rhinitis, asthma or both. It is currently thought that atopic dermatitis usually develops first and this then predisposes an individual to the production of IgE and the development of other atopic disorders
Extrinsic allergic alveolitis (EAA):	Shortness of breath, with or without cough, and in the acute phase there are usually muscular aches, fever and a lack of energy	Repeated or prolonged exposure to agents found in bacteria, animal products and chemicals	Non-IgE-mediated	EAA describes a group of lung disorders caused by an inflammation of the alveoli (air sacs in the lung)
Contact dermatitis	Redness, scaling and itching at sites of exposure to the irritant. Can lead to thickening of the skin (lichenification)	Most commonly due to an irritation caused by external substances, but may also result from non-atopic allergic sensitisation to substances in the workplace, or nickel, lanolin and cosmetics	Non-IgE-mediated	

Farmers' Lung		Bacteria found in straw, mouldy hay or grain		
Bird Fanciers' Lung		Bird droppings and feathers		
Animal Handlers' Lung		Dried urine, hair or animaldander		
Coeliac disease	Diarrhoea, failure to thrive (in infants and children), weight loss (in adults) and fatigue	Caused by an allergy to gliadin, a protein found in wheat, barley and rye	Non-IgE-mediated	Occurs in genetically predisposed individuals at all ages after infancy. It is an allergic disorder although the basic mechanism is autoimmune. Management requires a lifelong gluten-free diet

02.11 NIDANA

FACTORS RESPONSIBLE FOR DISEASE:

To understand the process of disease formation {vaishamya} it is essential to know the principle involving three factors in the production of disease described by Charaka in his nidana sthana[266].

1. Nidana

2. Dosha

3. Dusya

By the effect of causative factors Dosha first gets provoked or vitiated. Doshas when thus provoked and vitiated, vitiate the tissue of the body, which are referred in Ayurveda as Dusya. Another principle is that of Samanya and Vishesa that is, increase is caused by agents or causative factors similar to Doshas and Dushyas and decrease being caused by dissimilar ones is formulated. However, if the property of the Dosha is opposite to those of the causative factors agreement for co-operation in production of the disease is not possible. On the contrary these opposite properties counter the factor and the inroads of the disease are checked. In this way provoked Dosha does not find favorable response in the tissue, it finds hard to vitiate and the further process is checked or is delayed. Thus above three specific factors, which determine the ability or otherwise of the body to resist all types of disease is described [266]. Critical exposition by Cakrapani on above statement describes that the specific feature of etiological factors etc., which determine the ability to resist the disease is as under. [266]

1. When the etiological factors (here Viruddhahara/visha/asatmya) have properties homologous with Doshas and or Dhatus or when Doshas have properties homologous with Dhatus this leads up to the manifestation or aggravation of the disease.

2. If it is in a lesser degree and the combination is further subdued due to passage of time or due to repeated combination in still smaller degree, then it may lose strength and may result in the non-manifestation of the disease.
3. If at all a disease is so manifested then it will develop slowly or in a subdued form or all its symptoms may not be so manifested, as they should.

4. When the etiological factors Doshas and Dhatus are favourable moderately or strongly with each other then the result may be opposite. i.e. the disease may be slowly manifested, immediately manifested or all the symptoms may be well manifested.

The suppression or the incidence of the diseases can be tabulated as under (According to Caraka Samhita). Thus disease Pathogenesis depend upon association of causative factors, humors as well as elements of body.

Table 14- Role Of Nidana, Dosha And Dusshya In Samprapti

Nidana+Dosha +Dusshya	No association	No disease
Nidana+Dosha +Dusshya	Late association	Prolong course or delay in manifestation (chronic disease)
Nidana+Dosha +Dusshya	Weak or insufficient association	Mild disease or incomplete disease or irregular symptoms
Nidana+Dosha +Dusshya	Sudden association	Acute disease
Nidana+Dosha +Dusshya	Powerful association	Fatal (or) threatening disease

<u>Nidana for production of Asatmya condition (Hypersensitivity)</u>

I SAHAJA KARANAS (Sahajatena shareerena)

a. Beejadosha [267]
b. Satmyaja bhava abhava[268]
c. Poorva Karma

II AHARA & VIHARA

a. Tridoshanidanaas Ama{Ama-prathamadoshadustim ca (ma.ni) anye doshebyah evati dustebyo … … a.hri.soo)}

b. Viruddha ahara/vihara – Dhatudusti -Shonitadustinidana[269]

c. Kala – VataKapa samanya kopa kala –Sheeta (Samanyaguna for vata kapha)- ratri/season/vaya(early age - kapha; elderly-vata)

III VISHA NIMITTA

Visha = Anaphylactic; Gara visha/ Dooshi visha = Other 3
Immune reaction

Viruddha ahara/ vihara – Visha gopamam (a.hri.soo)

Visha=Anaphylactic; Gara = Other 3 immune reaction

B For the production of pranavaha sroto vikriti.

Table 15- Pranavaha stroto vikruti nidanas

S.N.	AHARA	C.S.	SS/ Ma.N	A.S	A.H
VATAPRAKOPAKA					
1	Ruksanna (fat free diet)	✓	✓		
2	Visamasana (irregular diet)	✓	✓		
3	Sitasana (cold food)		✓		
4	Anasana (fast)		✓		
5	Visa sevana (toxins)	✓	✓		✓
6	Sita ambu (cold water)	✓	✓	✓	✓
7	Vistambhibhojan(slowly digested food)	✓	✓		
8	Adhyasana (frequent meals)		✓		
9	Dvandvalayoga (mutually antagonistic)	✓			
KAPHAPRAKOPAKA					
1	Nispava (beans)	✓			
2	Pinyaka (tila paste)	✓			
3	Pistabhojan (paste preparation)	✓			
4	Jalaja mamsa (aquatic fish)	✓			
5	Guru bhojan (heavy diet)	✓			
6	Ama Kshira (unboiled milk)	✓	✓		
7	Dadhi (curd)	✓			
8	Anupa pisita	✓			
9	Tila taila	✓			
10	Abhisyandi Anna	✓			
11	Slesmala Dravya	✓	✓		
12	Saluka (lotus rhizome)	✓			
13	Utkledi Ahara	✓			
14	Masa (black gram)			✓	✓
Naidanik factors related to Vihara:					
VATAPRAKOPAKA					
1	Rajas (Dust)	✓	✓	✓	✓
2	Dhuma (Smoke)	✓	✓	✓	✓
3	Vata (wind)	✓	✓	✓	✓
4	Sita-Sthanasevana (residing in cold place)	✓	✓		
5	Sita ambu (Cold Water)	✓	✓	✓	✓
6	Vyayama (exercise)	✓	✓		

7	Gramya sevana (over indulgence in sex)	✓	✓		
8	Atyapatarpana (malnutrition)	✓			
9	Marmaghata (trauma over vital organ)	✓	✓		✓
10	Bhara vahan (excessive weight lifting)		✓		
11	Vega vidharana (suppression of urges)	✓	✓		✓
12	Suddhi atiyoga (excessive purification)	✓	✓	✓	✓
13	Kanta pratighata (throat trauma)	✓			
14	Urahpratighata (chest trauma)	✓			
15	Karmahata (exhausted)	✓	✓		
16	Ayasa				✓
17	Jagarana				✓
KAPHA PRAKOPAKA					
1	Abhisyandyupacaras	✓			
2	Divaswapna				✓
Vyanjaka hetu					
1	Megha (clouds)	✓			✓
2	Ambu (water)	✓			✓
3	Seeta (cold)	✓			✓
4	Sleshma Vardhaka (Kapha increasing issues)	✓			✓
Nidanarthakara rogas					
Vataja rogas					
1	Anaha	✓			
2	Daurbalya	✓			
3	Atisara	✓			
4	Ksaya		✓		
5	Ksataksaya	✓			
6	Udavarta	✓			
7	Visucika	✓		✓	✓
8	Panduroga	✓		✓	✓
9	Visa Sevana	✓			
10	Vibandha	✓			
11	Avarana				✓
12	Dhatu Ksaya	✓			
Pittaja rogas					
1	Rakta Pitta	✓			
2	Jvara	✓			
Kaphaja rogas					
1	Kasa			✓	✓
2	Amapradosa		✓		
3	Chardi	✓		✓	✓
4	Pratisyaya	✓			
5	Amatisara			✓	✓

Sl	**Aharaj (Dietary)**nidana	**C.S.**	**SS/Ma.N**	**A.S**

no				
1	Ajeerna	✓		
2	Atijalapana			✓
3	Ati Sheeta Ambupana	✓		
Viharaj (Behavioral)				
1	Ati Nariprasanga	✓	✓	
2	Ati Divaswapna	✓		✓
3	Ratri Jagarana	✓		✓
4	Vega Sandharana	✓	✓	✓
5	Ati Ashru Srava	✓		
6	Tapa Sevana		✓	
7	Dhuli, Rajah, Dhumra Sevana	✓	✓	
8	Sheetamati pratapa	✓	✓	✓
9	Ritu Vaishamya		✓	
10	Snana in Ajeerna	✓		✓
11	Snana with sheeta jala	✓		✓
12	Ati jala krida			✓
13	Ati Bhashana	✓		✓
14	Shirasobhitapa	✓	✓	
15	Shirovedana	✓		
Manasika				
1	Ati Krodha	✓		

ETIOLOGY

This includes factors involved in the development or onset of asthma and the factors (triggers) involved in the development of exacerbations of asthma and can be grouped as

1. Predisposing factors: the factor that gives an individual susceptibility to the disease and includes atopy and gender.

2. Causal factors: those factors that sensitize the airway and cause the onset of the disease. Allergens by far are the most common causal factors that is to be faced everywhere.

3Contributing factors: These augment the likelihood of asthma developing upon exposure to a causal factor; they may even increase susceptibility to asthma. The role of these is illustrated in the schematic diagrams below:

Table 16: Schematic sketch for contributing factors (A) to Asthma

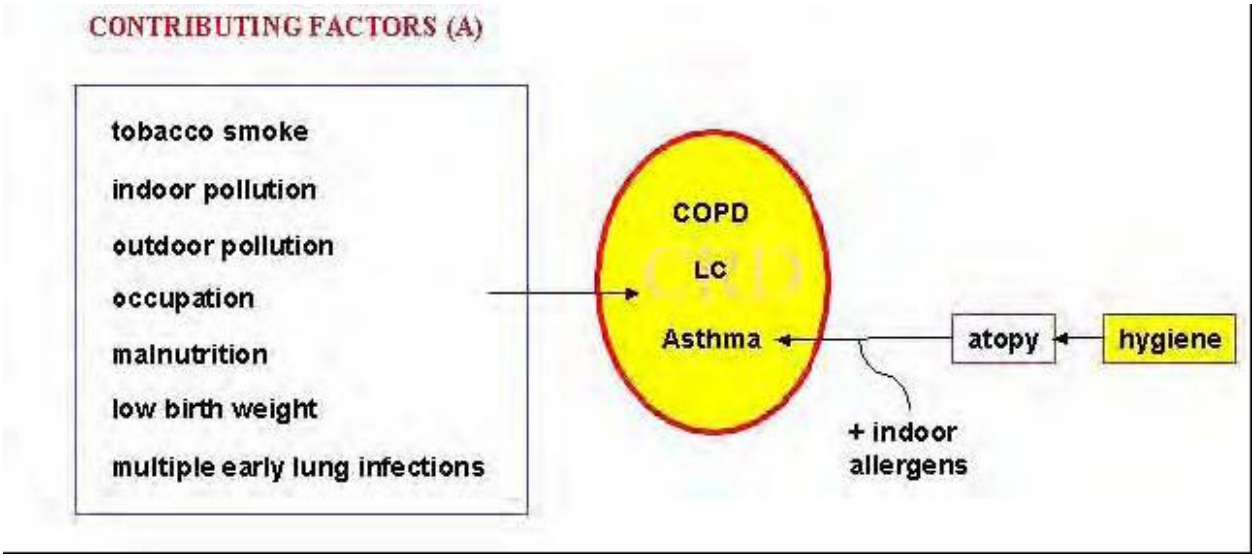
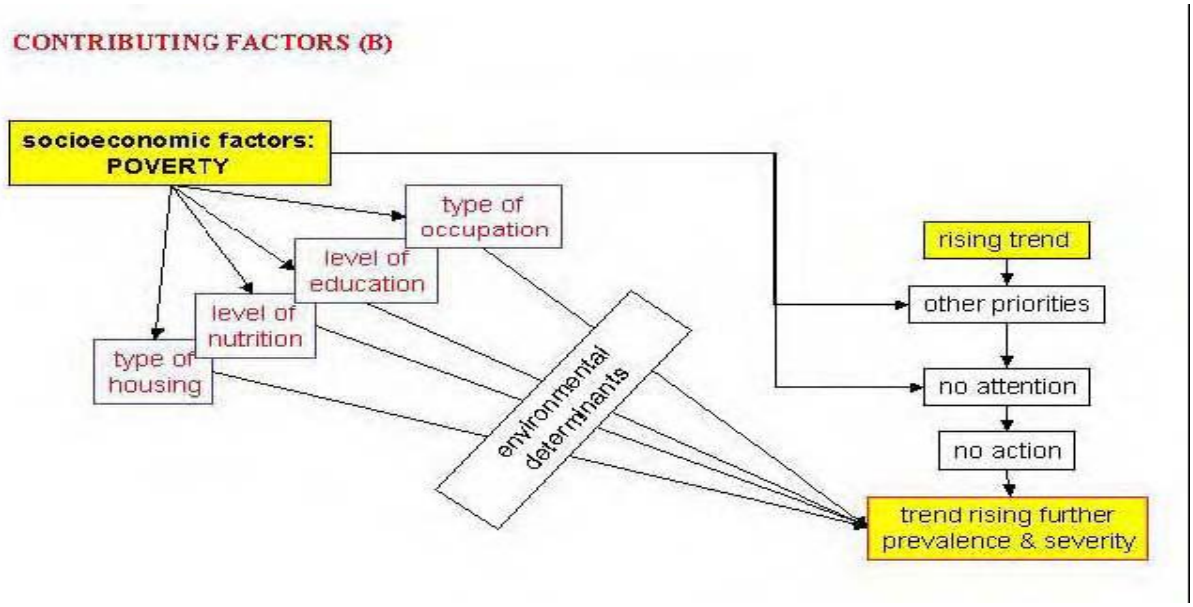


Table 17: Schematic sketch for contributing factors (B) to Asthma



(1) Age: Usually it affects young adults from the age of 15 years onwards, and tends to recede after the age of 40 to 50 years. It may affect young children also.

(2) Sex: Both sexes are affected.

(3) Predisposing Factors: The factors that predispose the tissues to allergy may be classified in the following way –

1. Hereditary (the most important single factor) & constitution
2. Infection & Intoxication
3. Endocrine factors
4. Factors interfering with the chemical & physical resistance of the skin or mucous membranes.
5. Trauma
6. Meteorological & seasonal conditions
7. Psychological

Hereditary & constitutional factors

50% of allergic patients give family histories of allergy, children with bilateral inheritance develop allergy in 75% of cases, those with unilateral inheritance who develop allergic amounts of 50% & those without a family history 7-12% (Urbach E, 1946, Allergy P. 75, London, Heineman).

Infection & intoxication

The direct action of bacteria & viruses, or their products on the tissue cells, constitutes one of the most important predisposing factors.

Endocrine factors

Menstruation, the menopause & ovarian dysfunction, all tends to increase allergic reactions; pregnancy usually reduces asthma, but increases nasal allergy. In hyperthyroidism, there is a heightened sensitivity of the sympathetic nervous system & a tendency to exudative reactions & allergy in the skin & mucous membranes.

Chemical & physical resistance of tissues

A deficiency of calcium & vitamin C & D increases capillary permeability & oedema. The evidence is conflicting, but suggests that the strength of the intercellular cement, & the resistance of the capillary endothelium, depends on adequate supply of calcium & vit. D. Severe

deficiency renders an individual more susceptible to allergy. There is evidence that a relative alkaloids develops in the tissue during allergic reactions (Buhrmester, Catherine, 1933, Annual Otol. Etc. St. Louis, 42, 1041).

Trauma

Trauma alone is rarely a major factor, although it obviously plays an important part when allergy develops after an operation on the nose.

Meteriological & seasonal condition

Apart from the importance of season in the incidence of pollen allergies, the factors of temperature, humidity, barometric pressure, sunshine, air movement & the electrical state of the air have an influence on the severity of allergic reactions particularly in the asthma, nasal allergy & migraine groups.

Geographical

Places in which the atmosphere is damp & stagnant & particularly where trees are numerous, are bad for cases of nasal & bronchial allergy. Attacks are often much less severe at high altitudes (over 5000 ft.) in areas where vegetation is scanty, & at sea at a distance beyond the usual reach of the dust, pollen & clouds.

Psychological factors

Psychological factors play a part in the majority of allergic subjects. They may act as the sole cause, as predisposing factors or an exciting factor.

Precipitating Factors (Allergens) -

The allergens are of two types:

(a) Exogenous (External agents)

(b) Endogenous (Within the body)

a. Exogenous factor (external agents) – The majority of cases of nasal allergy are due to exogenous allergens. In adults, the cause is usually due to inhalance; in early childhood, foods are the most common causative factor (Rackmann, 1931, Elimination diets & the patients allergies, London, Kimpton). The allergens may be single, but are usually multiple. The following allergens may be responsible for the allergic rhinitis.

Inhalants:

Which include such items as house dust, mattress & furniture stuffings, blankets & furnishing fabrics & clothing; animal emanations & scales; orris-root, soaps, creams & perfumes; odours of fish, eggs, coffee & citrus fruits. The most common of the inhalants are animal epidermals. Occasionally, allergic rhinitis may be the result of exposure to an occupational allergen.

Ingestants : such as wheat, milk, eggs, chocolates, fish & citrus fruits.

Contactants : such as nasal drops & sprays.

Physical agents : such as winds & draughts, changes in temperature & humidity; smokes & fumes of sulphur, gas, oil & especially burning anthracite & charcoal, microscopical particles such as barley grains & stone dust.

Bacterial allergens : of which the causative organisms are usually staphylococci, pneumococci or streptococci.

Drugs : of which those commonly causing nasal allergy are acetylsalicylic acid, iodides, quinine, amidopyrine & the sulphonamides.

Endocrine factors : such as occurs in pregnancy, menstruation & at the menopause.

Nervous & metabolic factors

b. Endogenous factors (within body)

These are classified as

a. Emotional & endocrine.

b. Bacteria, viruses, moulds & parasites-intestinal helminthes, such as round worms, pin worms, etc. multiplying within body.

c. Altered tissue proteins, exudates, transudates, inflammed tissues.

Critical analysis of Nidana.

Vyanjaka Hetu(s)-

These are stimulating, precipitating or aggravating factors. These also cause aggravation of the symptoms in an already generated disease or these cause the precipitation of the Samprapti of a disease. The knowledge of these Hetus is useful in preventing the actual formation of diseases by taking care to avoid such factors. Generally it is observed that in spite of similar stressful situation, different individuals develop different diseases it may be on one hand due to role of Prakriti i.e. the psychosomatic constitution of the person and on the other due to nature of the pre existing Khavaigunya predisposing specific Sthanasamshraya of vitiated Doshas during Kriyakala i.e. pathogenesis. Nidanas can generally be classified into Bahya Nidanas and Abhyanthara Nidanas i.e. extrinsic and intrinsic factors. Abhyanthara Nidanas or Nija Hetus are the intrinsic factors within the body.These are called the **Host factors.** These can be taken also as Pradhana Karana. The Bahya Nidanas are Aagantuja Nidana that cause irritation in the body e.g. raja, dhooma, Asatmya Ahara etc. These are the **Environmental factors.** Either these may be the main etiological factor i.e allergy or triggering factors and these triggering factors also can be considered as Vyanjaka Karana.

According to modern medical science, based on etiological factors pulmonary hypersensitivity can be

Classified into two types-

- ✓ **Atopic** (Allergic or extrinsic)
- ✓ **Non-atopic** (Idiopathic or intrinsic)

Atopy or allergic can be considered as a type of Prajyaparadha. (Vd. P Mali et al 2004) Non-atopic or idiopathic can be understood as Asatmyendriyartha Samyoga, Prajyaparadha Parinama[270]. These further include Vata & Kaphaprakopak Ahara, Vihar and Manasika Hetu and Kala.

ASAATMENDRIYARTHA SAMYOGA:

Asathmyendriyartha-samyoga is the Mithyayoga, Atiyoga and Ayoga of Indriya and Indriyartha. This is the incoherent, excessive and less contact between object of perception and concerned sense organ.

Srotrendriya (ear) - Shabda (sound)

Hearing of Bhishana-dhvani (terrifying words), Upaghata (news of accidents, murder, theft etc) Istavinasha-shabda (hearing words conveying loss of relatives, friends, money etc.) Parusha-vachana (abuse, rough or harsh words) may create fear in persons mind andm especially those persons, who are having less Satvaguna, may suffer from dyspnoea/ Shwasakashtata.As far as the disease Asatmyajanya pranavahasroto vikara is concerned, acute exacerbation of pulmonary hypersensitivity may occur due to Mithyayoga of Shabda as discussed above.This Shabda Mithyayoga may be considered as triggers i.e. it cannot cause Asthma to develop initially but can exacerbate pulmonary hypersensitivity once it is present.

Sparshanendriya (skin) - Sparsha (touch)

All the objects, which are present in surrounding atmosphere, are in continuous contact with our skin knowingly or unknowingly. Exposure to dust, irritant gaseous, domestic mites, and allergens cause irritation to respiratory mucosa and result into reflux bronchoconstriction. Another type of Sparsha explained by Acharya Charaka i.e. Manas Sparsha. Manasa Sparsha may be accepted as Manasa Pratyaksha. Any disturbance in Manasa Sparsha leads to Shwasa. Now a days number of cosmetic products are available e.g. body spray, lotion, creams etc. which are Asaatmya to some person leading to Asthma development. Thus, continuous exposure to dust, domestic mites, animal allergen, and air pollutants through Sparshana acts as Nidana due to Mithyayoga of Sparshanendriya (respiratory mucosa)

Ghranendriya (Nose) – Gandh (Smell)

Bronchial Asthma is chronic airway disease & these airways are in direct contact with nose. Irritants such as wood smoke, household sprays, volatile organic compounds (e.g. polishes and cooking oils) and air pollutants may also exacerbate Asthma. Active and passive smoking also increases incidence of pulmonary hypersensitivity in society.

Rasanendriya (tongue) - Rasa (taste)

Excessive indulgence of only one Rasa especially Madhura Rasa may lead to Shwasa. Vagbhata has mentioned Shwasa because of Madhura Rasa Atisevana. Tamaka Shwasa gets aggravated due to excessive use of Sheshmal Draya i.e. Madhura, Amla and Lavana Rasa. All these Rasa produces vitiation of Kapha in body and Kapha Vriddhi results into Shwasa. All the dietetic Nidana can be grouped under this heading.

Chakshurendriya (eye) – Rupa (Sights)

Sights which are Roudra (terrifying), Bhairava (fearful), Adbhuta (unusual, unprecedented), Dwista (annoying), Bibhastha (emotional), Vikrita (unnatural, abnormal) due to these Mithyayoga Alpa Satva person suffers from Kshudra Shwasa i.e. temporarily breathlessness. In Asthmatic persons it may aggravates preexisting condition.

PRAJNAPARADHA

Prajnaparadha is a conscious or unconscious indulgence in harmful activities. It is again of two types-

A Saririka Prajnaparadha, eg. Excessive indulgence in sex, excessive working and other like wise activities.

B Manasika Prajnaparadha are anxiety, excitement, fear, sorrow, anger, greed, pride etc.

Allergies and Prajnyaparadha-Although the references of allergy are not easily available in our text yet some scattered description of paroxysm of diseases for example Sannipatic Pratishyaya [271]and Kotha [271i]s available which can be considered due to allergic phenomenon.

According to Mali Pavan et al, 2004-Allergy has been defined as **"a foolishness of the body tissues".** Body tissues fail to recognize and accept common substances as wholesome. We all are endowed with power to make out the difference between harmful and safe substances of our daily use diet articles, clothing, perfumes, flower, books and papers, mattresses, pillow, bed sheets, things of personal use along with pets and their fur, hair, air, dust, smoke etc. We are freely using these and living almost with them without any discomfort. Our body tissues, nose, skin, lungs and G.I. tract are receptive to all these without any problem. Our surrounding atmosphere is also full of allergens such as domestic mite, animal allergens, pollens, irritant gases. Not all the persons are having discomfort or allergy with these but some of them suffer from allergic reaction. Nasal mucosa identified this as harmful subject's allergies and an alarm is sent in the form of a powerful strong rejection, which includes sneezing and profuse watering of nose. The reaction is sudden and powerful. Allergic reactions in the lungs, nose, skin and G.I. tract are usually sudden, strong alarming and dramatically resolved if the cause is detected and removed. However, this may not happen in every case. Onset in an allergic manifestation can go for a long spell of time, which happens in pulmonary hypersensitivity. Thus immune system of body, which is meant for performing protection against allergies, but an important undesirable side effect of

immunity, is the development under some conditions, of allergy or other types of hypersensitivity.

PARINAMA

Parinama stands for Kala. Seasonal and weather changes also play a crutial role in aggravating Tamaka Shwasa. Aggravation of symptoms takes place in early morning hours, Meghambu (cloudy atmosphere) and Sheeta Kala. In case of Pratamaka Shwasa, symptoms are aggravated in Ushna Kala i.e. Grishma ritu, where as during Ushna Kala symptom is subsided in case of Tamaka Shwasa. In winters and monsoons, most of the patients of Tamaka Shwasa find more episodes of exacerbations of the disease. As we know that Nidana act at 4 levels so an effort has been made to categorized them which to prevent the disease-

Table 18- Effect Of Nidanas Over Dosha, Dushya Strotas And Agni

NIDANA	DOSHA PROKAPAKA	DUSHYA / SROTODUSTIKARA / KHAVAIGUNYAKARA	AGNIVAIGUNYAKARA
AHARAJA	Ruksana(fat free diet) Aama Dosha, Vidahi, Vistambhi	Sheeta, Guru, Abhishyandi	Vishamasa (Irreg. diet) Apatarpana Dvandvatiyoga
VIHARAJA	Marmaghata Veganirodha Gramyadharma (ex. coitus) Adhva (exhausted)	Raja(dust),Dhumapan(smoke), Vata sevana(wind), Punkesara(pollers), Vyayama(exercise), Sita / Ardra Sthana (damp cold place)	Vegaghata
MANASIKA	Chinta, Shoka	Tamasa-vardhte, Emotions	
VYADHIVISHESHA	Dhatu Kshya	Jwara, Atisara, Chardi, Pandu Pratishaya, Urha kshata,Rakta-Pitta, Udavarta, Visuchika, Alsaka,	Dhatu-kshya

How these Nidan cause the pathology at different level in the body, can be understood by the virtue of Guna-

Vata-prakopaka Nidana-

Rukshanna & Sheetpana - Diet having more Ruksha & Sheet guna, due to Samanya Vriddhi Karanam increases the Ruksha & Sheet properties in the body and the Karma of Rukshna & Sheet Guna is Sankoch – bronchoconstriction.

Vyayama, Vyavaya, Adhva – These all-physical activities increae the Vata with more Chala Guna.

Kapha-prakopaka Nidana-

Dadhi & Aamksheer – Both are having Abhishayandi property and cause the Srotoavarodh – obstruction.

Jalaja Anup mansa – Having Kleda Guna, increases the Klinnata in Bronchial tree – mucous production.

Agni-vaishamyakara Nidana-

Vishamashana – Irregular dietary habit causes indigestion, indigestion at every level which forms abnormal Kapha which mainly causes the obstruction.

Khavaigunyakara Nidana-

Raja-Dhuma-Vata-these all external factors make the Srotas more susceptible due to weakness in Pranavaha Srotas. .

Daurbalya, Kshatakshina, Pandu, Atisara, Raktapitta – these all are the Dhatu Kshaya-janya Roga. Dhatukshaya Avastha cause Rukshata in the body (Dhatu) which further causes the Sankoch & Kathinya in Srotas.

Pratishyaya, Kasa- are the Nidanarthakar Roga for Tamaka Shwasa because if these are not treated well and timely, creat the Khavaigunya in the Srotas and make it susceptible for the disease.

Manasika Hetu-

Krodh, Bhaya, Shoka- These all-psychological factors cause more secretion of Adrenalin, Nor-adrenalin which creat bronchospasm and increase mucous production. According to Modern view, etiological factors for pulmonary hypersensitivity include:

HOST FACTORS (Khavaigunyakara) are those factors that predispose individuals to

Develop pulmonary hypersensitivity. Host factors include the genetic predisposition to the development of Asthma or allergic sensitization,

- Genetic predisposition
- Atopy or Airway hyper responsiveness to allergens
- Gender
- Race/ethnicity

RISK FACTORS (Khavaigunyakara & Dosha-prakopaka) are factors that influence the susceptibility to the development of pulmonary hypersensitivity in predisposed individuals, precipitate pulmonary hypersensitivity exacerbations and/or cause symptoms to persist.These factors are of two types i.e**. risk factors** involved in the development or onset

of pulmonary hypersensitivity and the **triggering factors** involved in the development of exacerbations.These factors (stimuli) can be grouped into seven major categories iv –

- ✓ Allergens – Indoor- House dust Animal dander Moulds Domestic mites
  Outdoor- Pollens
- ✓ Pharmacologic- Drugs and diets
- ✓ Environmental- Weather changes Outdoor pollution Indoor pollution- Smoking- Active Passive Family size Socioeconomic status
- ✓ Occupational
- ✓ Respiratory infections
- ✓ Exercise related
- ✓ Emotional factor

HOST FACTORS-

1. Genetic predisposition: There is good evidence to indicate that pulmonary hypersensitivity is a heritable disease. A number of studies show an increased prevalence of pulmonary hypersensitivity in the phenotype associated with pulmonary hypersensitivity among the offspring of subjects with pulmonary hypersensitivity compared to the offspring of subjects without pulmonary hypersensitivity. The phenotype associated with pulmonary hypersensitivity can be defined by subjective measures (e.g. symptoms), objective measures (e.g. airway hyper responsiveness or serum IgE level), or both. Because of the complex clinical presentation of pulmonary hypersensitivity, the genetic basis of the disease is often studied through intermediate phenotypes that can be measured objectively, such as the presence of atopy or airway hyper responsiveness, although these conditions are not specific to pulmonary hypersensitivity. This lack of a clear definition of the pulmonary hypersensitivity phenotype presents the biggest problem when reviewing studies of the genetic basis of Asthma and atopy, because multiple definitions of the same intermediate phenotype are used in different studies. Despite intensive effort and advances in molecular biology and genetics, no gene (or genes) involved in the heritability of atopy or Asthma has been identified with any certainty. The results of several studies provide an indication that multiple genes may be involved in the pathogenesis of pulmonary hypersensitivity, and chromosomal regions likely to harbor pulmonary hypersensitivity susceptibility genes have been identified.

2. Atopy- Atopic diseases are charecterised by the production of abnormal amounts of IgE antibodies in response to contact with environmental allergens. Atopy appears to be an important host factor that predisposes individuals to developing extrinsic Asthma. Allergic Asthma is often associated with a personal and/or family history of allergic diseases such as rhinitis, urticaria and eczema, with positive wheal and flare skin reaction to intradermal injection of extracts of airborne allergens, with increased level of IgE in serum and/or positive response to provocation test involving the inhalation of specific antigen.iv

3. Gender- Childhood pulmonary hypersensitivity is more prevalent in boys than girls. However, the increased risk for males in childhood seems not to be related to gender but to the narrower airways and this difference disappears after age 30 when airway diameter / length ratio is the same in both sexes probably because of changes in thoracic size that occurs with puberty in made but not in females.

4. Race- The increase of Asthma prevalence in developing countries in different parts of world suggests that environmental factors may be more important than genetic & racial factors for the development of Asthma. Even though there is slight difference in Asthma prevalence between different races. This difference may be attributable to socioeconomic conditions, allergen exposure and dietary factors than to racial predisposition.ii

RISK FACTORS-

1. Allergens

- ✓ **Indoor Allergens**
- ✓ **Outdoor Allergens**

Indoor Allergens - House dust- House dust is composed of several organic & inorganic compounds, including fibers, mould spores, pollen grains, insects, insect feces, mammalian dander, mites & mite feces. Domestic mites are the most common potential indoor allergen and a major cause of pulmonary hypersensitivity worldwide. Domestic mite allergens are present in mite body's secreta, excreta & constitute the main source of dust derived allergens. Although mite allergens are carried in particles too large to penetrate the airways, the most important mite allergens have proteolytic activity & thus they might have an easier access to the immuno competent cells.

Animal Allergens- Household animals (cats, dogs, rodents, and cockroach) release allergens in secretions, excretions & dander. Cats are potent sensitizers. The principle allergen, Fe Id is found in cat pelt. The sebaceous secretions and saliva are probably the most important

source. Allergen has also to be identified in voided urine from male cats, dust from house with cat contains - 10 - 1500 m /g of (at allergen Fe/dI). A dog allergen has been purified from dog hair & dender. This antigen Ca dI is present in large concentrations in saliva & can be measured in house dust. The allergen city of rodent antigens is well known is animal handlers, who became sensitized to urinary protein. In some locations among some ethnic groups, sensitization to cockroach allergen may even be more common than to domestic mite allergen from German, America, & Asia than to domestic mite. Molds yeasts can act as indoor air borne allergens. House humidifier provides a special risk for indoor fungal growth & air contamination. The most common indoor fungi are Penicillium Aspergillus, Alternaria, Candid, Claclosporium.

Outdoor Allergens - Pollens - Pollen allergens associated with development of pulmonary hypersensitivity come mainly from trees, grasses & weeds, micronics particles of starch granules are released from pollens particularly after rainfall seem to be responsible for pollen induced pulmonary hypersensitivity exacerbation. Molds and yeasts can be outdoor airborne allergens. Fungi tend to be seasonal allergens in temperate zones, where some fungi sporulate on warm, dry summer days, and others prefer the rainy nights of fall.

2. Drugs & Food Additives- Some food & other ingested substances such as salicylates, food preservatives, monosodium glutamate & some food coloring agents have a recognized effect of causing pulmonary hypersensitivity exacerbations. Some drugs such NSAID's are causal risk factors for pulmonary hypersensitivity.

3. Weather changes- Adverse weather conditions, such as freezing temperatures, high humidity, and episodes of acute pollution brought on by weather conditions that promote the concentration of atmospheric pollutant and antigen, have been associated with pulmonary hypersensitivity exacerbations.

4. Outdoor pollutants -There are two main types of outdoor pollution: industrial smog (sulfur dioxide particulate complex) and photochemical smog (ozone and nitrogen oxides), and they can coexist in a given area. Weather conditions and local geographic features affect levels of air pollutants. Environmental pollutants such as sulfur dioxide, ozone, and nitrogen oxides can (at concentrations found in heavily polluted cities) trigger bronchoconstriction, transiently increase airway responsiveness, and enhance allergic responses. Thus, in theory, pollution might indeed contribute to the development of pulmonary hypersensitivity. However, although pulmonary hypersensitivity seems to be

more frequent in industrialized countries, there is little, if any, evidence that air pollution is directly responsible for the increased prevalence of pulmonary hypersensitivity in these countries. Exposure to traffic, particularly to diesel exhaust, may exacerbate pre-existing allergic conditions but does not necessarily induce the development of new cases of Asthma and atopy. Diesel particles have also been shown to absorb allergens from grass pollen onto their surface and may therefore act as potential carriers to increase deposition of pollen allergens in the lung. In this way, both the allergen dose and the antigenicity of the pollen allergen may be enhanced by automobile-related pollution.

5. Indoor pollutants- The contaminants and atmospheric dynamics of indoor air pollution are different from those of outdoor air pollution. Modern construction techniques possibly contribute to greater indoor pollution by lowering the turnover of indoor air. An increased indoor pollutant load may be in addition to the increased antigen load (in particular, from the feces of domestic mites) produced by changes in house design and forms of heating and furnishing (especially the use of carpets and upholstered furniture). Major indoor pollutants are nitric oxide, nitrogen oxides, carbon monoxide, carbon dioxide, sulfur dioxide, formaldehyde, and biologicals such as endotoxin. Sources of these indoor pollutants include the following:

- ✓ Cooking with natural gas or liquid propane, which produces carbon monoxide, carbon dioxide, sulphur dioxide, nitric oxide, and nitrogen oxides.
- ✓ Cooking on wood, kerosene, or coal-burning stoves, which produce carbon monoxide, nitrogen oxides, and sulphur dioxide as well as respirable particles.
- ✓ Heating with gas, wood, coal, and kerosene units and fireplaces, which produce carbon monoxide, carbon dioxide, nitric oxide, nitrogen oxides, respirable particles, and particulate soot.
- ✓ Building and furnishing with foam installations, glues, fireboard, pressed board, plywood, particle board, carpet backing, and fabrics that contain the volatile organic compound formaldehyde, and using paints or other materials that release isocyanates.

6. Tobacco smoke-Tobacco burning, which is a ubiquitous source of indoor irritants, produces a large and complex mixture of gases, vapors, and particulate matter, among them respirable particles, polycyclic hydrocarbons, carbon monoxide, carbon dioxide, nitric oxide, nitrogen oxides, nicotine, and acrolein.

- ✓ **Passive smoking** There is evidence that exposure to environmental tobacco smoke (i.e., passive smoking) increases the risk of lower respiratory tract illnesses in utero, in infancy, and in childhood. Sidestream smoke, which burns hotter and is more toxic than the smoke inhaled by the tobacco user, is particularly irritating to the respiratory mucosa.
- ✓ **Active smoking** Active smoking is associated with accelerated decline of lung function in people swith pulmonary hypersensitivity, greater pulmonary hypersensitivity severity and poor response to its treatment, supporting the concept that active smoking may contribute to pulmonary hypersensitivity severity even without contributing to the development of pulmonary hypersensitivity.

7. Family size- Studies have indicated an inverse relationship between pulmonary hypersensitivity and family size, having no siblings or one sibling is associated with an increased risk of pulmonary hypersensitivity compared with having more than one sibling.iii

8. Socioeconomic-status- The socioeconomic status of families may be a surrogate measure of lifestyle characteristics rather than a measure of risk factors. These lifestyle characteristics may include dietary habits, family size, access to health care, passive smoking, allergen exposure, or other yet-unknown determinants.iii

9. Occupational sensitizers- are usually classified by high / low molecular weight. High molecular weight sensitizers probably sensitize subjects and causes Asthma exacerbations by the same mechanisms as allergens, but the mechanism of action of low molecular weight sensitizer remains largely unknown.

10. Infections- Respiratory infection is the most common of the stimuli that evoke acute exacerbations of pulmonary hypersensitivity. In young children, the most important infectious agents are respiratory syncytial virus and parainfluenza virus. In older children and adults, rhinovirus and influenza virus predominate as pathogen. The mechanism by which viruses induced exacerbation of Asthma may be related to the production of T-cell derived cytokines that potentiate the infiltration of inflammatory cells into already susceptible airways.

11. Exercise & Hyperventilation- Exercise is probably the most common trigger of brief episodes of symptoms. Exercise incites airflow limitation in most children & young adults who have pulmonary hypersensitivity. The mechanism of exercise induced airflow limitations are mainly related to changes of the airway mucosa induced by associated

hyperventilation either cooling or rewarming or to changes of osmolarity of fluid lining the airway mucosa. Hyperventilation with cold dry or even hot air can cause pulmonary hypersensitivity exacerbations. Through unknown mechanisms like exercise, hyperventilation seems to be a specific trigger for pulmonary hypersensitivity.

12. Extreme emotional expression-Emotional stress may be a trigger for Asthma exacerbations, primarily because extreme expressions of laughing, crying, anger or fear can lead to hyperventilation and hypocapnia, which can cause airway narrowing. Panic attacks that are rare but not exceptional in some patients with pulmonary hypersensitivity have a similar effect.

These all etilogical factors explained in modern medical science are very well similar and directly or indirectly fit in to the framework of Ayurvedic Nidana of Tamaka Shwasa.

02.12 SAMPRAPTI

In Ayurveda concept of allergy is scientifically explained under 'Asatmyaja Vyadhi' while its effects are explained in hereditary, viruddhahara, dushivisha & ritu sandhi. Sushruta has mentioned hereditary diseases [273]explaining that qualitative &quantitative proportion of Vata, Pitta & Kapha is fixed at the time of fertilization only. Accordingly one's immunity is also formed in that proportion. Hence probability of formation of IgE antibodies among some people can be explained. Similarly concept of Viruddhahara is indicated [274]that can be related with food allergens. Viruddhaharas may give rise to many chemical reactions in our body. They may also interfere with the normal metabolism of our body. Another important concept is of Dushivisha. Acharya Charaka has clearly mentioned[275] that Dushivisha leads to blood vitiating disorders like Kitibha, Kotha, etc. which can be compared with allergic reactions. Concept of environmental allergy is scientifically explained under the heading of 'Ritu Sandhi'. Vagbhata has mentioned that[276] if Ritu Charya of Ritu Sandhi is not observed it gives rise to Astamyaja Roga, Tridosha Prakopa and vitiation of Dhatus.

To treat any disease properly, it is necessary to know the causative factors as well as the disease process or the pathogenesis. As, though the disease manifested is of the same name with identical signs and symptoms, its treatment modality changes according to its Hetu and Samprapti. Again treatment is nothing but resurrecting the deranged process of formation of Dosha, Dhatu and Mala in the body, which is termed as Samprapti, Hence it is of vital importance to understand every facet of the disease including the Causative factors and pathogenesis before deciding the treatment plan.

VIRUDDHAHARA JANYA SAMPRAPTHI

Agni is responsible for the well being of physical and mental functioning of body. Ayur, varna,balam, utsaha and prana also depends on agni. Though poshana of deha, dhatu, ojo, bala, varna is due to ahara, the main reason for its conversion is agni itself. Because rasadi formation does not occur from apakwa ahara. The samana and kopana of doshas also depends upon agni and hence the normal agni is absolutely essential for health. Normal activity of agni help the mintainance of physiological activity while its abnormal state produces pathology and its absence causes death of the human being. Hence maintainace of human life itself depends on agni[277]

a.Virudhahara and Agnidushti:

Virudhahara will lead to agnimandyam which in turn leads to improper functioning of the body. Dosha dushti and agni dushti are interdependent. Virudhahara leads to dosha dushti which causes agnimandya [278.] if food is taken during agnimandya it leads to Apakwa and becomes shuktha which is similar to visha[279]. Due to agni mandya, ahara rasas not properly formed. Therefore absorption of initial product become sluggish and it is retained in the amasaya for a longer time which is termed as "ama"[280]. Ama due to its toxic nature is equated as visha [281]. virudhahara also causes ama formation.

b. Srothomargarodha

Dosha travel in the body through the channels called srotas and these srotases are formed by different dhatus. All the bodily function is entirely depends upon srotas. All the doshas, dhatus and malas depends on srotas for their formation and transportation. The pathology of srotas is one of the contributing factors of diseases. The causes of srotovaigunya are:

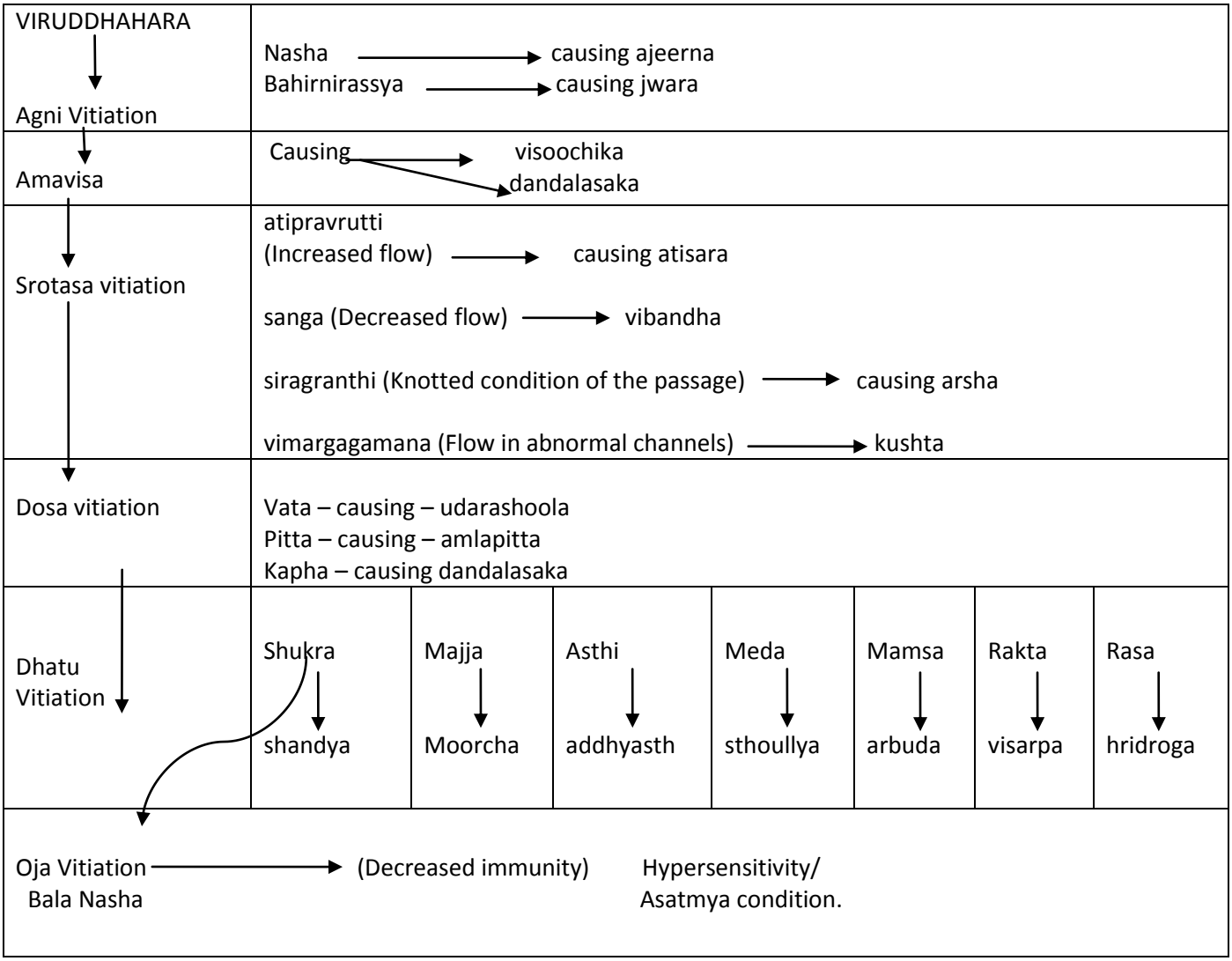
I] Diet and behaviour having qualities similar to those of doshas vitiate them. These vitiated doshas come in contact with dhatus, producing their vitiation. The site of such vitiated dhatu called as sthanavagunya or site of deformation

ii] Diet and behaviour having qualities contradictory to those of dhatus causes under nutrition and under development of dhatus. These dhatus cannot function properly. Vitiated doshas get easily lodged in under nourished and week dhatus or in such srotases and produce disease [282.]

Table no: 19 Effect of Virudha on different strotasas

No	Srotas	Virudhahara causing dushti	Type of Virudha
1	Annavaha	Atimatraakala	Matra Virudha Kalavirudha
2	Udakavaha	Ati Sushkanna sevanat	Vidhi Virudha
3	Rasavaha	Guru, Sita, ati snigdha, Atimatram	Matra Virudha
4	Raktha Vaha	Vidahinyanna Pana Snigdnoshnani dravani	Matra virudha
5	Mamsa Vaha	Abhisyandi, Sthula, guru	Vidhi Virudha
6	Medo Veha	Medyanam cati bhakshanat, Varunyaschati sevanat	Matra virudha
7	Asthivaha	Vatalanam ca sevanat	Vidhi virudha
8	Majja vaha	Atyabhishyandi	Vidhi Virudha
9	Mutra Vaha	Mutritodaka bhakshya	Krama virudha
10	Purishavaha	Atyasana ajirna	Matra virudha Vidhi Virudha
11	Sweda Vaha	Sitoshna akrama sevanat	Krama Virudha

Flow chart 1: Schematic presentation of viruddha nimitta samprapti



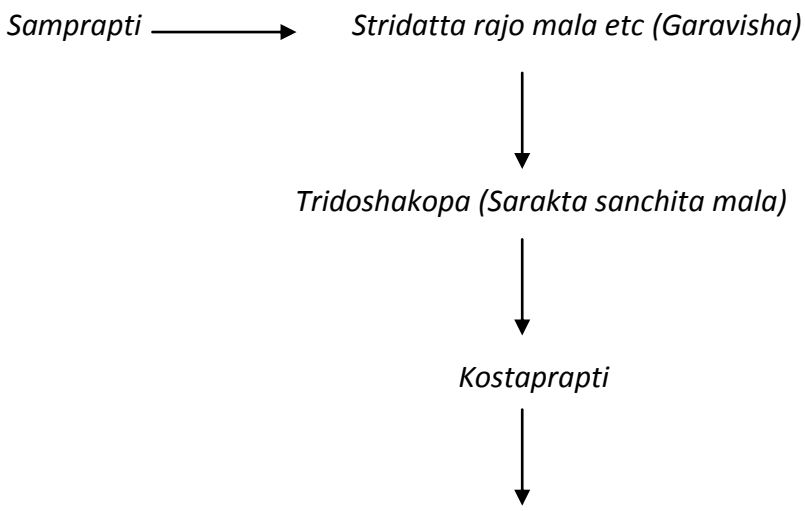
VISHA NIMTTA SAMPRAPTI

Action of *Gara Visha*

Action of Garavisha in samhita is explained in two ways according to the administration of poison both externally and internal. Action of Garavisha after intake or exposure to Garavisha mainly depends on combination of poisons, dose and root of administration. Action of Garavisha when it is applied through external skin by different churnas (Keeta deha Churnas) mixed with yogas, symptoms caused, are explained in Sushruta samhita in Keeta kalpa chapter.[283]. In this condition action of Garavisha may be through transdermal absorption of Garayogas causing symptoms of Visha.

Action of *Garavisha* after entering our body explained in the pathogenesis of *Udara* roga *samprapthi* of *Sannipatodar*a, *baddodara* and *chidrodhara* respectively. Here in *Udara roga* main *nidana* is intake of *Gara* (*stridatta rajo mala, pakshmavalai*) along with food.[284]

Flow chart 2 : Schematic representation of action of Gara Visha



Charaka said same opinion of *Vaghbhata* in *Udara roga Nidana -Garashanath*. The *Gara,* which is combination of different drugs, can be considered as unwholesome. According to *Indu* commentary *Gara* includes "*Sakalamap*i *vasthu jaatam*", and it is special type of poison unlike *Jangama* and *Sthavara Visha*.[285]

Some act adversely due to their mutually contradictory properties, some by combination, and some by method of preparations, some by virtue of place, time and dose and some by nature. The idea of incompatibility is so intimately connected with intrinsic poison. The development of toxin within the body mainly depends upon the digestive capacity, properties of food, frequency of ingestion and mental condition.

…Apachyamanam shukthatvam yatyannam visharupatam…

Accumulation of this toxin in specific tissue leads to specific diseases. After entering the body the toxin may be detoxified in different manners, oxidation, hydrolysis and conjugation. The biotransformation of toxins is a complete or a partial process of detoxification. Artificial poisons are manmade by compounding various substances that are toxic and non toxic. Artificial poisons are compounded from non toxic substance as they are not metabolized normally (*Apaki*) they attain the property of visha and produce defective biotransformation triggering on sequential pathological reactions. This toxin which is not metabolized normally can be correlated to *Ama* which is produced by improper digestion by deranged *Agni* in gut and tissues. This *jataragni mandya* causes *dhatvagnimandya* and deranges the proper *dhatuparinama* and in term causes *dosha datu* and *srotodusti*. The excellence of all the tissues is *ojus*. The depletion of *ojus leads to* depletion of normal health of person and causes diseases. This depends on affinity of some poison to specific tissue. In other words each toxin has specific affinity to particular organ or system in the body. For example in broader sense, *Upavishas* are considered as slow poison and comparatively less toxic. Among these *Upavishas, Vishamushti* (Strychnus nuxvomica) will mainly influence on nervous system and is neurotoxic. Above influence of Vishamushti depends on dose. The drug *Dhattura* (Dhattura stramonium) which has influence on Respiratory system is also sedative in action. There is a specific poison affinity to different systems in the body. This depends on particular toxin ingredients. Action of *Visha* in the body also depends on *Prabhava,* the main factor in Visha *vriddhi* and *Kshaya. Indhu*, Commentary on *Astanga sangraha* explained, by giving *Sirisha* and *Haridra* poison will pacify, opposite to this by *Swapna* and *Meghavarana Visha* will increases in the body. This is because *Prabhavakarma*. The reason behind this action may be *Panchamahabhutas* special action; no body should doubt this action Prabhava.[286]

Psychological actions of Garavisha

As the definition of visha indicates that which produces Vishaada. Here, vishaada meaning manovikaara or chittaavasaada. Any visha dravyas having ten qualities specially vyavahi, vikaasi, suksma and tiksna etc these qualities swift in action affects the body and mind quickly. These visha gunas are opposite to ojus. Poisons like Gara Visha (artificial poisons) can influence human mind very intensely.

This can be broadly classified into two

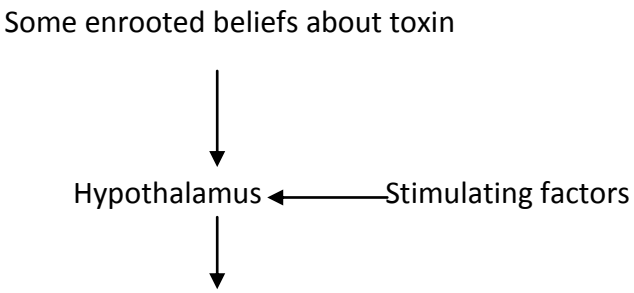
3. Direct toxic effects
4. Indirect toxic effect

Direct toxic effects

Generally, poisons are potent and they can be absorbed quickly. Upavishas, comparatively slow poisons in broader sense can be considered under Gara Visha have action on CNS which will affect the function of mind. Euphoria, confusional psychosis and non specific personality changes etc are some of the examples. In Ayurvedic samhita there is explanation of treatment Hridayaavarana which is mainly for Chitte visha vyapthi prtisheda according to Dalhana.[86] This shows Samhitas give more importance to psychological aspect while treating poisons.

Indirect toxic effects

Some enrooted beliefs of people such as poison given to them by others etc. along with some stimulating factors, causes stimulation of hypothalamus. There are some specific centers in hypothalamus to regulate or to modulate different activities in our body. They have crucial role in body functions like circulation, fluid and electrolyte balance, temperature regulations, feeling of hunger and thirst. So, stimulation of nuclei may result in various somatic manifestations, as a general body mechanism. Some patients may have false belief that their disease is due to ingestion of toxic materials. They are obsessed that they were poisoned in deceit. In many cases, more than toxic effect, fear and anxiety causes much severe symptoms.



In Ayurveda it is clearly explained that psychological action of poison in the context of Shanka Visha (Dubious poison) is a clinical condition generated by fear alone when a person is pricked by something in darkness, as he assumes that he is bitten by a snake and exhibits signs and symptoms accordingly. The appearance of symptoms of dubious poisoning may be psychological reaction. Same mechanism holds well in Gara Visha aspect also. In this condition treatment is psychological.

Cumulative period

Yogaratnakara mentioned Garavisha will affect the person after fifteen days or month period. same opinion is told by Basavarajeeyam in 23rd chapter.

Action of Dooshi Visha

Acharya Sushruta has described Dushivisha in kalpasthana[287] According to him, when an Artificial or Natural toxins afflict a person, he is treated with antitoxic treatments. As a result, a complete elimination of these toxins from within the body is not possible. They may remain in a dormant state for quiet long period.

Acharya Charaka Similar meaning can be elicited from chikitsasthana [288]He has opined that intake of toxic drugs, which are less potent (Hina Veerya) remains in a dormant state within the body for years together, without causing any harm to the body. It remains in the latent stage due to avarana of Kapha.

Dhatu Dushti

Chakrapani has commented upon this statement of Acharya Charaka, “Dushivisha vitiates the Dhatus after the lapse of time, on obtaining favorable conditions.”To this Sushruta[289] has mentioned “When causative factors like Desha, Kala, Anna, which add to the potency of the Visha; & Divaswapna are indulged in, that Dushivisha which is lying dormant will become more potent vitiating the Dhatus leading to the manifestation of a disease. It is seen

commonly that Allergic disorders are not affecting every individual similarly in presence of same causative factors.

Desha

It is commonly observed, that People of same Area, Colony, City, State, etc. are not affected equally on an exposure to a foreign body. Though living in the same environmental conditions, some people remains healthy while only few of them will be allergic to certain things. Change of colony, city, etc. also plays an important role in the production of allergy.

Kala

This factor can be seen easily in our day-to-day life. It is generally observed, that often patients with rhinitis have maximum presentation of signs & symptoms inspiring & rainy seasons, it is with other allergic disorders too. The detailed description of it is given in Ritu Sandhi point.

Anna

It is already described in Viruddhahara.

Divaswapna

Day sleep is known as Divaswapna that results to Agnimandya, which is the root cause of all the diseases.

Conditions developed due to Dooshi visha are as follows,
(Allergic disorders described in Ayurveda)

Table No: 20 Dushi Visha Lakshanas

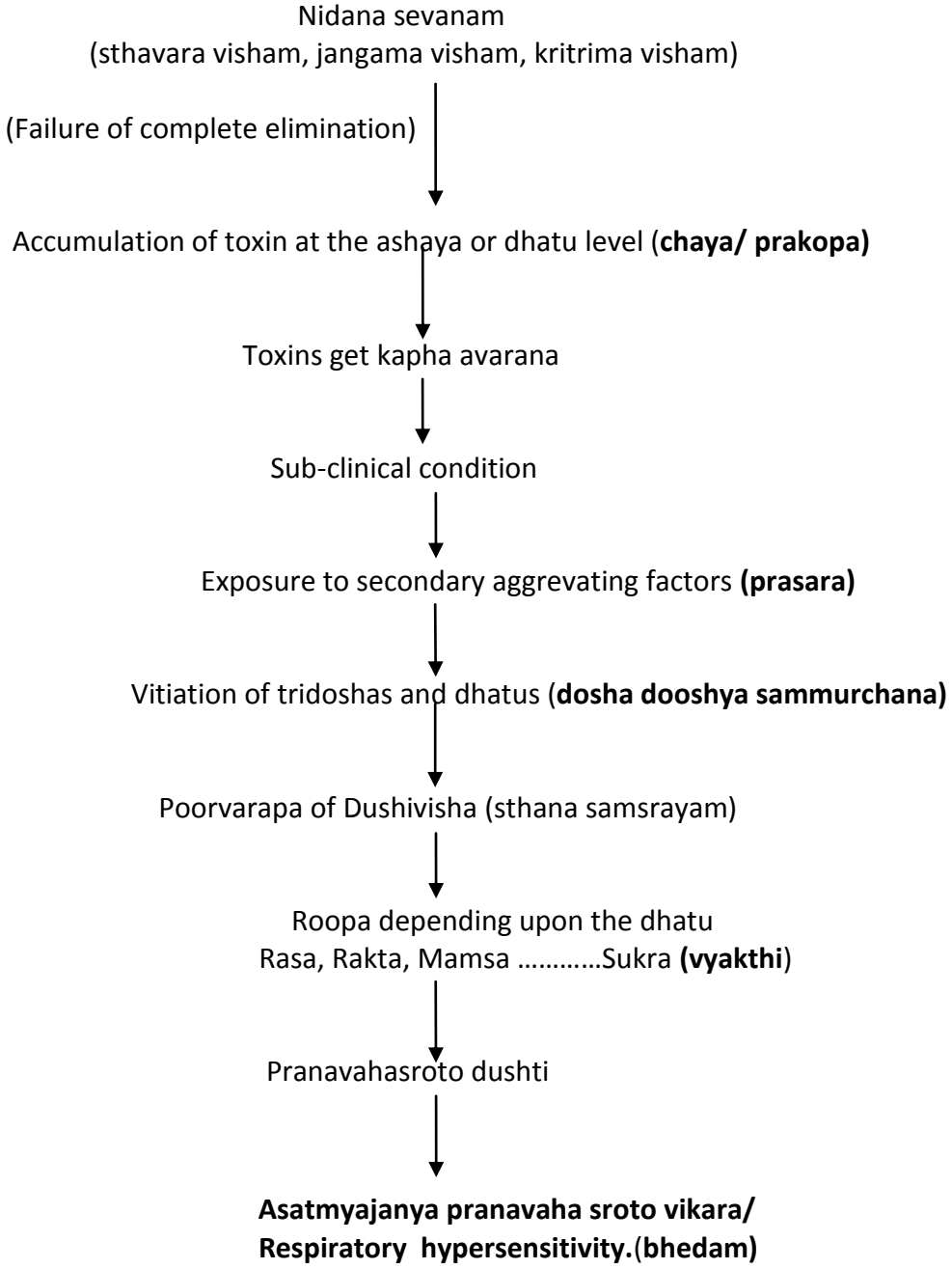
Sl no	LAKSHANAS	CHARAKA Ch.Chi.23/31	SUSHRUTA S.Ka.2/30)
1	• Shonita Dushti	✓	
2	• Kitibha	✓	
3	• Kotha	✓	✓
4	• Avipaka		✓
5	• Arochaka		✓
6	• Mandala		✓
7	• Shotha		✓
8	• Vamana		✓
9	• Atisara		✓
10	• Jalodara		✓
11	• Mandala		✓

Many of the diseases mentioned above has allergy as one of the causative factor.

Relation of Dushivisha & Histamine

- Histamine is also present in the body in latent form similar to Dushivisha.
- It remains in an inactive form within the covering of cell wall.
- Cell wall can be called Kapha Dravya.
- Histamine is active only by its chemical reaction with Histaminaze.

Flow chart 3: Schematic representation of pathogenesis of Dooshi Visha



Visha and Gara visha in the context of virudhahara

Virudhahara is compared to visha and gara. Hemadri [290] in his commentary on Ashtanga Hridaya says that virudhahara is said as visha because it causes instantaneous death. Sushruthacharya has specifically mentioned that dravyas like pinyaka, kavuka, karira, on combination with payas becomes vishatulya [291]

Virudhahara is compared to gara visha because it may cause delayed death or leads to diseases.[292] Many diseases caused by virudhahara like jwara, kshaya, pandu, sotha, adhmana, Arsa, udara unmanda etc., are same as that occurs due to gara [293]

Mechanism of Action of Visha and Virudhahara

When visha is brought into contact with the body, it will at first cause the dushti of raktha and then the three doshas later it reaches hridaya [294]. Hridaya is the seat of ojas. Visha is having qualities opposite to that of ojas. If the visha gunas predominate, the patient dies on the contrary when ojo gunas are superior to those of visha gunas the patient survives.

Virudhahara is one of the important causes for rakthadushti [295.] It also causes the utklesha of tridoshas [296] Rakta in its dhatu form are responsible for bala, varnam and sukham. It is the basis of life. Thus raktha is very closely related to ojas in its function. Sufficient amount of food with appropriate quality will be digested properly and converted into consecutive dhatus. When virudhahara is followed dhatu parinama process will be deranged and affect the ojas. If virudhahara combination is, very toxic death will occur instantly if the derangement is not much stronger, it will lead to decreased bala; vyadhikshamata of the body depends upon bala [297] and the deterioration of it will lead to various diseases

Thus, the total samprapti can be summarized in the following manner,

Samprapti -- Etiopathogenesis

Sahaja + Ahra,vihara (viruddha) + Visha (Gara visha/ Dooshi visha) = Allergy

A Sahaja = Poor balavriddhikara bhava = Susceptible/ Diseased

B Ahara, vihara

1. Ahrara, vihara - Dosha hetu

A) Dosha (Anyonya moorchita dusta dosha) = Ama

e.g :

Tamaka swasa – Vatakapha{pittasthana} =Ama

Amavata – Kaphavata {tridosha-vatapittakapo bhuyo dushitah so annajo rasah} =Ama

Sheetapitta – Vatakapha {pittena saha sambhuya}=Ama

Kusta – Tridosha = Ama

B) Anyonyamurchita dosha = Ama[298]= Mala[299] =

Altered immune reaction.

In universe opposite qualities fight each other –

Viruddha gunanaam tu paraspara upaghato bhavati, yathaa-

Vahnitoyayoh[300] Doshas are sahaja satmya, though there is opposite gunas of dosha[301] – that is health.

But, sometime rarely doasha guna start fight each other[302] **Parasparagunaupaghatastu yadyapi doshaanam praayo naasti eva, tathaa api adrushtavashat kvachit bhavateeti bhaavah.**

Body immune don't destroy body but sometimes it itself destroys body = Altered

C) Ama – dosha – Amavisha

– saamavata,saamapitta,saamakapa [303]

– Amahetu [304]

– **virudda adyashana ajeernashana seelinah punar aamadoshah amavishamityacakshate bhishajah**[305]

Viruddham visha garopamam[306] - sudden/gradual effects

Visha – 1.sadya (savisha sarpa)

2. Kalantara (garavisha, doosheevisha)

Kalantara visha – effects[307]

Amashaya – kapavataroga (amavata,tamakasvasa)

Pakvashaya – anilapittaroga (raktatisara = ulcerative colitis)

Rasadi – rasadi vikara(all systemic diseases)

Gara /dooshee visha = asatmya,virudda

D) Viruddha =Nidana =dhatu dusti without dosha[308]

- **Rasadishu ayathartham vaa tadvikaaraaya kalpate** |[309]

– dosha dushtikaaritvena

dhaatudooshanakaratvam na labhyate, vishishta shakti

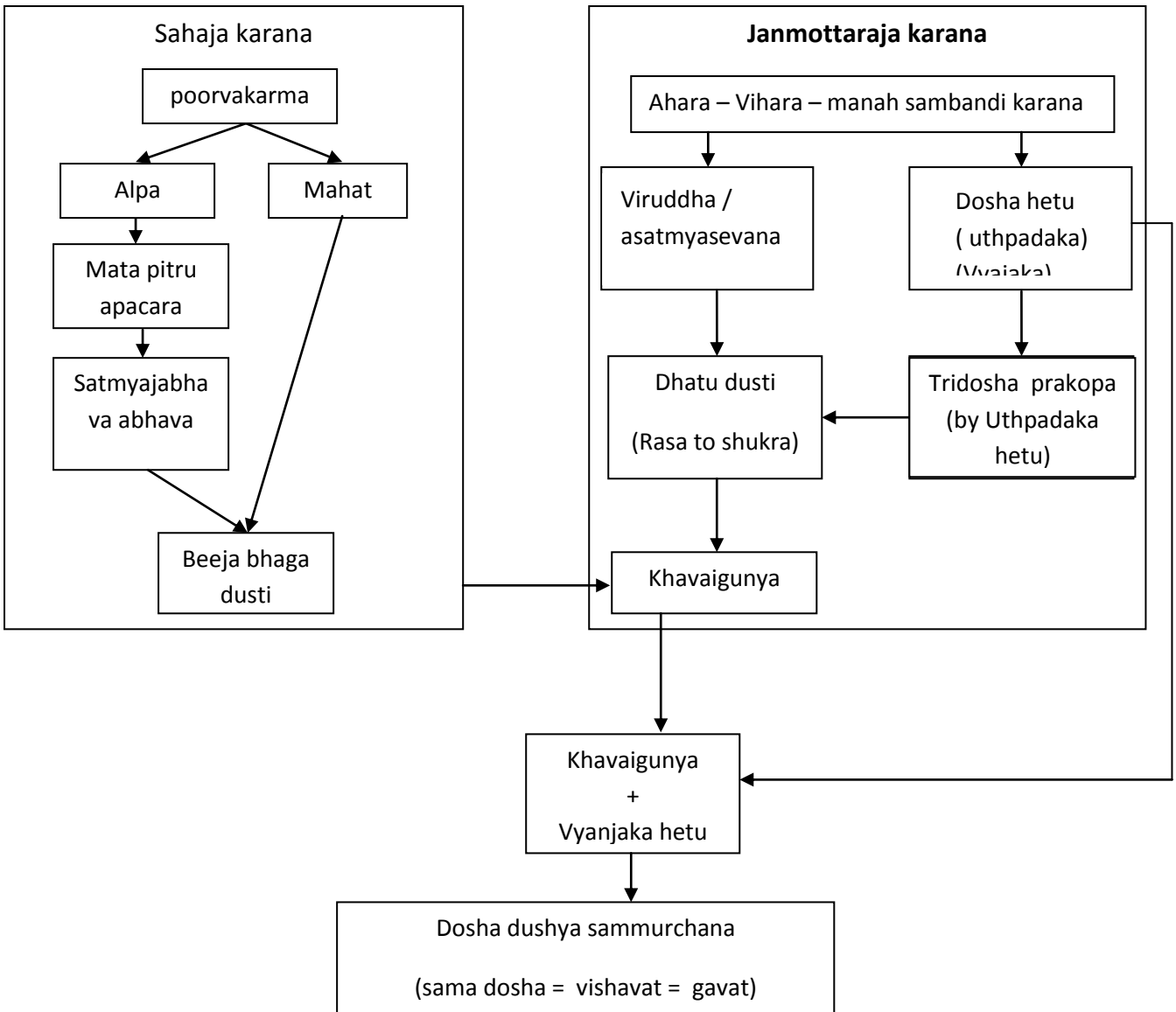
dravyaanaam; tathaahi- kaanicit doshadushtikaraani, kaanicit

dhaatudushtikaraaniti;

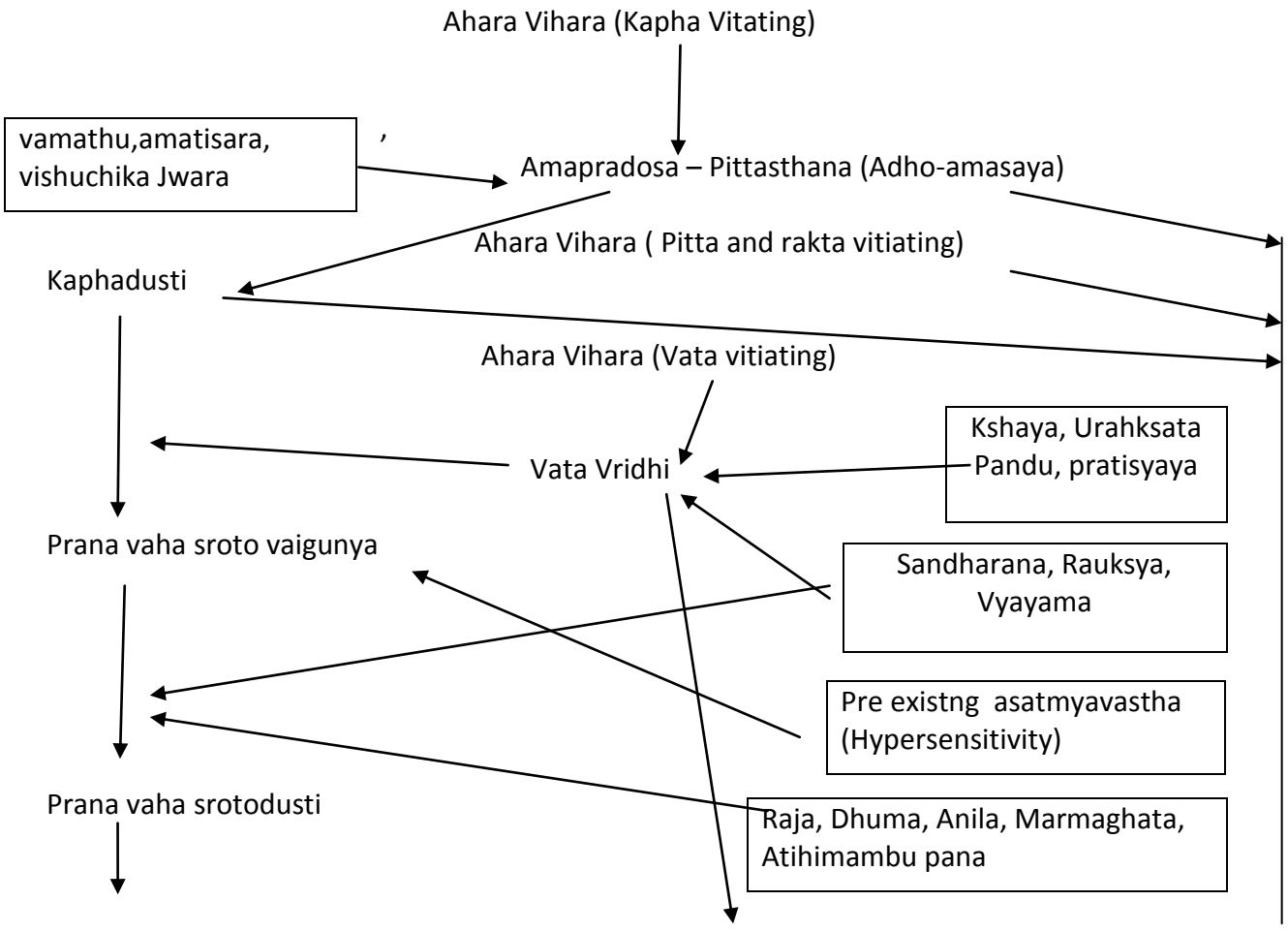
Dravyas may vitiate dosha may disturb dhatu. Some dravyas are having the capacity to damage the specific dhatu (tissue) directly with out the involvement of dosha there by permanent damage &disease.

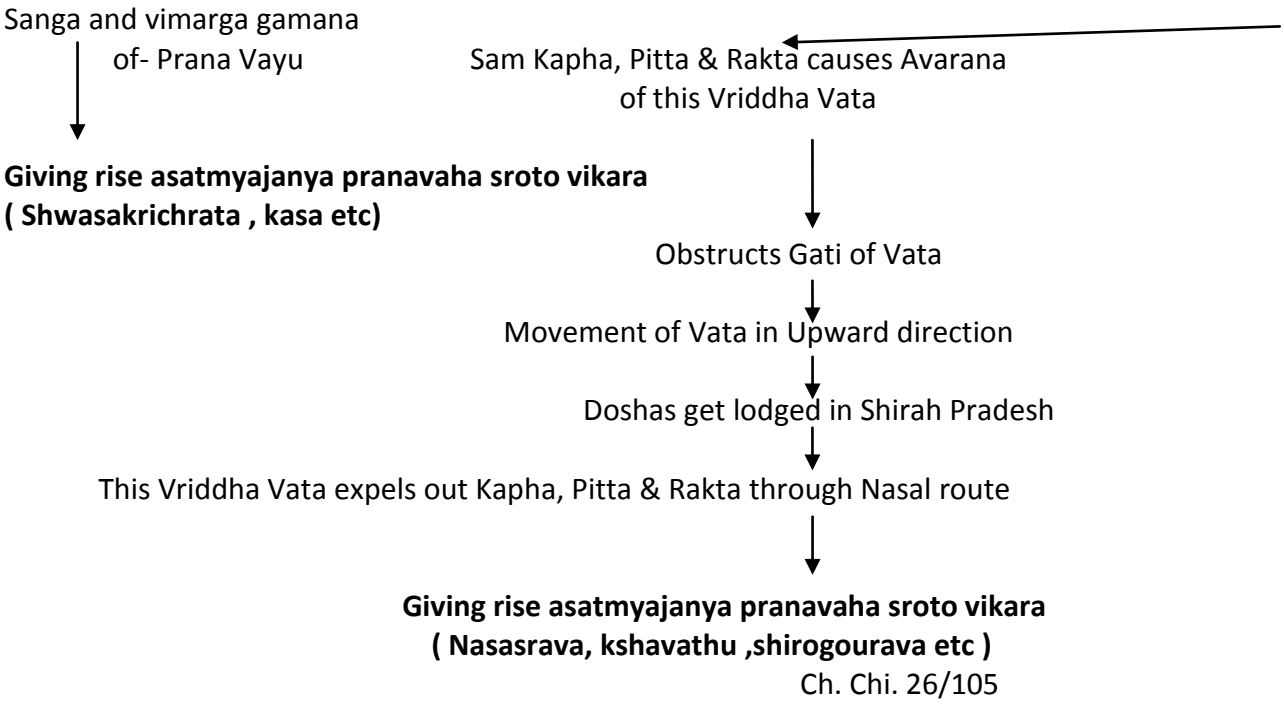
– vishadi bahudrushataantena[310] …. &which can be compared to 4 types of hypersensitivity reaction.

Flow chart 4: Diagrammatic presentation of samprapti of asatmyavastha (Hypersensitivity)



Schematic representation of Samprapti of Pranavaha Sroto Dushti





Samprapti Ghatakas

1. Nidana - Kapha Vata Prakopaka Ahara Vihara, etc.
2. Dosha - Kapha Vata Pradhana, Alpa Pitta
3. Dushya - Rasa, Rakta
4. Agni - Jatharagni, Rasadhatwagni
5. Ama - Rasadhatwagnimandya
6. Srotas - Rasavaha, Raktavaha, Pranavaha ,*Anna vaha Srotas* .
7. Srotodushti - Sanga, Vimargagamana, Atipravriti,
8. Udbhavasthana - Amashaya
9. Sancharisthana - Gala, Jatrurdhwa, Nasa, Ganda, Pradesha
10. Vyaktasthana - Nasa
11. Rogamarga – *Abhyantara* Bahya
12. Dosha Marga - Shakha
13. Vyadhiswabhava - Ashukari
14. Vyadhi Avastha - Vyakta
15. Adhisthana - Urah Pradesha (phuphusa) Nasa, Shiras

The disease process starts by the aggravation of Tridoshas by multiferous factors i.e. exogenic & endogenic. The proper functioning of Dosha, Agni, Malas & balanced status of Atma, Mana & Indriya bring about health, whereas disturbance of equilibrium lead to disease. Samprapti plays the most important role to detects the disease or Bhedas of them, (among the 5 aspects of diagnosis) after Hetusevana till the disease appears[311]. (The process leading to the disease is called the Samprapti.

This Sampratpti takes place when causative factors are manily Vata vitiating. Such causative factors vitiates Vata, leading totis Vriddhi. Here Kapha, Pitta & Rakta are in Sam Avastha. But they'll obstruct the Gtai of this Vata causing Avarana of Vayu. Doshas get lodged in Shirah Pradesh. This Vriddha Vata expels out Kapha, Pitta & Rakta through nasal route, giving rise to symptoms of Pratishyaya.

Here Vata gets vitiated with its own etiological factors & Kapha, Pitta & Rakta gets vitiated with their etiological factors. Both are individually vitiated. This vitiated Kapha, Pitta & Rakta will obstruct the Gati of Vata causing its 'Avarana' which leads to the Urdhavagamana of Vata. Sthana Sanshraya in Shirah Pradesh, giving rise to the disease Pratishyaya. The above given brief description of Pathogenesis of Pratishyaya described by various Acharyas may further be elaborated on the basis of

Shada Vidha Kriya Kala given by Sushruta.

Sanchaya

Doshas when in a balanced state shall remain in their own Ashayas as a normal body phenomena, but any disturbance results to an inequilibrium between them. There is over accumulation, though remaining within their original limited spaces. This is characterized by vague symptomatology. In this stage an over all features of an aversion towards similar (to the Doshas aggravating factors) & attraction towards contraries (Viprita Gunechha) is usually observed. Predominance of Vata Recognized by Stabdha Kostha & Purna Kostha Predominance of Pitta Peetavabhasata Predominance of Kapha Gaurav & Alasya Acharya Dalhan clarifies phenomenas of each doshas in relation to head (Urdhavajatrugata). Pranavayu Its field of activity is related to almost all the functions of head Alochaka Pitta Its field of activity is related with vision Tarpaka Kapha Its field of activity is related with all sense organs (Indriyas) Shonita (Rakta) Circulating through blood vessels All these get accumulated at their sites in Sanchaya stage.

Prakopa

When the provocative factors for the previous stage are allowed to act further or the physician could not advise the proper measures to be taken in the first stage, then the previously accumulated Doshas which were in accumulative stage gets vitiated. Still seated in their own original sites. Thus sufficiently vitiated Doshas will give rise to additional generalized Doshika symptoms which were seen in the Sanchaya stage. The signs & symptoms of this stage will be generalized as well as localized.

Prasara

Acharya Sushruta says that the Doshas which have become Prakupita, due to causes already mentioned, expand & overflow from the limits of their respective locations. Further he points out that Vayu which possess the power of locomotion or extreme motility should be looked into as the cause of the expansion or overflowing of Prakrut Doshas either alone (Eka), double (Samyoga Dwanda) or in the triple (all the three, Sannipata) together with Rakta expand & over run the body and in all directions. If the person continues to indulge in unhealthy foods etc., or if the treatment is ineffective, the abnormality continues further to the onset of the fourth stage. During the first three stages, the unhealthy foods and activities not only bring about increase of the Doshas but at the same time, also bring about mild abnormalities in the Kosthagni (digestive activity in the alimentary tract), the Dhatus (tissues), the Srotas (pores, channels of the Dhatus, cell pores, ducts.) and the Ojas (the vital fluid material present in every Dhatu and responsible for its strength vis-a-vis the strength of the body). The Kosthagni, which digests the food, becomes abnormal (Agni Vaishamya) by the action of the unhealthy foods etc. and also by the increase of the Doshas. Increase of Vata causes Vishamagni (irregular, unpredictable, erratic) making digestion of food variable from time to time, day to day etc. Increase of Pitta causes Tikshnagni (excessively keen, strong) making digestion unusually quick and changing of food materials, and increase of Kapha causes Mandagni (weak, poor) making inadequate, and delayed digestion of food. In all these abnormal states, the food does not undergo perfect digestion and undigested materials - Ama - (improperly processed, over processed or inadequately processed intermediary products of digestion) remain over in the Ahara Rasa (essence of food). The quantity of such

materials is more incase of Mandagni, moderate in case of Vishamagni and very little in case of Tikshnagni. In Prasara Avastha the vitiated Dosha through Rasa and Raktavaha Channels circulates through out the body.

Sthana Samsraya

The circulating Doshas mixed with the circulating Rasa Dhatu, now tend to settle at certain place in the Dhatus (Sthana Samsraya) and bring about abnormalities there, especially the Srotas (pores, channels of cells of tissues) The Dhatus (tissues) may not fall on easy prey to the onslaught of the Dosha. They have their defense, in a fluid material known as Ojas which is responsible for their Bala (strength), to carryout their functions (Karya Shakti) and to prevent diseases (Vyadhi Utpada-Pratibandhakatva). As long as the Ojas is normal in its Pramana (quantity) and Gunas (qualities), the Doshas cannot vitiate the Dhatus or the Srotas. The Ojas undergoes Kshaya (decrease) due to many causes such as lack of food, physical strain, injury to vital organs, excess indulgence in alcohol and such other substances of poisonous nature; anger, grief, worry and other mental emotions; loss of blood, semen and other tissues etc. The decrease of Ojas makes the Dhatus poor in strength and succeptable to the bad effect of the increased Doshas. The Srotas may undergo following four kinds of abnormal changes

(Sroto Dushti or Khavaigunya).

(a) Atipravriti – increased functioning.

(b) Sanga or Rodha – obstruction, blockage, decreased functioning and consequent increase in size.

(c) Granthi – growths, thickening, etc.

(d) Vimargagamana – movement of material in wrong direction, passage or place.

The place or site (organ) where one or more of these Srotodusti/ Khavaigunya has taken place, becomes the site of origin of the disease. The Ama, which was formed by Mandagni accumulates in the Rasa Dhatu and brings about changes in it. Its normal Tanutva (thinness) changes to Bahalatva (thickness), Visadatva (non-sticky nature) to Pichilatva (sticky, slimy) and normal Pramana (quantity) to increased quantity (Vriddhi). This kind of Sama Rasa (Rasa mixed with Ama) circulating all over the body finds difficulty in entering into the minute Srotas, which have also becomeabnormal by this time. Sama Rasa blocks the Srotas, accumulates outside the Dhatu Pramanas (tissue cells) and makes for Dhatu Vriddhi. Every Dhatu has its own specific Agni anologus to the Kosthagni (digestive activity in the

alimentary tract); these Dhatvagnis derive strengthfrom the Kosthagni and work similarly. They also become Tikshna (strong) and the latter causes Vriddhi (increase) or Kshaya (decrease) of the Dhatus have been considered as Vaishamya (abnormalities). Even the four kinds of Srotodushti also form part of Dhatu Vaishamya; hence Dhatu Vaishamya itself is termed as the disease. Thus, in the fourth stage, important abnormalities occurring inside the body are further increase of the Doshas, their localization at certain place, (Sthanasamshraya), decrease of Ojas (Ojas Kshaya), vitiation of Srotas (Srotodushti, Khavaigunya), accumulation of Ama (Ama Sanchaya) and union of abnormal Doshas with Dushyas (Dosha-Dushya Sammurchana); all these act as essential prerequisites for the onset of the disease. This Kriyakala is the stage of actual commencement of the disease. It is characterized by appearing of Purvarupa/Pragrupa (premonitory, prodromal symptoms) which are produced by each one of the above said abnormalities.

This Prana, Kapha, Pitta Avrita Udana Vata gets lodged in the Pranavaha Srotasa, specially in Nasa, where Khavaigunya is available. The premonitory symptoms of the disease can be demonstrated in this stage. In this stage patient gets following premonitory symptoms of Pratishyaya.

(i) Shirogurutvam(Heaviness of the head)

(ii) Kshavathu (Sneezing)

(iii) Parihrishtaromata (Generalized horripilation)

Vyaktavastha

This is the fifth Kriyakala and characterized by the full manifestation of the disease (Vyadhivyakti) with all its symptoms and signs (Rupa). Each one of the aforesaid abnormalities contribute to its own symptoms and signs, which are clearly recognizable. They vary in number and strength from one patient to the other, depending upon the age, sex, constitution, strength of the causes and many other factors. The diseases are given specific names based on the chief symptom/sign or the organ affected and many other factors. They are even classified as arising for many one of the Dosha (Ekadoshaja), two of them together (Dwidoshaja, Dwandvaja, or Samsargaja) or by all the three of them together (Tridoshaja, Sannipataja). The abnormalities, though profound, can be brought to normal easily when effective treatment and all other favourable factors are present and with difficulty in the presence of unfavourable factors. Some times the disease is uncontrolable

and progresses further to the sixth and final stage. In the process of Vyaktavastha the following symptoms of Pratishyaya may be present.

- Shirashula- Kaphotklesha- Ghrana Viplava- Nasa Avarodha- Swarabheda etc.

Bhedavastha

During the sixth Kriyakala all the abnormalities become still more profound and irreversible. In spite of the best treatment, they continue to persist and make the patient very debilitated, by loss or depletion of the Dhatus, give rise to one or more Upadrava (complications). Some times even Arishta Lakshanas (signs and symptoms which herald death) might also manifest. All these grave symptoms and signs differentiate this person from others. Hence, this stage is called as Bheda. In case of the disease Pratishyaya, one can easily conclude that the disease is either chronic or complicated on the symptomatology of aneamia, deafness etc. It may lead to production of, Dushta Pratishyaya and Kasa, Svasa, Kshaya also. Hence the concept of Shada Kriyakala in reference to the disease Pratishyaya seems to be more scientific both from the understanding of the disease process, as well as its treatment view point.

Probable Effect of Nidanas on Doshas :

1) Intake of Atisheeta Jala (Consumption of Chilled Water):

Excessive consumption of Sheeta Jala vitiates Vata Dosha. Due to Sheeta Guna, this vitiated or Vriddha Vata when reaches Shirah Pradesh Kapha, Pitta Rakta Doshas do Avarana of the Gati or the movement of the Vayu Doshas get lodged in Shirah Pradesh Vriddha Vata expels out Kapha, Pitta andRakta through nasal route manifesting the disease Pratishyaya. This Samprapti is Sadyah in nature as mentioned by Sushruta[312]. On the other hand if same Nidanas are consumed for a longer duration, it vitiates Kapha Dosha which leads to Mandagni in turn produces Amavisha which creates Adya Dhatu Rasa Dhatu Dushti through Hridaya it circulates throughout the body through Rasavahi and Raktavahi Srotasas Sama Doshas obstruct the movement of Vata, thus creating Pratiloma Gati of Vayu takes Sthana Sanshraya in Shirah Pradesh (in Khavaigunya) Dosha Dushya Sammurchhana takes place, which manifests the diseases Pratishyaya (Rogotpatti) by expelling Kapha, Pitta and Rakta through nasal route.

2) Intake of Atisheeta Jala in Sleshma Prakriti (Consumption of Chilled Water In Shleshma Prakriti Person) :

Same pathogenesis will take place but, with more severity of symptoms due to Shleshma Prakriti. Because along with Vata Prakopa, Kapha is also being triggered or vitiated it due to Sheeta Guna of Jala and Kapha Prakriti of the person. So along with Vata vitiation Kapha also gets vitiated and the pathogenesis of the disease Pratishyaya may take place, in either of the ways, mentioned above.

3) Atimatra Guru-Sheeta-Madhura Ahara (Consumption of Heavy, Cold and Sweet Meals in Excessive Quantity) :

As mentioned by Acharya Charaka [313] With excessive intake of Guru, Sheeta and Madhura Ahara, Vata and Kapha both Doshas get aggravated due to the principle "Sam-Guna Vriddhi" and these aggravated Vata and Kapha follow the same vicious cycle of pathogenesis, as mentioned above to manifest the disease Pratishyaya.

4) Snana with Atisheeta Jala (To Take Bath With Too Cold Water):

5) Ati Jala Krida (Excessive Bath/Swimming in Cold Water) :

Snana with Sheeta Jala and Ati Jala Krida, both vitiate Vata Dosha. This vitiated Vata when reaches Shirah Pradesha, where it is obstructed by the Avarana of Kapha, Pitta and Rakta. Thus, the Vriddha Vata expels out the Doshas through nose giving rise the disease Pratishyaya, as mentioned above. When the same etiology continuous for a longer duration, it may vitiate Kapha along with Vata also, leading to same pathogenesis of Pratishyaya due to Agnimandya, as mentioned in Nidana one.

6) Nidra After Dugdha Pana (To Sleep After Taking Milk) :

Increases Snigdha Guna Kapha Vriddhi vitiates Agni leads to Agnimandya Amotpatti Rasa Dhatu Dushti and so on, the manifestation of the disease Pratishyaya.

7) Sheetam Atipratapa (Exposure to Cold of Head) :

Excessive exposure to cold of head [314] may cause a local lesion in Shirah Pradesha (Khavaigunya) by vitiating Vata, Kapha, Pitta and Rakta, that too particularly in person having low immunity. In such cases any further exposure leads to Sadyah or Acute manifestation of the disease i.e. Achaya Prakopa (without pathogenesis). Kapha, Pitta and Rakta obstruct the movement of Vata, which leads to Pratiloma Gati of Vayu and following the same pathogenesis creates the same sympatomatology of disease Pratishyaya.

8) Mandagni :

Due to Mandagni there is indigestion of ingested food leading to production of Amavisha vitiates Rasa Dhatu vitiates Doshas while circulating through out the body through Srotasas

takes Sthana Sanshraya at Khavaigunya in Shirah Pradesha Avarana of Vata with Sama Dosha (hampering the Gati of Vayu) leading to Pratiloma Gati (here, Udana Vayu gets Avarana) manifestation of the disease Pratishyaya.

9) Ajirna :

Acharya Charaka endorses the same etiopathogenesis in the production of disease Pratishyaya with Ajirna, where production of Amavisha takes place with indigestion which results Mandagni and the same pathogenesis takes place to produce the disease Pratishyaya as mentioned above [315]

10) Vishamashana :

Acharya Charaka again cites another causative factor, Vishamashana, for the manifestation of the disease Pratishyaya. The same pathogenesis participates in the manifestation of disease by production of Ama.[316]

11) Snana in Ajirna (To Take Bath in Ajirna) :

Snana, which is contraindicated in Ajirna, when patient ignores the precaution then much more production of Ama, takes place leading to Mandagni and same Samprapti takes place for the disease.

12) Ati Nari Prasanga (Excessive Indulgence in Sex) :

According to Sushruta, there is a Sadyah manifestation of disease Pratishyaya with Nidanas like Ati Nari Prasanga, Mala, Mutra, Vega Sandharana etc. by vitiation of Vata Dosha[317]. Vitiated Vata when reaches Shirah Pradesha it manifests the disease as already described. Otherwise also this Nidana creates Dhatu Kshaya Vata Prakopa leads to Vishamagni Mandagni increases Amotpatti Amavisha vitiates Rasa Dhatu. Thus vitiating Doshas i.e. Kapha, Pitta and Rakta Sama Doshas obstruct the movement of Vayu manifestation of the disease Pratishyaya[318]

13) Vega Sandharana (To Control The Urge to Pass Stool/Urine):

Same pathogenesis takes place to produce the disease due to Vata Prakopa and due to Kapha leading to Mandagni, as mentioned by Achayra Charaka[319] **14) Ati Bhashana (Excessive Talking) :**

Due to Vata Vriddhi, Vata gets vitiated Avarana by Kapha, Pitta and Rakta in Shirah Pradesh obstruction in Gati Pratiloma Gati and same symptomatology i.e. expulsion of
Doshas through nasal route, manifesting the disease Pratishyaya.

15) Ratrijagarana (Not to Sleep During Night) [320] **:**

Ratri Jagarana creates Rukshata increases Vata Dosha and same pathogenesis takes place mentioned above. In the case of prolongation of same Nidana leads to Vata Dushti and manifestation of disease Pratishyaya.

16) Divaswapna (To Sleep During Day) :

Increases Kapha Dosha which leads to Mandagni. Indigestion takes place with the production of Ama and Adya Dhatu Rasa Dhatu Dushti amalgamation with Amavisha
Vitiated Rasa Dhatu manifests the disease, after lodging in Shirah Pradesha [321]

17) Shiraso-Abhitapa (Exposure to Heat of Head) :

Excessive heat vitiates Pitta Dosha Ushna Guna of Pitta dries up Snigdhata in Shirah Pradesh and melts Kapha Dosha (Vilayana) Decrease in Snigdhata, increases Rukshatva and Kharatva results in Vata Vriddhi in Shirah Pradesha vitiated Vata Dosha expels out Kapha through Nose manifesting disease Pratishyaya. Sushruta labels it as Nidan for Sadyah or acute pathogenesis of the disease[322]

18) Ritu Vaishamya :

According to Charaka, Ritu Vaishamya has a direct impact on Jatharagni[323] Vitiation of Jatharagni produces Amavisha, resulting improper digestion of ingested food. This Ama vitiates Rasa Dhatu and manifests pathogenesis for the disease Pratishyaya. In another reference Charaka mentions, while describing the Nidanas of Pratishyaya, due to Ritu Vaishamya there is Tridosha Prakopa and excessive increase of Vata which manifests the disease Pratishyaya, where pathogenesis starts with the obstruction of movement of Vayu [324]. As practically we see that due to certain climatic changes there is a natural phenomenon of body to maintain homeostasis or to acclimatize accordingly. Body adjusts the temperature against any temperature variation. In case of sudden variation in temperature there is immediate vasoconstriction or dilatation resulting in local inflammatory conditions, increased secretion, local injury etc. Such condition is quite susceptible to manifest the disease with subsequent exposures. As it becomes favorable condition for the manifestation of the disease with such exposures, due to creation of Kha-vaigunya (weakest point). It only

happens with the persons having low immunity, some anatomical deformity, pathological or psychological predisposing factors, already in hand.

19) Dhuli-Rajah-Dhuma (Exposure to Dust, Smoke etc.) :

Same pathogenesis has been described regarding Dhuli- Rajah Sevana in Charaka[325]. Due to such etiological factors there is a Sadyah Prakopa of Vata leading to manifestation of disease, with Achaya Prakopa. Such pathogenesis requires presence of any predisposing factor mentioned i.e. family history of allergy, sensitivity towards any particular allergen, weak immunity, anatomical, pathological or psychological reasons. Frequent exposures convert this disease from acute to chronic stage, if these predisposing factors, along with etiological factors, are not handled properly.

Sadyah Nidana (Acute Causative Factors) :

`Dhuli-Rajah-Dhuma etc. work only in the presence of predisposing factors i.e. nasal polyp, DNS, family history, anatomical deformity, pathological or psychological reasons. In such conditions after exposure to any allergen only nasal mucus glands, goblet cells participate actively to protect from or block the dust particles or to trap the allergens. Inhaled allergens produce specific IgE antibody in the genetically predisposed individuals. This antibody becomes fixed to the blood basophiles or tissue mast cells by its Fcend. On subsequent exposures, antigen combines with IgE antibody at its Fcend. This reaction produces degeneration of mast cells which release several chemical mediators i.e. histamine, leacotriene, eosinophilic chemotactic factors, neutrophil chemotactic factor, bradykinin etc. which are responsible for the symptomatology of the disease, depending upon the tissue involved. There may be vasodilatation, mucosal oedema, excessive secretion, infiltration with eosinophils. Mucosa earlier sensitized to an allergen will react to smaller doses of subsequent specific allergen i.e. Priming effect. Also gets “Primed” to other nonspecific antigens to which patient was not exposed. Sneezing is the reaction of autonomic nervous system to remove allergen. Circulation increases, leading to hypertrophy of turbinates, in acute stage. In advance stage when the disease leads to chronicity the mucus membrane becomes bluish. Epithelium of the nose is of two types. Nasal mucus membrane as well as the lining of the nasal sinus is by respiratory epithelium. Mucus and serum glands under lie the mucus membrane and the olfactory epithelium covers the ` turbinates of the upper 1/3rd of the nose. In acute phase, the mucus secretion is from respiratory epithelium. But in chronic stage, there is involvement of olfactory epithelium also, which leads to anosmia.

20) **Nitya Anupahita Shayana (Not to sleep in proper posture) :**

21) **Apavrita Mukha Shayana (To sleep covering face) :**

22) **Ati Parshwa Shayana (To sleep on one side) :**

According to Kashyapa, all these factors, in a due course of time, vitiate Vata Dosha and then same pathogenesis takes place, in the manifestation of the disease, after getting obstructed by Kapha Dosha in Shirah Pradesha, as already described[326]. When a person sleeps continuously covering his face, the air inside the covering Sheet etc. becomes hot and impure, because of, not getting fresh air from outside. Vitiation of Vata results in manifestation of the disease. When a person continuously sleeps on one side, the opposite side of the nose gets obstructed by Kapha, being heavier than other Doshas, so it moves to the dependant part more quickly and easily. This obstruction vitiates the Dosas in Shirah Pradesh leading to manifestation of the disease.

23) Manasika (Role of Psychic Factors) :

While defining the concept of good health, Acharya Sushruta has clearly cited the role of Atma, Indriya and Manah in attaining the health. He defined health as[327] Health is a psycho-biological whole i.e. a state of equilibrium and a contented state of consciousness, senses and mind. First three elements i.e. Dosha, Dhatu and Mala are related to Sharirika Bhava and "Prasannatmendriya Manah" implies the Manasika Bhavas. It throws a light to the inseparable association between mind, body and spirit in the maintenance of health. Any disturbance in this equilibrium leads to diseased state of person. Almost same etiopathogenesis has been mentioned in the vasomotor type of Chronic Rhinitis, where along with rhinoria, nasal obstruction and sneezing, there is a clear cut indication of role of Manasa Bhava in the occurrence of vasomotor type of rhinitis, because of in this case autonomic nervous system is unstable which is under the control of hypothalamus. Therefore, emotions (Krodha etc. anger, fear, anxiety) play a great role in the manifestation of this disease. As demonstrated and endorsed by Harold G. Wolff (M. D.) in his book "Stress and Disease" that nasal dysfunctions are the basis of man's commonest complaints. Much nasal diseases are related to stressful life situations. He mentioned a patient observed to have normal structure and size, with minimal secretions. She was caught in an unhappy marriage. In her angry and frustrated state she burst into tears and her nasal structure became redden, swollen and wet. When the subject expressed her rage and desperation and was on the verge of tears, her nasal mucosa became hyperemic and swollen and there

was profuse secretion and obstruction. This demonstration indicates that the psychic factors play a major role in the manifestation of Pratishyaya. Charaka also emphasizes the same being the main reason behind the occurrence of many diseases right from the time of Treta Yuga [328]. According to Acharyas Krodha, Shoka, Chinta, Ashruvega Sandharana, Atiashrusrava are the prime psychic etiological factors participating in the manifestation disease Pratishyaya [329,330]

Impact of Manasika Bhavas On Sharirika Dosas :

Krodha (anger) vitiates Pitta and Vata[331] Shoka (depression) and Bhaya (fear) vitiate Vata[332,]Chinta (tension) leads to Vata and Rasavahi Srotodushti[333]. Manasika Bhavas get disturbed due to Nidana Sevana, like Krodha, Shoka etc. Due to Nidana Sevana, Doshas specially Vata and Pitta are provoked. These provoked Doshas, as well as disturbed Manasika Bhavas hamper the Agni leading to Agni Vaishamya or Agni Dushti Rasa Dhatu Dushti Dosha Prakopa Doshas get lodged in Shirah Pradesha, manifesting the disease. According to Sushruta, Manasika Bhavas like Krodha, Shoka, Bhaya etc. are the etiological factors of Ojakshaya i.e. immunity gets weak, which leads a person to become more susceptible or prone to get diseased.

Atiahsru Srava (Excessive shading of tears) :

Due to Nidana Sevana, there may be Atipravritti or Dushti of Udana Vayu, leading to Vatakapha Prakopa in Shirah Pradesh, thus manifesting the disease Pratishyaya. Lacrimal apparatus is a group of structures that produces and drains lacrimal fluid or tears. From lacrimal glands, a tear pass medially over anterior surface of eye ball, enters in lacrimal punctum and then passes to lacrimal canal, which leads to lacrimal sac and then to nasolacrimal duct, which carries lacrimal fluid into nasal cavity, just inferior to inferior nasal concha. If an irritating substance makes contact with the conjuntiva, lacrimal glands are stimulated to over secrete. Lacrimination is a protective mechanism for any irritant, as it dilutes and washes away the irritant, watery eyes also occur in inflammation of nasal mucosa i.e. in cold, there is obstruction in the nasolacrimal duct and blocks the drainage of tears. Humans are unique in expressing emotions, both happiness and sadness, by crying. In response to parasympathetic stimulation, the lacrimal glands produce excessive lacrimal fluid that may spill over the edges of the eye lids and even fill the nasal cavity with fluid. Thus, crying produces a runny nose (Tortora, Principles of Anatomy and Physiology, Chapter 16), as demonstrated by Harold G. Wolff, mentioned above.

Ashru Vegasandharana (To Control the Urge to Cry) :

When a person, in depression wants to cry, but is not able to do so, may be due to any reason, then the tears inspite of coming out from the eyes, find their way through nose via nasolacrimal duct; leading to runny nose. According to Ayurveda, this could be described as due to Vegasandharana Vata Prakopa (Udana Vayu) when reaches Shirah Pradesha (Kapha Sthana) Avarana by Kapha of Vata expulsion of Doshas through nose. Thus manifests the disease[334] .

Pathophysiology

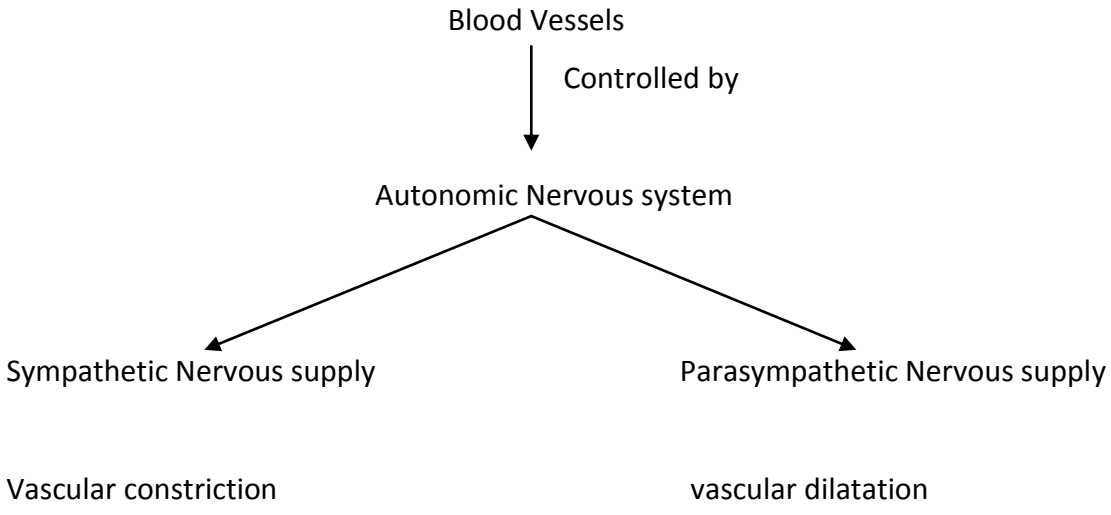
For proper understanding the Pathophysiology of hypersensitive respiratory disorders can be studied under following headings,

- ✓ Upper respiratory tract pathology.
- ✓ Lower respiratory tract pathology.

Upper respiratory tract pathology

The heating & humidificiation of inspired air is an important function of the nasal mucosa. The highly vascularized mucosa of the turbinates & septum provide an effective structure of heat & humid air as it passes over them.

Flow chart 6: Schematic representation of upper respiratory tract pathology



Reduction in secretion increase in secretion

- ✓ The protecting & cleansing role of the nasal mucosa is also a very important function
- ✓ Relatively large particles are filtered out of the inspired air by the hair within the nostrils.
- ✓ The cilio mucus transport system of the nasal mucosa provides a mechanism which helps to keep the nose clean & thus to supply relatively pure air to lungs.
- ✓ A thin, tenacious, & adhesive mucous blanket covering the nasal mucosa is produced by mucous & serous glands & epithelial goblet cells in the mucosa.

Cilia

- ✓ The major portions of the nose, septum & paranasal sinuses are lined by ciliated cells.
- ✓ The cilia beat at a frequency 10 to 15 beats per minute, producing a streaming movement of the mucous blanket at an approximate rate of 2.5 to 7.5 mm per minute.
- ✓ This blanket containing the filtered materials is moved toward the pharynx to be expectorated or swallowed.

Nasal secretions

- ✓ Contains an enzyme lysosomes, which is bacteriostatic
- ✓ . pH – 7
- ✓ Lysosome activity & ciliary action are optimal at this pH

Like all allergic reactions, Allergic Rhinitis involves a special set of cells in the immune system known as mast cells. Mast cells, found in the lining of the nasal passages and eyelids, display a special type of antibody, called immunoglobulin type E (IgE), on their surface. Inside, mast cells store reactive chemicals in small packets, called granules. After initial exposure to an antigen, antigen–processing cells (macrophages) present the processed peptides to T–helper cells. Upon subsequent exposure to the same antigen, these cells are stimulated to differentiate into either more T helper cells or B cells. The B cells may further differentiate into plasma cells and produce immunoglobulin E (IgE) specific to that antigen. Allergen-specific IgE molecules then bind to the surface of mast cells, sensitizing them. Further exposures resulting the bridging of 2 adjacent IgE molecules, leading to the release of

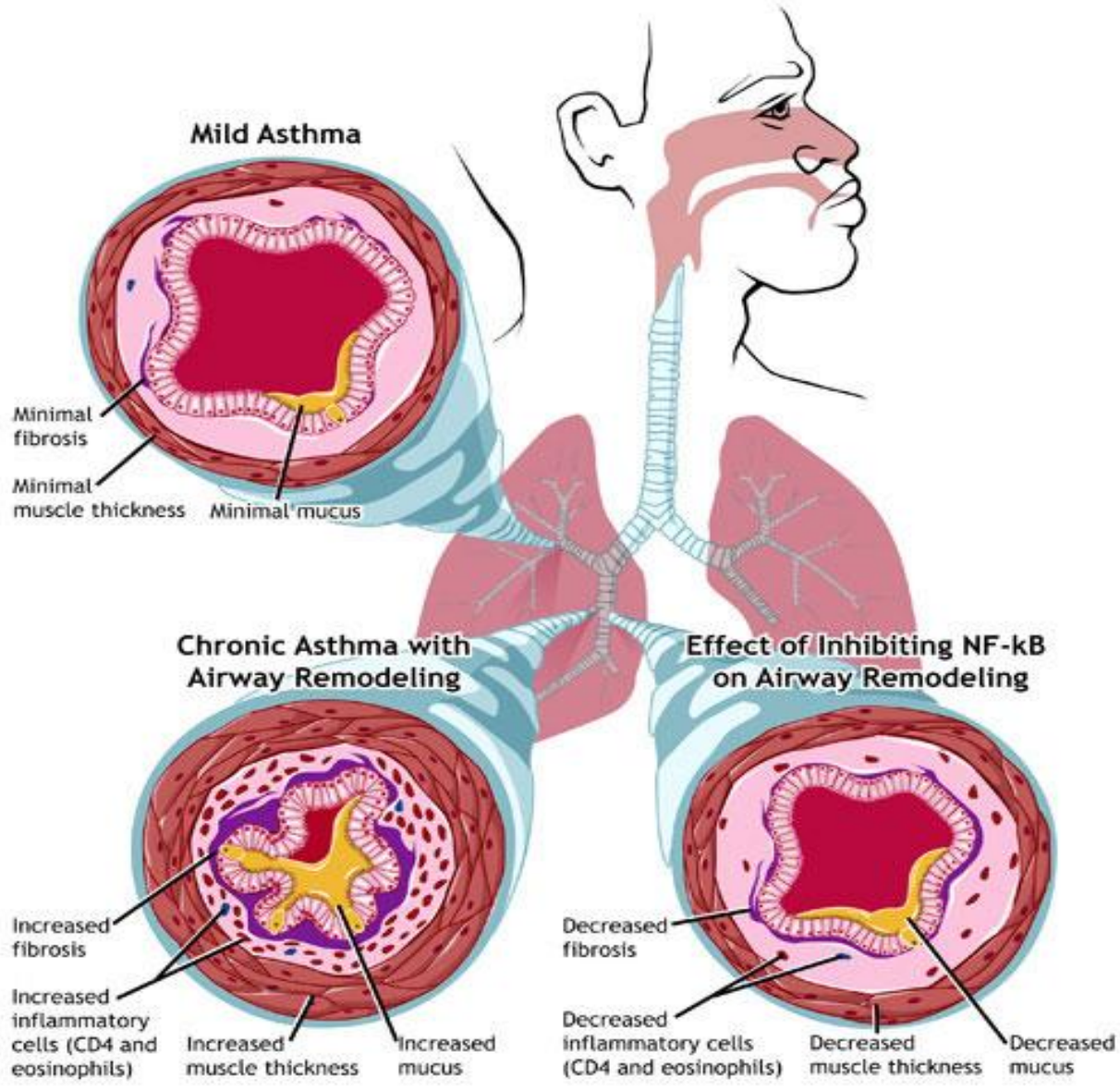
preformed mediators from mast cell granules. These mediators (i.e. histamine, leukotrienes, kinins) cause early phase symptoms such as sneezing, rhinorrhea and congestion. Late-phase reactions begin 2-4 hours later and are caused by newly arrived inflammatory cells. Mediators released by these cells prolong the earlier reactions and lead to chronic inflammations.

Histological changes

- ✓ Dilatation of the vascular bed with oedema formation.
- ✓ Enlarged active mucous glands & goblets cells.
- ✓ Infiltration of the sub mucosa & mucosa with large number of eosinophils.
- ✓ Plasma cells & lymphocytes may also be seen.

These changes are completely reversible, for when the specific pollinating season is over the mucosa returns to normal. If the patient's symptomatology remains limited to a seasonal pattern, there may be no permanent alterations in the nasal mucosa & nasal function.

Diagram 3: Lower respiratory tract pathology



The current concept of asthma and other hypersensitive disorders pathogenesis is that a characteristic chronic inflammatory process involving the airway wall, causing the development of airflow limitation and increased responsiveness, thereby predisposing the airways to narrow in response to a variety of stimuli. Characteristic feature of the airway inflammation are increased number of activated eosinophils, mast cells and T lymphocytes in the airway mucosa and lumen and an increased thickness of the reticular layer of the basement membrane (sub epithelial fibrosis). These changes may be present even when asthma and other hypersensitive disorders is asymptomatic. Following inhalation of an allergen in a subject with atopic asthma, obstruction is seen in a matter of minutes, with a peak airflow obstruction in 20 to 30 minutes. This is early asthma and other hypersensitive

disorders response and is primarily the result of the direct action of mast cell (IgE mediated degranulation) derived mediators and their metabolites, in the hyper responsive bronchial smooth muscle. Local effects are also seen in nerves and blood vessels near the site of initial insult. In some cases the reaction soon terminates, with resolution accompanying a decrease in short-lived mediator concentrations. In many cases however, a second phase of airflow obstruction occurs 6 to 10 hours later. This is termed late asthmatic response.

The late response is postulated to be the result of specific cytokines released along with, or soon after the short-lived mediators causing the early reaction. Owing partly to longer half-lives and high biological potencies, cytokine molecules are able to diffuse from airway blood vessels to the general circulation. Here, they summon classical effector cells of the inflammatory response to the site of injury and in the process activate these cells for what will prove to be a 'battle'. The cytokines also serve to alert the endothelial cells to the barrage of cells which were summoned. The vascular endothelial cells then express specific adhesion molecules on their effector cell egress. Soon effector cells appear, chiefly of the eosinophils lineage, but also representatives of Th2 class of lymphocytes, neutrophils and monocytes. The process of cellular adhesion and diapedesis ensues, along with some final up regulation, partly as a result of adhesion. Activated Th2 CD4 – helper cells produce IL-4, which in presence of signals such as those provided by ligation of the cell surface molecule CD4, cause B lymphocyte immunoglobulin gene subclass switching from IgE and IgM in favor of IgE. Once the endothelial barrier has been traversed, the cells are free to converge on the inflammatory focus and release preformed and newly synthesized factors designed to isolate, combat or even lyse the provocative trigger. Local release of mediators including super oxides, cytokines, cationic proteins and enzymes occurs and it is at this point that the clinical manifestations of asthma (airway hyper responsiveness, broncho constriction, epithelial cell sloughing, and edema) are observed.

02.13 PURVA RUPA

It is the warming sign of manifesting disease[335].

Prodromal symptoms are the manifestations of a disease when the vitiated Doshas get localized at a place where there is impairment in the channels [Sthana Samsraya]. When the manifestations get converted into Vyaktavastha, they are termed as Rupa. In acute disease condition manifested by exposure to triggering factors it is not necessary that Purvarupas be present. But in the disease produced by Kalantarajanaka Nidanas a full-blown picture of Purvarupas will be associated. Purvarupa is the sign of the disease[336]. It also can be divided into two types i.e.

1) Samanya Purvarupa (General prodromal symptoms)

2) Vishishta Purvarupa (Specific prodromal symptoms)

By Samanya Purvarupa, we can assume about the arrival of the disease, whereas by Vishishta Purvarupa, we can assume about the specific types of the disease, like Vataja or Pittaja, etc. Hence both types of Purvarupas have their own importance. According to Acharya Charaka when the symptoms of the disease are less in intensity then they are called Purvarupa.

In the present context of asatmyajanya pranavahasrotovikara (respiratory hypersensitivity) the poorvaroopa mentioned in context of pratishyaya can be considered, as these clinical features are observed almost smilar to respiratory hypersensitivity reactions.

The Purvarupas described by Sushruta include [337],

- ✓ Sirogurutwa (Heaviness of head)
- ✓ . Ksavathu (Sneezing)
- ✓ . Angamarda (Body ache)
- ✓ . Parihrishtaromata (Generalized horripilation)
- ✓ Stambha (Stiffness)

In Ashtanga Hridaya, Charaka Samhita[338] & Kashyapa Samhita, there is no mention of the Purvarupa of the disease Pratishyaya. Madhava Nidana[339] ,Bhavaprakash[340], Vaidya Kalpadruma[341], Gadanigraha, Yogaratnakara all have mentioned the Purvarupa same as that of Sushruta. But the only difference between them & Sushruta is that, they have replaced Shiropoornata instead of Shirogurutva. In Videha Samhita the following Purvarupas are mentioned [342].

- ✓ Kshavathu (Sneezing)
- ✓ . Sirogurutwa (Heaviness of head)

- ✓ Nasa Dhumayana (Burning sensation in nose)
- ✓ Talu Vidarana (Irritation in the pallate)
- ✓ Kanthodhwamsa (Blockage in the throat)
- ✓ Mukha Srava (Excessive secretion)
- ✓ Manthana

An overview of the Purvarupas highlights the generalized vitiation of Doshas. Moreover they are suggestive of the dominance of Kapha and Vata in the initial stage of etiopathogenesis.

02.14 RUPA

Rupa is the full blown clinical manifestation of the disease when the Shadkriyakala (Samprapti Prakriya) reaches the level of Vyakta Avastha[343].

These clinical manifestations or Lakshanas are divided in two groups i.e.

1) Samanya (General) and

2) Vishesha (Specific)

The Samanya or general symptoms are found in all types of diseases but Vishesha Lakshanas are found only in one of the specific type of disease.

Samanya Lakshana (General Symptoms)

Asaatmyajanya pranavahasroto vikara is condition in which the lakshanas of pratishyay, kasa, and shwasa are observed. Where in pratishyaya lakshanas are predominantly present. Hence the lakshanas of pratishyaya, shwasa and kasa which are similar to respiratory hypersensitive reactions can be considered.

Acharya Charaka has not mentioned specifically about the Samanya Lakshana but in the disease Rajayakshma, while mentioning symptoms he has given the Samanya Lakshana of Pratishyaya as follows[344] –

- ✓ Shirahashoola
- ✓ Shirogaurava
- ✓ Nasaviplava
- ✓ Jwara
- ✓ Kasa
- ✓ Kaphotklesha
- ✓ Swarabheda
- ✓ Aruchi

- ✓ Klama
- ✓ Yakshma
- ✓ Indriya Asamarthata

In Sushruta Samhita[345] & Astanga Hridaya, there is no mention of Samanya Lakshana of Pratishyaya. Acharya Vagbhata described the general symptoms of all Nasa Rogas[346.]

- ✓ Shwasa Krichhrata (Nasal obstruction)
- ✓ Peenas
- ✓ Prathatam Kshavathu (Frequent sneezing)
- ✓ Sanunasika Vaditvam (Nasal speech)
- ✓ Putinasa (Bad smell in breadth)
- ✓ Shirovyadha

In general clinical pictures Kashyapa mentions that the face, head and nose of the patient becomes just as if obstructed and there will be anosmia or impairment to olfactory sensation[347].

Table 21 Vishesha Lakshanas of asatmyajanya pranavaha sroto dusti vikara.

Sl.no	Pratishyaya	Kasa	Shwasa	Pulmonary hypersensitivity.
1	Kshavathu (V) (K) (c. s.)	X	X	Sneezing
2	Nasavarodha (V) (C. S.) (Su.S) (As Hr)	X	X	Nasal obstruction
3	Tanunasasrava (V) (C. S.) (Su.S) Pittakapha Srava (P) Snigdhakaphasrava(K) (As Hr) Shitasrava (K) (Su.S) Ghanasrava(K) (C. S.)	✓	Pinasam (T*J*) (C. S.) (As Hr)	Rhinorrhoea
4	Kasa (K) (C. S.) (Su.S) (As Hr)	✓	Kasa (T*J*) (C. S.) (Su.S) (As Hr) Ativegatana Murhurmuhu Kasa (T*J*) (C. S.)	Cough
5	Shirahshoola(V) (C. S.) (As Hr) Sankhapradesha-vedana(V) (Su.S) (As Hr)	✓	✓	Headache
6	Nasakandu (K) (C. S.)	X	X	Nasal Itching
7			Muhurmuhu Shwasa (T*J*) (C. S.)	Repeated attacks
8	Aruchi (K) (C. S.) (As Hr)	✓	Aruci (T*J*) (As Hr)	Anorexia
9	Swarabheda(V) (As Hr) Swaropaghata (V) (C. S.) (Su.S)	✓	✓	Hoarseness of voice

10	Jwara (P) (C. S.) (As Hr)	✓	✓	Fever
11	Shirogaurava(K) (Su.S)	X	X	Heaviness in head
12	Shwasa (K) (As Hr)	✓	✓	Breathlessness
13	Gandhagyata (D) (C. S.) (Su.S)	X	✓	Anosmia
14	Shosha (D) (C. S.)	✓	✓	Fatigue
15		✓	X	Mental depression
16	Nasavarodha (V) (C. S.) (As Hr) (Su.S) Nasaagrapaka (P) (C. S.) (As Hr)	X	X	Nasal congestion
17	Nasakandu (K) (C. S.)	X	X	Nasal itching
18	Netrashotha (K) (C. S.) Akshikandu (R) (As Hr)	X	X	Eye symptoms itching& lacrimation
19	Srotrakandu (R) (As Hr)	X	X	Ear symptoms itching of the ears,
20	Talukandu (K) (Su.S) Talushosha (V) (Su.S)	✓	X	Itchingof the palate,

The above table shows that symptoms of Vataja Pratishyaya, kasa and shwasa like Kshavathu, Nasavarodha, Tanusrava, Gandha hani etc. are more related with symptoms of Respiratory hypersensitivity / allergy.So, Respiratory hypersensitivity / allergy can be correlated with condition developed either due to Vataja Pratishyaya or kasa or shwasa,individually or in combination.

Clinical Features

The diagnosis of allergic respiratory condition is usually not difficult in fact; it is often made by the patient or by the parents of the allergic child.

Allergic History:

For the physician who treats patients with allergic respiratory condition, nothing is more crucial than the allergic history, it is important not only in identifying an allergy but also in guiding the treatment plan. In assessing the relative importance of different allergens in patients with allergic respiratory condition, it is essential first to take a detailed environmental history, which includes:

- ✓ A clear definition of the geographical location of the patient.
- ✓ Whether the patient has moved or not.
- ✓ Place of birth.
- ✓ Home environment, such as : carpeting, pets, smoking, trees, plants, grass, hobbies.
- ✓ Seasonality of symptoms

- ✓ Relationship of symptoms to : Working conditions, Diet Indoors V/S. outdoors

Family History:

- ✓ Children of individual with allergies have been shown to have a higher incidence of allergies than that of other children.
- ✓ If both parents have allergies, their child has a 50% chance of having the same problem.
- ✓ As Allergic respiratory condition commonly occurs in families, it is important to determine a family background of rhinitis or of the other allergic conditions (asthma, eczema, urticaria).

Past Medical History:

- ✓ In children, a history of recurrent otitis media, upper respiratory tract infection, asthma & chronic rashes are suggestive of allergies.
- ✓ Other pertinent medical problems (e.g. asthma, aspirin hypersensitivity) and the use of medications (e.g. beta-blockers, tranquilizers) that could interfere with the treatment for allergies should be evaluated.
- ✓ Inquire about the results of previous allergy tests and treatment

General Symptoms:

The major symptoms of allergic respiratory condition are –

- ✓ Sneezing
- ✓ Rhinorrhea
- ✓ Nasal congestion
- ✓ Nasal itching

Some patients may not have the complete symptom complex.

- ✓ Sneezing is the most characteristic symptom.
- ✓ A paroxysm of 10 to 20 sneezes in rapid succession.
- ✓ These episodes are especially apt to occur in the morning, & may leave a patient exhausted.
- ✓ Sneezing episodes may arise without warning, or they may be preceded by an uncomfortable itching or irritated feeling in the nose.
- ✓ Rhinorrhea is typically a watery, thin discharge which may be quite profuse & continuous.
- ✓ Because of the copious nature of the rhinorrhea, the skin covering

- ✓ The external nares & the upper lip may become irritated & tender.
- ✓ Purulent discharge is never seen in uncomplicated allergic rhinitis; its
- ✓ Presence indicates secondary infection.
- ✓ Nasal congestion, due to swollen turbinate's, is a prominent complaint.
- ✓ The nasal obstruction may be intermittent, or more troublesome in the
- ✓ Evening & at night.
- ✓ If the nasal obstruction is severe, interference with aeration &
- ✓ Drainage of the par nasal sinuses or Eustachian tube may occur, resulting in the complaints of headache or earache.
- ✓ The headache is of the so called vacuum type, presumed to be caused by the development of negative pressure as air is absorbed from the obstructed sinus or middle ear.
- ✓ Patients may also complain that their hearing is decreased & that sounds muffled.
- ✓ With continuous severe nasal congestion, the sense of smell & taste may be lost.
- ✓ . Nasal itching also be a prominent feature, inducing frequent rubbing of the nose, particularly in children.

Eye symptoms

- ✓ Consists of itching & lacrimation occasionally there may be marked itching of the ears, palate, throat or face which may be extremely annoying. Because of the irritating sensation in the throat & the posterior drainage of the nasal secretion, a hacking, non productive cough may be present.

Systemic symptoms

- ✓ Weakness
- ✓ Malaise
- ✓ Mental depression
- ✓ Irritability
- ✓ Fatigue
- ✓ Anorexia

The symptoms of perennial allergic rhinitis are those of seasonal allergic rhinitis, although they are frequently less severe. This is due to the almost constant exposure to low concentrations of an allergen such as house dust. Symptoms may lead the patients to "Sinus trouble" or "Frequent cold". The chronic nasal obstruction may cause –

1. Mouth breathing
2. Snoring
3. Almost constant sniffing
4. Nasal twang to speech
5. Dry, irritated or sore throat
6. Loss of smell & taste

Nasavarodha is the cardinal feature of **Vatika Pratishyaya.** Due to Nasavarodha the patient is forced to breath through mouth resulting in Oshta Shosha, Gala Shosha, Talu Shosha and Mukha Shosha. In most of the cases the obstruction in upper airways leads to sleep disturbances (Jagatyabhikshnam). Nisha Jagarana again contributes to aggravation of Vata and these form a vicious cycle. All the other features could be better explained in terms of the cardinal clinical features (Atmarupa) of Vata which indicates the predominance of Vata in the disease of Pratishyaya[348].

The features like sneezing ,headache, nasal obstruction etc. points towards the allergic rhinitis,

This is usually characterized by spamsmodic attacks by severe sneezing and rhinorrhea. Nasal

discharge is of watery and copious, nasal blockage leads to the mouth breathing and this may lead to laryngitis, pharyngitis, tonsillitis and causing Mukha Shosha, Swaropaghata, Swarabheda etc. Psychological features like stress also plays a major role in this type of Pratishyaya. Headache mainly localized in temporal region. All these characteristics indicate towards a very close association of Vatika Pratishyaya with chronic Allergic and vasomotor Rhinitis.

The symptoms of **Pittaja Pratishyaya** are easy to be understood in terms of the cardinal clinical

features of aggravated Pitta, the secondary infective manifestations resulting from vitiation of Pitta and subsequent vitiation of Rakta and systemic manifestations due to chronicity of the disease[349]. High grade chronic inflammation leads to secondary infections like furunculosis, vestibulities and symptoms like Kasa, Nasa Paka, Nasagra Paka, Ghranapidika and associated inflammatory signs like fever, pain i.e. Jwara, Ushnabhitapa etc. The discharge becomes thick and purulent along with atrophy of nasal mucosa and turbinates, with foul smelling crusts leading to Pitta Srava, Tamra Varna Srava, Ruksha Srava and other

symptoms like Akshipaka, Karshya, Pandu, Pipasa, Trishna etc. are suggestive of secondary infection and correlates with the signs and symptoms of Pittaja Pratishyaya with Atrophic Rhinitis.

In **Kaphaja Pratishyaya** the symptoms are indicating longstanding Samprapti. Affection of lowerrespiratory passages, paranasal sinuses and subsequent involvement of central nervous system are evident from the spectrum of symptoms described in the classics[350.]. Asthma, chronic cough (Kasa, Shwasa) are suggestive of long standing infection and involvement of lower respiratory tract. There is thick copious, foul smelling nasal discharge (Ghranasrava with Gatra Gaurava etc.) and due to involvement of surrounding structures there is Netra Shotha and itching (a peculiar sign) on Ostha, Mukah, Shira, Nasa etc., again points towards the resemblance of clinical picture of Kaphaja Pratishyaya with Allergic Rhinitis.

The symptoms of **Raktaja Pratishyaya** could be considered as produced by a long-standing infection and probably as a secondary condition to a trauma or injury to airways or adjacent structures[351]. Nasal bleeding (epistaxis i.e. Raktasrava) may be due to acute inflammation and marked vasodilation. The infection may spread to eye causing red eye (Tamrakshi) and to lower respiratory tract causing Uroghata, Urahsuptata and eye, ear, nose infections etc. The clinical picture again indicates towards almost a similar picture of severe condition of Atrophic Rhinitis with Raktaja Pratishyaya.

When we assess these all signs and symptoms, we can see that all three Doshas are involved in the pathogenesis. So this Dushta peenasa is also Sannipathika in nature. Lower respiratory tract involvement is the reason for the presence of symptoms like Kasa, Shwasa and Chest pain. The Nasal discharge becomes thick and foul smelling. The symptoms of Dushta Pratishyaya could be considered as produced by a long-standing infection and probably negligence of patient, also indicating to words the chronicity & serious condition of the disease and includes the maximum number of complications mentioned in different types of chronic rhinitis, which involves all the sense organs[352].

02.15 UPASHAYA AND ANUPASHAYA

UPASHAYA AND ANUPASHAYA

Upashaya and Anupashaya may be considered as a therapeutic test. The diet, drugs & activity which increase symptoms and attack, are known as Anupashaya. & that relieve the symptoms, are known as Upashaya. Here in the context of asatmyajanya pranavaha srotovikara the upashaya and anupashaya mentioned in the context of pratishyaya, shwasa,and kasa can be considered.

Upashaya

- ✓ Shlesma Vimokshante Saukhyam
- ✓ Ushnabhinandati
- ✓ Asino labhate Saukhyam
- ✓ Bronchodilators
- ✓ Oral intake of Ghrita, (warm) containing sour ingredients
- ✓ Various kinds of Swedana & Vamana.
- ✓ Nasya with the squeezed juices at appropriate time
- ✓ Dhumapana & Gandusha should be done depending upon the types of Dosha involved.
- ✓ Snigdha, Ushna, Lavana & Amla Padartha Sevana

Anupashaya

- ✓ Durdina, Meghambu, Sheeta Ritu
- ✓ Sheetambu, Pragvaten
- ✓ Kaphavardhaka Ahara Vihar
- ✓ Excessive intake of Guru, Madhura, Sheeta substance
- ✓ Excessive intake of cold water
- ✓ Dhul, Rajaha Sevana
- ✓ Atidrava Sevana after meal
- ✓ Vishamashana

02.16 CLINICAL EXAMINATIONS

Face : Patients with Allergic Rhinitis do not have the so-called 'allergic facies' - rather, they often have red, puffy faces with reddened, watery eyes. Their conjunctiva are hyperaemic and granular.

- ✓ Mouth breathing - many of these patients are constant mouth breathers, sometimes displaying the so-called 'allergic gape'.
- ✓ Patients can develop rather long, mournful faces. The palate is often high-arched and narrow in these patients and dental crowding is common, especially in young children. These features form the long face syndrome.
- ✓ Patients often pull their faces in various ways, often like rabbits, in an attempt to open up their nasal passages.
- ✓ Patients with Allergic Rhinitis frequently grimace and twitch their face, in general, and nose, in particular, because of itchy mucous membranes.
- ✓ Chronic mouth breathing secondary to nasal congestion can result in the typical adenoid faces.

Eyes

- ✓ Patients may have injected conjunctiva; increased lacrimation; and long, silky eyelashes.
- ✓ Dennie-Morgan lines (creases in the lower eyelid skin) and allergic shiners (dark discoloration below the lower eyelids) caused by venous stasis may be present.

Ears

- ✓ Ears frequently are unremarkable.
- ✓ Eczematoid otitis externa and middle ear effusion may be present.

Nose

Nose - often appears swollen, reddened and shiny from constant rubbing.

- ✓ Nasal mucosa - examination of the nasal passages usually shows severe swelling of the nasal mucous membrane and lower turbinates with profuse secretions which range from clear to thick mucoid
- ✓ Some text books often describe the colour of the mucous membranes as grey or greyish-pink but this is extremely variable.
- ✓ In many cases there is long-standing chronic inflammation of these mucous membranes, and the colour is often dark red.

- ✓ It is not unusual for the swollen anterior turbinate to obstruct the nostril completely.
- ✓ The 'allergic salute' may also be characterised by a side wards rubbing of the nose.
- ✓ Younger children cannot complain about their early symptoms, they frequently present with later sequelae such as otitis media with effusion. It is consequently important to recognise the presence of allergy at this age. Very young allergic children are often seen rubbing their noses on sheets and their mother's shoulders, even before the hands can find their way to the nose.

Mouth

- ✓ A high arched palate, narrow premaxilla, and receding chin may be present secondary to long-term mouth breathing.

Throat

- ✓ Usually appears reddened with prominent lymphoid follicles on the posterior pharyngeal wall.

Speech

- ✓ May have a nasal quality and there may be loss of taste and smell.

02.17 LABORATORY INVESTIGATIONS

Skin Test :

- ✓ Skin testing generally is considered to be the criterion standard of allergy workup. The classic wheal-and-flare responses result from the interaction between the antigen and sensitized mast cells in the skin.
- ✓ In general, the acute phase starts within 2-4 minutes and reaches a maximum in 10-20 minutes. It may be followed by a late phase 4-6 hours later.
- ✓ A number of factors affect the responses; these include the following:
- ✓ Volume and potency of the antigen
- ✓ Reactivity of the skin
- ✓ Age and race of the patient
- ✓ Area of body tested
- ✓ Distance between the injections and time of day of testing
- ✓ Medications (eg, antihistamines and tricyclic antidepressants)
- ✓ Because of these variables, positive and negative controls must be usedto ensure the validity of the results.
- ✓ In addition, patients receiving beta-blocker therapy are at risk for severe reactions, and the drugs should be switched to another class of medication before testing is initiated.
- ✓ Currently, 3 types of skin tests are in use.

1) **Prick testing:** is rapid and safe, and scores are graded from 0-4 according to both wheal and flare responses. However, low-grade sensitivities can be missed. Therefore, the test often is used as a screening tool, which is followed by intradermal testing if necessary. It tests for immediate hypersensitivity and demonstrates an IgEmediated allergic reaction. Certain factors such as drugs, age and the season may influence the results. In performing an SPT, the use of a large number of allergens is expensive, time-consuming and usually not necessary. SPT should initially be limited to the common aero-allergens in the patient's environment.

2) **Single-dilution intradermal testing** involves injecting 0.01-0.05 ml. of antigen into the epidermis. The resulting wheal and flare are measured after 10-20 minutes and graded as in prick testing. This test can be used to detect most low-degree atopies if a 1:500

concentration is used. However, as with prick testing, it does not permit accurate quantitation of the sensitivity to the antigen involved.

3) **Progressive-dilution intradermal testing** (skin endpoint titration) involves a series of 5-fold dilutions, starting with a concentration that is sufficiently dilute to be nonreactive. Progressively stronger concentrations are injected until a wheal forms. The endpoint is confirmed when the wheal with the next stronger dilution is 2 mm larger than the previous wheal. This endpoint indicates the relative sensitivity of the patient to the allergen and designates the starting point for immunotherapy. This method allows both qualitative and quantitative assessment of sensitivity to the antigen in question.

Total Serum IgE :

- ✓ In normal subject, levels of IgE increase from birth to adolescence and then decrease slowly to reach a plateau after the age of 20 – 30 years.
- ✓ In contrast to total IgE, which has a poor clinical correlation, antigenspecific IgE antibodies are important in the diagnosis of inhalant allergy.
- ✓ Compared with skin testing, in vitro testing is more specific, and it is not affected by skin reactivity or medications. It also has no risk of systemic reaction and is better tolerated, because it is less traumatic. However, in vitro testing is less sensitive than skin testing, especially in regard to molds. Also, the results are not available immediately and must be verified with skin testing before immunotherapy can be started.
- ✓ The original method for obtaining an IgE count, the radio allergosorbent test (RAST), has evolved from a radioimmunoassay to a test that involves enzymatic or fluorometric processes (eg, enzyme-linked immunosorbent assay [ELISA]). Fadal and Nalebuff have modified the test to increase its sensitivity and to improve the correlation of its findings to those obtained with the skin endpoint titration method.

Phadiat op Test :

- ✓ The Phadiatop test is a screening test using several inhalant allergens on a single solid phase and therefore will detect specific IgE in a single assay.
- ✓ It has an efficiency of over 95%, defining those individuals who need more detailed investigation.
- ✓ It is less useful in children under 3 years old, in whom the CAP RAST F x 5 (6-foods test) is generally more useful, particularly if combined with total serum IgE.

Cap Rast

- ✓ This test is useful when SPT are not available, e.g. in young children.
- ✓ Doctors are advised not to send blood for this test without specifying which allergens they require and are appropriate for their region.
- ✓ Laboratories offer an extremely wide range of CAP RAST tests, and failure to specify can make the cost to the patient unnecessarily high.

Nasal Smears :

- ✓ Nasal smears may differentiate between allergic and infective rhinitis.
- ✓ Eosinophils are characteristic of allergic rhinitis with eosinophilia (NARES), where neutrophils imply bacterial infection.

Imaging Studies :

- ✓ No radiologic studies are necessary in the evaluation of patients with allergies because the diagnosis is made on the basis of the history and confirmed with relevant physical findings and test results.
- ✓ Imaging findings, if available for other reasons, usually are nonspecific and may be the same as those in other types of rhinosinusitis (eg. mucosal thickening, turbinate hypertrophy).

1) Plain x-rays

- ✓ Despite advances in imaging technology, plain films still have a limited role to play.
- ✓ They are of most value in detecting the presence of acute infective processes.
- ✓ The main limitations of plain films lie in the poor visualisation of the ethmoid air cells, and the difficulty in distinguishing between infection, polyps and tumours in a completely opacified sinus.
- ✓ The standard sub-mento vertical (Waters) view will clearly show an air/fluid level as well as a complicated septal deviation.
- ✓ The frontal and ethmoid sinuses are more clearly shown in the posteroanterior (Caldwell) view.
- ✓ The density of the orbital cavity correlates with the normal frontal and ethmoid sinuses.
- ✓ Problematic areas like the ostiomeatal complexes are not shown in any of these views.

- ✓ They are also not of use in very young children as the sinuses are not fully aerated - 75% of children aged under 1 year will therefore have opaque maxillary sinuses.
- ✓ The adenoidal fat pad is best visualised in the lateral view, but plain Xrays are not adequate to assess the posterior nasal space.

2) CT Scanning

- ✓ CT scanning should be reserved for complicated sinusitis and as part of surgical planning.
- ✓ It has become the imaging modality of choice for clearly viewing the anatomical and pathological changes present in the sinuses.
- ✓ The key area, the ostiomeatal complex, is clearly shown, as are all the other changes in the sinuses if coronal views are requested.
- ✓ Concern has been expressed about the radiation exposure during CT scanning. Although the radiation dose to the lens of the eye is higher than that for plain films, it is still well within safety limits.

Endoscopy of the Nose in Allergic Rhinitis :

- ✓ Endoscopes can either be rigid or flexible. Most otolaryngologypractices are fitted out with either a rigid scope or a flexible scope.
- ✓ Endoscopy is relatively easy in adults but is difficult in children, inwhom its use is therefore limited.
- ✓ Endoscopy of the nose in allergic rhinitis not only helps to show the typical mucosal findings but also allows determination of additional pathology, e.g. polyps in the middle meatus or associated anatomical factors which may affect the treatment of allergic rhinitis, e.g. septal deviation.

Cytotoxic Test:

Nasal cytologic studies may be needed.

- ✓ Nasal secretions are stained with hematoxylin and eosin.
- ✓ In general, the presence of eosinophils and goblet cells is suggestive of allergy, whereas the presence of neutrophils and bacteria is characteristic of infection.

Other Tests :

Many other alternative tests for allergies are available, but they have not been fully validated yet.

i) Basophilic histamine – release test.

ii) Leukocyte antibody test for related antigens.

02.18 UPADRAVA

Upadrava are the secondary symptoms (Vikara) occurring due to further progression of same process of main disease. The later is often pacified when, the main disease is treated. As it appears later, it becomes more afflicting because of the patient being already suffering from the disease. Here in the context of asatmyajanya pranavaha srotovikara the upadrava mentioned in the context of pratishyaya can be considered. Acharya Sushruta states[387] that all types of Pratishyaya lead to vitiated condition without proper treatment & give rise to following complications –

- Badhirya- Andhata- Aghranam- Ghoranayanam- Kasa- Agnimandya- Shopha, etc.Madhava Nidana, Bhava Prakash, Gadanigraha, Vaidya Kalpadrum,Yogaratnakara, etc. have accepted above mentioned Upadravas of Pratishyaya. However, Bhavaprakash & Gadanigraha has replaced Shosha in the place of Shotha. Acharya Charaka has not given the direct reference of Upadravas of Pratishyaya[388] but while describing Dushta condition of Pratishyaya, he has described that if Dushta condition is neglected, it gives rise to various Nidanarthakara disorders like –- Arbuda- Arunshika- Shiroroga- Karnaroga- Khalitya- Kapishaloma- Shosha, etc.

This is suggestive of if asatmyajanya pranavaha srotovikara not treted properly then it may lead to nasa gata,netragata, karnagata and sarvadaihika upadravas.

While in the contempory science, following explanation regarding pulmonary hypersensitivity is available.

An allergic reaction occurs when a person's immune system does not recognize a particular substance such as pollen, venom from an insect bite or a particular food protein. The body releases antibodies and histamine in order to attack the perceived intruder. The heightened levels of histamine cause various reactions in the body such as respiratory issues, hives and asthma. While most allergies are treated with over-the-counter (OTC) drugs, some allergies may result in further complications such as upper respiratory infections, asthma, anaphylactic shock and eczema.

Upper Respiratory Infections

Upper respiratory infections are a common complication of seasonal allergies. Seasonal allergies typically result in nasal congestion, promoting upper respiratory congestion. The body overproduces mucus in the sinus cavity, causing inflammation in the sinuses and

leading to postnasal drip and sinusitis (sinus infection). Postnasal drip is a condition where the sinuses drip excess mucus in the back of the throat, which leads to chest and throat congestion. The excess mucus creates a perfect moist environment for the growth of bacteria, viruses and fungi, producing infection.

Asthma

The Mayo Clinic claims that a person with allergies is more prone to develop asthma. Asthma is a result of an immune response that triggers the lungs and airways to constrict, causing difficulty breathing. Asthma is commonly treated with a doctor's prescription for a medicated inhaler to relax the airways, allowing proper breathing. If you suffer from severe asthma, seek medical advice on how to avoid and treat allergies. A severe asthmatic attack can lead to major complications and even death.

Anaphylactic Shock

Anaphylactic shock is a rare allergic reaction in which the entire body responds to the allergen. It is the most common for people who are allergic to insect bites and stings and should be taken very seriously. Anaphylactic shock comes on suddenly, as the entire body begins to release extreme levels of histamine. A person may experience shortness of breath, abdominal pain, wheezing, dizziness, vomiting and other reactions. If you think you are experiencing anaphylactic shock, call 9-1-1 and get medical attention immediately. Anaphylactic shock can result in death.

Eczema

Eczema is an allergic skin reaction commonly triggered by allergic reactions. Eczema is common in babies and young children who mostly grow out of the condition later in life. Eczema begins as small, red dots on the skin that become itchy and irritated. They soon turn into raised bumps that commonly ooze and crust over. If not treated, eczema can leave permanent scaring. Eczema is treated with oral antihistamines, if related to seasonal allergies, and with prescribed topical steroidal lotions.

O2.19 SADHYASADHYATA

As Asatmyajanya pranavaha srotovikara exhibits prtishyaya, Tamaka Shvasa and Kasa lakshanas in permutation and in combination, hence sadhyasadhyata of above said diseases can be considered. Tamaka Shvasa in general is described as Yapya (palliable) disease. However in individual with recent origin of disease, person of Pravara bala or both said to be Sadhya (Ch. Chi. 17/.62). Vagbhata also follows the Charaka. Sushruta opines that it is a disease, which can be cured with much difficulty (Krichasadhya). If it is appears in a debilitated individuals its prognosis becomes very difficult (Su. Ut. 51/14). In disease Tamaka Shvasa Kapha and Vata Dosha are involved primarily. Both of them exhibit opposite qualities. Hence management will be also difficult as factors, which excite Vata, alleviate the Kapha Dosha and vice Versa. Pranavaha Srotasa is mainly involved. This Srotas is having direct exposure to the environment hence persons are more exposed to Nidana like Raja, Dhuma etc.

Disease Tamaka Shvasa is having Multifactorial origin along with diet, environmental and meteorological factors like rains, cloudy weather, chilly wind etc & patients can not avoid these Nidana. While discussing disease Pratishyaya none of the Acharyas have mentioned Sadhya-Asadhyata, whereas almost all the authorities mentioned that neglected case or improperly treated cases may take the shape of Dushta condition of Pratishyaya, which is a Krichchra Sadhya (Su. Utt. 24/16). Apart from this Madhava has added one more condition of Krimija Shiroroga resulting out of Dushta Pratishyaya.

Along with this, there are other factors, which plays an important role in assessing the prognosis of the disease. Bijadosha,

02.20 CHIKITSA

Chikitsa is defined as Nidana parivarjana[353] or avoidance of causative factors. Ayurveda basically being emphatic about swasthya rakshana give priority to prophylactic management. This is very much applicable in the case of asatya janya pranavaha srotodushti vikara. The agantuja hetu, triggering factors have to be avoided in the first place. Charaka has rightly said, primary importance in Shwasa Chikitsa is the avoidance of causative factors[354]. Both Ayurveda and Modern scientist agree to this fact.

There are many instances in the text books of Ayurveda, where we may identify the features, treatment, and causes about Allergy. Only thing is that they are described in different parts of the Samhitas. The concept of *Satmya* and *Asatmya* is clearly an indication of immunity and allergy. Here *Satmya* means Compatibility. The matter of *Asatmya* means non compatible to body. I.e. if a medicine or food is not acceptable to the body, body tries to through it away in different fashions. It may be diarrhea, vomiting, and skin manifestations and so on. Hence the treatment runs according to the presenting complaint. Whenever the combination of two drugs or food is going to take place it acts as either *Samyoga* or *Viruddha.* The *Viruddha* is the causative factor for *Atma/Shareera asathmya* in many instances. Even visha either Gara or Dooshi can manifest such conditions i.e., asatmya avastha.

Hence while planning the treatment of asatmyajanya pranavaha srotovikaras we may require to consider the treatment principles of visha either Gara or Dooshi,viruddha, satmya asatmyavikaras along with pranavahasrotovikara.

Treatment of Virudhahara

The comparison of virudhahara to visha is to show the need of the proper diagnosis and prompt treatment. The shodhana therapy aims at purification of body from toxins. It involves vamana and virechana to expel the utklishta doshas [355]

If doshas are not aggravated then shamana can be performed. This is by utilizing dravyas which are opposite in nature to that of virudhahara. Observing hita ahara and viharas and taking vishahara oushadha help to achieve shamana and body has to be made samskruta with such dravyas [356]

To strengthen the body against the harmful effects of virudhahara 'hithanna' has to be observed. This is an effect of virudhahara and called as abhi samskarana. Vyayama and snigdhahara should be observed. Chakrapani has included rasayana therapy along with it Dravyas like triphala can be utilized for this purpose in vatatapika method [357].

Apathya Thyagavidhi

Acharyas says that the habits which have been followed for long should not be stopped abruptly because it will lead to remissions. So virudhahara should be discarded in a gradual phase and whole some foods should be taken in a slow and steady manner. In the initial stage 1/16 or ¼ of apathya has to be discarded [For abala 1/16 has to discard first] and pathyahara has to adopt in its place. Thus within 16 or 64 days person will be able to reach the stage of wholesome food[358].

Chikitsa (Management) of Dooshivisha.

Nidana parivarjana

Factors like pragvata, ajeerna etc which aggrevate Dushivisha should be avoided.

Principle

Dushivisha patient should be subjected first to proper swedana and then to sodhana – vamana and virechana. Then Dushivishari agada mixed with honey should be given. The recipe of this agada is as follows Pippali, Dhyamaka, Mamsi, Lodhra, Ela, Suvarchika, Kutanata, Natam, Kushtam, Madhuyashti, Chandanam and Gairikam.[359]

Snehapana:

In the treatment of Dushivisha snehapana is not mentioned. It may be because of the fact that doshas are in utklesha state and poorva karma by snehana is not required.

Swedana

Swedana as such is contra indicated in Visha but only in Dushivisha it is specifically recommended.[360]

Importance of swedana

Pandit C.K. Vasudevasharma in his commentary to Sushruta samhitha has emphasized on the use of swedana in Dushivisha. By the application of swedana, kapha which encapsulates the visha will be liquiefied and removed and is brought to koshta which can be removed by purification process.

- ✓ Sodhana : In sodhana knowledge of visha sthana is important. In amashaya sthana, vamana is indicated.[361]
- ✓ Due to predominance of vitiation of raktha in Dushi visha, Acharya Charaka prescribes raktha mokshana and also panchakarma**.**[362]
- ✓ According to the involvement of vitiated doshas, treatment should be given.
- ✓ Use of sudha (Euphorbia nerrifolia) is mentioned in Kushta, Dushivisha etc.This drug causes tikshna virechana and is given for virechana, only whenthe dosha accumulation is more in the body.[363]
- ✓ The prakriti, satmya, ritu, vishastana and bala of the patient should be considered in case of Dushivisha

Importance of ghrita

The anti-ojus property of Visha is counteracted by ghrita. It is also useful in hridayavaranam. It is useful in all stages of visha vegas especially in vata vitiated conditions.

Importance of rasa

Madhura, tikta, kashaya and katu rasa are said to be antitoxic. Katu rasa is having the properties of deepana, pachana and ruchya. It is kaphahara and sroto sodaka.

Virechana yoga

Haritaki, Lavana, Magadhi and Maricha together is a purgative recipe for patients of Dushivisha as told by Kashyapa.[364] The recipe called Vyoshadi yoga should be administered by the physicaian for purgation in diseases produced by Dushi visha.[365]

Prognosis

A case of Dushi visha poisoning in a prudent and judicious person, and of recent growth is easily cured, while palliation is the only relief that can be offered in a case of more than a year's standing. In an enfeebled and intemperate patient it should be considered as incurable.[366]

Pathyapathya

- ✓ Rakthasali, sashtika, priyangu (a type of grain)
- ✓ Yusha, mamsarasa of ena (a kind of deer), sikhi (peacock), tittiri, prasata (cock)
- ✓ Saindhava, tanduleeyaka (Amaranthus spinosus), jivanti (Leptadenia reticulata), vartaka (Mersalia minuta), sunishannaka (Solanum melogena)

Apathya

- ✓ Navadhanya
- ✓ Kulattha (Dolichos biflorus), tila, souvira, phanitam, sura.
- ✓ Food and drugs which are teekshna, ushna, vidahi
- ✓ Ajeerna, adhyasana, divaswapna, vyavaya, krodha, atapa[367]
- ✓ Tailam, tambulam, lavanam, guda, amla,sarshapa, narikela, takra, kshara dravyas, mamsa dravyas, sura, dadhi, masha, panasa[368]

Vishavimuktha Lakshanas

- ✓ Prasannata of dosha (Normalcy of dosha)
- ✓ Prakriti of dhatus (Normalcy of dhatus)
- ✓ Abhikamksha of anna (Desirous of food)
- ✓ Samata of mootra and vit (Normalcy of urine and faeces)
- ✓ Prasannata of varna, indriya, chitta and cheshta (Normalcy of colour, sense organs, mind, activities)

Management of *Garavisha*

The line of treatment of Garavisha (Artificial poison) includes:

Vamana is the first choice of treatment told by vaghbhata followed by pathya paana bhojana and Sutrashanavidhi is to be followed[369].

Sutrasthana vidhi

This procedure is important in the management of Garavisha. Here it is explained that in Vishabuktha (who has taken poison along with food) should undergo Ubhayavishuddhi (Vamana and Virechanna).Then he should take Suksma Tamra rajah along with Madhu for Hrudayavishuddhi (purification of heart). After confirming Hrudayavishuddhi, intake of Hema churna (Gold) is advised. [370]

Here in the above procedure a doubt arises whether again shodhana is to be advised before giving Hrudayavishuddhi. Commentators of Vaghbhata have clarified the doubt that there is no need to give again Vamana. After Ubhayavishuddhi the word 'Kaale' had been used, which means after Samsarjanakrama, considering desha, kaala etc one should do

Hruday avishuddhi. Above treatment tells that if there is a doubt of intake of visha one should immediately do Ubhayavishuddhi.

Importance of Hridayaavarana

Susruta has mentioned in the context of Hridayavarana that the intelligent person takes medicated Ghritas to protect his Hridaya from all the ill effects of possible induction of Visha [371]. Charaka has explained Hridayaavarana is foremost of all the Upakramas in all types of poison [372]. There is thin distinction between Hrudayavishuddhi and Hridayaavarana treatment. In Garavisha Hridayaavarana treatment can be considered as preventive and Hrudayavishuddhi as a main treatment. According to Dalhana commentary, in the context of Susruta, Hridayaavarana is treatment for... Chitte visha vyapthi pratisheda... Here controversy is whether Hrudaya is to be considered as Chitta or Manas. Hridayaavarana treatment is more beneficial in intake of visham. Because intake of visha will directly spread to Hrudaya i.e. vital organ. This treatment acts as protective covering membrane over vital organs. This treatment not only gives protection to Hridaya, but also those vital organs in the body which are doing similar functions like Hridaya, example lungs, kidney, brain etc. So, the treatment of Hridayaavarana has wider application. It includes Ghrithakalpas like Ajeyaghritha, Amrita ghritha etc. These drugs can cross even blood brain barriers and can act on mind to preserve higher mental functions.

Role of Swarna in Garavisha

Acharyas explained agroushadi for Garavisha is Swarna (Gold) .Qualities of Suvarna is Brihmana, Snigdha, Madhurarasa, Sheetaveerya and best Rasayana. These qualities are opposite to visha gunas and equal to Ojus gunas[373].Suvarna included under madhurarasagana by Astanga Hridaya. This drug is mainly …Pitta anila vishapaha… [374]

Qualities of Swarna in total are as follows -

- ✓ Ojovardhaka
- ✓ Pittaanila vishapaha
- ✓ Rasayana

Many practitioners of Ayurveda today are using Swarna kalpas in various classical disorders. They are getting results; reason may be Garavisha is one of the Nidana for many classical diseases.

When visha is brought into contact with the body, it will at first cause the dushti of raktha and then the three doshas later it reaches hridaya [375]. Hridaya is the seat of ojas. Visha is having qualities opposite to that of ojas. If the visha gunas predominate, the patient dies on the contrary when ojo gunas are superior to those of visha gunas the patient survives. Virudhahara is one of the important causes for rakthadushti [376.] It also causes the utklesha of tridoshas [377] Rakta in its dhatu form are responsible for bala, varnam and sukham. It is the basis of life. Thus raktha is very closely related to ojas in its function. Sufficient amount of food with appropriate quality will be digested properly and converted into consecutive dhatus. When virudhahara is followed dhatu parinama process will be deranged and affect the ojas. If virudhahara combination is very toxic death will occur instantly if the derangement is not much stronger, it will lead to decreased bala; vyadhikshamata of the body depends upon bala [378] and the deterioration of it will lead to various diseases

Hence it can be said that asatmyajanya pranavaha srotovikara is the condition produced due to tridoshas and rakta dahtu . Vamana and virechana are the choice of shodhana treatment which will help in removing vitiated pitta dosha ,the toxins which are responsible for allergy. As the bala is decreased vyadhikshamatva will be less, Rasayana medicines play an important role in increasing the dhtus / oja/bala/ vyadhikshamatva.

Prognosis

Most people with Allergic Rhinitis can achieve adequate relief with a combination of preventive strategies and treatment. While allergies may improve over time, they may also get worse or expand to include new allergens. Early treatment can help prevent an increased sensitization to other allergens.

Medical Care

The 3 basic approaches for the treatment of allergies are (1) avoidance, (2) pharmacotherapy, and (3) immunotherapy. Treatment should start with avoidance of allergens and environmental controls. In almost all cases, however, some pharmacotherapy is needed because the patient is either unwilling or unable to avoid allergens and control

occasional exacerbations of symptoms. For patients with a severe allergy that is not responsive to environmental controls and pharmacotherapy or for those who do not wish to use medication for a lifetime, immunotherapy may be offered.

1) **Avoidance of Allergens and Environmental controls**

- ✓ Patients who have seasonal allergies should avoid outdoor activities when allergens are in the air. The patient's house and workplace should be kept as clean as possible.
- ✓ House dust mites thrive in warm humid conditions, and the antigen is found in their feces. Control measures include removing reservoirs (eg, stuffed animals, carpets, heavy drapes), covering bedding with dustmite– proof covers, and washing potential reservoirs in hot water. Frequent vaccuming with a high-efficiency particulate-arresting (HEPA) vaccum and use of acaricides (eg, benzyl benzoate) and products that denature dust mite antigen (eg, tannic acid) are encouraged. In addition, lowering the relative humidity to less than 50% and lowering the temperature to less than 70°F are helpful in controlling the dust mite population.
- ✓ If removing pets is not feasible, they should be kept outdoors or, at least, out of the bedroom. Also, frequent vaccuming with an HEPA vaccum and washing the animals are helpful in decreasing the allergen load.
- ✓ Molds are present throughout the year in damp areas, both indoors and outdoors. Attention should be paid to reservoirs such as refrigerator drip pans, areas around air conditioner condensers and under sinks, indoor plants, and decaying vegetation in the yard. The use of a dehumidifier and an HEPA air-filtration system also is encouraged.

Prevention

Reducing exposure to pollen may improve symptoms of seasonal Allergic Rhinitis. Strategies include the following:

- ✓ Stay indoors with windows closed during the morning hours, when pollen levels are highest.
- ✓ Keep car windows up while driving.
- ✓ Use a surgical face mask when outside.
- ✓ Avoid uncut fields.
- ✓ Learn which trees are producing pollen in which seasons, and avoid forests at the height of pollen season.

- ✓ Wash clothes and hair after being outside.
- ✓ Clean air conditioner filters in the home regularly.
- ✓ Use electrostatic filters for central air conditioning. Moving to a region with lower pollen levels is rarely effective, since new allergies often develop. Preventing perennial Allergic Rhinitis requires identification of the responsible allergens.

Mould spores:

- ✓ Keep the house dry through ventilation and use of dehumidifiers.
- ✓ Use a disinfectant such as dilute bleach to clean surfaces such as bathroom floors and walls.
- ✓ Have ducts cleaned and disinfected.
- ✓ Clean and disinfect air conditioners and coolers.
- ✓ Throw out mouldy or mildewed books, shoes, pillows, or furniture.

House dust:

- ✓ Vaccum frequently, and change the bag regularly. Use a bag with small pores to catch extra-fine particles.
- ✓ Clean floors and walls with a damp mop.
- ✓ Install electrostatic filters in heating and cooling ducts, and change all filters regularly.

Animal dander :

- ✓ Avoid contact if possible.
- ✓ Wash hands after contact.
- ✓ Vaccum frequently.
- ✓ Keep pets out of the bedroom, and off furniture, rugs, and other dander-catching surfaces.
- ✓ Have your pets bathed and groomed frequently.

2) Pharmacotherapy

Antihistamines :

Antihistamines block the histamine receptors on nasal tissue, decreasing the effect of histamine release by mast cells. They may be used after symptoms appear, though they may be even more effective when used preventively, before symptoms appear. A wide variety of

antihistamines are available. Older antihistamines often produce drowsiness as a major side effect. Such antihistamines include the following:

- ✓ Diphenhydramine (Benadryl and generics)
- ✓ Chlorpheniramine (Chlor-trimeton and generics)
- ✓ . Brompheniramine (Dimetane and generics)
- ✓ Clemastine (Tavist and generics).

Newer antihistamines that do not cause drowsiness are available by prescription and include the following:

- ✓ Astemizole (Hismanal)
- ✓ Loratidine (Claritin)
- ✓ Fexofenadine (Allegra)

Decongestants :

Decongestants constrict blood vessels to counteract the effects of histamine. Nasal sprays are available that can be applied directly to the nasal lining and oral systemic preparations are available. Decongestants are stimulants and may cause increased heart rate and blood pressure, headaches, and agitation. Use of topical decongestants for longer than several days can cause loss of effectiveness and rebound congestion, in which nasal passages become more severely swollen than before treatment.

Topical corticosteroids :

Topical corticosteroids reduce mucous membrane inflammation and are available by prescription. Allergies tend to become worse as the season progresses because the immune system becomes sensitized to particular antigens and can produce a faster, stronger response. Topical corticosteroids are especially effective at reducing this seasonal sensitization because they work more slowly and last longer than most other medication types. As a result, they are best started before allergy season begins. Side effects are usually mild, but may include headaches, nosebleeds, and unpleasant taste sensations.

Mast cell stabilizers :

Cromolyn sodium prevents the release of mast cell granules, thereby preventing release of histamine and the other chemicals contained in them. It acts as a preventive treatment if it is begun several weeks before the onset of the allergy season. It can be used for perennial Allergic Rhinitis as well.

Alternative treatment:

Alternative treatments for AR often focus on modulation of the body's immune response, and frequently center around diet and lifestyle adjustments. Chinese herbal medicine can help rebalance a person's system, as can both acute and constitutional homeopathic treatment. Vitamin C in substantial amounts can help stabilize the mucous membrane response. For symptom relief, western herbal remedies including eyebright(Euphrasia officinalis) and nettle (Urtica dioica) may be helpful. Bee pollen may also be effective in alleviating or eliminating Allergic Rhinitis symptoms.

Immunotherapy

Immunotherapy, also known as desensitization or allergy shots, alters the balance of antibody types in the body, thereby reducing the ability of IgE to cause allergic reactions. Immunotherapy is preceded by allergy testing to determine the precise allergens responsible. Injections involve very small but gradually increasing amounts of allergen, over several

weeks or months, with periodic boosters. Full benefits may take up to several years to achieve and are not seen at all in about one in five patients. Individuals receiving all shots will be monitored closely following each shot because of the small risk of anaphylaxis, a condition that can result in difficulty breathing and a sharp drop in blood pressure.

- ✓ Immunotherapy is indicated in patients whose symptoms are not well controlled with avoidance measures and pharmacotherapy. It also is appropriate for those with symptoms lasting more than 1 season and documented allergen-specific IgE antibodies.
- ✓ Immunotherapy should be considered only in individuals who can comply with weekly injections for approximately 3 years.
- ✓ Immunotherapy should be avoided in those receiving beta-blockers and those who have poorly controlled asthma, autoimmune disorders, or immunodeficiency disorders.
- ✓ During pregnancy, injections should not be initiated, and doses should not be increased.
- ✓ Although the exact mechanisms of immunotherapy are not known, they are associated with decreased allergen-specific IgE levels and increased allergen-specific

immunoglobulin G (IgG) levels. These IgG molecules are thought to be blocking antibodies that are important in impeding the allergic reaction.

- ✓ Immunotherapy involves regular injections (every 5-7 d) of increasing amounts of each reacting allergen until the symptoms are relieved or the maximum tolerated dose is reached, at which time a maintenance dose is given every 2-4 weeks. This dose is maintained until symptoms are controlled for 2-3 seasons and then tapered.
- ✓ Although systemic reactions are rare when immunotherapy is properly administered, only qualified personnel should give injections, and resuscitative equipment should be available.

Surgical Care :

Although Allergic Rhinitis is a medical condition, adjunctive surgery may be offered to alleviate obstructive symptoms in appropriate individuals. Examples are nasal polypectomy in the patients who have severe polyposis and various inferior turbinate reduction maneuvers in patients who have nasal obstruction caused by persistent turbinate hypertrophy that persists despite maximal medical therapy.

Diet :

Food allergies can cause nasal symptoms similar to those caused by inhalant allergies. Therefore, a workup for possible food allergies should be considered if the patient has a history of food reactions, if findings of the inhalant allergy evaluation are negative, and if appropriate treatments fail to yield improvement.

02.11 PATHYA-APATHYA

According to Charakacharya Pathya means the wholesome drugs & regimen, which do not adversely affect the body & mind. While those, which adversely affect the body & mind are considered as Apathya. [Ch. su. 25/45-46] Well-known chikitsa grantha of medeval period "Vaidyajivan" by V. D. Lolimbraj had stated the importance of Pathya – Apathya in chikitsa. i.e. If a person follows the rules for particular diseases there is very little significance of drug treatment & when a person is exposed to Apathya then drug treatment has no value because taken drug can't be able to cure the disease. According to ayurvedic texts, virudha (Apathya ahara and vihar) are stated as the chief etiological factors of the Swasa roga and can be considered for Asatmyajanya pranavaha srotovikara. Nidan parivarjana is one of the important part of the chikitsa. Thus, avoidance of etiological factors is one of the parts of chikitsa. This stops the further progression of the disease by restricting vitiation of doshas & dushyas.

PATHYA

- ✓ Annavarga: - Purana sashtik, Raktashalidhanya, wheat, Yava, Mudga, Kullath
- ✓ Shakavarga: - Parval, Jivanti, Baingan, Chollai
- ✓ Phalavarga: - Bimbiphala, Jambeeri phala, Nimbu, Draksha, Amalaki, Amlavetasa, Bilva, Amla rasa pradhanaphala, Pakva Kushmanda.
- ✓ Dugdhavarga: - Ajadughdha, ghrita, Puranaghrita
- ✓ Mamsavarga: - Mamsa of Deer, Tittar, Peacock, Hen, Lava, Shuka, Rabit, Jangala Mamsa and mamsa rasa.
- ✓ Peya: - Warm water, Honey, Arista (Alcohal) Gomutra (Cow's urine), Sauviraka.
- ✓ Others: - Trikatu, Hingu, Jeera, Kapur (Campher), Saindhava, Elaichi.
- ✓ Vihara: - Devaswapa, Pranayam, Ushnajala snana, Avagaha swedana, Abhyanga, Medicated Cigars (Dhumapana)

APATHYA

- ✓ Anna (Aharavarga): - Rukshanna, Guruanna, Vistambhi ahara, Nishpava, Masha & Kapha-Vata vardhaka ahara.
- ✓ Phala varga: - Banana, Apakva kushmanda
- ✓ Dugdhavarga: - Dahi, unboiled milk.
- ✓ Mamsa varga: - Matsya, Anupa Mamsa

- ✓ Peya: - Dushitajala, Cold water
- ✓ Shakavarga: - Kanda shaka, Saursava.
- ✓ Vihar: - Exposure to cold, dust and polluted environment, weight lifting, exercise, indulgence in sexual activities (ghramyadharma), Chinta, Long journey, Suppression of mutravega etc.

GENERAL INSTRUCTION TO BE FOLLOWED:

- ✓ To take light diet according to Agni Bala.
- ✓ To avoid occupational Asthma, worker should use face mask, when they are working in factories, cotton mills or at places where there is Dust, fumes etc.
- ✓ Deep breathing exercise should be followed.
- ✓ Diet during evening should be taken three hours before going to bed.
- ✓ Avoid direct exposure to external environment after use of Air conditioning.
- ✓ Use of warm clothes in winter season.

NOT TO BE FOLLOWED:

- ✓ Over eating and taking milk at bed time. Fried, chilly, too cold, sour, heavy preparations. Cold and damp places.
- ✓ Fasting for a longer period.
- ✓ Seating in frosty, smoky and congested places for a longer period.
- ✓ Rukshanna particularly toast, popcorn etc.
- ✓ Jalaja, Anupa, Mansa, Dadhi, Aamaksheera, due to Guru and Abhisyandi property.
- ✓ Bread, Burger, Pizza, Cheezes, Paneera etc is used which are having Srotorodhaka property.
- ✓ Contact with those pet animals, which do not suit the individual. Direct exposure to Prag-vata.

DRUG REVIEW

The present study comprises of both shodhana and shaman treatments. For Shodhana Virechana treatment is given, starting with shodhananga snehapana with Kanthakari ghrita[389] as a poorvakarma after confirming niramavastha and for Virechana Trivrit lehya[390] was used. For Shamana Chikitsa Shirisharishta[391]and Bhrigu haritaki[392] yogas were administered.

Table No.155: List of drugs used for clinical trial

Sl no	**Oushadhi yoga.**	**Purpose**
1	shirisharishta	Shamanachikitsa
2	Bhrigu haritaki	Shamanachikitsa

Table No.156: List of drugs used for virechana

Sl no	**Oushadhi yoga.**	**Purpose**
1	Kanthakari ghrita.	Shodhananga snehapana
2	Moorchita tila taila	Sarvanga abhyanga
3	Trivrit lehya	Virechanartha

Table No.157: Ingredients of Kantakari gritha

Sl .No	**Ingredients**	**Part**	**Quantity**
1	Kantakari (Solanum xanthocarpum)	Panchanga	
2	Guduchi (Tinospora cadifolia).	Kanda , patra .	
3	Ghrita	----------	

Table No.158: Ingridients of shirishaarista

Sl .No	Ingredients	Part	Quantity
1	Shirisha (Albizzia lebbeck)	twak	Tulardha
2	Pippali (Piper longum)	Phala	1 pala
3	Priyangu (Callicarpa macrophylla)	Beeja ,pushpa	1 pala
4	Kushta (Saussurea lappa)	Moola.	1 pala
5	Ela (Elettaria cardamomum)	Beeja	1 pala
6	Nilini (Indigofera tinctoria)	Patra, moola	1 pala
7	Nagakeshara (Mesua ferrea)	Pushpa kesara	1 pala
8	Haridra (Curcuma longa)	Kanda	1 pala
9	Daruharidra (Berberis aristata)	Kanda twak.	1 pala
10	Shunthi (Zingiber officinale)	Kanda	1 pala

Table No.159: Ingridients of brighuharitaki avaleha:

Sl .No	Ingredients	Part	Quantity
1	Kantakari (Solanum xanthocarpum)	Panchanga	100 pala.
2	Haritaki (Terninalia chebula)	Phala	100 numbers.
3	Shunthi (zingiber officinale)	Kanda	04 tola.
4	Mareecha (Piper nigrum)	Phala	04 tola.
5	Pippali (Piper longum)	Phala	04 tola.
6	Ela (Elettaria cardamomum)	Phala	04 tola.
7	Twak (Cinnamomum zeylanicum)	Kanda twak	04 tola.
8	Nagakeshara (Mesua ferrea)	Pushpa kesara	04 tola.
9	Talisha patra (Abies webbiana)	Patra	04 tola.
10	Madhu		24 tola.
11	Guda.		400 tola.
12	Water		1024 tola

Individual drug review

02.12 SHIRISHA

- **Botanical source**: *Albizzia lebbeck* Benth.
- **Family**: Leguminoseae
- **Synonyms**: Viasha hanta, Madhupushpa, Kapitana, Shyamala, Shukataru, Uddanaka.
- **Vernacular name**:
    - **Hindi**: Siras, Sirsa
    - **Gujarati**: Sirsadiyo
    - **Kannada**: Bage mara
    - **English**: Indian walnut, Parrot tree, Woman's tongue tree
- **Classification**:

According to Caraka Samhita: Shirovirechana, Swedahara, Vishaghna, Vedanasthapana[223]Saraasava[224]Kashayaskandha[225]According to Susruta Samhita : Avasadaka [226] Sirovirecana[227] Pitta- nashana[228] Vishahara[229] According to Vagbhata:Kashayagama [230]According to Kashyapa and Bhela: Siro virechana[231] According to Bhaishajya Ratnavali: Nyagrodhadi gana [232]According to Bhavaprakasa : Vatadi varga

Citations from Sanskrit literature – The delicacy, nicety and the sensitivity of the Shirisha flowers are compared with Sita. Similarly in Kumarasambhava of Kalidasa, soukumaryta of Goddess Parvati is exemplified by the Shirisha flowers. Shirisha was also used for beautification in lieu of ear ornaments. Shirisha flowers were also extensively used in Astrological practices to predict the good crop of Priyangu and Kanguni.

Habitat: The tree prefers the moist situations and is common through out India except on Sindh.

Habit and general features: A large erect deciduous tree with an umbrella shaped crown. It has a clear bole of 20-30' height and attains 6' in girth. The rate of growth is very rapid (Wealth of India.p.no.43).

Bark: Appreciably thick and rough, dark brown to grayish black with vertical & transverse deep fissures. The rind or outer bark comprises nearly a third or more of the thickness of the entire bark. On the trunks and older branches, the bark has a composite structure composed of discontinuous alternating strata of 'woody' and sub serous layers. Excluding the corky layer, the middle and inner barks which comprises the officinal tissue is nearly two thirds the thickness of the entire bark. Its outer part has a characteristic reddish brown tinge.

Leaf: about 9' long, alternate, stipulate, evenly bipinnate, grooves on upper side, tapering.

Leaf lets: 4 – 8 pairs, opposite, short stalked, 1-2' long, ½ -3/4' broad, entire, oblong and pale.

Flower: Sessile or short pedicelled, all bisexual, regular, whitish or yellowish white, fragrant,

Calyx -0.125' long, petalw-5, connate below the middle to form funnel shaped corolla, stamens indefinite.

Fruit: 6"-1', straight or slightly curved, ¾ - 1 ½' broad, thin but firm, straw to yellowish brown.

Seeds: Non endospermic, yellowish brown, ¼ - ¾ ' long, ovate, horse shaped compression near margin.

Varieties : 2 types – Sweta and Krishna (P.V.Sharma)

Rasa Pancaka : Rasa : Madhura, Tikta, Kasaya.

Virya : Anushna

Vipaka : Katu

Guna : Grahi; Bija – Stambhaka

Dosha shamana : Tridosha

Vyadhi : Visa, Twagroga, Shwasa, Shotha, Agrya Oushadha for Vishas

Officinal parts: Panchanga, stem-bark, flowers, seeds.

Chemical constituents:

Echinocystic acid and β-sitosterol identified in bark and seeds (Indian J. Applied Chem.1969, 32, 73, Chem. Abstr. 1971, 75, 16035z); Saponin mixture from pods and seeds on hydrolysis yielded echinocystic acid, oleanolic acid, albegenin and albigenic acid (Bull. Chem. Soc. Jpn. 1970, 43, 446; Indian J. Appl. Chem. 1971, 34, 214; Chem. Abstr 1972, 77, 85583 e); a saponin – lebbekenin C – on acid hydrolysis yielded echinocystic acid, glucose and rhamnose (Planta Med. 1973, 24, 183); friedelan -3-one (friedelin) and γ-sitosterol from bark (Curr. Sci. 1974, 43, 46); a triterpene Saponin –lebbakinin A m.p. 205°-from seeds composed of glucose, galactose, arabinose, xylose, fructose and rhamnose in ratio 5:1:1:1:1:2 and echinocystic acid (Indian J. Chem. 1973, 11, 1094); Another Saponin lebbakinin D – on acid hydrolysis yielded echinocystic acid glucose, galactose, arabinose, xylose, and rhamnose (J. Indian. Chem.Soc.1975, 52,1202); lebbakanin E m.p. 125°, isolated and shown to consist of acacic acid and glucose, arabinose, xylose and rhamnose in ratio of 4:2:1:1 (J. Indian. Chem.Soc.1976, 53, 859; Nat. Acad. Sci. Lett. 1979, 2, 135, Chem. Abstr. 1979, 91, 120369 c); in addition to melacacidin and melanoxetin, two new compounds – (-)2,3-cis-3,4-cis-3,Ω-methyl-melacacidin as its methyl ether and 3'-O-methylmelonoxetin-isolated from heartwood (Indian J. Chem. 1977,15B, 201); plant contained three non-protein sulphur aminoacids; mature leaves contained ketoacids including phosphoenolpyruvate, glyoxalate, oxalacetate and α- oxoglutarate (Plant Biochem. J. 1977, 4, 34; Chem. Abstr. 1977, 87,148762 s); vicenin-2, reynoutrin, rutin, myricitrin and robinin from leaves (Shoyakugaku Zasshi 1977, 31, 172; Chem. Abstr. 1978, 88, 148947 b). Lebbakinin C 3' -O-Methylmelanoxetin Lebbakinin E

Pharmacologic action:

Bark and flowers decoctions protected guinea pig against histamine and acetyl-choline induced bronchospasm. Chronic treatment with bark decoction also protected sensitized guinea pig against antigen challenge. Drug showed antiasthmatic and antianaphylactic

activities due to inhibition of phenomenon of sensitization (Tripathy & Das, Indian J. Pharmacol. 1977, 9, 189), (J. Res. Indian Med.1973, 8, 29).

The bark is used as one of the ingredient of an Ayurvedic Kada or decoction used for treating asthma. Pharmacologically it was found to show antitussive action and the ability to prevent allergy induced bronchospasm. Bark is also useful in treatment of allergic conjunctivitis [Iyengar et al, Indian Drugs, 1994, 31, 183, 187; Mukhopadhyay et al, J Res Educ Ind Med, 1992, 11 (4), 17]. From the alcoholic extract of stem bark were isolated cardenolide glycosides, anthraquinone glycosides & CHCl3R (antidermatophytic) which showed antibacterial & antifungal activity. They also showed activity against other aerobes, yeast and protozoans, Trichomonas vaginalis (Ganguli & Bhatt, Indian J Exp Biol, 1993, 31, 125).

Analysis of the plant revealed the presence of flavonoids, triterpenoids and triterpenoid saponins (Agrawal & Singh, Indian J Pharm Sci, 1991, 53, 24). The bark yields tannins (7-11%) of condensed type, viz. D-catechin, isomers of leucocyanidin (5,7,3',4'-tetrahydroxyflavan-3,4-diol); and (-)-melacacidin (7,8,3',4'-tetrahydroxyflavan-3,4-diol); and a new leucoanthocyanidin, lebbecacidin (8,3',4'-trihydroxyflavan-3,4-diol). It also gives friedelin and ß-sitosterol. Extract of the bark possesses anthelmintic activity and expectorant action (Rayudu & Rajadurai, Leath Sci, 1965, 12, 21; Rayudu, ibid, 1967, 14, 234; Tripathi & Das Gupta, Curr Sci, 1974, 43, 46; Shah & Bhattacharyya, loc. cit.).

Research works:

1) Saponin fraction & seed extract of plant significantly reduces the number of ruptured mast cells, in both mesenteric buds and peritoneal fluid obtained from sensitized rats and this effect was identical in both types of systemic Anaphylaxis (Active & Passive) [233]

2) Shirisha twak kwath administered in patients of Tamaka shvasa shows significant increase in the Lung vital capacity. Eonsinophil count, W.B.C. count, & E.S.R. were reduced significantly at the end of treatment.[234]

3) Stabilizing effect on mast cells reduces Histamine level, suppresses T Lymphocyte activity, reduces allergy inducing Antibodies. Antiallergic & Anti-inflammatory action may be due to action on Adrenal.[235]

4) Oral administration of Albizia bark (ethanol extract) provided adrenal protection against histamine-induced bronchospasm. Tripathi et al. studied asthmatic patients who were treated with this plant and showed reduced histamine levels and elevated cortisol levels.[236]

5) Albizzia lebbeck- Inhibits degranulation of mast cells, synthesizes reaginic type antibodies and has a pharmacological action like Disodium Cromoglycate.[237]

6) The effect of crude extract of *Albizzia lebbeck* from the seeds and a pure saponin fraction on the mast cells in peritoneal fluid was studied in rats subjected to anaphylaxis. The results showed that the extract as well as the pure saponin fraction had a stabilizing effect on the mast cell membrane.[238]

7) Iyengar, M.A.et al (1994) studies on an antiasthma kada-A proprietary herbal combination. Part I Clinical study, Indian drug vol. 31(5) pp 183-186 [239]

8) Iyengar, M.A.et al (1994) studies on antiasthma kada-A proprietary herbal combination. Part II. Pharmacological studies, Indian drugs vol.31(5)pp 187-191[239]

9) Pandya, M.M.(1993), Clinical trial of anti-inflammatory Ayurvedic formulation as an external application. Sachitra Ayurveda vol 45(10) pp 764-768 [239]

10) Shaw, B.P. & Bamkin, Bera (1986) Treatment of T.P.E. with Shireesh flowers churna, Nagarjuna vol 29(6) pp 1-3 [239]

11) Tripathi, R.M. & Das, P.K.(1977) Studies on antiasthmatic and antianaphylactic activity of Abizzia lebbeck, Ind. Journ. Pharmacology, vol 9 pp 189-194[239]

12) Oral administration of Albizia bark (ethanol extract) provided adrenal protection against histamine-induced bronchospasm.[240]

13)In an uncontrolled study in 60 patients with asthma it was found that clinical response depended on the duration of the disease. Response was excellent for asthma of recent onset (less than two years). Results were less predictable in older cases. After treatment with Albizia the patients' elevated histamine levels were decreased, plasma cortisol was increased and plasma catecholamine decreased. [241]

14) Plant Sirish is reported to have antiseptic, anti-dysenteric and anti- tubercular properties. The bark has acrid taste. It is recommended for bronchitis, leprosy, and paralysis and helminthes infections.[242]

15) The liquid extract of A. lebbeck on histamine-induced bronchospasm in guinea pig showed significant rise in plasma cortisole, which shows the protective action of A. lebbeck in bronchospasm and other allergic conditions. The anti anaphylactic activity of A. lebbeck is due to mast cell stabilizing property and wholly due to antihistaminic or smooth muscle relaxant activity.[243]

16) Tripathi S et al Ethnophar 1 (4):385-396, 1979. Chromoglycate like action on mast cells.[244]

Previous MD/PhD Research works on the antiasthmatic activity of Shireesh (*Albezia lebback*) xvii

(a) Sharma O.D. (1977) – Clinical and experimental study on Tamaka Shwasa and its management with Albezia lebback. BHU

(b) Tripathi Pratibha (1980) – Effect of Albezia lebback on Adrenal gland w.s.r. to its role in the management of Bronchial Asthma. BHU

(c) Trivedi C.S. (1995) - Tamaka Shwasa Me Shirishadi Kashaya ka Prayogika adhyana. Nagpur

(d) Kaliya Kamaleshwar (2004) – Study on Shireesh (Albezia lebback) and its efficacy in Tamaka Shwasa. UD

(e) Muralidhar R. (2004) - A comparative pharmaceutico-pharmaco-clinical study of Shirisharishta and its Shwasahara effect Jamnagar.

(f) Jaiswal Mandeep (2007) - A comparative Pharmaceutico-Pharmaco-Clinical Study of Shirisharishta prepared by Twaka and Sara Kastha of Shirisha w.s.r. to it's Shwasahara Effect. Jamnagar.

Plate 1: Shireesha kandatwak.

Plate 2: shirsha patra,pushpa and phala

Shireesh (*Albezia lebback*)

02.13 Pippali:

Latin name: *Piper longum.*linn

Family: Piperaceae

Synonyms : Magadhi, Vaidehi, Kana

Vernacular Names:

Hindi : Pipal Gujarathi : Pipal

Kannada: Pipli English: Long pepper

Part used : Root, fruit

Classification: Kasahara, Hiccanigrahana, Deepaniya[245] Pippalyadi, Urdhabhagahara[246]

Haritakyadi Varga[247]

Rasa Pancaka :

Rasa : Katu

Guna : Laghu, Snigdha

Virya : Ushna

Vipaka : Katu

Doshakarma : Vata-Kaphahara

Officinal Part: Fruits

Chemical Composition:

It has 4-5% Piperine, fragmented oil 0.7%, Piplartine, Sesamin and Piplasterol Alkaloids. The plant contains essential oil consisting of long chain hydrocarbons, mono and sesauiter pens caryophyllene being the main product. Other constituents are piplartine, piper longumine, piperlonguminine and its dihydro-derivative, pipernonaline, piperundecalidine,pipercide and guineensine, sesamin, dieudesmin, ß– sitosterol and dihydrostiqmasterol. It also consists of four aristolactams (cepharadione B, aristolactam A, norcepharadione B,piperadione) and aminoacids. (Medicinal plants of India, G . V. Satyavati et al 1987, 2, New Delhi). Dried Pippali consists of an essential oil - 7% with spicy odour. Also Tricontane, dihydrostigmasterol, Piplasterol, and un- identified steroid reducing sugar and glycosides are present in dried Pippali (Alexander H.L. Bronchial Asthma - its diagnosis and treatment. Philadelphia.Lea, P.105 1972)

Pharmacological Action:

Lot of pharmacological studies establishes the effect of Pippali in asthma. Some of which are shown below:

(i.) Evaluation of anti–allergic activity of piper longum (Bahanlear S.A. Dept. pharmaco.Seth G.S. Medical coll. Bombay pub. in Indian drug 1984)

(ii.) A marked anti-inflammatory activity of piper longum fruit decoction against carageenin induced rat paw edema. (Sharwell singh, 1986)

(iii.) Piperlongumine, an amide alkaloid, is used in the treatment of asthma.

(iv.) Its milk extract effectively reduced passive coetaneous anaphylaxis in rats, protected guinea pigs against antigen induced bronchospasm. It did not have significant effect on total quantity of histamine in lungs, trachea and intestines or on release of histamine on antigenic challenge. (Dahanukar et al 1981, Dahanukar and Karandikar, 1984)

(v.) The respiratory depression induced by pentobarbitone and morphine in anesthetized dogs was also antagonized by Piperine (Singh. et al 1973a). Piperine reversed respiratory depression induced by nalorphine, but unlike nalorphine Piperine did not antagonize morphine induced analgesia in rats (Singh et al 1973b)

(vi.) Piperlongumine as well as extract of P. longum showed marked antispasmodic action on isolated tissue (Fernandez et.al 1980)

(vii.) The crude extract of P. longum as well as piplartine suppressed the ciliary movements of the esophagus of frog. These findings suggest that therapeutic efficacy in relieving cough could be due to the suppression of cough reflex (Banga et al. 1964).

(viii.) Alcoholic extracts of dry fruits showed activity against Micrococcus pyogens, Var. aureus and Escherichia coli (George et al, J. Sci. Industri. Res. 1947).

Plate 3-Pippali: *Piper longum*

Plate 4 - Pippali: *Piper longum* Fruits.

02.14 Priyangu

Botanical source: *Caliparpa marcrophylla*

Family: Verbenaceae

Synonym : Phalini, Gandha priyangu, Viswaksenakanta.

Officinal Part: Flower

Classification: Purisa sangrahaniya, Mutra virajaniya[248]

Rasa Pancaka :

Rasa : Tikta, Kashaya, Madhura

Guna : Guru, Ruksha

Virya : Shita

Vipaka : Katu

Doshaghnata : Vata, Pittahara

Karma : Dipana, Anulomana.

Chemical constituents: Aromatic oil

Plate 5 : Priyangu- Infloroscence.

Priyangu: ***Caliparpa marcrophylla***

PRIYANGU.

02.15Kushta

Botanical source: *Saussurea lappa*.

Family: Compositae (Myrsinaceae)

Synonyms: Kashmiraja, Pakala, Vapya, Ruk

Vernacular names :

Hindi : Kut, Kuth. Gujarati : Uplet, Kat. Kannada: Kankushta English: Costus root.

Classification: Lekhaniya, Sukrasodhana, Asthapanopaga[249]

Officinal part: Root

Rasa Pancaka :

Rasa : Tikta, Katu, Madhura

Guna : Laghu , Ruksha, Tikshna

Virya : Ushna

Vipaka : Katu

Doshaghnata : Kapha, Vatahara

Karma : Dipana, Pachaka, Anulomana, Vrishya, Artavajanana, Garbhashayottejaka.

Chemical Constituents : Essential oil, Saussurine alkaloid, bitter resin, Volatile oil (1.5 - 2.5%),Costulonide, a new sesquiterpene lactone – methoxy dihydro costulonide. Kuth roots contain resinoids (6%), essential oil (1.5%), and alkaloid (0.05%), inulin (18%), a fixed oil and other minor constituents like tannins and sugars. Roots from aged plants contain a higher percentage of oil, and samples collected during September-October give higher yields than those collected during earlier months (Rao & Verma, J. sci. industr. Res., 1951, 10B, 166). Alcoholic extract of the root containing both the essential oil and the alkaloid has been found very useful in the treatment of bronchial asthma, particularly of the vagotonic type. The total alkaloidal preparation of the drug has been used with great success both subcutaneously and orally and the relief obtained is said to be comparable to that of conventional bronchodilators without any side effect. Also, a useful drug for chronic bronchitis and asthma. (Chopra, 1958, 405-06; Raghavan et al., J. Post-Grad. Med.,Bombay, 1962, 8, 158; Sastry & Dutta, lndian J. Pharm., 1961, 23, 247). A parasticide, containing

costunolide and dehydrocostus lactone isolated from the plant, is effective in controlling Anisakis infestaton in human digestive tract (Bhattacharya, Indian J Exp Biol, 1994, 32, 31).

PLATE NO: 6 KUSTA

02.16 Ela

Botanical source : *Elettaria Cardamomum*

Family : Zingiberaceae

Synonyms : Triputa, Truti, Dravidi, Kapotaparnika, Sukshmaila, Candrabala.

Vernacular Names :

Hindi : Elayaci Gujarathi : Elci Kannada : Elakki English : Cardamum

Officinal Part : Fruit

Classification : Katukaskandha, Shwasahara, Angamarda Prasamana & Sirovirecana[25] Eladi Gana[251]

Rasapancaka :

Rasa : Katu, Madhura

Guna : Laghu, Ruksha

Virya : Shita

Vipaka : Madhura

Doshaghnata: Tridoshahara

Karma : Rocana, Anulomana, Mutrala, Kasa-Shwasahara, Kaphanissaraka

Chemical Constituents: 3-8% volatile oil, which contains Terpine, Terpinyl acetate and cineol.3-4% starch etc. Volatile components of cardamom exhibit antimicrobial activity. Oil has antiaflatoxin substances. It has inhibitory properties against aflatoxins synthesis and caused 90% drop in aflatoxin elaboration. Thus, oil can be successfully utilized against the danger of aflatoxin on food commodities [Kubo et al, J Agric Food Chem, 1991, 39, 1984; Ranjan et al, Geobios, 1992, 19(1), 39]. Terpineol and acetyl terpineol, the active principles of cardamom seeds showed greater penetration enhancing capacities than Azone, which was used as a comparative penetration enhancer for the diffusion of prednisolone through mouse skin in vitro (Yamahara et al, Chem Pharm Bull, 1989, 37, 855). Cardamom tincture is

used in the slimming preparations containing ephederine. Cardamom is used in preparation of antioxidants that control ageing (Chem Abstr, 1991, 115, 189780; 1991, 114, 60843).

Plate 7 :Ela

02.17 Nilini

Botanical source: *Indigofera tinctoria*.

Family : Papillionaceae

Synonyms : Nilini, Ranjini, Sthiraraga, Anjana keshika.

Vernacular names :

Hindi : Nil Lil, Guli. Gujarati : Gali. Kannada : Nili English : Indigo

Rasapancaka :

Rasa : Katu, Tikta

Virya : Ushna

Vipaka : Katu

Doshaghnata :Vata-,Kapha –hara.

Karma : Keshya, Visa, Krimi-, Jwara-, Kasa - hara Recani, Mohahara.

Officinal part: Root, fruit.

Chemical constituents : Following are the analytical values of leaves (dry basis): nitrogen (N),5.11; phosphoric acid (P_2O_5), 0.78; potash (K_2O), 1.67; and lime (CaO), 5.35%. It is a rich source of potash, the ash (4.4%) containing as much as 9.5% of soluble potassium salts. (Yegna Narayan Aiyer, 613; Whyte et al., 281; A Manual of Green Manuring, 100; Idnani & Chibber, Sci. & Cult., 1952-53, 18, 362; Prasad & Dange, Indian For. Leafl ., No. 95, 1947, 4; Mem. Dep. Agric. Madras, No. 36, 1954, 837).

Pharmacological action:

Protective effect on tissue anti-oxidant defense system against D-galactosamine, and endotoxin induced hepatopathy in rats (J. on Natural Remedies V 31 (1), 2002). Alcoholic extract of the aerial parts showed hepatoprotective activity in experimental animals. The extract (1000 mg/kg a i) also increased bile flow and liver weight in rats, suggesting stimulation of microsomal enzymes of the liver. The effect was, however, more pronounced

in male rats as compared to female rats (De et al, Indian Drugs, 1993, 30, 355; Tyagi et al, Acta Clin Scient, 1991, 1, 79). Toxicological studies of the extract in vivo and in vitro against the Pulse beetle (Callosobrucus chinensis) and Mosquito (Anopheles stephensi) larvae showed that the rotenoids were more effective against mosquito larvae. Extracts from callus was more effective against both insects (Kamal & Mangla, J Biosci, 1993, 18, 93). Maximum histamine content (5.0mg/g dry wt) was found in 8 week old tissues in culture. Cells under actively dividing stage, contain more histamine (Kamal & Mangla, Indian Drugs, 1992, 29, 179).

Plate 8:Nilini.

02.18 Nagakesara

Botanical source: *Mesua Ferrea*

Family: Guttifereae

Synonyms: Nagapuspa, Campeya

Vernacular Names:

Hindi : Pilakesar. Gujarathi : Pilu Nagakesara Kannada : Nagasampige.

English: Assam iron wood, Cobras saffron.

Officinal Part: Fruit, male stamens.

Classification : Eladi, Vacadi, Priyangavadi and Anjanadi[252]

Rasapancaka :

Rasa : Kasaya ,Tikta

Guna : Laghu, Ruksa

Virya : Usna (Slight)

Vipaka : Katu

Dosaghnata : Kaphapittasamaka

Karma : Amapacaka, Visahara, , Kandu, Hrillasa Nashaka, Kaphaghna, KasaShwasahara.

Chemical Constituents: Oily resins, Volatile oil and meusol.Chemical constituents: Mammeisin is reported from the seeds, while stamens afforded two novel biflavones designated as mesuaferrone – A and mesuaferrone –B, mesuanic acid, α and β – amyrine, β-sitosterol, Fatty acids from the seed oil is reported. Other constituents isolated are: mesuol, mesuaferrol, leucoanthocyanidin, mesuone, mammeigin, mesuagin, mesuaxanthone – A and – B, euxanthone, other xanthone derivatives, ferrrol – A and – B, a triterpene called guttiferol, ferrxanthone derivative and essential oil from various parts.

Pharmacologic activities:

Ethanolic extract of the whole plant excluding roots showed antibacterial activity. Other pharmacological activities reported are – antifungal, anthelmentic, hypotensive, antispasmodic,antianaphylactic, antiasthmatic, antiimplantation, anti-inflammatory, juvenomimetic, insecticidal.The LD50 of ethanolic extract of whole plant in mice was 500mg/kg i.p, LD50 of acetone extract of stamens in mice was 400 mg/kg i.v, and nontoxic upto 1600mg/kg p.o. Potentiates bronchodilator activity and also posses' antihistaminic property.

Plate 9: Nagakesara

02.19 Haridra

Botanical source : *Curcuma longa L*.

Family : Zingiberaceae.

Synonyms : Bhadralata, Dirghranga, Hattavilasini, Krmighni, Visaghni, Yositpriya.

Vernacular names :

Hindi : Haladi Gujarati : Haladar Kannada : Arashina English : Turmeric

Classification : Lekhaniya, Kushthaghna, Vishaghna, Tikta Skandha[253] Haridradi-, Mustadi Gana, Lakshadi Gana, Tikta Varga[254]

Rasapancaka :

Rasa : Katu, Tikta

Guna : Ruksha

Vipaka : Katu

Virya : Ushna

Doshaghnata : Kaphaghna, Vataghna.

Karma : Krimighna, Sandhanakrit, Vishaghna. Shothahara, Anulomana, Garbhashaya Sodhana.

Active constituents: curcuminoids – the non-volatile coloring matter -6% of which curcumin constitutes about 50-60%, essential oils - 2-7% and the minor components include diferuloylmethane desmethoxy curcumin, dicinnamoylmethane, bidesmehtoxycurcumin.Volatile oils (5%) – Sesquiterpenes (60%) like 1-cycloisoprenmyrcene, Zingiberebe (25%),tumerone, ar-tumerone, alpha atlantone, gamma atlantone, -Phellandrene, Sabinene and also, cineole, borneol & curcumone. Sugars – arabinose (1%), fructose (12%) and glucose (28%).Also contains bitter substances, fixed oils and acids. (-Study of crude drugs, M.A.Iyengar).

Pharmacological activities:

Antibacterial, cholagogue, insecticidal, antifungal, anti-inflammatory,antiprotozoal, CNS depressant, anti-fertility, antiarthritic, hypocholesteremic, antihistaminic,antihepatotoxic. Toxicity of the rhizome in rats, guinea pigs and monkeys were reported. LD50 value of aqueous suspension of volatile oil was found to be 3.25 ml/kg; while LD50 of volatile oil emulsified in Twin 80 was found 0.533 ml/kg. cytotoxic effects of curcuminoids have been observed in cell culture. Effect of essential oil & Curcumin are antibacterial and antifungal.

Plate 10:Haridra

02.20 Daruharidra

Botanical source*: Berberis Arishtata*

Family : Berberidaceae

Officinal part : Root stem, fruit, bark and wood.

Rasapancaka:

Rasa : Tikta, Kashaya

Guna : Laghu, Ruksha

Virya : Ushna

Vipaka : Katu

Doshakarma : Kapha-, Pitta-, Shamaka

Karma : Dipana, Pittasaraka, Rakta Shodhaka, Swedajanana and Varnya.

Chemical Composition: The root has 2-3 % Berberine. Others - oxyberberine, berbamine,aromolilne, karachine, palmatine, oxycanthine and taxilamine are reported from various parts.

Pharmacologic activities:

Hypoglycaemic, anticancer, gastroirritant, antifatigue, anticoagulant, antipyretic, local anaesthetic, antiprotozoal, anti-T.B, anti-bacterial, anti-inflammatory, antitrachoma,hypotensive, CNS depressant. LD50 value of berbarine in mice was found to be 25.3mg/kg i.p.Drug was found free from any serious toxicity in human beings.

Plate 11:Daruharidra

02.21 Sunthi

Botanical source : *Zingiber Officinale*

Family : Zingiberaceae

Synonyms : Nagara, Mahaausadham, Katubhadra

Vernacular Names :

Hindi: Sonth. Gujarathi : Sunth Kannada : Sunthi. English: Dry Ginger

Classification: Triptighna, Arsoghna, Deepaniya [255] Pippalyadi gana[256]

Rasapancaka:

Rasa : Katu

Guna : Laghu, Snigdha

Virya : Ushna

Vipaka : Madhura

Doshakarma : Kapha Vatahara

Karma : Rocaka, Amavataghna, Dipana, Pacaka, Vrishya and Hridya.

Officinal Part: Rhizome

Chemical Composition:

It has 10.6% water content, 15.4% protein, 5.3% starch and 1-2.7% Volatile oil. Addition of aqueous extract of rhizome inhibited aflatoxin synthesis by 67 and 68% in SMKY – liquid medium and maize composite respectively. The extract also exhibited nematicidal activity against the burrowing nematode - Radopholus similis. Ethanolic extract of rhizome showed low antifungal activity against Aspergillus niger, A. flavus and Penicillium citrinum. The leaf extract is found to be toxic to ringworm causing fungi - Epidermophyton floccosum, Trichophyton mentagrophytes and Microsporum gypseum (Ranjan et al, Geobios, 1992, 19, 39; Gnanapragasm & Prematunge, Sri Lanka J Tea Sci, 1991, 59, 82; Rizki et al, Pakist J Sci

Indutr Res, 1989, 32, 608; Mishra et al, Indian Drugs, 1991, 28, 300). Ginger oil induced the detoxifying enzyme system – glutathione S-transferase, active against chemical carcinogens, in small intestine mucosa and liver of female A/J mice. The active principles [6]- Shogaol (at 2.5 mg/kg), and [6]-[8]-and [10]-gingeral (at 5mg/kg) are found to enhance gastro-intestinal motility in mice. Zingerone is reported to scavange superoxide anions in xanthin-xanthin oxidase systems (Lam & Zheng, J Agric Fd Chem, 1991, 39, 660; Yamahara et al, Chem Pharm Bull, 1990, 38, 430; Krishnakantha & Lokesh, IndianJBiochem Biophys, 1993, 30, 133). Ginger oil can be utilized as a seed protectant to control post harvest spoilage, as it completely inhibited the growth of a number of storage fungi and it is sensitive to many others including ringworm causing fungi. Gingerone A exhibited moderate growth inhibition activity against some plant pathogenic fungi. Repellent property of the oil is also demonstrated against cockroaches and the pea weevil - Bruchus pisorum (Mishra, IndianPerfum, 1990, 34, 266; Kaur & Sinha, J Res Ayur Siddha, 1991, 12, 200; Endo et al, Phytochemistry, 1990, 29, 797; Garg & Jain, J Econ Bot Phytochem, 1991, 2, 25).

Plate 12:Sunthi

Bagasse:

Bagasse is the fibrous residue of the cane stalks (Saccharum officinarum) left after crushingand extraction of the juice. It forms about 28-36 per cent of the cane.The composition of bagasse differs according to the variety and maturity of cane, method of harvesting, and efficiency of milling. It analyses to: 105 moisture, 46-52; fibre, 43-52; and soluble solids (mostly sugar), 2-6%. The two main fractions of bagasse, viz. the fibrous and pithy parts consist on a dry basis of c.25 per cent of pentosans which are mainly hemicelluloses of the xylan type. Hydrolysis of the hemicellulose fractions revealed the ccurrence of xylose, arabinose, and also glucose, galactose, xylulose, and glucuronic acid (Paturau, 25; Pathak, Proc. 11th Congr. ISSCT, 1962, 1211; Banerjee et al., Proc. Indian Sci. Congr., 1960, pt III, 218; Banerjee et al., Sci. & Cult., 1961, 27, 498; Pathak & Srinivasan,Indian Pulp Pap., 1957-58, 12, 429; Guha & Pant, ibid., 1964-65, 19, 327). The amino acids present in bagasse are (mg. % in the protein): aspartic acid, 13.25; threonine, 5.58; methionine,7.84; valine, 3.33; leucine, 5-75; tyrosine, 1.51; and alanine, 3.56. Antitumour substances have been reported in bagasse (0.1%); they are probably polysaccharides consisting mainly ofhexose and pentose (Viswanathan et al., Indian J. med. Res., 1963, 51, 563; Chem. Abstr.,1966, 65, 12558). (Wealth of India).

Table no 12 Showing comparative Rasapanchaka of the ingredients of Shirisharishta:

Content	Rasa	Guna	Virya	Vipak	Dosaghnata
Shirisha	Madura,Tikta Kasaya	Laghu, Ruksha, Tikshna	Anu-U	K	Kapha, Pitta, Vata
Pippali	Katu	Laghu Snigdha, Tikshna	U	K	Kapha, Vata, Pittavardhak
Priyangu	Kasaya	Guru , Ruksha	S	K	Kapha, Pitta , Vata
Kusta	Katu, Tikta Madhur,	Laghu, Ruksa, Snigdha	U	K	Kapha, Vata
Ela	Katu, Madhura	Laghu, Sukshma	S	M	Kapha, Pitta, Vata
Nili	Katu, Tikta	Laghu, Ruksha	U	K	Kapha, Vata
Haridra	Katu, Tikta	Laghu, Ruksha	U	K	Kapha, Pitta

Daruharidra	Kasaya, Tikta	Laghu, Ruksha	U	K	Kapha, Pitta
Nagakesar	Kashaya, Tikta	Laghu, Ruksha	U	K	Kapha, Pitta, Vatavardhak
Shunthi	Katu	Laghu, Snigdha	U	M	Kapha, Vata

14) Yeast:

Yeasts and yeast-like fungi are widely distributed in nature (about 500 species). They are present in orchards and vineyards, in the air, the soil and the intestinal tract of animals.Most yeasts are larger than most bacteria. Like bacteria and moulds, they can have beneficial and non-beneficial effects in foods.Although there is a large diversity of yeasts and yeast-like fungi, only a few are commonly associated with the production of fermented foods. They are all either Ascomycetous yeasts or members of the genus Candida. Varieties of the Saccharomyces cerevisiae genus are the most common yeasts in fermented foods and beverages based on fruit and vegetables. All strains of this genus ferment glucose and many ferment other plant derived carbohydrates such as sucrose, maltose and raffinose. In the tropics, Saccharomyces pombe is the dominant yeast in the production of traditional fermented beverages, especially those derived from maize and millet (Adams and Moss, 1995).Yeast is an single-celled fungi belonging to the vegetable family, which reproduces asexually by budding or division. The yeast doesn't itself take part in the fermentation process,but it secretes a complex set of enzymes that act upon the sugars and convert it to alcohol and carbon dioxide gas. The genus Saccharomyces is the one most commonly used due to its efficient alcohol production and tolerance of high alcohol levels. The bakers' yeast available at grocers shop may also be used but, the alcohol yield associated with the brewers yeast will not be available as the bakers' yeast is not bred for alcohol tolerance. Yeasts can break down simple sugars only (monosaccharide) into CO_2 and ethanol. So, enzymes that break down starch and disaccharides (sucrose = glucose + fructose is most common sugar in plants) must be added before adding yeast except for honey and some fruit juices.

02.22 HARITAKI

Botanical Name: *Terminalia Chebula*

Natural order: Combretaceae

Classification Charaka : Jvaraghna, Kushtaghna, Kasaghna, Arshoghna, Prajasthapana

Sushruta : Triphala, Amalakyadi, Parushakadi

Classical Names :

Haritaki, Abhaya, Pathya, Kayastha, Putana, Chetaki, Shiva, Rohini

Vernacular name :

Eng: Chebulic myrobalan Hindi : Hara, Harad Guj : Hardo Marathi -Hirada

Parts used: Fruit

Chemical Constituents:

Anthraquinone glycoside, Chebulinic acid, Chebulagic acid, Tannic acid, Terchebulin, Tetrachebulin, Vitamin C, Arachidic, Behenic, Linoleic, Oleic, Palmitic & Stearic acids (fruit kernels), Chebulin (flowers), 2 α hydroxymicromeric acid, maslinic acid & 2 α hydroxyursolic acid (leaves) Gallic acid & Syringic acid isolated from fruit.Bulletin of Med. Ethno bot-Ris V-10-1989 Tannin containing plants are having carcinogenic and also Metagenic effect. Advances in Plant Sci. V-1991 Punicalgin, Punicallin, Terchebulic acid, Flavogallonic acid, Gallic acid esters. Extract inhibit ATPase activity in Cardiac muscle of frog on dose dependant manner. Conf. of Pharmacy & Symph. on herb drugs Mar-1991.

Research Works:

On oral administraion T. Chebula increases gastric emptying time & can serves as an useful alternative to prokinetic drugs available today.Dahanukar Thorat et al Dept of Pharmac; G.S.college, Mumbai.Gallic acid & Chebulagic acid from T. Chebula fruit inhibits cytotoxic T lhymphocyte - mediated cytotoxicity with 50 to 30 micro m. Hamada 1997

A crude extract of T. Chebula is reported to have potent & broad spectrum Antibacterial activity against human pathogenic gram +ve & gram -ve bacteria (Phadake & Kulkarni et al 1989)

Cancer: A Tannin fraction from the dried fruit pulp of T. chebula is reported to have Antimutagenic activity in vitro (kaur et al. 1998). A methanol extract of T. Chebula is reported to have a potential for inhibiting growth of Leukemia cells attributed to Aarjunglucoside- I & Arjunegenin [Crenncia et al 1996]

Antibacterial effect was found to be stable over wide range of temp. Geobios V-26-1999 A new Triterpine, 2 α hydroxy micromeric acid, Mastinic acid, 2 α hydroxyursolic acid have been isolated from T. Chebula. Phytochem. V-29-1990 Extract shows Cardiotonic activity and increases tone of contraction & cardiac output without altering heart rate. Steriod Sapogenins, Saponins, Anthraquinone derivative & Tannis were defected in the extract. Fitoterapia V-61-1990 Gallic acid & Syringic acid isolated from fruit. Bulletin of Med. Ethno bot-Ris V-10-1989 Tannin containing plants are having carcinogenic and also Metagenic effect. Advances in Plant Sci. V-1991Punicalgin, Punicallin, Terchebulic acid, Flavogallonic acid, Gallic acid esters. Extract inhibit ATPase activity in Cardiac muscle of frog on dose dependant manner. Conf. of Pharmacy & Symph. on herb drugs Mar-1991. Oral Administration of female albino rats in 580 mg/kg from 1st to 5thday of pregnancy, exhibit anti-implantation activity with 61% foetal loss.It has strong effect against herpes simplex virus HSV, HIV , Antibacterial activity & exhibits strong Cardiotonic properties. It also has Antioxidant components, which indicates it can increase the life of tissues (www.sssbiotic.com) It is mild purgative due to glycoside present in it. It renders irregular peristalsis movement uniformly progressive. (www.gorkhaexim.com) Japanese study shows that it is of potential value in treating AIDS,Herpes & Acyclovir resistant herpes. A korean study shows that an extract of the fruits is more Antioxidant than either BHA/BHT two strong Antioxidants long used as food preservatives & now called as "Anti-aging' supplements. http://www.healthymagnets.com

C.V.S.: T. Chebula significantly reduces serum cholesterol, aortic Sudanophilia & the cholesterol contents of liver & aorta in Cholesterol fed rabbits.(Thakar et al 1988)

Antifungal : A water extract of T. Chebula was found to have an Antifungal activity. [Dutta et. al. 1998]

Antiviral : A hot water extract of T. chebula inhibites replication of cytomegalovirus & murine cytomegalovirus in vitro & in vivo in the immune suppressed cyclosproine treated mice (Yukawa et al. 1996). It showed a significant inhibitary activity on HIV 1 reverse transcriptase.(Cl. Mekkaway et. al 1995)

Digestive : T. chebula is reported to improve the secretory status of brunner's gland invovled in the protection against duodenal ulcer. [Nadar & Pillai 1989]T. Chebula has been found to possess Cardiotonic and hypocholesterolemic effect. Ind. Journal of nutrition & Dietetics V 38(53) P. 83-88, 2000

Plate13: Haritaki

02.23 KANTAKARI

Botanical Name: *Solanum Xanthocarpum*

Natural Order: Solanaceae

Classification: Charaka - Kasahara, Kanthya, Hikkanigrahana Shothahara, Sheeta prashamana, Angamarda prashamana

Classical Name: Kantakari, Dushparsha, Kshudra, Vyaghri, Nidigdhika

Vernacular Name : Eng - Yellow berried night shade Hindi - Choti Kateri, Bhat Kateriya

Chemical Constituents:

A gluco alkaloid termed Solanocarpine is found in the fruits. Solanocarpidine & Sterol known as Carpesterol are also present, Potassium nitrate, Fatty acid, Resinous & Phenolic substance, Diosgenin & Sitosterol re present. http//www.modern-natural.com

Pharmacological Action:

The crude drug extract causes transient Hypotensive effect, which is partly inhibited by Atropine. The gluco alkoloid Saponin & Resin fraction increases the force of contraction of isolated frog's heart & causes gradual rise in blood pressure levels. The Alcoholic leaf extract causes contraction of dog tracheal chain while the glucoalkaloid & alcoholic stem extract after initial potentiation causes refractoriness to constrictor responses of Acetylcholine & Histamine. Histamine releasing effects have been shown.http://www.modern-natural.com

A significant increase in cardiac muscle contraction with methanol extract whereas significant decrease in contractility was caused by petroleum ether & it also causes lowering of temprature showing Antipyretic activity.Bangladesh journal of Biohem. Vol. 2 No. 1 69-74, Dec. 1997.Leaves contains Solasodine It is an important Phytosterol alkaloid which is versatile substitute of Disogenin for different steroid.Ind. drug Aug. 1995. Solasodine - Steroidal alkaloid causes decrease in the motility of human & bovine spermatozoa as evident from reduction in motile sperm count in dose & duration dependant manner. It also inhibit activity of glucose 6 phosphate,fructose 6, Diphosphatase, glycogen phosphoxyelose, glucose 6 phosphateisomerase & Amylase in spermatozoal homogenates.Joun. of Pharmacy

V 28 - 1990Alcoholic extract of Xanthocarpus seeds on adult male at the dose of 20,60, 100 mg/kg body wt/ day for 30 & 60 day shows -Cauda epididymal sperm count & motitily reduces extract manifest spermicidal activity on rat epididymal spermotozoa. The probable Androgen deprivation effect of extract is explained by decreased level of circulating Testosterone level, seminal vesicle fructose, prostatic acid, hosphatase and elavated cholesterol in rats. Ind. Journal of Expt Bio V. 26 – 1988.

Plate 14: KANTAKARI- ***Solanum Xanthocarpum***

02.24 GUDUCHI

Botanical name: *Tinospora cordifolia Miers*.

Family: Menispermaceae

Classical names: Amrita, Amritavallri, Madhuparni, Chhina, Guduchi Chhinnaruha, Vatsadani, jeevanti, Chakalakshani, Rasayani, Tantrika,

Rasa panchaka:

Rasa : Tikta, Kasaya.

Guna : Guru, snigdha.

Veerya : Ushna.

Vipaka : Madhura.

Doshghanta : Tridosasamaka.

Part of used: Stems.

Chemical constituent:

Tinosporin and a furanoid diterpene dilactone identical withcolumbin, have been isolated (CSMDBIA, M). The other constituents reportedfrom stem are: tinosporide, cordifolide andunosporin, tinosporin, tinosporic acid and tinosporol, heptacosanol, cordifol, B-sitosterol andtinosporidine, tinosporide, octacosanol and a crystalline compound (C13 H16O5)6 and a newditerpenoid furanolactone. The quaternary alkaloids, magnoflorine and tembetarine have been indentified. A new hypoglycemic agent was isolated and it was found to be 1,2 - substituted pyrrolidine. A new phenolic lignan 3 - (x, 4 - dihydroxy -3- methoxy benzyl) - 4- (4-hydroxy - 3- ethoxybenzyl0 –etrahydrofuran alongwith octacosanol, nonacosan - 15- one and p-sitosterol were isolated.

Roghnata: Vishamajvara, Kamala, Vami, Vatrakta, Arsha, Vatajvara, Prameha,Kushta, Pandu Shvasa Kasa Prameha, Raktavikara, Agnimandhya,

Karma: Kaphaghna, Balya Vrisya, Dipana Pachan Sangrahi, Visaghan, Anulomana, Pittasaraka Bhutaghna .Hridya, Rasayana.

Action and uses: The stem is bitter, anti spasmodic, anti inflammatory, expectorant,

tonic, appetizer, digestive, diuretic, potent aphrodisiac. Useful in skin

Infections, jaundice, diabetes, chronic diarrhoea and dysentery, heart disease,Hypertension, leprosy helminthiasis rheumatoid arthritis.

Pharmacological activities:

Anti inflammatory, Anti bacterial, Anti allergic, Anti stress, Anti oxidant, immune - stimulant & analgesic. Hepato-protective, Anti diabitic,Anti tumour, Anti endocrine.

Plate15: GUDUCHI -*Tinospora cordifolia Miers*.

02.25 MARICHA

Botanical name: *Piper nigrum Linn.*

Family: Piperacea

Classification: Deepanaiya, Krimighna, Shiro-virechana, Shoola prashamana Shoolprashamana, Shirovirechana[257]Pipplyadi, Trayushana [258]Haritakyadi[259] Pipplyadi Varga[260]

Classical name: Vellaja, Krishna, Usana, Dharmapatana.[261]

Rasa panchaka:

Rasa: Katu.

Guna : Laghu, Tiksna.

Veerya : Ushna.

Vipaka : Katu.

Doshghanta : Vatakaphasamaka

Part of used: Fruit

Chemical Constituent:

Analysis of black pepper(dried) gave following ranges of values:moisture8.7-14.1, total nitrogen 1.55-2.60,nitrogen in non volatile ether extract 0.7-4.22, volatile ether extract 0.3-4.2,non volatile extract 3.9-11.5,alcohol extract 4.4-12.0, starch (by acid hydrolysis) 28.0-49.0,crude fibre 8.7-18.0,crude piperine 2.8-9.0,piperine (spectrometrically)1.7-7.4,total ash 3.6-5.7, and acid insol. Ash(sand)0.03-0.55.fruits mainly contain piperine 5-10%,piperdine 5%,piperttine and chavicine. Fruits also yield oil of pepper.

Rogaghnata: Kapha-Vata janya Vikara, Ajirna, Yakritavikara. Agni andhya Pratishya Kasa, Shvasa, Hikka, Shoola Krimi, Hdridaurbaliya.

Karma: Vata-Kaphashamaka, Lekhana, Deepana, Pachana, Srotoshodhana, Kaphanissarka Srothosodhana Jwaraghna.**Action and uses:**

The fruits acrid bitter, carminative, digestive, Asthma, fever, cough,catarrhal, hoarseness. They are useful in throat -trouble colic pain Hiccough,Cholera.

Pharmacological activities: Anti oxidant, Analgesic, Muscle relaxant, Antipyritic, Anti inflammatory, Anti bacterial, Cychlo oxygenosee inhibitory activity.

Plate 16: MARICHA- ***Piper nigrum Linn***

02.26 Madhu (Honey)

Rasa panchaka –

Rasa - Madhura, Kashaya

Guna - Laghu, Ruksha, Pichchhila

Virya - Shita

Vipaka - Katu

Doshaghnata –Tridoshahara[262] Kapha-Pittahara[263]

Karma - Deepana, Lekhana, Yogavahi, Balya, Brihana, Hridya, Ropana, Sangrahi, Chakshushya, Prasadana, Medoghna

Karma - Hikka, Shwasa, Kasa, Atisara, Chhardi, Trishna, Krimi, Vishaghna

Types of Madhu :

The classical classification of Madhu is eight types.

Namely, 1. Pauttika 2. Chhatra 3. Bhramara 4. Ardhya 5. Kshaudra 6. Auddhalaka 7. Makshika 8. Dala[264]

Makshika is best among Madhu varga. It has Laghu guna helps alleviating Netramaya, Arsha, Kamala, Shwasa, Kasa, Kshaya[265]

Chemistry:

It has a characteristic odour and sweet acrid taste, which may very somewhat depend upon the floral source of the product. It is mainly an equimolar of mixture of dextrose and fructose known as invert sugars (50-90%) and water. It also contains 0.1-10 % of sucrose and small quantities of other carbohydrates, volatile oil, pigments and plant parts especially pollen grains. It also contains Vitamin B and C.

Actions / Uses:

Honey is pharmaceutics agent; it possesses nutrient and demulcent properties. It is also used as vehicle similar to syrup, although honey possesses more of laxative action than syrup; it is also used as a pill.

02.27 TALISH PATRA

Botanical Name*: Abies webbiana Lindl.*

Natural order : Pinaceae

Varga: Sushruta : Sirovirechana

Chemical Constituents: A biflavonoid, abiesin, n-triacontanol, beta-sitosterol and betuloside are present in the leaves. The essential oil from leaves contains alpha-pinene,l-limonene, deltacarene, dipentene, l-bornyl acetate and l-cardinene as major constituents.

Toxicology: Higher dose of drug caused tachycardiac arrhythmia, nausea, dizziness, diffuse abdominal pain, unconciousness, weak breathing, brief ventricular flutter and slow pulse.vi

Pharmacological action: Expectorant vii, bronchial sedative, decongestant,anticatarrhal, antiseptic, carminative. The leaves are astringent, carminative, stomachic and tonic. These are used in the treatment of asthma, bronchitis etc.i viii

Reseach works:

1) Plant saponin has been shown to protect sensitized mast cells from degranulation on antigen shock thus confirming the immunosuppressive and memb. Stabilizing effect like sod. Chromoglycate. (Modern clinical research)

2) The methanol extract of A. webbiana Lindl was evaluated for its effect on a cough model induced by sulphur dioxide gas in mice. When administered orally it exhibited significant antitussive activity compared with the control in a dose dependent manner. ii

3) The methanol extract of the dried leaves of Abies webbiana was evaluated for antimicrobial activity. The methanol extract showed a broad spectrum of antibacterial activity.iii

4) The antiinflammatory effect was established in preliminary studies. Fractional extraction of the leaves indicated uneven predominance of flavonoids, glycosides, terpenoids and steroids. An antiinflammatory effect in the 5 h carrageenin oedema model was exhibited by all fractions, indicating multiple active principles.

5) The methanol extract of the dried leaves of Abies webbiana was evaluated for antimicrobial activity. The methanol extract showed a broad spectrum of antibacterial activity.

Plate 17-Taalispatra

02.28 Guda

Synonyms : Matsyandika (in Gouda desa)

Vernacular names : Hindi : Guda Gujarathi : God Kannada : Bella English : Treacle

Rasapancaka:

Rasa : Madhura.

Guna : Guru

Virya : Sita.

Vipaka : Madhura.

Doshakarma : Vata and Pitta hara.

Nava Guda : Kapha-, Krimi- kara and Agnikara.

Purana Guda : Anabhisyandi, Pathya, Agnipusthikara,Vrishya, Asrik prasadana.

Chemical constituents:

Jaggery is rich in minerals, iron and instant glucose. It is not only easily digestible but has various minerals and vitamins in right proportion, which is extremely useful for our body. Unlike white sugar which consists almost entirely of sucrose, Jaggery contains some proteins, fat, minerals and vitamins, and hence considered to be more nutritious and moreover free of many of the detrimental chemicals which the sugar has to bear and possess in order to get its fine color and odor. Generally, good quality Jaggery has a light color, good flavor, hardness, crystalline structure and good keeping quality. It is reported that a part of the tannins present in the juice reacts with iron during crushing and boiling, and imparts a dark color to the gur, and that the remaining portion reacts with the iron present in the gur during storage and intensifies the color of the stored product (Wealth of India).

Table No: 13 Nutrient Content of jaggery (per 100 g) (Source: Wealth of India)

Sl no	Ingredients	Percentage
1	Moisture	3.80 g
2	Protein	0.40 g
3	Carbohydrate	95.00 g
4	Phosphorous	40.20 mg
5	Total Minerals	0.60 g
6	Thiamine	0.02 mg
7	Vitamin C	0.50 mg
8	Fat	0.10 g
9	Calcium	80.20 mg
10	Iron	11.4 mg
11	Carotene	168 mcg
12	Riboflavin :	0.05 mg
13	Energy :	183 k. calories

Pharmacological Characters of Guda:

Identification of fermenting organism in Jaggery revealed that Bacillus Sp. was present in both the new and old jaggery. Among the Bacillus Sp. B. acetoethylicus and B. Polymyxa are reported to bring about alcohol production (Prescott and Runn, 1959). Old and new jaggery yield almost equal percentage of alcohol.

Guda helps in translocation of the particles ingested dust particles from the lungs to the tracheobroncial lymph nodes– A.P.Sahu and A.K.Saxena, Environ Health Perspect 102(Suppl 6):211-214 (1994).

03 MATERIALS AND METHODS

MATERIALS

Trial drug

The present study comprises of both shodhana and shaman treatments. For Shodhana Virechana treatment is given, starting with shodhananga snehapana with Kanthakari ghrita[389] as a poorvakarma after confirming niramavastha and for Virechana Trivrit lehya[390] was used. For Shamana Chikitsa Shirisharishta[391]and Bhrigu haritaki[392] yogas were administered.

Table No.155: List of drugs used for clinical trial

Sl no	Oushadhi yoga.	Purpose
1	shirisharishta	Shamanachikitsa
2	Bhrigu haritaki	Shamanachikitsa

Table No.156: List of drugs used for virechana

Sl no	Oushadhi yoga.	Purpose
1	Kanthakari ghrita.	Shodhananga snehapana
2	Moorchita tila taila	Sarvanga abhyanga
3	Trivrit lehya	Virechanartha

Most of the raw drugs were procured from western ghat and few were purchased from the market. Drugs authentification was done with the help of Botanist. Kantakarigrita, Bhriguharitaki were prepared. Where as Trivritlehya was purchased from Aryavaidyashala Kottakkal pharmacy and Shirisharishta was prepared in Swadeshi Oushadha Bhandar, Udupi, Karnataka

3.1 METHOD OF PREPERATION OF KANTHAKARI GHRITA

Table No.157: **Preparation of Kanthkari ghrita**

Sl .No	**Ingredients**	**Part**	**Quantity**
1	Kantakari (Solanum xanthocarpum)	Panchanga	
2	Guduchi (Tinospora cadifolia).	Kanda , patra .	
3	Ghrita	----------	

The raw drugs were collected from nearby natural habitat, and authentification was done with the help of a botanist. Cleaned, dries and properly weighed ingredients were used to prepare kantakari gritha. Kantakari gritha was prepared with the help of bhaishajya kalpana department. Gritha was stored in a well-protected airtight container.

3.2 Preparation of shireesharishta.

Table No.158: Ingredients of Shireesharishta.

Sl .No	**Ingredients**	**Part**	**Quantity**
1	Shirisha (Albizzia lebbeck)	twak	Tulardha
2	Pippali (Piper longum)	Phala	1 pala
3	Priyangu (Callicarpa macrophylla)	Beeja ,pushpa	1 pala
4	Kushta (Saussurea lappa)	Moola.	1 pala
5	Ela (Elettaria cardamomum)	Beeja	1 pala
6	Nilini (Indigofera tinctoria)	Patra, moola	1 pala
7	Nagakeshara (Mesua ferrea)	Pushpa kesara	1 pala
8	Haridra (Curcuma longa)	Kanda	1 pala
9	Daruharidra (Berberis aristata)	Kanda twak.	1 pala
10	Shunthi (Zingiber officinale)	Kanda	1 pala

After proper authetification the above said ingredients were given to Swadeshi Aushad Bahandar,Udupi, Karnataka. Sirisha was prepared in accordance with Sandhana Kalpana mentioned in the classics.

03.3 METHOD OF PREPERATION OF BHRIGU HARITAKI

Table No.159: Preparation of Bhriguharitaki

Sl .No	Ingredients	Part	Quantity
1	Kantakari (Solanum xanthocarpum)	Panchanga	100 pala.
2	Haritaki (Terninalia chebula)	Phala	100 numbers.
3	Shunthi (zingiber officinale)	Kanda	04 tola.
4	Mareecha (Piper nigrum)	Phala	04 tola.
5	Pippali (Piper longum)	Phala	04 tola.
6	Ela (Elettaria cardamomum)	Phala	04 tola.
7	Twak (Cinnamomum zeylanicum)	Kanda twak	04 tola.
8	Nagakeshara (Mesua ferrea)	Pushpa kesara	04 tola.
9	Talisha patra (Abies webbiana)	Patra	04 tola.
10	Madhu		24 tola.
11	Guda.		400 tola.
12	Water		1024 tola

The raw drugs were collected from nearby natural habitat, and authentification was done. After proper authetification the above said ingredients were given to Baishajya Kalpana department. Bhriguharitaki avaleha was prepared in accordance with avaleha kalpana mentioned in the classics.

03.4 Research Approach.

Research is ongoing process. Since its evolution, newer invention replaces the older one. Every day one has to think new approach. Hence, in the present study the objective was to see the effect of shirisharishta1and Bhrigu haritaki[2]in the management of pulmonary hypersensitivity when used in combination over when used single.

03.5 Research Design.

The present study is a randomized single blind clinical trial on pulmonary hypersensitivities with pretest & posttest design. 150 patients were selected for present study and received treatment in the following manner.

Table No.160: Research Design

Duration	Group A	Group B	Group C
7 to 10 days.	**Arohana karma snehapana with Kanthakari ghrita Virechana with trivrit lehya.**	**Arohana karma snehapana with Kanthakari ghrita Virechana with trivrit lehya.**	**Arohana karma snehapana with Kanthakari ghrita Virechana with trivrit lehya.**
30 days after (samsarjana karma)	**Shirisharishta** was given in the dosage of 20ml three times in a day with ushnajala as anupan after food.	**Bhriguharitaki Leha** was given in the dosage of 10ml three times in a day with Sukhoshna dugdha as anupan before food.	**Shirisharishta** was given in the dosage of 20ml three times in a day with ushnajala as anupan after food. **Bhriguharitaki Leha** was given in the dosage of 10ml three times in a day with Sukhoshna dugdha as anupan before food.

03.6 SETTINGS

The Investigator selected the Out Patient Department of Kayacikitsā, Bharati Vidyapeeth University's & KLEs Āyurveda Hospital, Pune/Belgaum as the setting for the study.

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other.

03.7 STUDY POPULATION

Patients diagnosed as pulmonary hypersensitivities with underlined parameters attending the O.P.D. of Āyurveda Hospital, hailing from city and nearby districts were included in the study. A total 150 patients of clinically and in Laboratory well established pulmonary hypersensitivities of either sex were considered through well-organized selection from O.P.D. / I.P.D., clinical referrals and through village camps.

03.8 Sample

The sample for the present study was selected from the population consisting of adult patients of either sex with features of pulmonary hypersensitivities satisfying the Inclusion criteria.

03.9 HYPOTHESIS

Null Hypothesis

The Shamana (Shirisharishta and Bhriguharitaki) therapies do not have any role in the management of pulmonary hypersensitivities.

Alternative Hypothesis

The Shamana (Shirisharishta and Bhriguharitaki) therapies do have a significant role in the management of pulmonary hypersensitivities.

Ho –There is no difference in means of readings in group A, group B and group C.

H1- The mean of readings of at least one group is different from that of other.

Reasons for selecting the research design

Always research needs well-planned and appropriate design and is necessary for conducting a scientific trial. Investigator here has selected 150 subjects for study. To meet the objective and to conclude the efficacy of Shamana (Shirisharishta and Bhriguharitaki) therapy, this type of design was opted.

We live in a world full of microorganisms. Every facet of our existence exposes us to bacteria, viruses, fungi & numerous parasites. We have a natural micro flora on all over the body, within all orifices & throughout the GI tract. The respiratory tract while performing its physiological function is exposed to a wide variety of air-borne environmental antigens. The lungs are working as filter for the entire circulating blood volume. Thus, these are constantly exposed to various blood & air-borne agents that possess potential to accelerate inflammation, infection or immune processes. pulmonary hypersensitivities can be named as Atopic disease. Atopy is defined as familial tendency to sensitization to environmental allergens. Atopic allergy is a type 1 hypersensitivity reaction that produces IgE antibodies to allergens viz. pollen, dust, etc. pulmonary hypersensitivities has always been proved to be a problematic ailment to the doctors. The magnitude of the condition can be understood by the fact that, though it is known from the ancient era & inspite of worldwide efforts to combat this impediment, still there is no definite solution for the problem. There is only symptomatic treatment in modern medicine & so many measures have been adopted to check this disease.

There are many approaches for the management of pulmonary hypersensitivities. However, most of them are symptomatic relievers. As the prevalence of pulmonary hypersensitivities is increasing day by day due to globalization, there was a need of some permanent solution. This was the basic assumption hence to test this assumption present design was adopted.

All the 150 patients received 30 days therapy, where in Investigator idea is was to assess the results properly. Any duration less than this would not have been sufficient to draw even a tentative conclusion, as pulmonary hypersensitivities being a Cirakāri Vyādhi and episodic in nature which needs constant therapy. It requires a long-standing therapy, but Investigator knowing the time limitation of the present study has restricted to 30 days only.

Investigator aimed at small of150 patients because; in such a small duration as well as small Investment project, it will not be possible to include excess number of study subjects. All the 150 patients were followed for six months to observe the sustenance of the therapy even when the therapy is stopped.

Selection Criteria

03.10 INCLUSION CRITERIA

Patients from both sexes in between 16 to 60 yrs age group presenting with following clinical and laboratory findings were selected for present clinical study.

1. Kasa with or without Kapha nishtheevan. (Cough)
2. Shwaskrichrata (Dyspnoea)
3. Nasa srava (Running nose)
4. Nasa avarodha (Nose block)
5. Kshavathu (Sneezing)
6. History of Atopy.
7. Nasavarodha

03.11 EXCLUSION CEITERIA

1. Patients below the age group of 16yrs and above 60 yrs
2. Pulmonary hypersensitivities with other systemic diseases like Diabetes Malites Hypertension, IHD etc.
3. Pulmonary hypersensitivities with other chronic respiratory diseases like Tuberculosis, Bronchiectitis, Emphysema, COPD etc.
4. Pulmonary hypersensitivities in pregnant and lactating woman.

03.12 LABORATORY INVESTIGATIONS

Table No.161: LABORATORY INVESTIGATIONS

S l no	Sample	Name of investigation	Before treatment	After treatment
1	Blood Examinations	CBC	✓	✓
		AEC	✓	✓
		Ser. Total IgE	✓	✓
2	Sputum Examination **If required**	AFB	**If required**	**If required**
		Cytology	**If required**	**If required**
		Culture & sensitivity.	**If required**	**If required**
3	Chest 'X' ray - **If required**		**If required**	**If required**

03.13 Time and Duration of the study

Total duration of study was 36 months.

Study duration of each case was after **virechan & Samsarjana Krama** 30 days + 6months follow up.

03.14 INTERVENTION / TREATMENT

After obtaining the written informed consent, the selected patients were assigned into **three groups.** i.e. **Group A, Group B, Group C.** of **50 patients** each.
All patients of these three groups were given Arohanakrama Snehapana with Kantakarighritha after confirming the niramavastha, and Is continued till the appearance of samyaksnigdha lakshanas. Duiring vishramakala sarvanga abhyanga was done with moorchita tilataila and bhashpasweda was given for three days. Virechana was administerd with Trivrit lehya. After assessing the type of shuddhi samsarjanakrama was fixed.

After Virechana,

For Group a patient – Shirisharishta was given in the dosage of 20ml three times in a day with ushnajala as anupan after food.

For Group B patient – Bhriguharitaki Leha was given in the dosage of 10ml three times in a day with Sukhoshna dugdha as anupan before food.

For Group C patient- Shirisharishta was given in the dosage of 20ml three times in a day with ushnajala as anupan after food. **Bhriguharitaki Leha** was given in the dosage of 10ml three times in a day with Sukhoshna dugdha as anupan before food.

DURATION: After **virechan & Samsarjana Krama** 30 days.

FOLLOW UPS

Patients were followed for every 7^{th} day during medication & every 15^{th} day after medication for 6 months.

ASSESSMENT PARAMETERS

03.16 Assessment Criteria

In the present Clinical study all the cases of pulmonary hypersensitivities were assessed with the specific Subjective and Objective parameters on every 7^{th} day during medication & every 15^{th} day after medication for 6 months.

Subjective parameters

Shwaskrichrata, Shiraha shoola, Shirogouravata, Nasa srava, Kasa, Nasavarodha, Kshavathu, Nasa kandu

These Lakṣaṇa were assessed with the help of following Clinical Grading.

Table No.162: Investigations – Blood Examinations

Blood Examinations	CBC
	AEC
	Ser. Total IgE

Subjective & Objective Parameters were assessed by giving grading to them before and after treatment and during follow up period and statistical analysis was done by calculating 'P' value t test & ANOVA

1. Nasavarodha (Nasal obstruction)

Table No.163: Nasavarodha Grading

Sl no	Grading	
1	**0** – ABSENT	No obstruction
2	**1** – Mild	Inhalation & exhalation with effort with feeling of mild obstruction
3	**2** – Moderate	Inhalation & exhalation with effort with feeling of moderate obstruction Inhalation & exhalation to be supplemented with mouth breathing
4	**3** – Severe	Inhalation & exhalation with effort with feeling of severe obstruction Inhalation & exhalation to be supplemented with mouth breathing
5	**4** - Very severe	Complete blockage with total mouth breathing

2. Nasa srava.

Table No.164: Nasa Srava Grading

Sl no	Grading	
1	**0** – ABSENT	No discharge
2	**1** – Mild	Occasional Nasa srava. with a feeling of running nose without visible fluid
3	**2** – Moderate	Nasa srava with occasional running nose with visible fluid
4	**3** – Severe	Nasa srava with running nose which needs moping but controllable
5	**4** - Very severe	Severe Nasa srava with copious fluid needs continuously moped

3. Kshavathu (sneezing)

Table No.165: Kshavathu Grading

Sl no	Grading	
1	**0** – ABSENT	No sneezing
2	**1** – Mild	1 – 10 sneezing in entire day.
3	**2** – Moderate	10 – 15 sneezing in entire day.
4	**3** – Severe	15 – 20 sneezing in entire day.
5	**4** - Very severe	> 20 sneezing in entire day.

4. Shirah shoola (Head ache).

Table No.166: Shirah Shoola Grading

Sl no	Grading	
1	**0** – ABSENT	No headache
2	**1** – Mild	Mild headache
3	**2** – Moderate	Moderate headache
4	**3** – Severe	Severe headache patient restless & able to carry routine work with great difficulty
5	**4** - Very severe	Severe cripping, headache which renders patient bed ridden

5. **Kasa (Cough)**

Table No.167 Kasa Grading

Sl no	Grading	
1	**0** – ABSENT	No cough.
2	**1** – Mild	Occasional cough.
3	**2** – Moderate	Moderate Cough .
4	**3** – Severe	Continuous cough with throat & chest pain .
5	**4** - Very severe	Severe continuous cough with throat & chest pain.

6. **Nasa kandu (nasal itching)**

Table No.168: NASA Kandu Grading

Sl no	Grading	
1	**0** – ABSENT	No Nasa kandu.
2	**1** – Mild	Mild/not clear.
3	**2** – Moderate	Moderate /slightly understable.
4	**3** – Severe	Unable to speak /cannot produce any sound.
5	**4** - Very severe	Severe and continuous Nasa kandu with throat & chest pain.

Laboratory findings were assessed before and after treatment and statistical analysis was done by calculating 'P' value t test & ANOVA

Before treatment (BT), After virechana (A), After 7^{th} day of treatment (B), After 15^{th} day of treatment (C) , After 21^{st} day of treatment (D), After treatment (AT) , 1^{St} FOLLOW-UP (P), 2^{Nd} FOLLOW-UP (Q), 3^{Rd} FOLLOW-UP (R) & 4^{Th} FOLLOW-UP (S).

Based on above grading the response was assessed as

CURE

Complete reduction in all signs and symptoms.

Serum IgE becomes normal

Esinophil & AbsoluteEsinophil become normal.

SIGNIFICANT IMPROVEMENT

Complete reduction in all signs and symptoms.

Serum IgE nearly normal (less than 200 iu/dl

Esinophil&AbsoluteEsinophil become normal.

IMPROVEMENT

Presence of signs and symptoms in mild degree

Serum IgE nearly normal (less than 200 iu/dl)

Esinophil & AbsoluteEsinophil nearly normal

INSIGNIFICANTLY IMPROOVEMENT

Persistence of signs and symptoms in moderate degree

Serum IgE moderately high (less than 300 iu/dl)

Esinophil & AbsoluteEsinophil moderately high (5-7cells/ dl)

NO IMPROOVEMENT

Persistence of signs and symptoms in moderate to severe degree

Serum IgE high (more than 300 iu/dl)

Esinophil & AbsoluteEsinophil unchanged.

Trial Drugs

The detailed information regarding Trial drugs of both shaman yogas are described in drug review chapter.

Plan for Data Analysis –

The statistical analysis of this study was planned to carry Frequencies, Percentages, Means, Standard Deviation and Error for different parameters. The data are presented in tables and graphs. The statistical significance of the difference between the means of various study parameters were derived using‘t’ test.

04 OBSERVATIONS AND RESULTS

04.1 GENERAL OBSERVATIONS

For the present clinical trial 156 patients were selected incidentally. There were 6 drop outs during the course of clinical study. The study was completed with the remaining 150 patients.

Table No.41

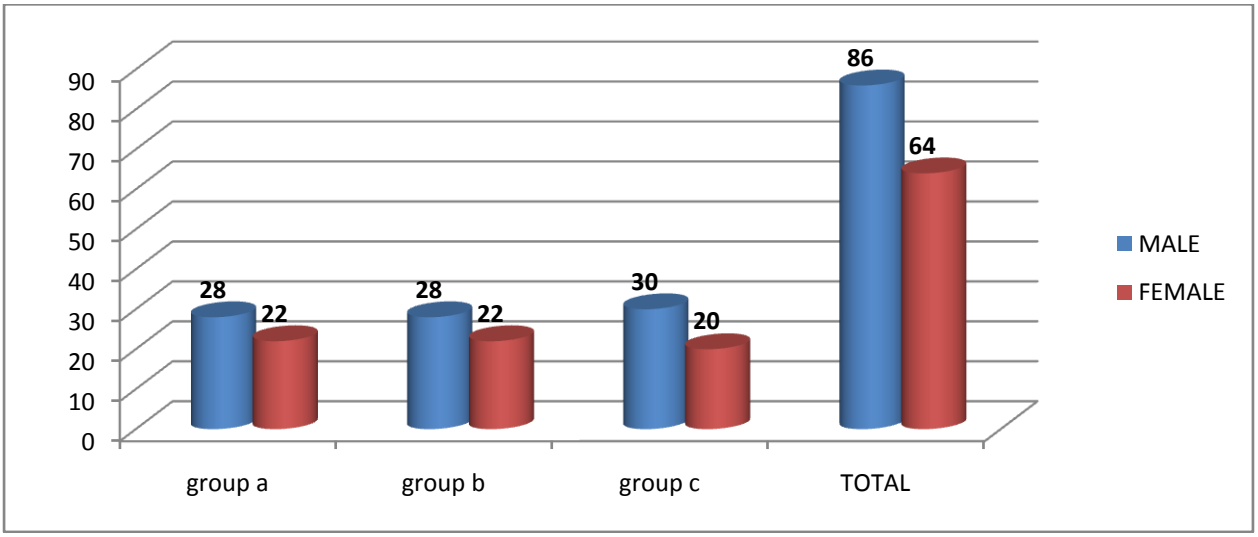
SEX WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY

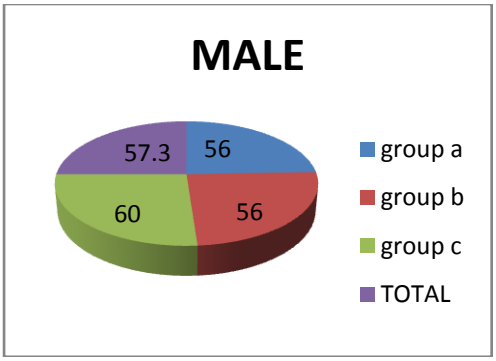
SL.NO	SEX	GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	MALE	28	56	28	56	30	60	86	57.3
2	FEMALE	22	44	22	44	20	40	64	42.7

The selected population comprisedof 86 (57.3%) males and 64 (42.7%) females, who were further divided equally into three groups (each of 50) as follows.
Group A -28 (56%) males and 22 (44%) females
Group B -28 (56%) males and 22 (44%) females
Group C -30 (60%) males and 20 (40%) females

Graph: 1

Sex wise distribution of 150 patients with pulmonary hypersensitivity.





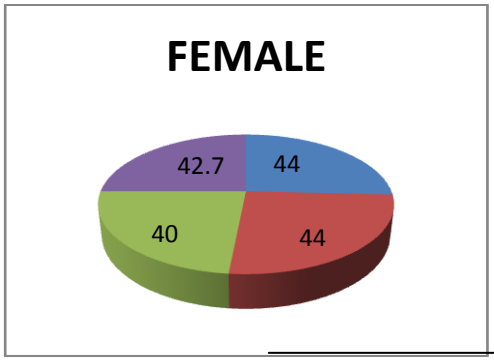


Table No.42

AGE WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO	AGE	GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	16-25	10	20	11	22	10	20	31	20.66
2	26-35	20	40	18	36	19	38	57	38
3	36-45	12	24	16	32	17	34	45	30
4	46-55	06	12	02	04	03	06	11	7.33
5	56-60	02	04	03	06	01	02	06	4

The patients within the age group of 16-60 years were selected for the clinical trial.

Among whom 31 (20.66%) patients were within the age group of 16- 25 years, in which 10(20%) patients were in group A, 11(22%) patients were in group B and 10(20%) patients were in group C.

57 (38 %) patients were within the age group of 26- 35 years, among whom 20(40%) patients were in group A, 18(36%) patients were in group B, and 19(38%) were in group C.

45 (30%) patients were within the age group of 36-45 years, among whom 12(24%) patients were in group a, 16 (32%) patients were in group B and 17(34%) patients were in group C.

11(7.33%) patients were within the age group of 46-55 years, among whom 6(12%) patients were in group A,2 (4%) patients were in group B and 3(6%) patients were in group C.

6(4%) patientswere within the age group of 56-60 years, among whom 2(4%) patients were in group a, 3(6%) patients were in group B and 1(2%) patients were in group C.

Table No.43

RELIGION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO	RELIGION	GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	HINDU	46	92	43	86	47	94	136	90.66
2	MUSLIM	03	6	04	8	01	2	08	5.33
3	CHRISTIAN	01	2	03	6	02	4	06	4

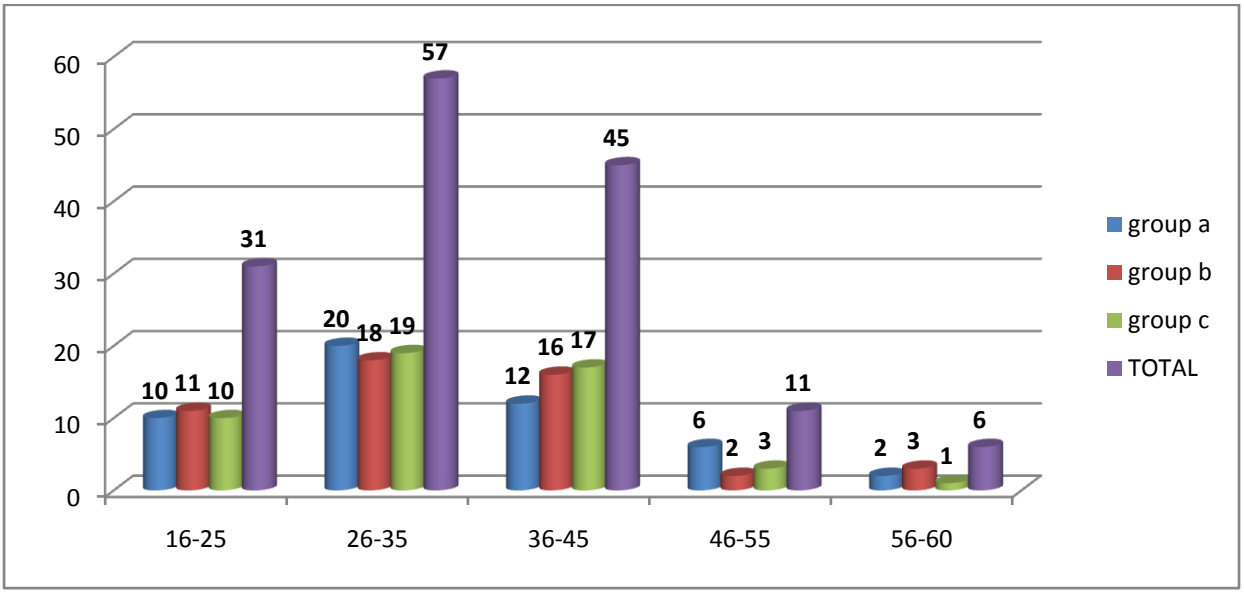
The patients selected for the clinical trial were of different castes.

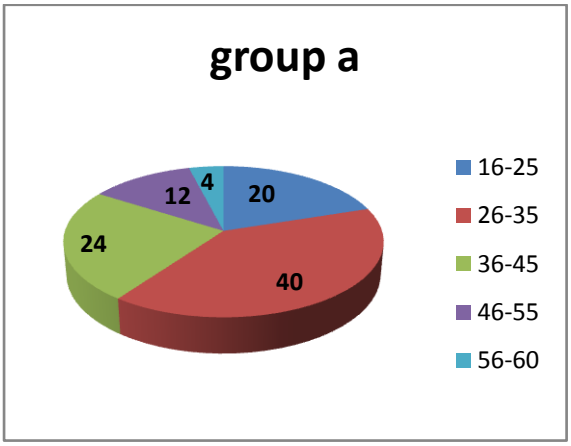
Among them 136 (90.66%) patients were Hindus, inwhom 46(92%) patients were ingroup A, 43(86%) patients were ingroup B, and 4 (94%)patients were ingroup C.

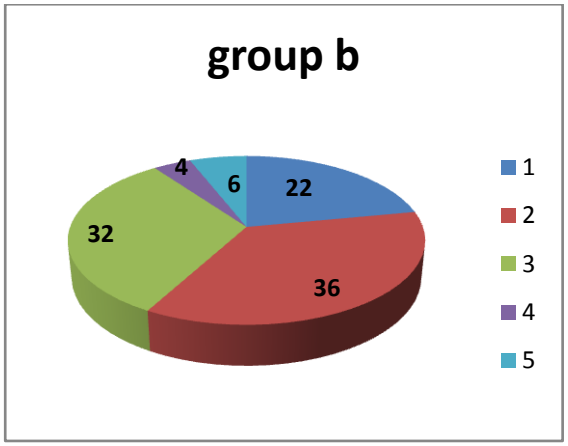
08 (5.33 %) patients were Muslims, among whom, 03(06%)patients were ingroup A, 04(08%) patients were ingroup B, and 01 (02%) patients were ingroup C.

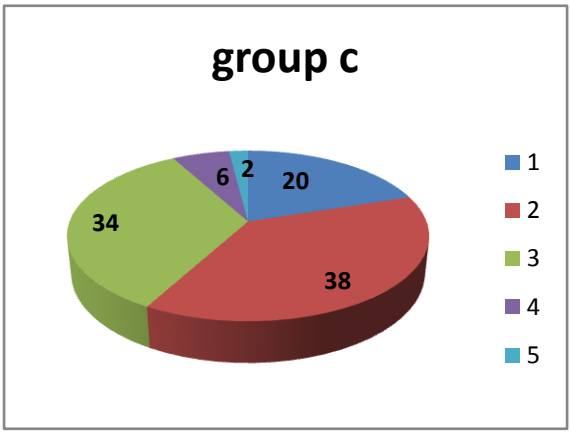
06 (4 %) patients were Christians among whom, 01(02%)patient was ingroup A 03(06%) patients were ingroup B, and 02 (04%)patients were ingroup C.

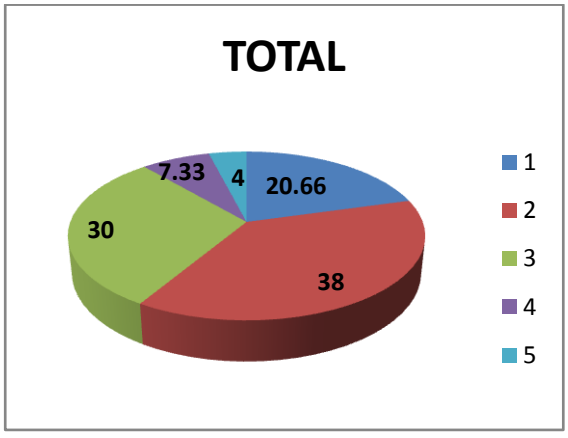
Graph: 2 Age wise distribution of 150 patients with pulmonary hypersensitivity.



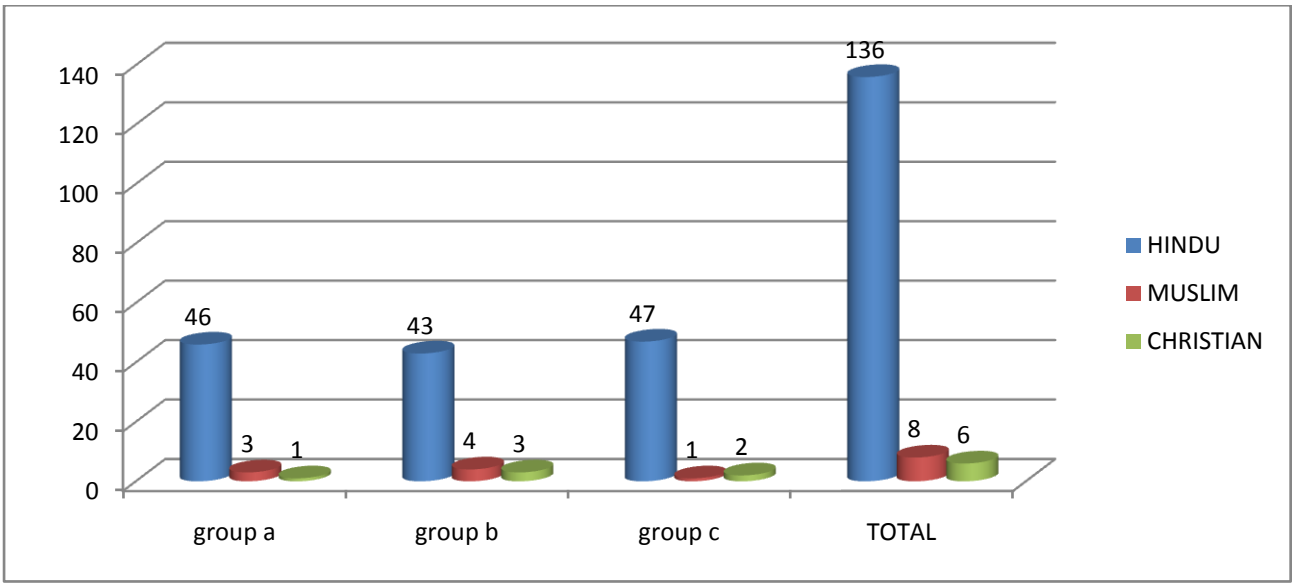


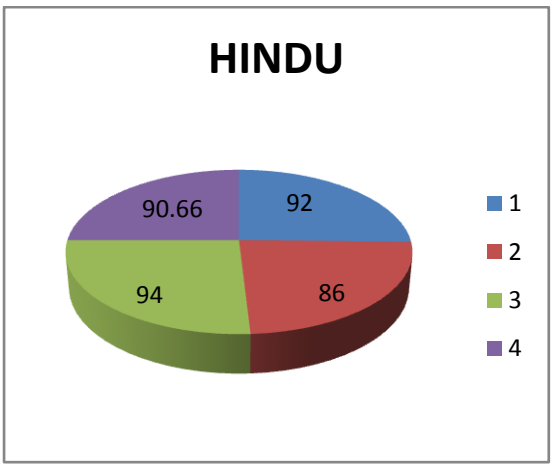


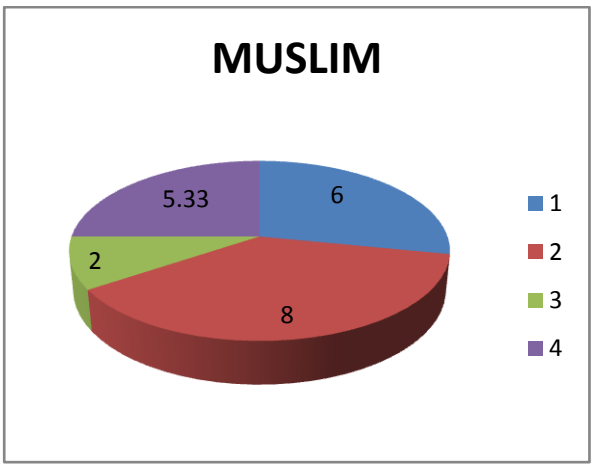




Graph: 3 Religion wise distribution of 150 patients with pulmonary hypersensitivity.







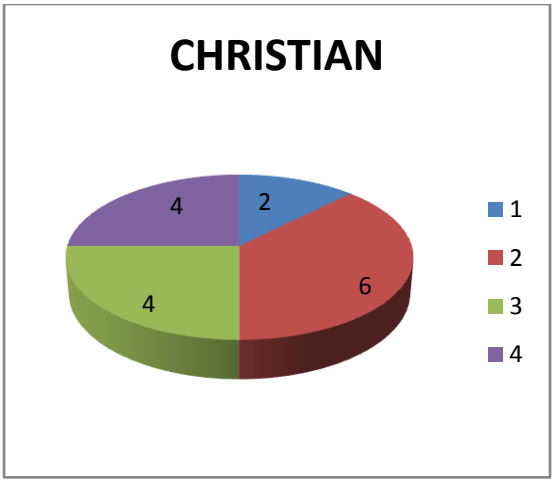


Table No.44

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SOCIO-ECONOMIC STATUS

SL.NO	ECONOMIC STATUS	GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	UPPER MIDDLE CLASS	10	20	18	36	14	28	42	28
2	MIDDLE CLASS	25	50	17	34	26	52	68	45.33
3	LOWER CLASS	15	30	15	30	10	20	40	26.66

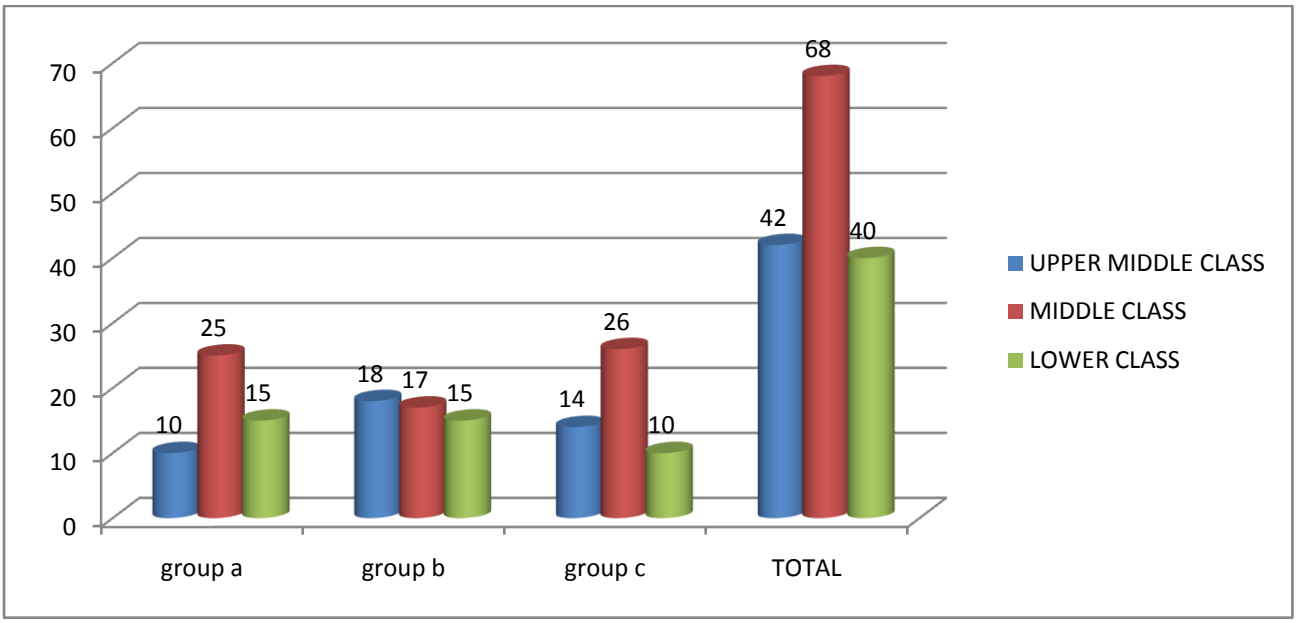
The patients selected for the clinical trial were of different socio-economic status.

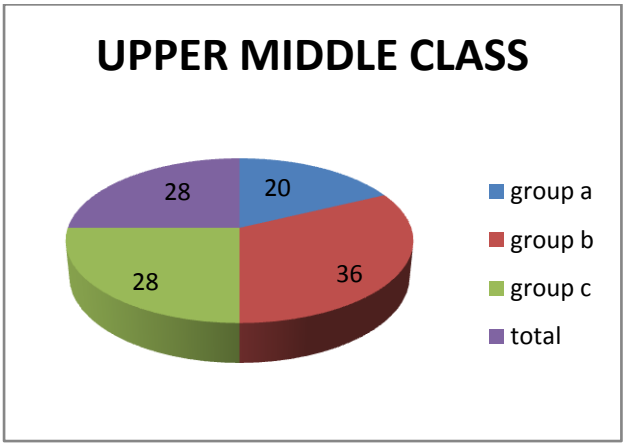
Among them 42 (28%) patients belonged to UPPER MIDDLE CLASS, in whom 10(20%) patients were ingroup A, 18(36%) patients were ingroup B, and 14(28%) patients were ingroup C.

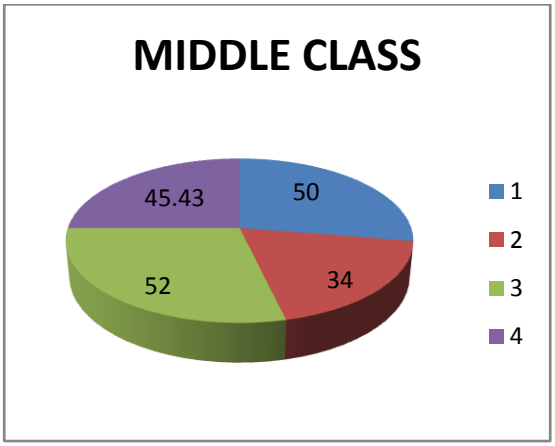
68 (45.33 %) patients belonged to MIDDLE CLASS, among whom 25(50%) patients were ingroup A, 17(34%) patients were ingroup B, and 26(52%) patients were ingroup C.

40(26.66 %) patients belonged to LOWER CLASS, among whom 15(30%) patients were ingroup A, 15(30%) patients were ingroup B, and 10 (20%)patients were ingroup C.

Graph: 4 Socio-economic status wise distributions of 150 patients with pulmonary hypersensitivity.







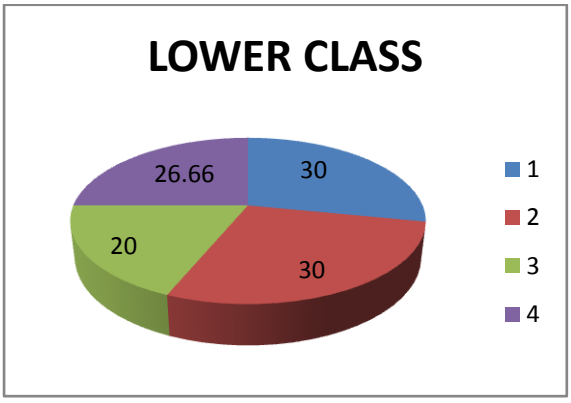


Table No.45

OCCUPATION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	STUDENT	07	14	09	18	07	14	23	15.33
2	HOUSE WIFE	08	16	05	10	06	12	19	12.66
3	AGRICULTURE	04	8	03	6	04	8	11	7.33
4	TEACHER	05	10	03	06	02	04	10	6.66
5	BUSINESS	--	00	01	02	01	02	02	1.33
6	WEAVER	05	10	06	12	06	12	17	11.33
7	PAINTER	06	12	09	18	07	14	22	14.66
8	MINE WORKER	01	02	04	08	06	12	11	7.33
9	BANK EMPLOYEE	06	12	02	04	01	02	09	6
10	FACTORY	08	16	08	16	10	20	26	17.33

The patients selected for the clinical trial were of different OCCUPATION

23 (15.33%) patients were STUDENTS, among whom 07(14%) patients were ingroup A, 09(18%) patients were ingroup B, and 07(14%) patients were ingroup C.

19 (12.66 %) patients were HOUSE WIVES, among whom 08(16%)patients were ingroup A, 05(10%) patients were ingroup B, and 06(12%) patients were ingroup C.

11(7.33 %) patients were AGRICULTURISTS, among whom 04(08%) patients were ingroup A, 03(06%) patients were ingroup B, 04 (08%) patients were ingroup C.

10(6.66 %) patients were TEACHERS, among whom, 05(10%) patients were ingroup A, 03(06%)patients were ingroup B, and 02 (04%) patients were ingroup C.

02(1.33 %) patients were BUSINESSMEN, among whom, 00(0%) patients were ingroup A, 01(02%)patients were ingroup B, and 01 (02%)patients were ingroup C.

17(11.33 %) patients were WEAVERS, among whom 05(10%) patients were ingroup A, 06(12%) patients were ingroup B, and 06 (12%) patients were ingroup C.

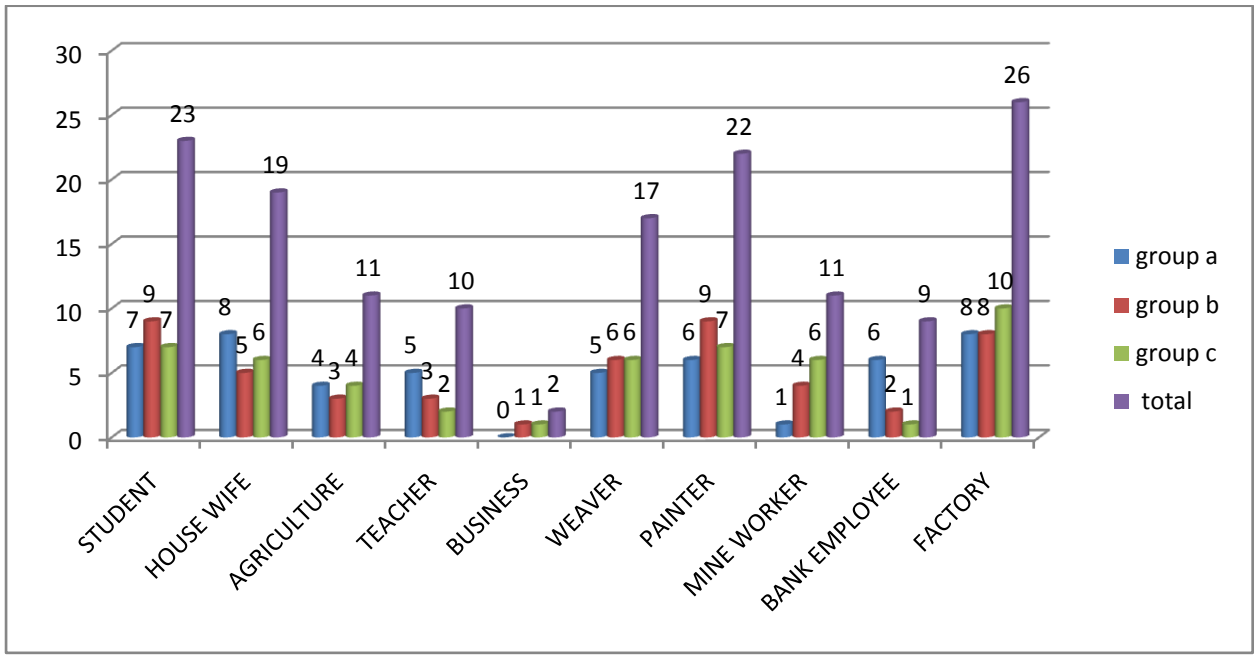
22(14.66 %) patients were PAINTERS, among whom 06(12%) patients were ingroup A, 09(18%) patients were ingroup B,and 07 (14%) patients were ingroup C.

11(7.33 %) patients were MINE WORKERS, among whom 1(2%) patients were ingroup A, 4(8%)patients were ingroup B, and 6 (12%)patients were ingroup C.

9(6 %) patients were BANK EMPLOYEES, among whom 6(12%) patients were ingroup A, 2(4%) patients were ingroup B, and 1 (2%) patients were ingroup C.

26(17.33 %) patients wereFACTORY Workers, among whom 8(16%) patients were ingroup A, 8(16%) patients were ingroup B, and 10 (20%) patients were ingroup C.

Graph: 5 Occupation wise distributions of 150 patients with pulmonary hypersensitivity.



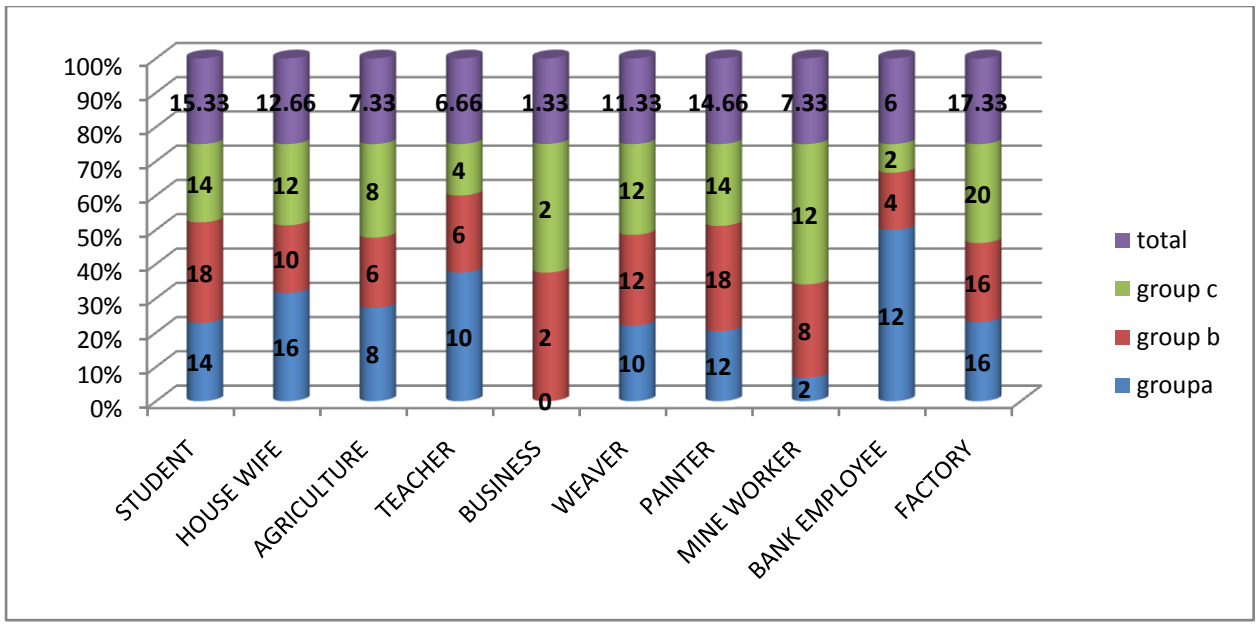


Table No.46

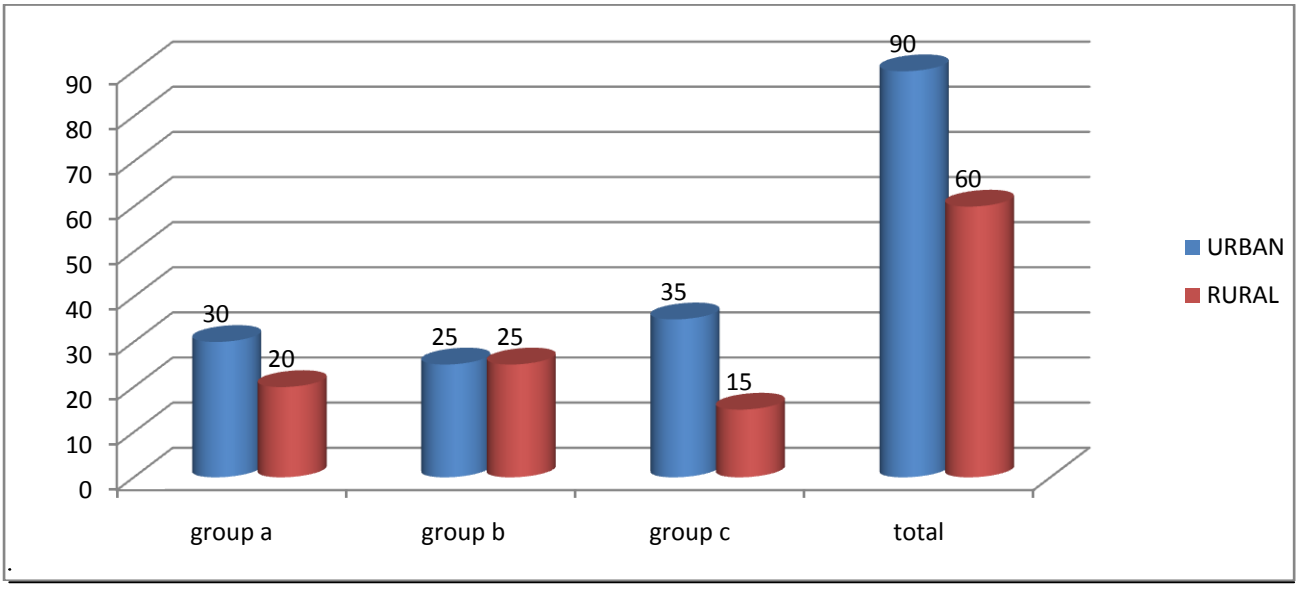
HABITAT WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	URBAN	30	60	25	50	35	70	90	60
2	RURAL	20	40	25	50	15	30	60	40

The patients were from different places; rural and urban. People residing in urban area were 90(60%) and in rural were 60(40%). Among urban, 30 (60%)patients were ingroup A, 25

(50%)patients were ingroup B,and 35 (70%)patients were ingroup C. Among rural, 20 (40%)patients were ingroup A, 25 (50%)patients were ingroup B and, 15(30%)patients were ingroup C.

Graph: 6 Habitat wise distributions of 150 patients with pulmonary hypersensitivity.



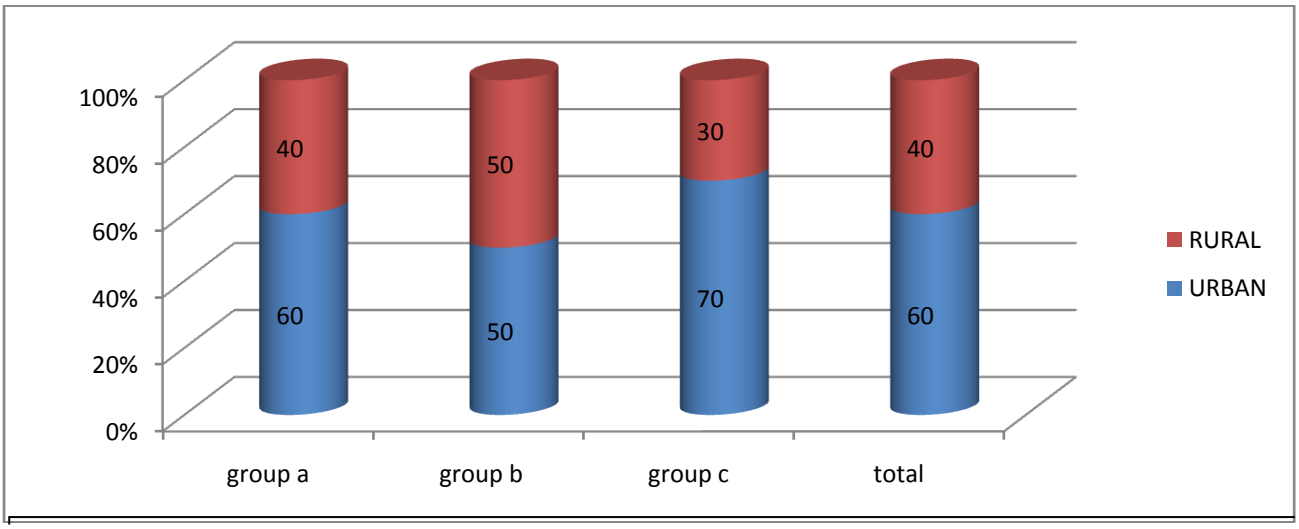


Table No.47

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO THEIR HABITS

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	ALCOHOL	05	10	06	12	09	18	20	13.33
2	SMOKING	15	30	12	24	20	40	47	31.33
3	BOTH 1 &2	04	08	05	10	09	18	18	12
4	TOBACCO CHEWING	07	14	09	18	03	06	19	12.66
5	GUTKA CHEWING	08	16	04	08	06	12	18	12
6	TEA /COFFEE	46	92	49	98	48	96	143	95.33

The patients of the trial had various habits-

20 (13.33%) patientswerealcoholics, in whom there were 5(10%) patients in group A, 6(12%)patients in group B, and 9(18%)patients in group C.

47(31.33%)patients were smokers, inwhom there were 15(30%)patients in group A, 12(24%)patients in group B, and 20(40%)patients in group C.

18(12 %)patients were both smokers and alcoholics, inwhom there were 4(8%)patients in group A, 5(10%)patients in group B, and 9(18%)patients in group C.

19(12.66%)patients were tobacco chewers, inwhom there were07(14%)patients in group A, 09(18%)patients in group B, and 3(6%).patients in group C.

18(12%)patients were gutka chewers, inwhom there were 8(16%)patients in group A, 04(8%)patients in group B, and 6(12%)patients in group C.

143(95.33%) patientswere consuming tea and coffee, inwhom there were46(92%)patients in group A, 49(98%)patients in group B, and 48 (96%)patients in group C.

Table No.48

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO THEIR DIETARY HABITS

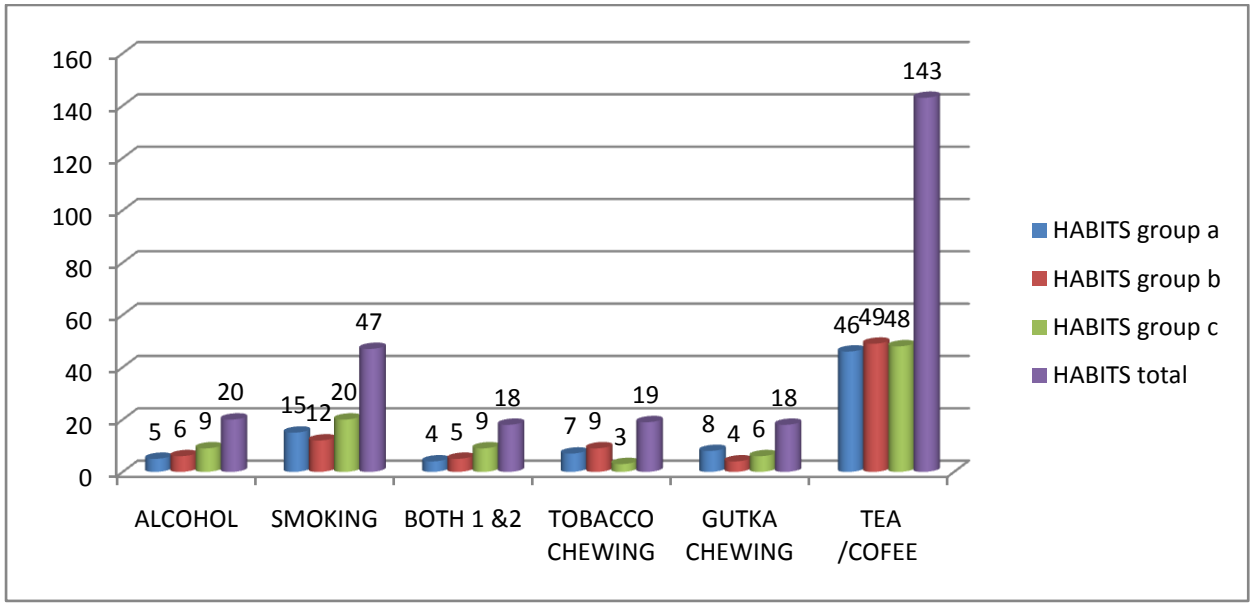
The Patients had different dietary habits,

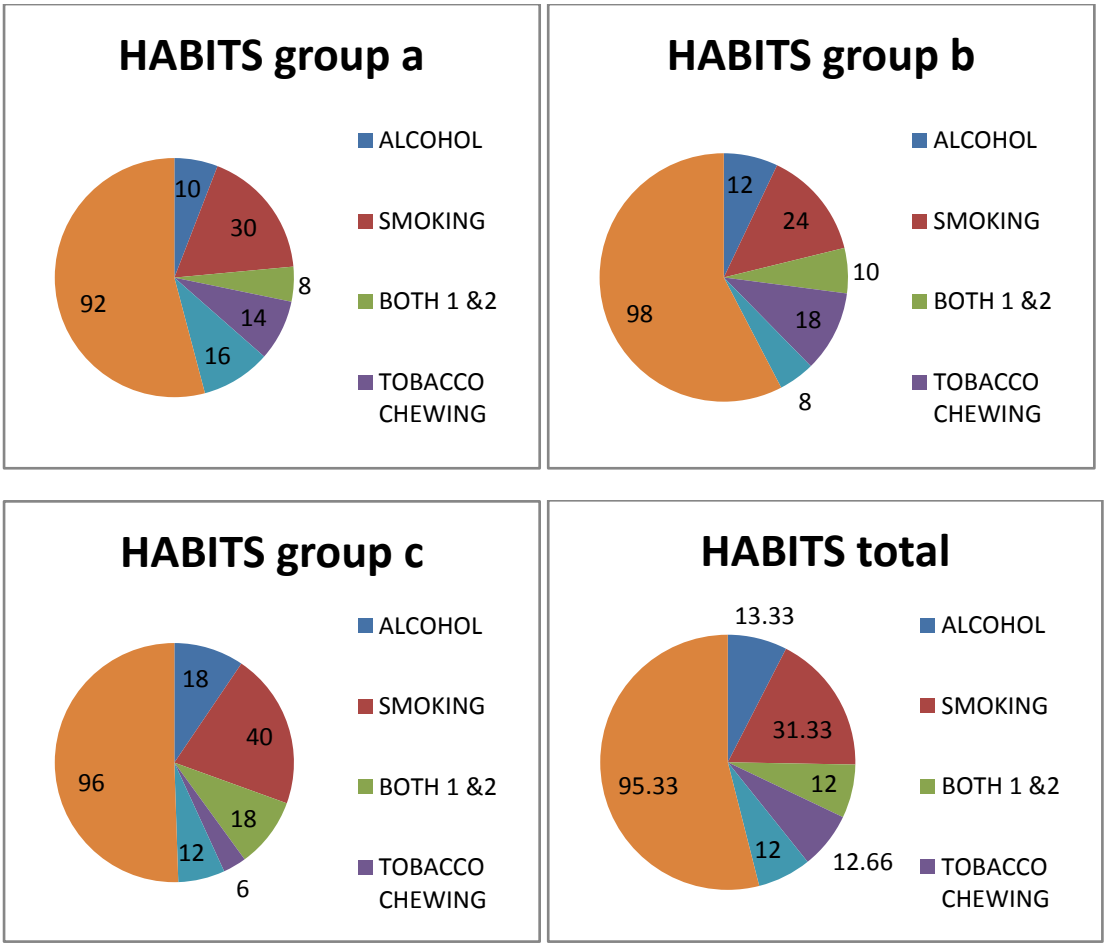
SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	PURELY VEG	32	64	27	54	29	58	88	58.66
2	MIXED	18	36	23	46	21	42	62	41.33

Among them 88 (58.66%)werepure vegetarians, inwhom there were32(64%)patients in group A, 27(54%)patients in group B,and 29(58%)patients in group C.

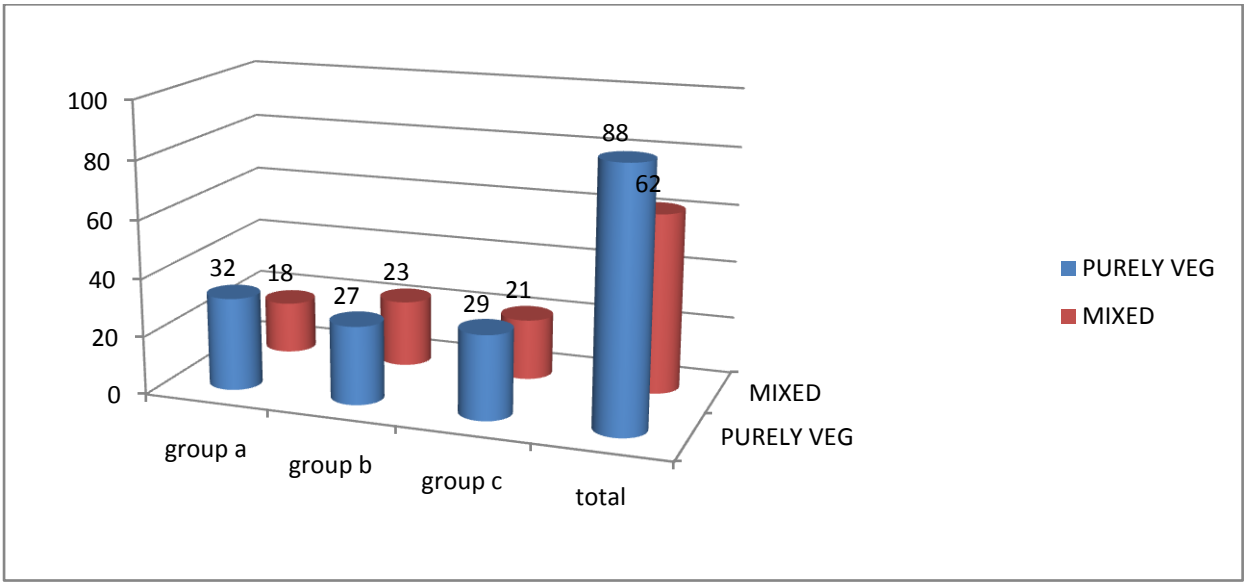
62 (41.33%) consumed mixed diet, inwhom there were 18(36%)patients in group A, 23(46%)patients in group B,and 21(42%)patients in group C.

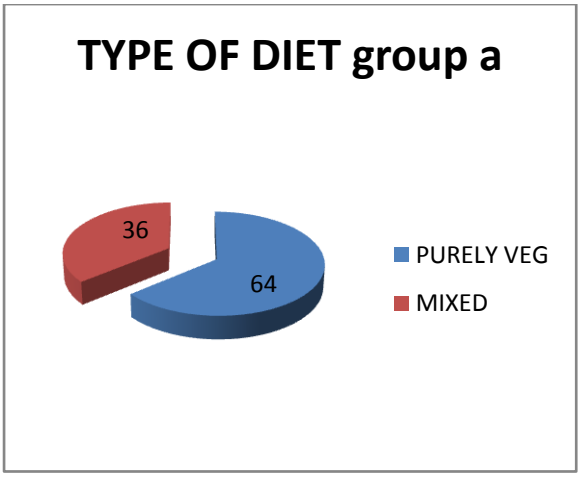
Graph: 7 Habit wise distributions of 150 patients with pulmonary hypersensitivity.

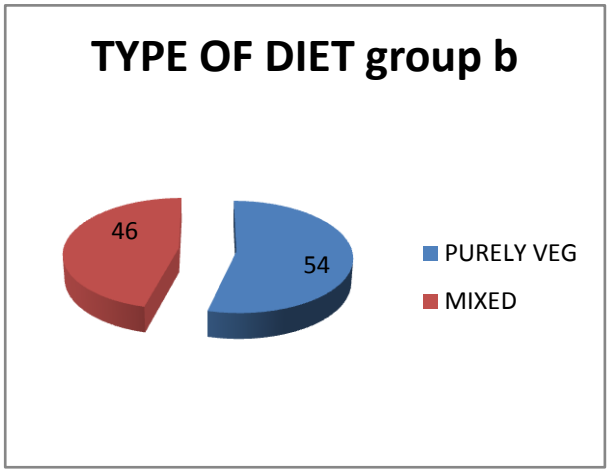


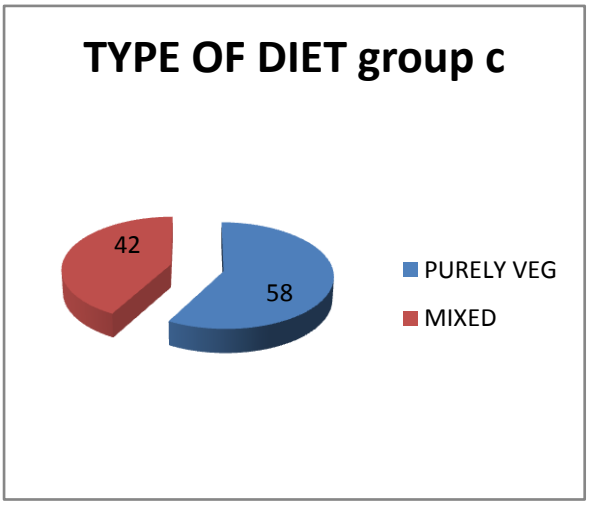


Graph: 8 Dietary habit wise distributions of 150 patients with pulmonary hypersensitivity.









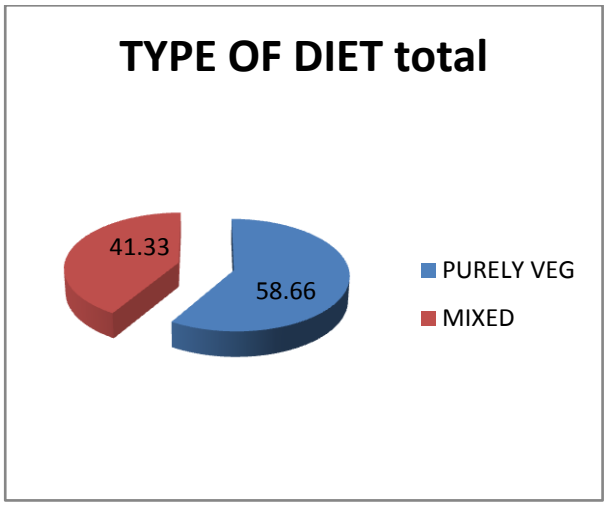


Table No.49

AGNI WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	MANDA	12	24	15	30	17	34	44	29.33
2	VISHAMA	15	30	18	36	19	38	52	34.66
3	TEEKSHNA	13	26	17	34	14	28	44	29.33

The agnibala of the patients was different,

Among them 44 (29.33%) patients were with mandaagni, inwhom 12(24%)were in group A, 15(30%)were in group B, and 17(34%)were in group C.

52 (34.66%)patients were withvishamaagni, among whom 15(30%)were in group A, 18(36%)were in group B, and 19(38%)were in group C.

44 (29.33%)patients were withteekshnaagni, among whom 13(26%)were in group A, 17(34%)were in group B,and 14(28%)were in group C.

Table No.50

KOSHTA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY

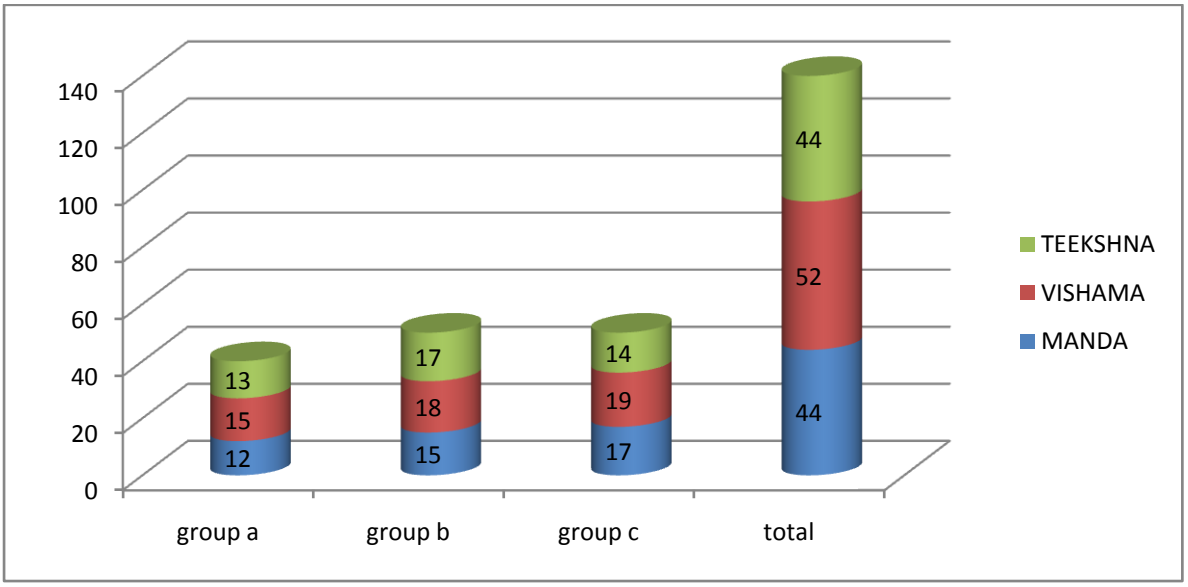
SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	MRUDU	13	26	17	34	14	28	44	29.33
2	MADHYAMA	12	24	15	30	17	34	44	29.33
3	KROORA	15	30	18	36	19	38	52	34.66

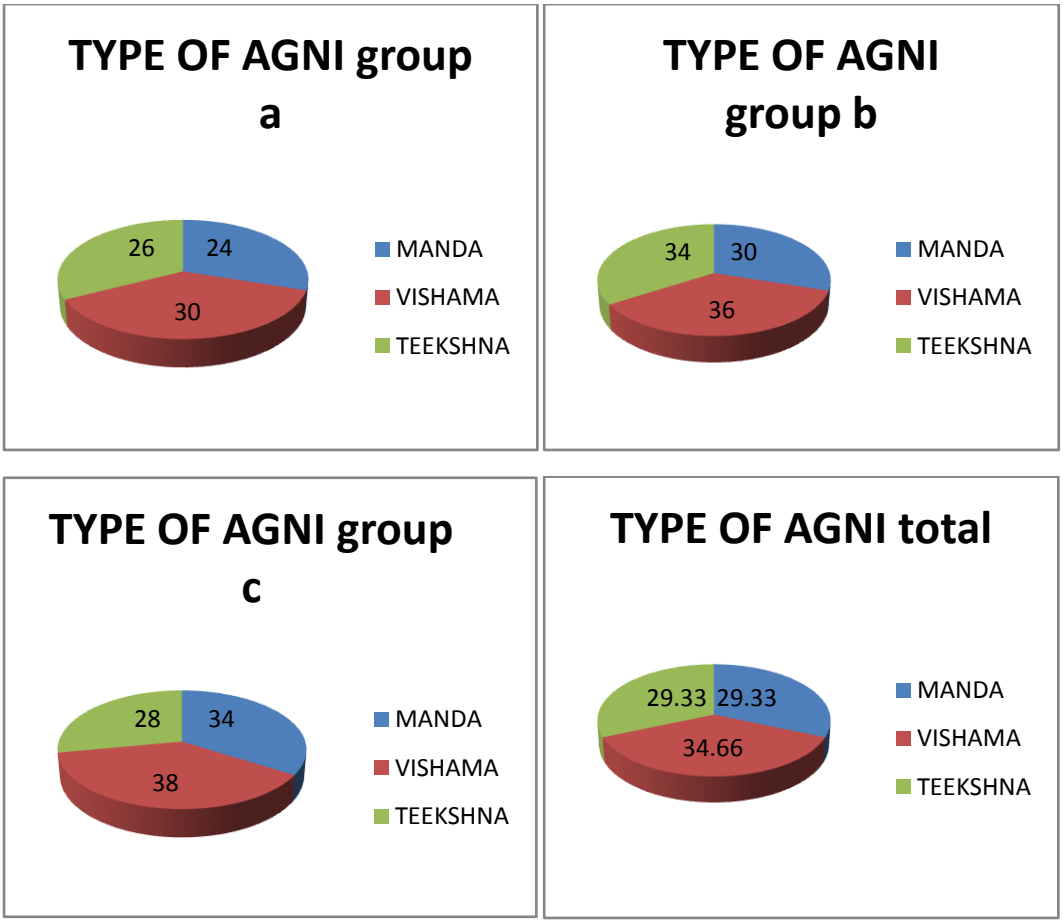
The koshta of the patients was accessed and Among them 44 (29.33%)patients were with mrudukoshta, in whom 13(26%)were in group A, 17(34%)were in group B,and 14(28%)were in group C.

44 (29.33%)patients were with madhyamakostha, among whom 12(24%)were in group A, 15(30%)were in group B, abd 17(34%)were in group C.

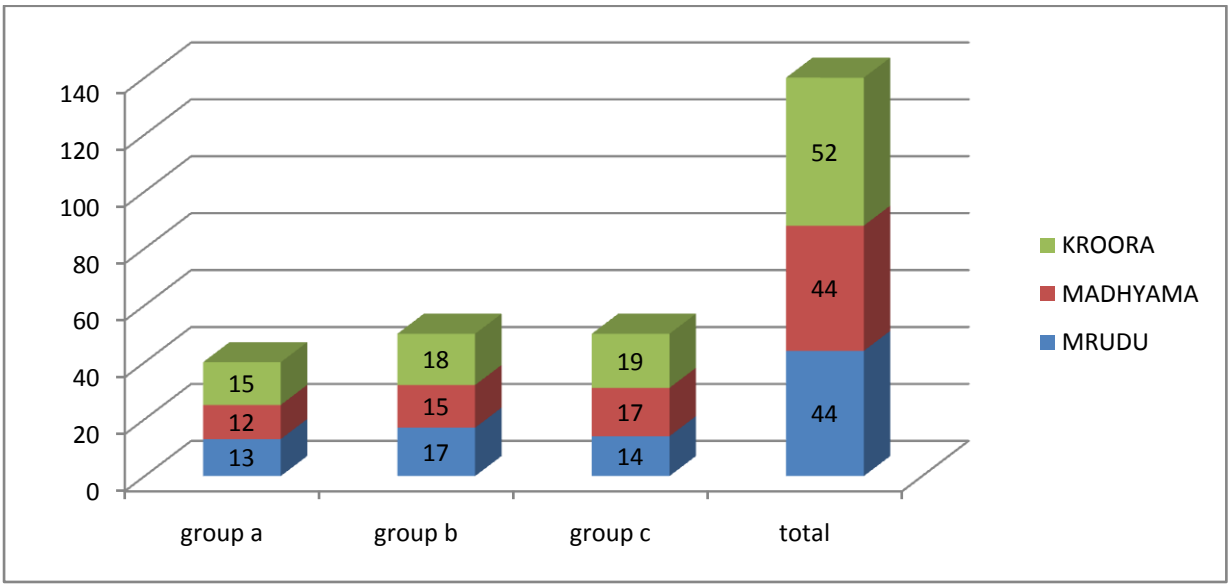
52 (34.66%)patients were with kroorakosta, among whom15(30%)were in group A, 18(36%)were in group B, and 19(38%)were in group C.

Graph: 9 Agni wise distributions of 150 patients with pulmonary hypersensitivity.





Graph: 10 Koshta wise distributions of 150 patients with pulmonary hypersensitivity.



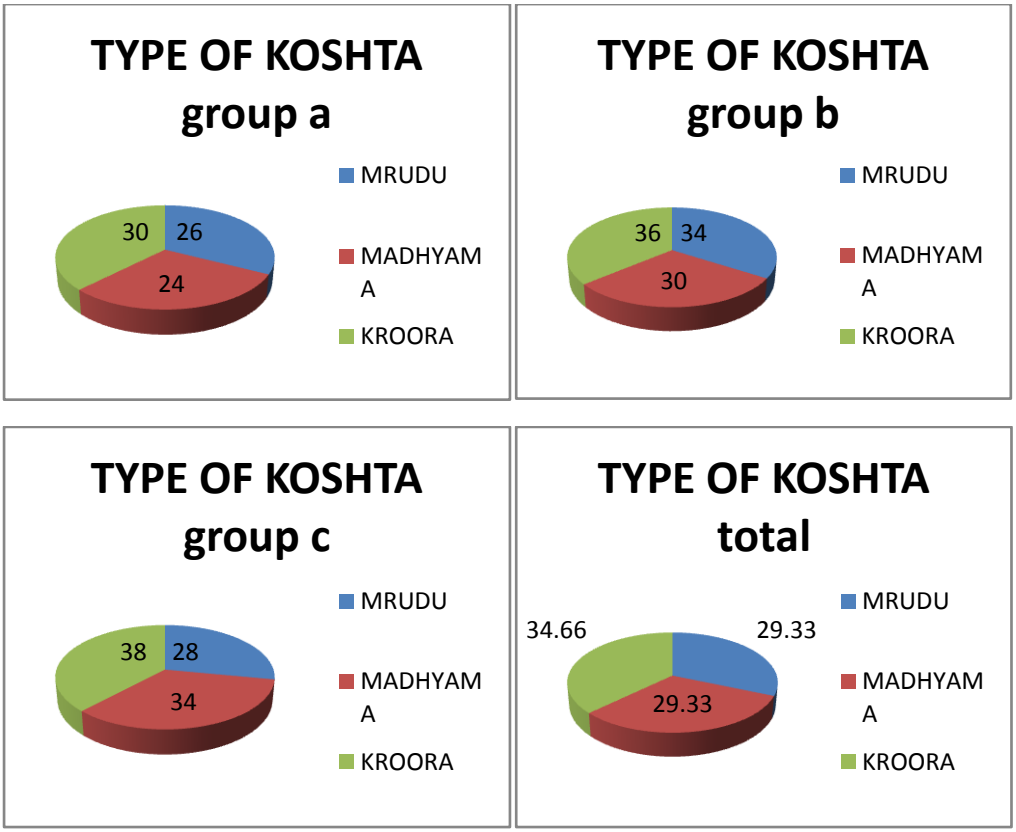


Table No.51

PRAKRITI WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	VATA	01	02	-	00	-	00	01	0.6
2	PITTA	-	00	-	00	01	02	01	0.6
3	KAPHA	06	12	07	14	03	06	16	10.66
4	PITTAKAPHA	15	30	14	28	12	24	41	27.33
5	VATAPITTA	07	14	05	10	06	12	18	12.0
6	KAPHAVATA	21	42	24	48	28	56	73	48.66
7	TRIDOSHAJA	-	00	-	00	00	00	-	00

The patients in the trial had dirrerent prakritis.

Among them patients with vataprakriti were 1 (0.6%), among whom,1(2%)were in group A 0(0%)were in group B,and 0(0%)in group C.

Patients with pitta prakritiwere 1 (0.6%) ,among whom 0(0%)were in group A (0%)were in group B,and 1(2%)werein group C.

Patients withkaphaprakriti were 16(10.66%),among whom6(12%)werein group A, 7(14%)werein group B,and 3(6%)were in group C.

Patients with pitta kaphaprakriti were 41 (27.33%),among whom15(30%)werein group A, 14(28%)werein group B,and12(24%)werein group C.

Patients withvata pittaprakriti were 18 (012.0%),among whom 7(14%)were in group A, 5(10%)werein group B,and 6(12%)were in group c.

Patients withkaphavataprakriti were 73(48.66%),among whom 21(42%)werein group A, 24(48%)werein group B, and28(56%)were in group C.

There were no patients with tridoshajaprakriti.

Table No.52

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO THEIR SATVA

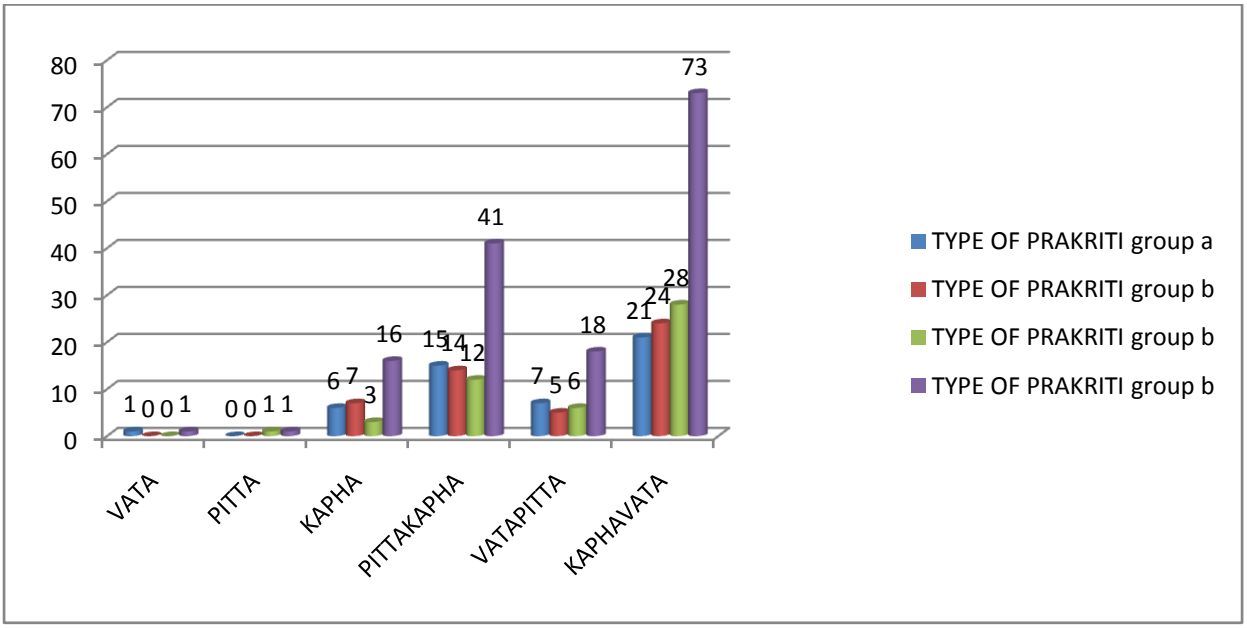
SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	PRAVARA	05	10	06	12	03	06	14	9.33
2	MADHYAMA	37	74	34	68	34	68	105	70
3	AVARA	08	16	10	20	13	26	31	20.66

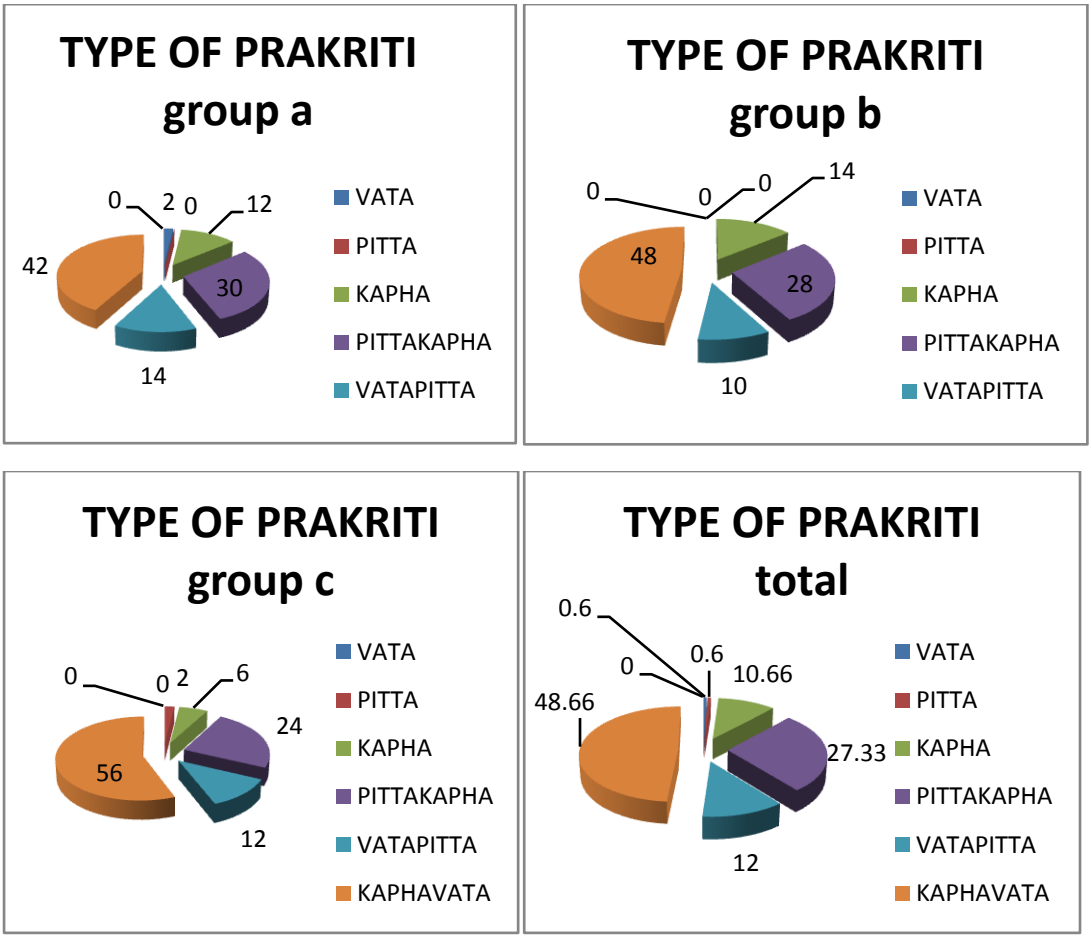
The satwa of 150 patients was accessed and Patients withpravarasatwa were 14 (9.33%),among whom5(10%)werein group A, 6(12%)werein group B, and 3(6%)were in group C.

Patients withmadhyamasatwa were 105 (70%) ,among whom37(74%)werein group A, 34(68%)were in group B ,and 34(68%)were in group C.

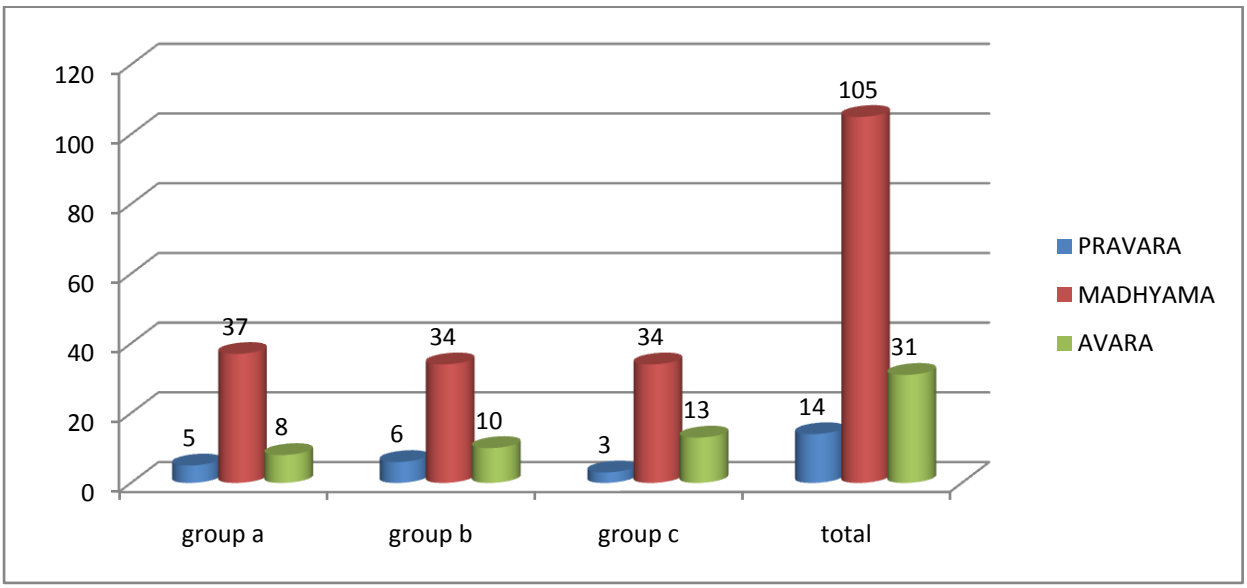
Patients withavarasatwa were 31 (20.66%),among whom8(16%)werein group A, 10(20%)werein group B,and 13(26%)werein group C.

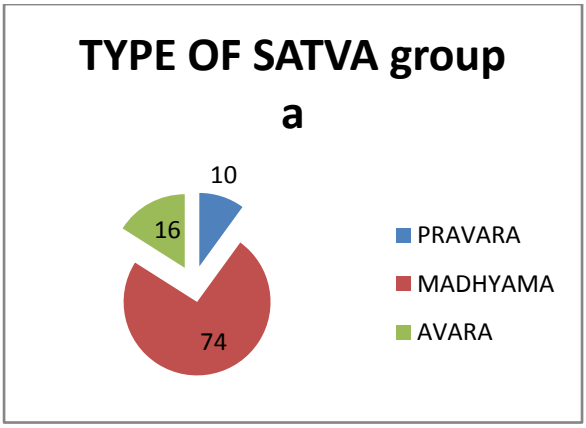
Graph: 11 Prakriti wise distributions of 150 patients with pulmonary hypersensitivity.

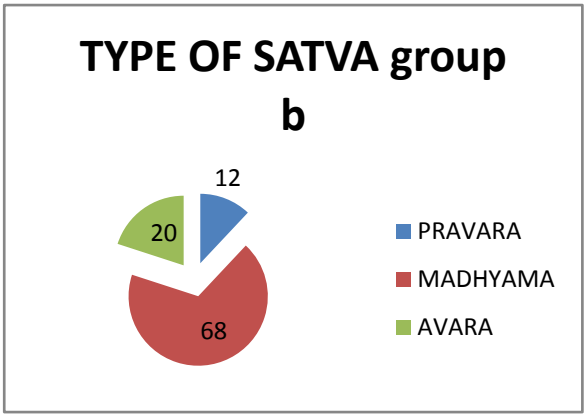


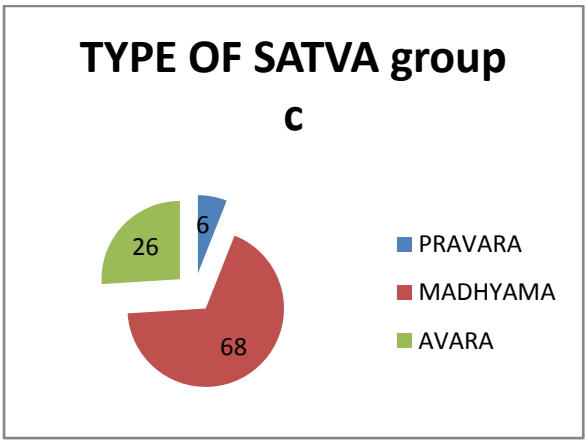


Graph: 12 Deha satva wise distributions of 150 patients with pulmonary hypersensitivity.









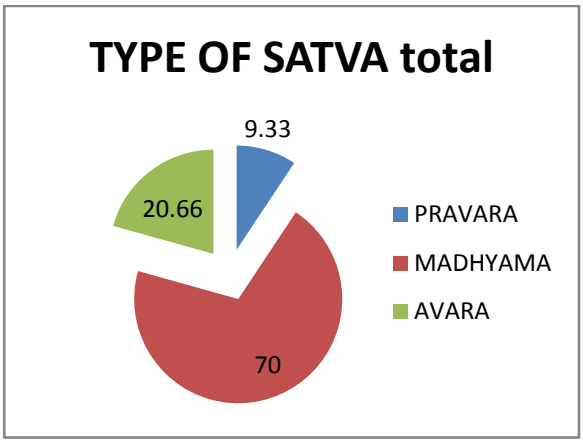


Table No.52

SARA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	PRAVARA	05	10	04	08	07	14	16	10.66
2	MADHYAMA	37	74	40	80	38	76	115	76.66
3	AVARA	08	16	06	12	05	10	19	12.66

The sara of all the patients was accessed and Patients withpravarasara were 16 (10.66%),among whom 05(10%)were in group A, 4(8%)werein group B,and 7(14%)werein groupC.

Patients withmadhyamasara were 115 (76.66%),among whom 37(74%)werein group A, 40(80%)werein group B,and 38(76%)werein groupC.

Patients withavarasarawere 19 (12.66%),among whom 8(16%)werein group A, 6(12%)werein group B,and 5(10%)werein groupC.

Table No.53

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO FAMILY HISTORY.

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	PRESENT	43	86	41	82	45	90	129	86.0
2	ABSENT	07	14	09	18	05	10	21	14

Few patients in the trial had a positive family history,

Among all, 129 (86%)patients were with positive family history,among whom 43(86%)werein group A, 41(82%)werein group B,and 45(90%)werein groupC.

21 (14%)patients werewithnegative family history,among whom07(14%)werein group A, 9(18%)werein group B,and 5(10%)werein groupC.

Table No.54

DESHA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SL.NO		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
1	ANUPA	29	58	31	62	30	60	90	60
2	SADHARANA	16	32	13	26	15	30	44	29.33
3	JANGALA	05	10	06	12	05	10	16	10.66

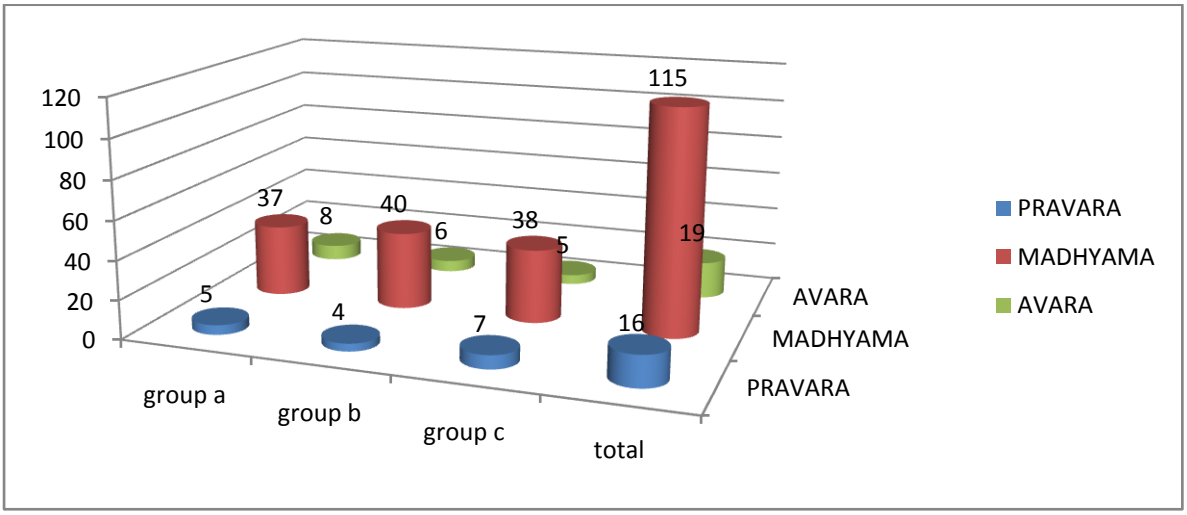
The patients of the trial were from different desha

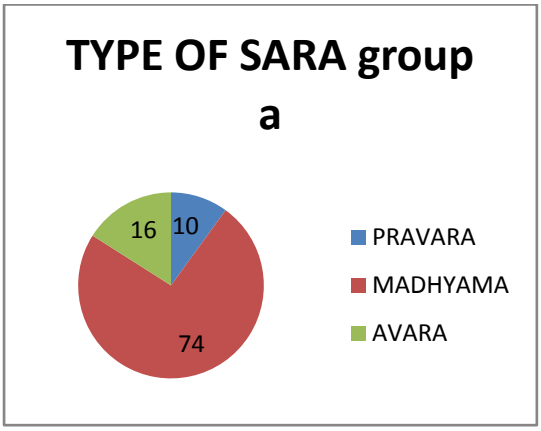
Patients fromanupadesha were 90 (60%),in whom 29(58%)werein group A, 31(62%)werein group B,and 30(60%)werein groupC.

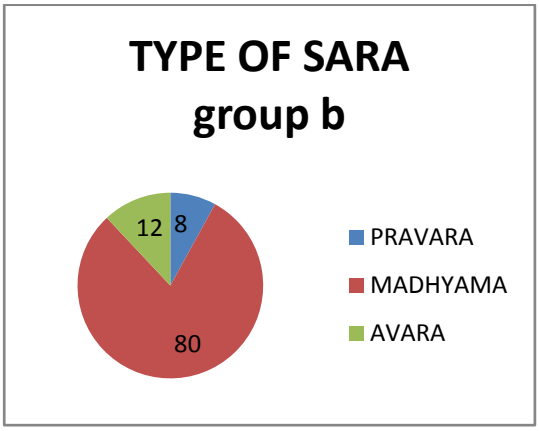
Patients fromsadharanadeshawere 44 (29.33%),among whom 16(32%)werein group A, 13(26%)werein group B,and 15(30%)werein groupC.

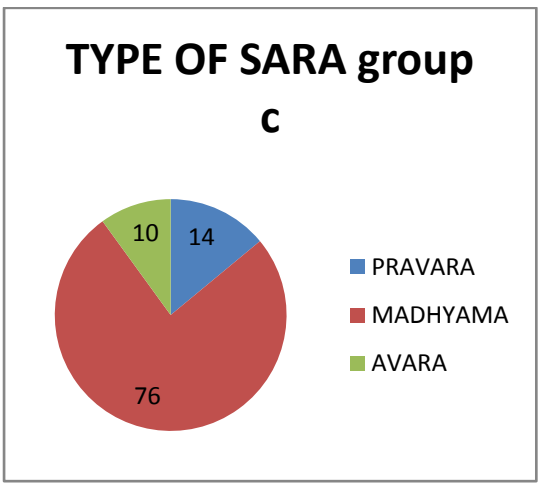
Patients fromjangaladesha were 16 (10.66%),among whom 05(10%)werein group A, 06(12%)werein group B,and 05(10%)werein groupC.

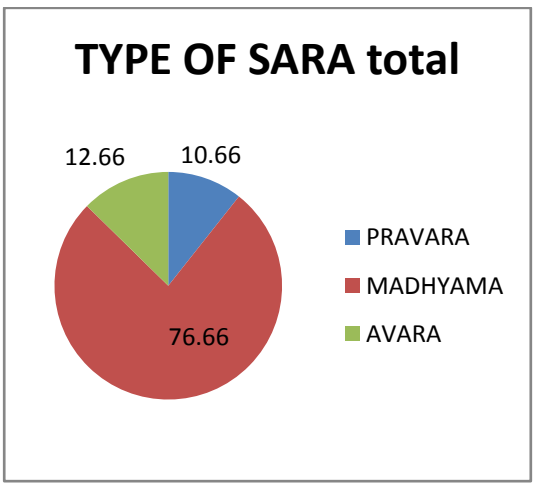
Graph: 13 Sara wise distributions of 150 patients with pulmonary hypersensitivity.



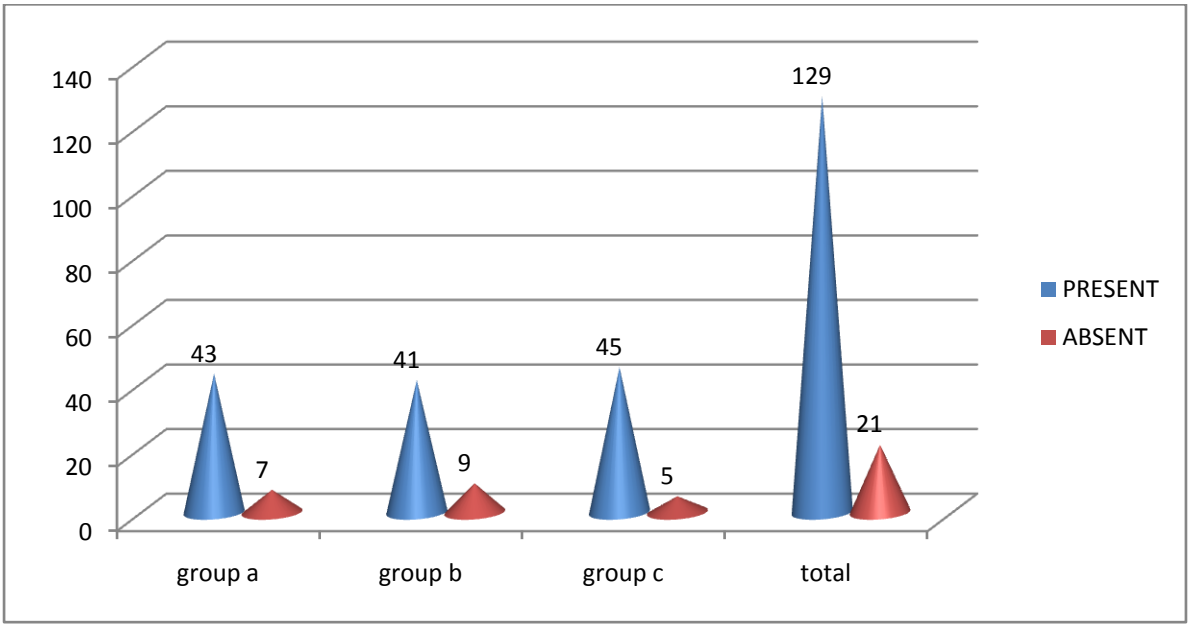


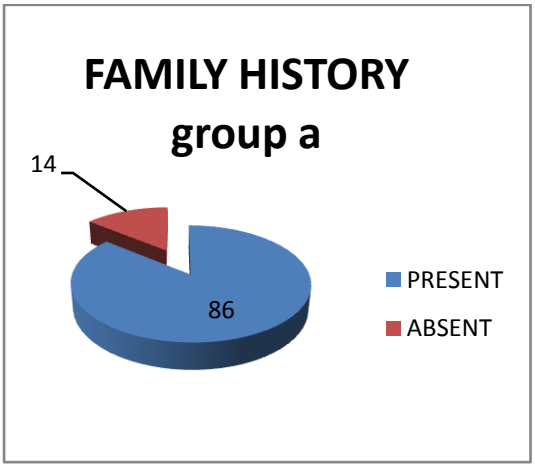


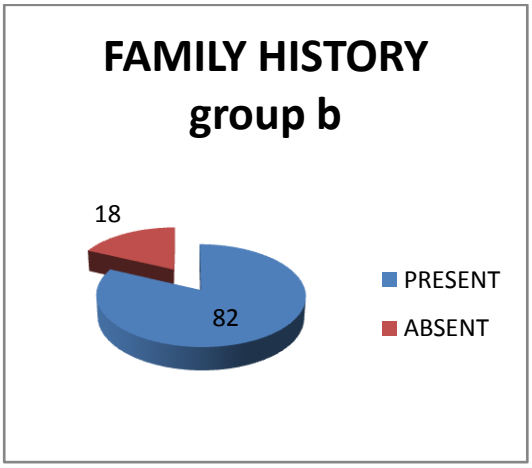


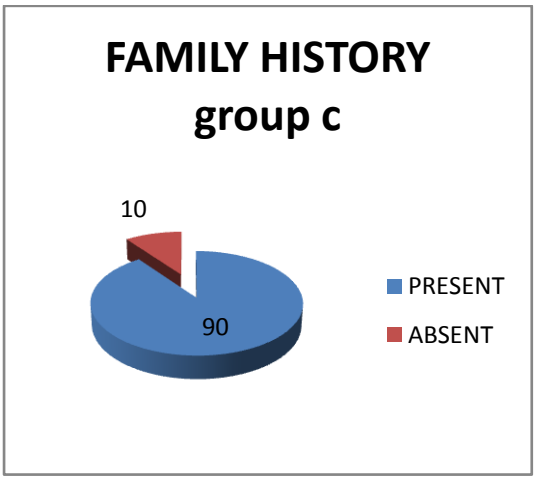


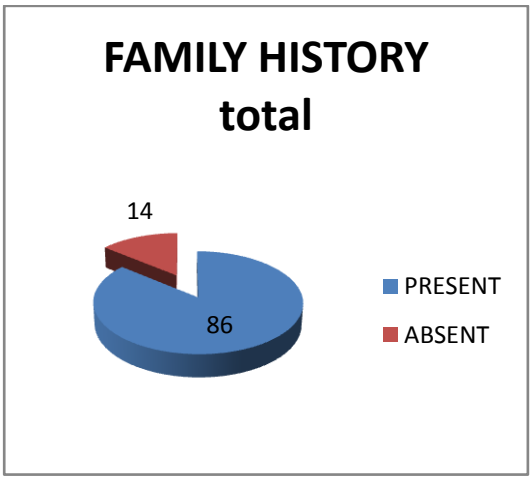
Graph: 14 Family history wise distributions of 150 patients with pulmonary hypersensitivity.



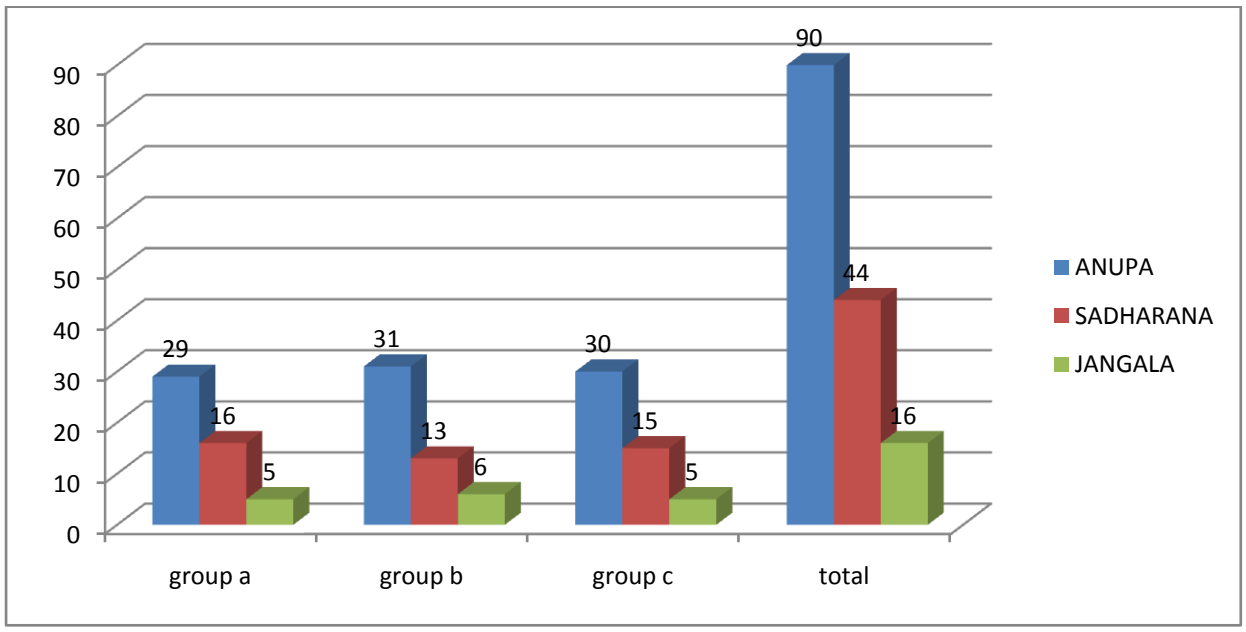


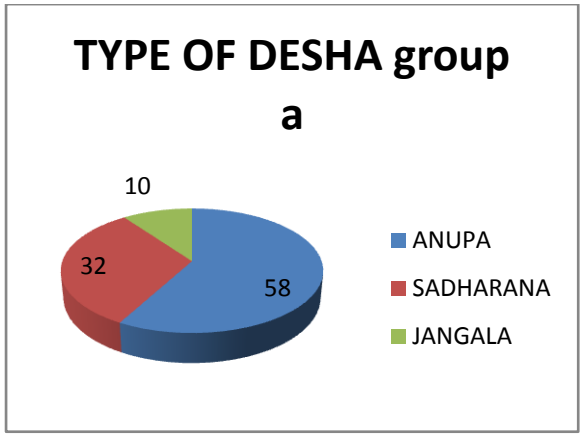


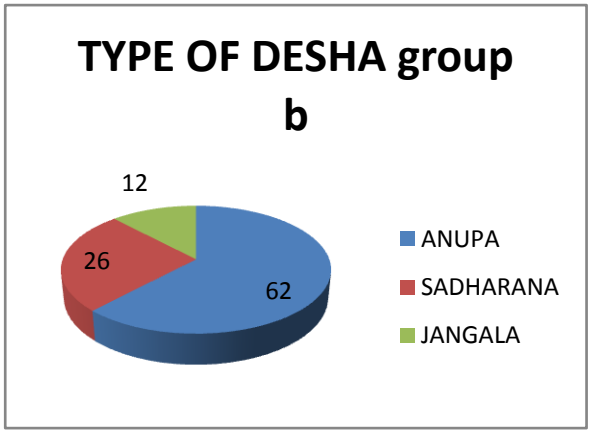


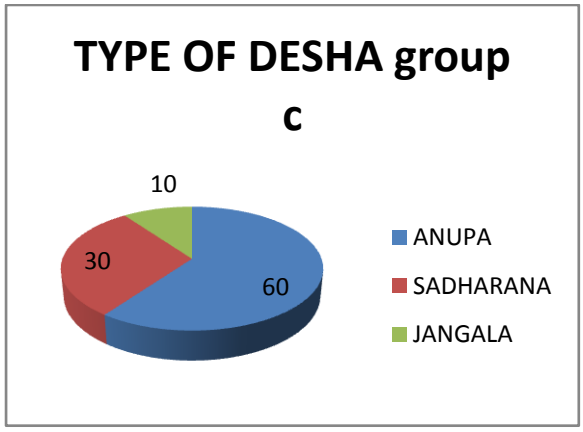


Graph: 15 Desha wise distributions of 150 patients with pulmonary hypersensitivity.









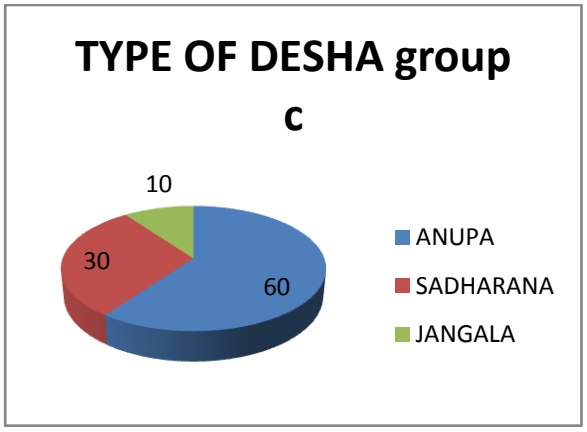


Table No. 56

NASASRAVA (NASAL DISCHARGE) WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

DURATION	YEARS	GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	8-10YRS	14	28	15	30	19	36	48	32
	6-8 YRS	6	12	4	8	9	18	19	12.66
	4-6 YRS	16	32	11	22	8	16	35	23.33
	2-4 YRS	8	16	17	34	12	24	23	15.33
	1-2 YRS	6	12	7	14	2	4	15	10

Out of 150 patients,48(32%) patientshad nasasravaFrom 8- 10 yrs, among whom 14(28%)were in group A, 15(30%)werein group B,and 19(36%)werein groupC.

19(12.66%)patientshad nasasravaFrom 6-8 yrs,among whom6(12%)werein group A, 4(8%)werein group B,and 9(18%)werein groupC.

35(23.33%)patientshad nasasravaFrom 4-6 yrs,among whom16(32%)werein group A, 11(22%)werein group B,and 08(16%)werein groupC.

23(15.33%)patients had nasasravaFrom 2-4 yrs,among whom8(16%)werein group A, 17(34%)werein group B, and 12(24%)werein groupC.

15(10%)patients had nasasravaFrom 1-2 yrs,among whom6(12%)werein group A, 7(14%)werein group B,and 2(4%)werein groupC.

Table No.57

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO MODE OF ONSET OF NASASRAVA

MODE OF ONSET		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	INSIDIOUS	2	4	8	16	11	22	21	14
	GRADUAL	35	70	39	78	29	58	103	68.66
	SUB ACUTE	11	22	01	2	6	12	18	12
	ACUTE	02	04	02	04	04	08	08	5.33

The patients had nasasrava of different modes of onset

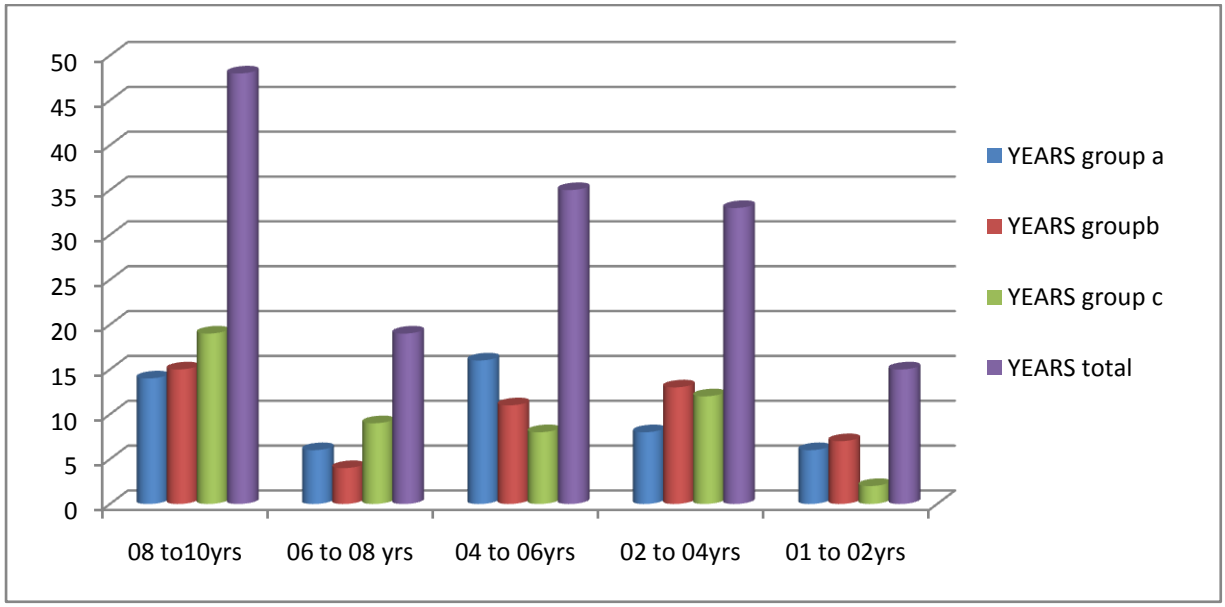
Among all 150 patients,21 (14%) patients hadinsidiousonset,among whom 2(4%)werein group A, 8(16%)werein group B,and11(22%)werein groupC.

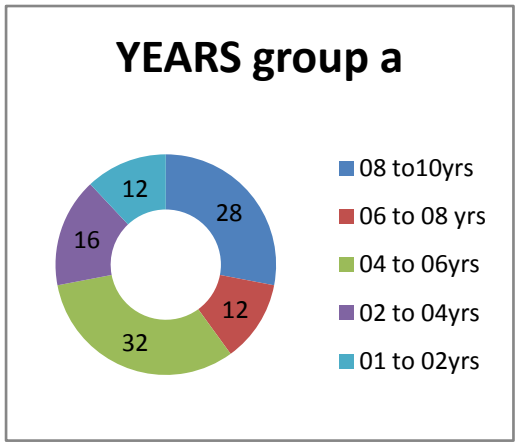
103 (68.66%)patients hadGradualonset,among whom 35(70%)werein group A, 39(78%)werein group B,and 29(58%)werein groupC.

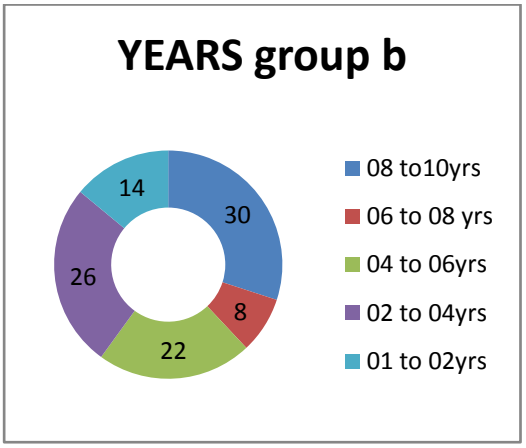
18 (12%)patients hadsub-acuteonset,among whom 11(22%)werein group A, 1(2%)werein group B,and 6(12%)werein groupC.

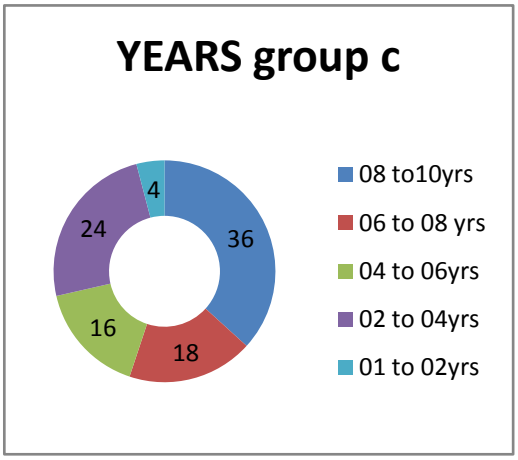
8 (5.33%)patients hadacuteonset,among whom 02(04%)werein group A, 2(4%)werein group B,and 4(8%)werein groupC.

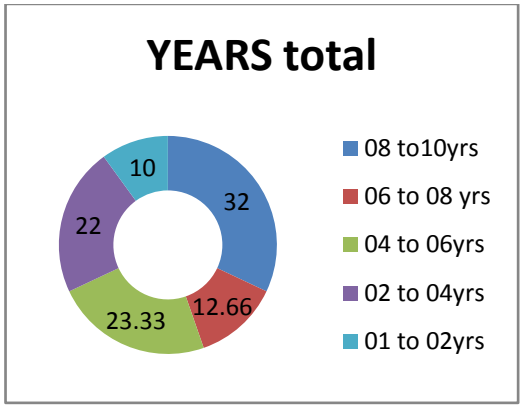
Graph: 16 Nasasrava duration wise distributions of 150 patients with pulmonary hypersensitivity.



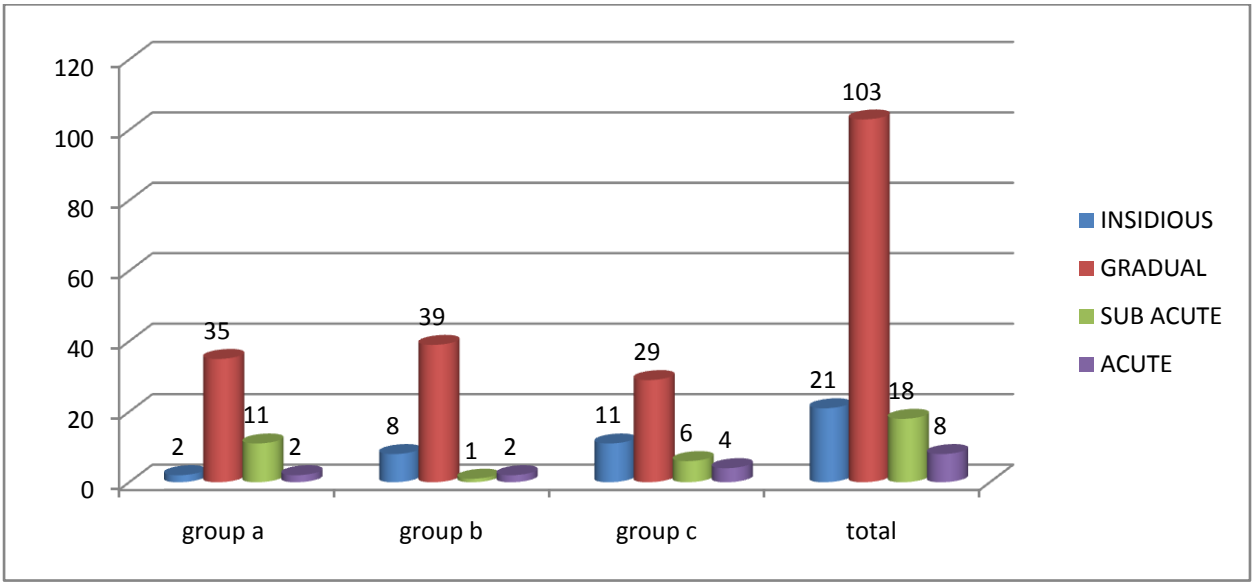


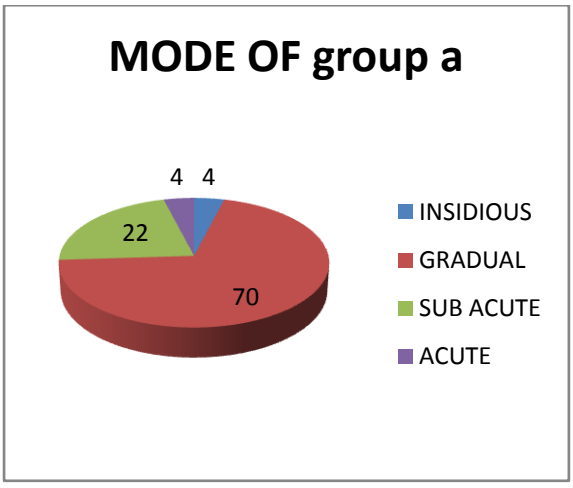


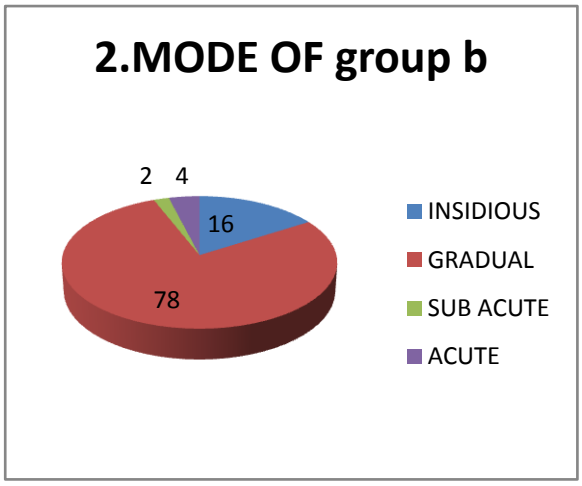


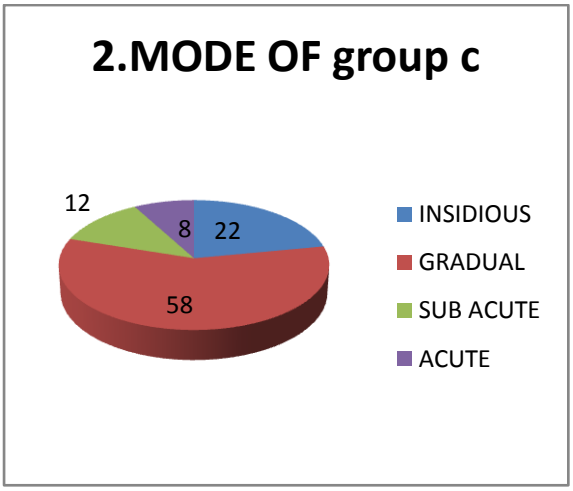


Graph: 17 Nasasrava mode of onset wise distributions of 150 patients with pulmonary hypersensitivity.









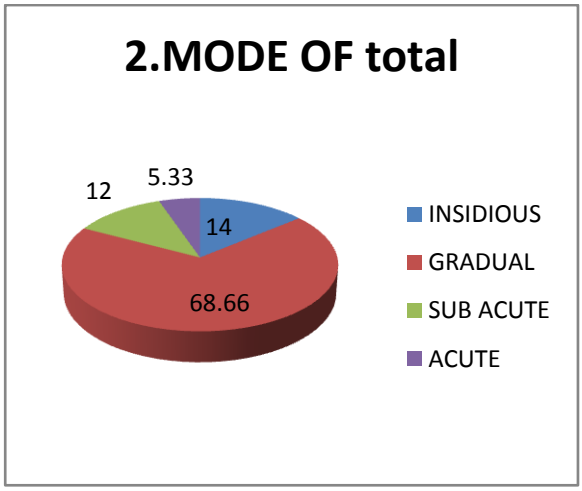


Table No.58

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF NASASRAVA

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	28	56	21	42	12	24	61	40.66
	RECEEDING	03	06	08	16	11	22	22	14.66
	RELAPSING	03	06	11	22	09	18	23	15.33
	STATIONARY	16	32	10	20	18	36	44	99.33

The patients had nasasrava of different pattern

Among 150 patients, 61 (40.66%)had progressive nasasrava,among whom 28(56%)were in group A, 21(42%)were ingroup B, and 12(24%)werein group C

22 (14.66%)had preceding nasasrava,among whom03(06%)werein group A, 8(16%)wereIn group B,and 11(22%)werein group C

23 (15.33%)had relapsing nasasrava,among whom 03(06%)werein group A, 11(22%)werein group B,and 9(18%)werein group C

44 (99.33%) had stationary nasasrava,among whom 16(32%)werein group A, 10(20%)were in group B,and 18(36%)werein group

Table No. 59

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY OF NASASRAVA

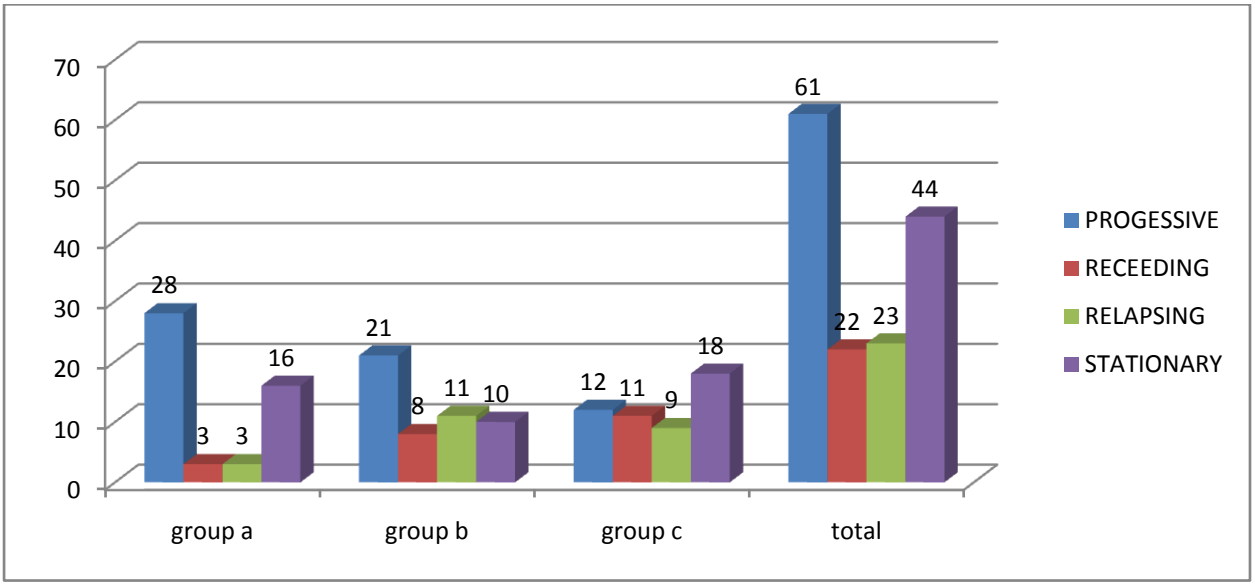
SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	08	16	10	20	05	10	23	15.33
	MODERATE	12	24	09	18	11	22	32	21.33
	SEVERE	30	60	31	62	34	38	95	63.33

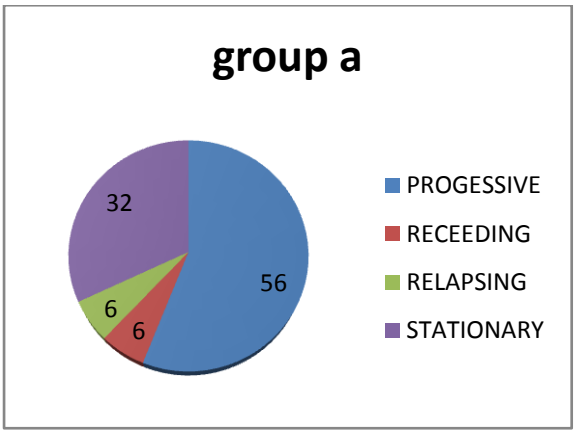
Among all of the patients, 23 (15.33%) patients had mild srava,among whom 8(16%)werein group A, 10(20%)were in group B,and 05(10%)werein group C

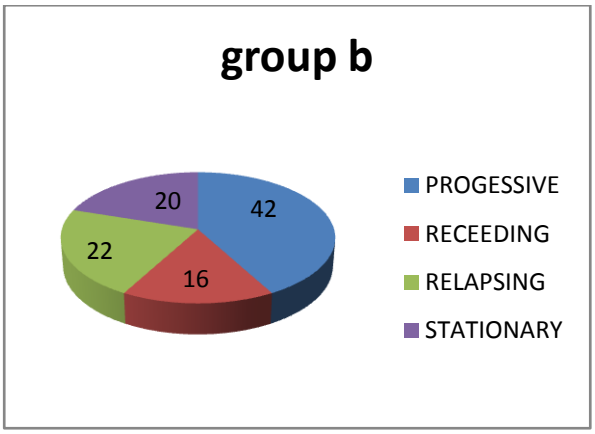
32 (21.33%)patients had moderatesrava, among whom 12(24%)werein group A, 9(18%)werein group B,and 11(22%)werein group C

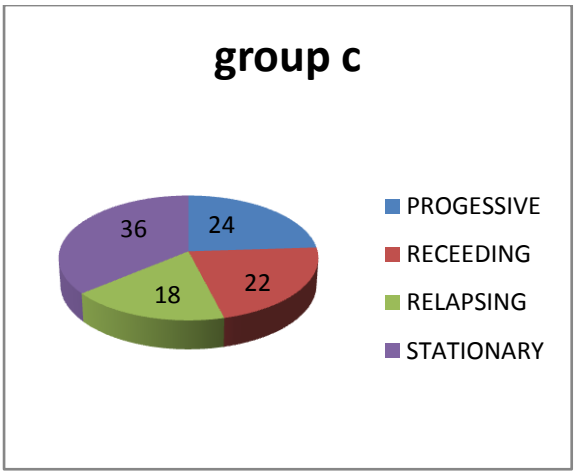
95 (63.33%)patients had severesrava,among whom 30(60%)werein group A, 31(62%)werein group B,and34(38%)werein group C

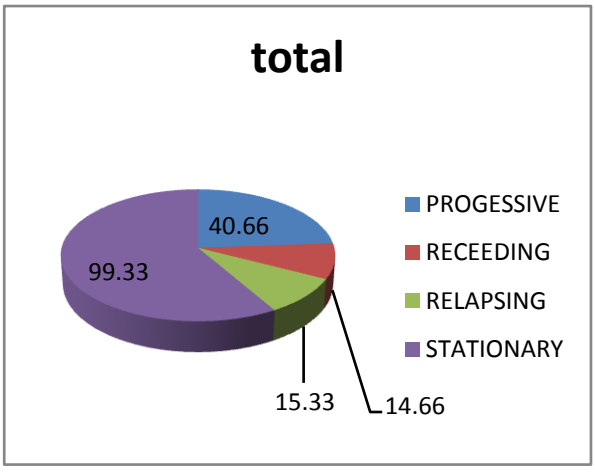
Graph: 18 Nasasrsva pattern wise distributions of 150 patients with pulmonary hypersensitivity.



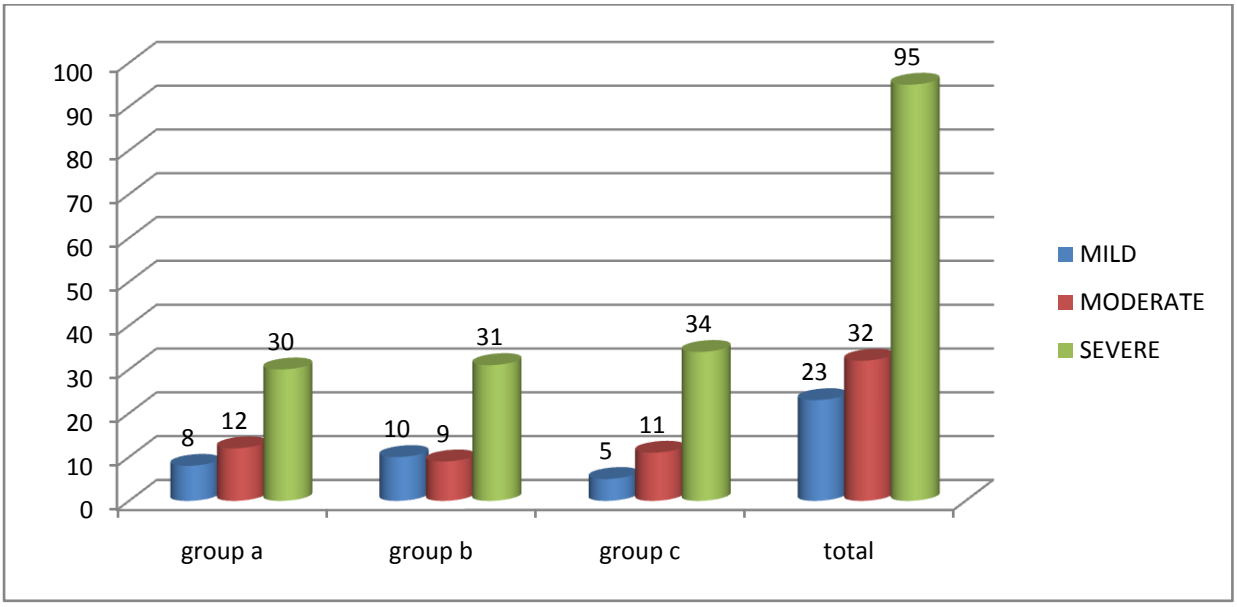


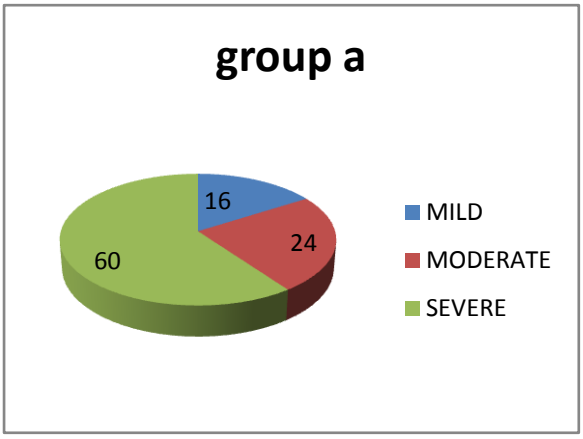


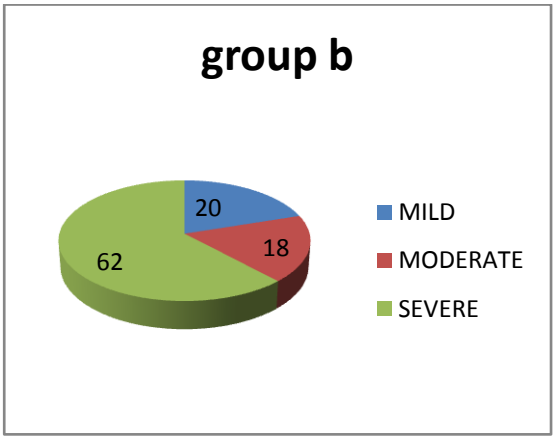


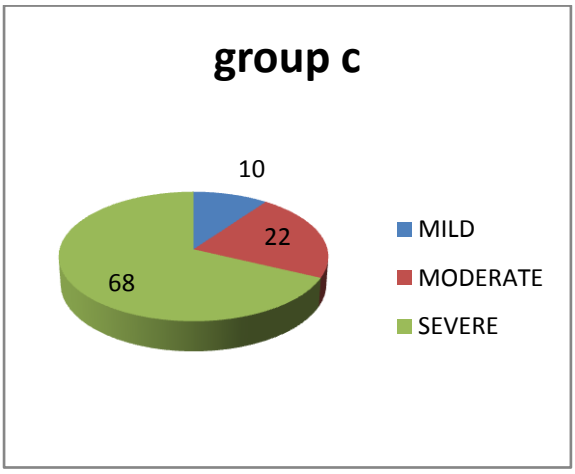


Graph: 19 Nasasrsva severity wise distributions of 150 patients with pulmonary hypersensitivity.









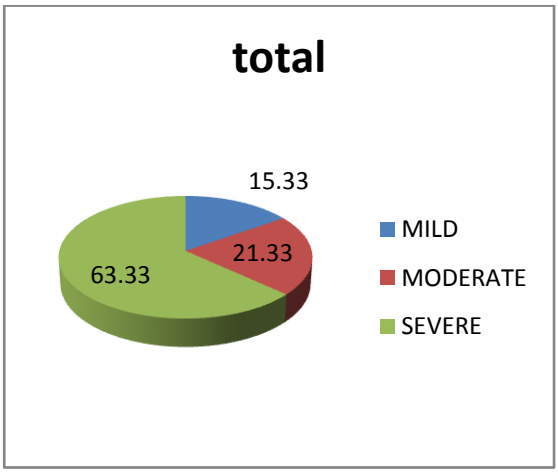


Table No.60

NASASRAVA KALA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SRAVA KALA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	EARLY MORNING	48	96	48	96	49	98	145	96.66
	MORNING	02	04	01	02	01	02	04	2.66
	AFTERNOON	-	-	-	-	-	-	-	-
	EVENING	20	40	15	30	00	00	35	23.33
	NIGHT	06	12	01	02	-	-	07	4.66
	MID-NIGHT	-	--	-	-	-	-	-	-
	IMMIDIATELY AFTER KSHAVATHU	50	100	50	100	50	100	150	100

The patients had nasasrava at different kaala.

Among all,patients with srava in early morning were 145 (96.66%)among whom ,48(96%) were in group A, 48(96%)were in group B,and 49(98%)were in group C-

Patients with srava in morning were 04 (2.66%), among whom ,2(4%)were in group A,1(2%),were in group Band,1(2%)were in group C

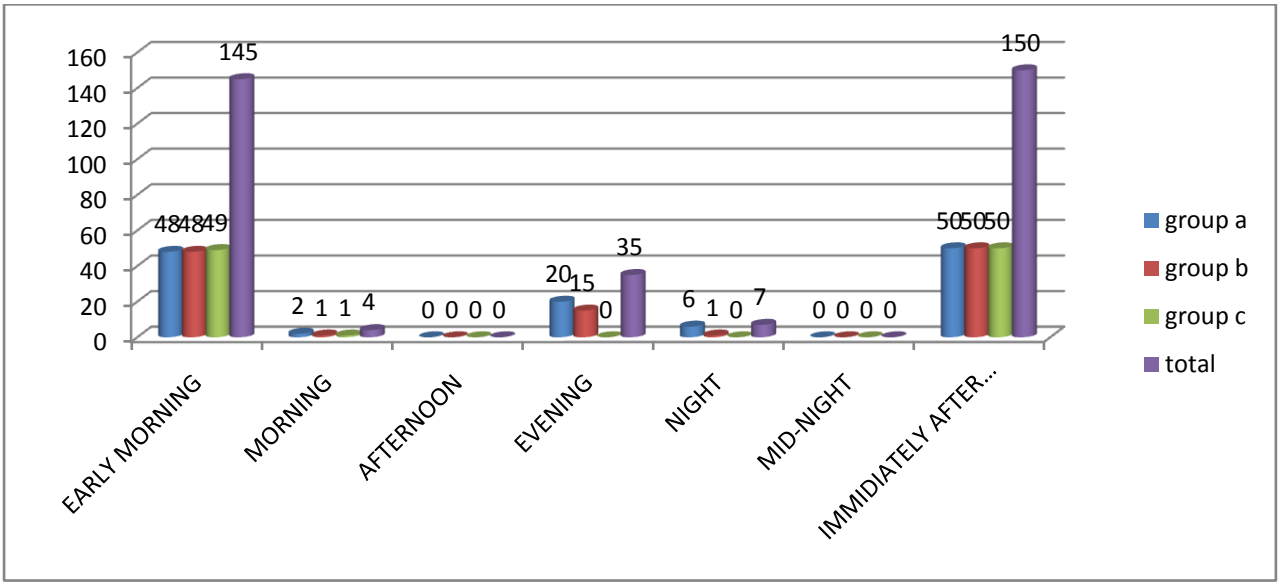
There were no patients with srava in afternoon.

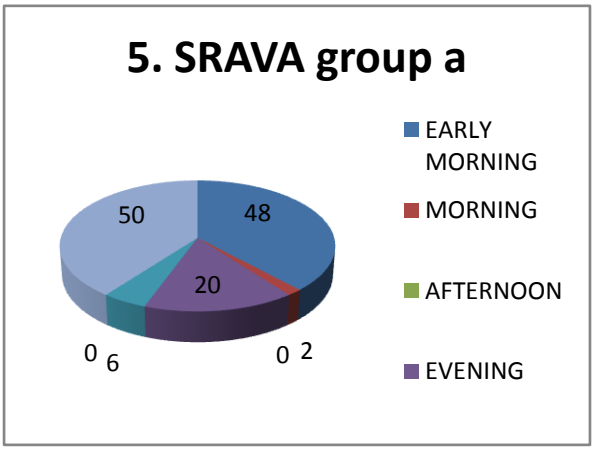
Patients with sravain evening were 35 (23.33%)among whom,20(40%)were in group A, 15(30%) were in group B, nonewere in group C.

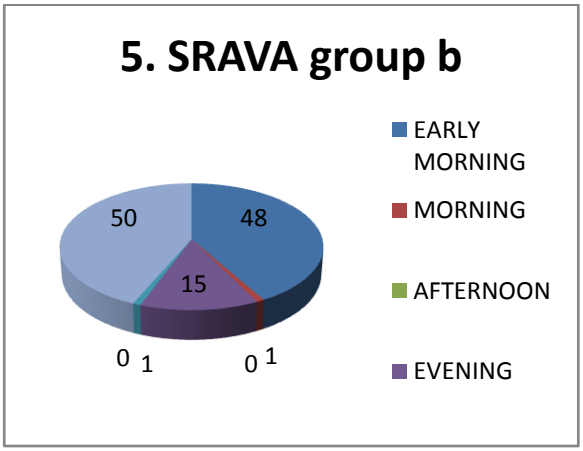
There were no patients with srava at midight.

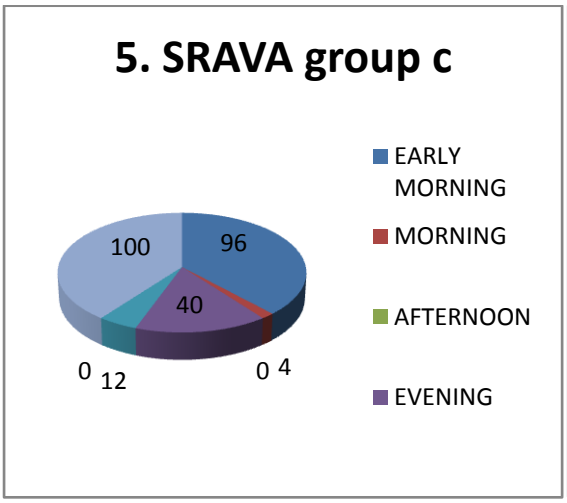
Patients with sravaimmediately after kshavathu were 150 (100%)among whom, 50(100%) were in group A, 50(100%) werein group B,and50(100%)were in group C-.

Graph: 20 Nasasrsva kala wise distributions of 150 patients with pulmonary hypersensitivity.









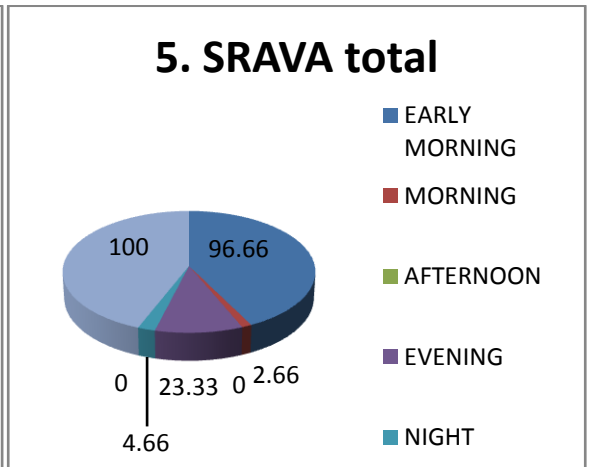


Table No.61

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO TYPE

OF NASASRAVA

TYPES OF SRAVA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	WATERY	48	96	48	96	49	98	145	96.66
	MUCOID	02	04	02	04	01	02	05	3.33
	PURULENT	-	-	-	-	-	-	-	-

Among 150 patients,145 (96.66%) had watery discharge in whom 48(96%) were in group A,were 48(96%)in group B,and49(98%)were in group C.

Patients withmucoid discharge were 5 (3.33%),in whom 2(4%) were in group A, 2(4%)were in group B, and1(2%)were in group C

There were no patients with purulent discharge.

Table No.62

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO GANDHA OF NASASRAVA

GANDHA OF SRAVA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	NIRGANDHA	50	100	50	100	50	100	150	100
	DURGANDHA	-	-	-	-	-	-	-	-

All the 150 (100%)patients had nirgandhasrava,among whom, 50(100%) were in group A,50(100%) were in group B,50(100%) werein group C.

There were no patients with durgandhitasrava.

Table No.63

NASAVARODHA (NASAL OBSTRUCTION) DURATION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

DURATION		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	08- 10 YRS	14	28	15	30	13	26	42	28
	06-08 YRS	08	16	06	12	09	18	23	15.33
	04- 06 YRS	16	32	11	22	12	24	39	26
	02-04 YRS	08	16	15	30	09	18	32	21.33
	01-02 YRS	04	08	03	06	06	12	13	8.66

Among all 150 patient,s42(28%)patients had Nasavarodha from From 8- 10 yrs among whom, 14(28%)were in group A, were in 15(30%)group B,and13(26%)were in group C

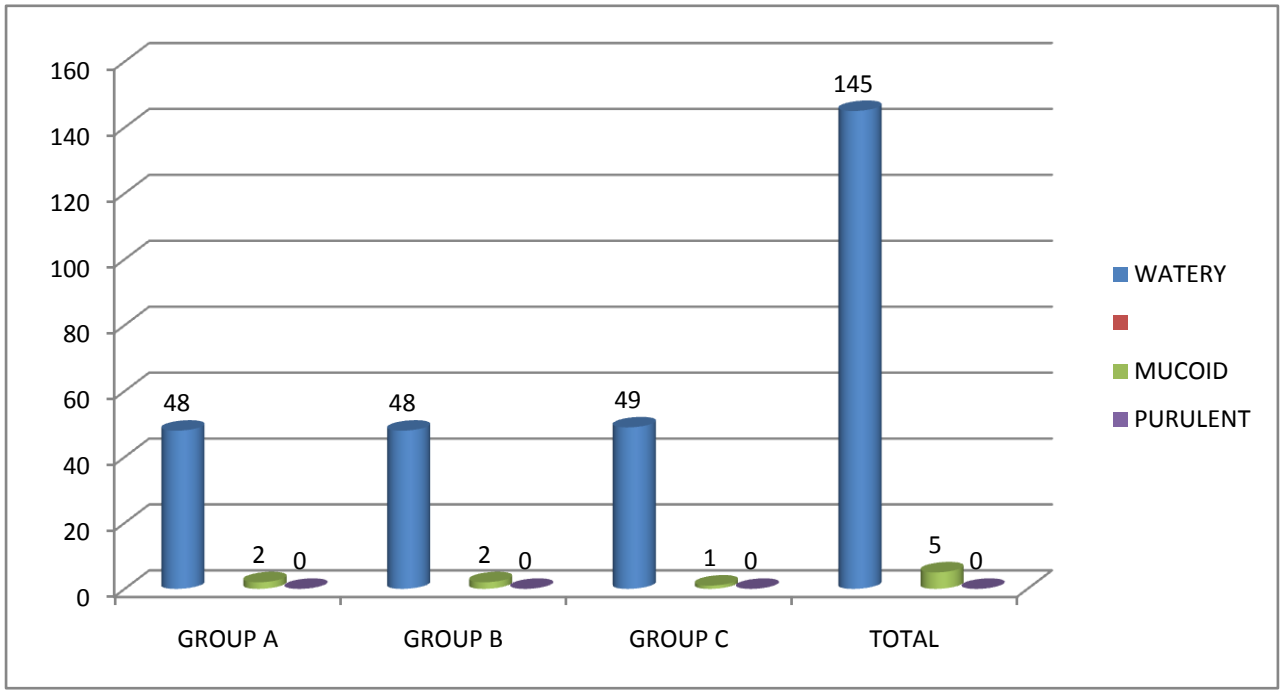
23(15.33 %)patients had NasavarodhaFrom 6-8 yrsamong whom, 8(16%)were in group A, 6(12%)were ingroup B,and9(18%)were ingroup C

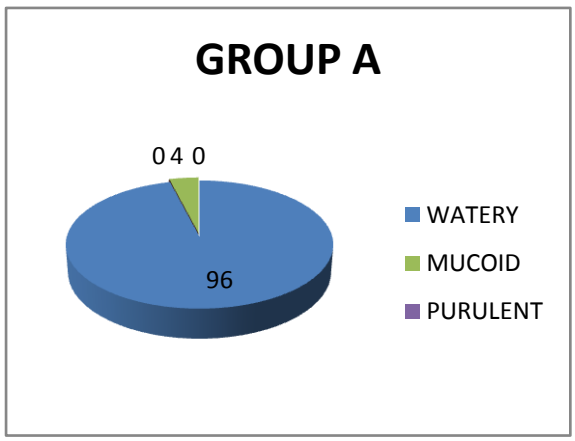
39(26%)patients hadNasavarodhaFrom 4-6 yrsamong whom, 16(32%)were in group A, 11(22%)were ingroup B,and12(24%)were ingroup C

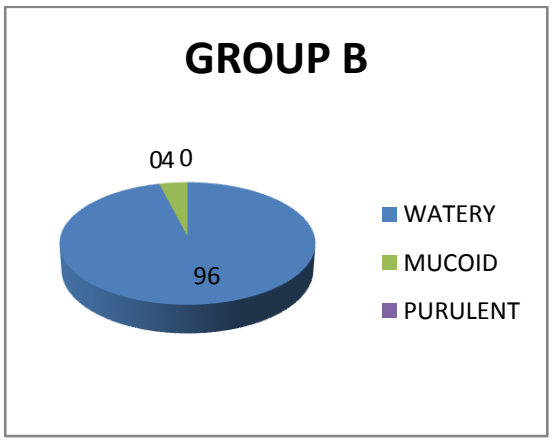
32(21.33%)patients had NasavarodhaFrom 2-4 yrsamong whom, 8(16%)were in group A, 15(30%)were ingroup B,and9(18%)were ingroup C

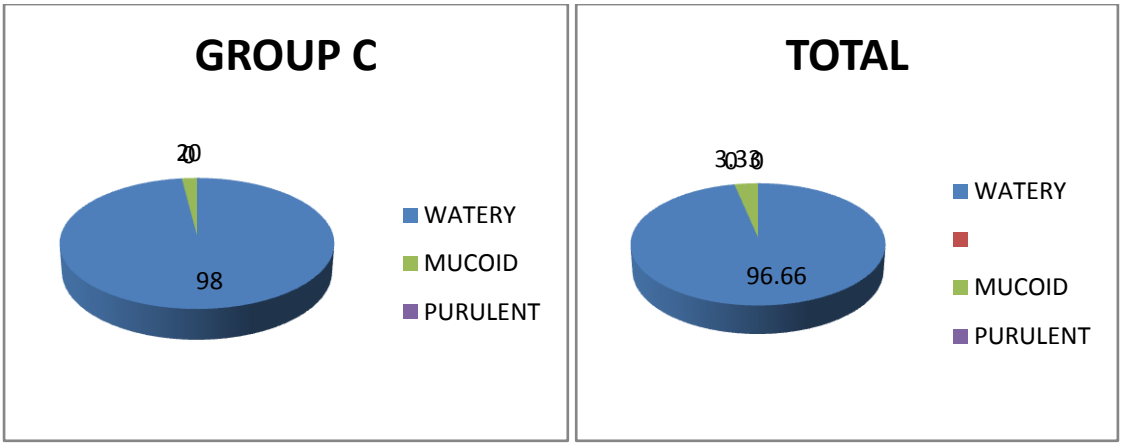
13(8.66%)patients had NasavarodhaFrom 1-2 yrsamong whom,4(08%)were in group A,3(6%)were ingroup B,and6(12%)were in group C

Graph: 21 Nasasrsva type wise distributions of patients with pulmonary hypersensitivity.

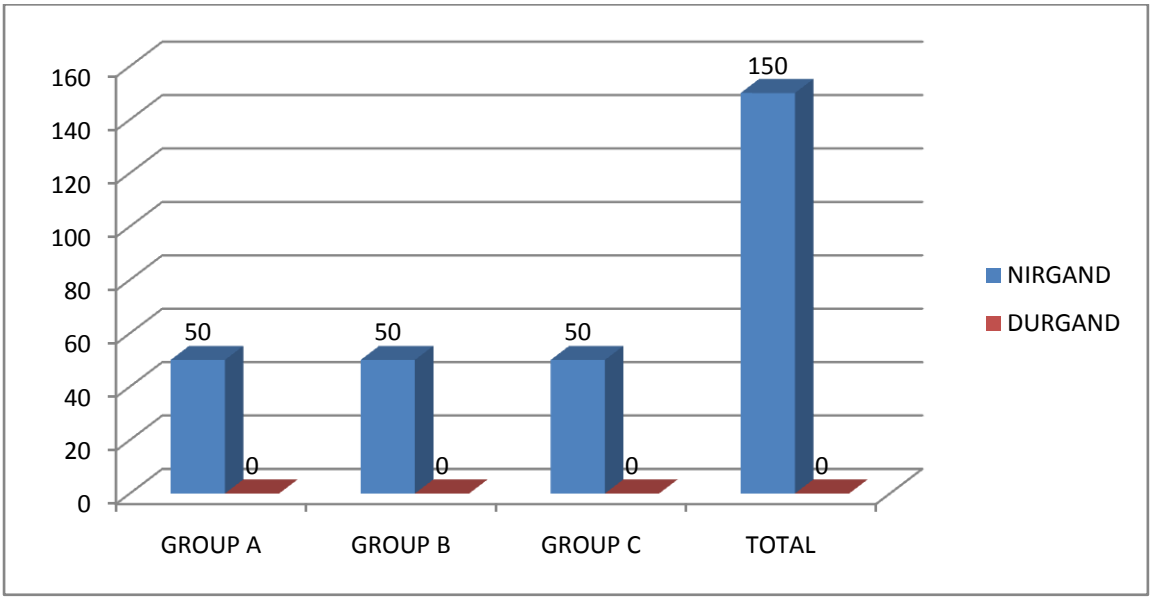


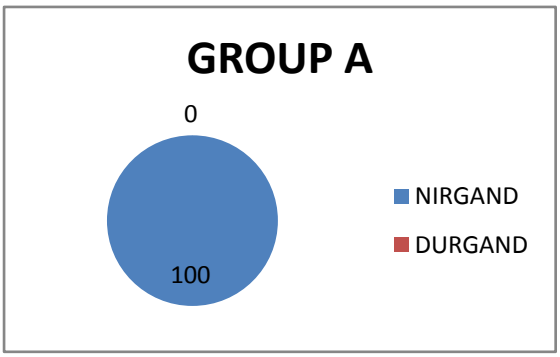


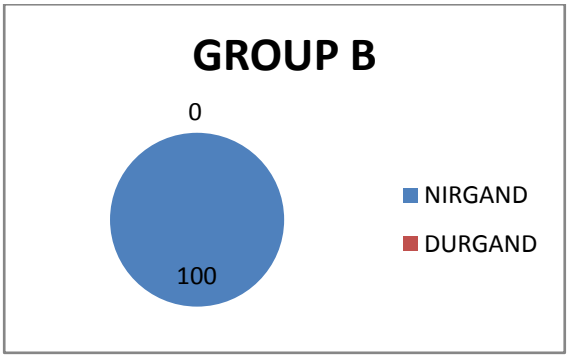


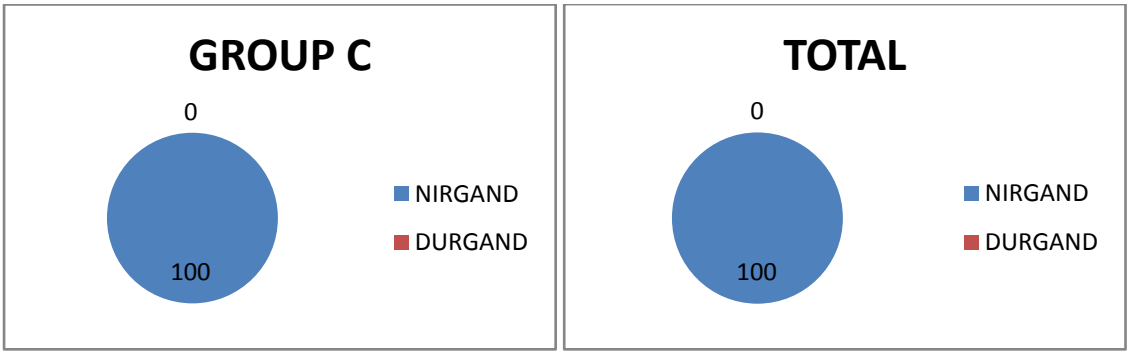


Graph: 22 Nasasrasva gandha wise distributions of patients with pulmonary hypersensitivity.

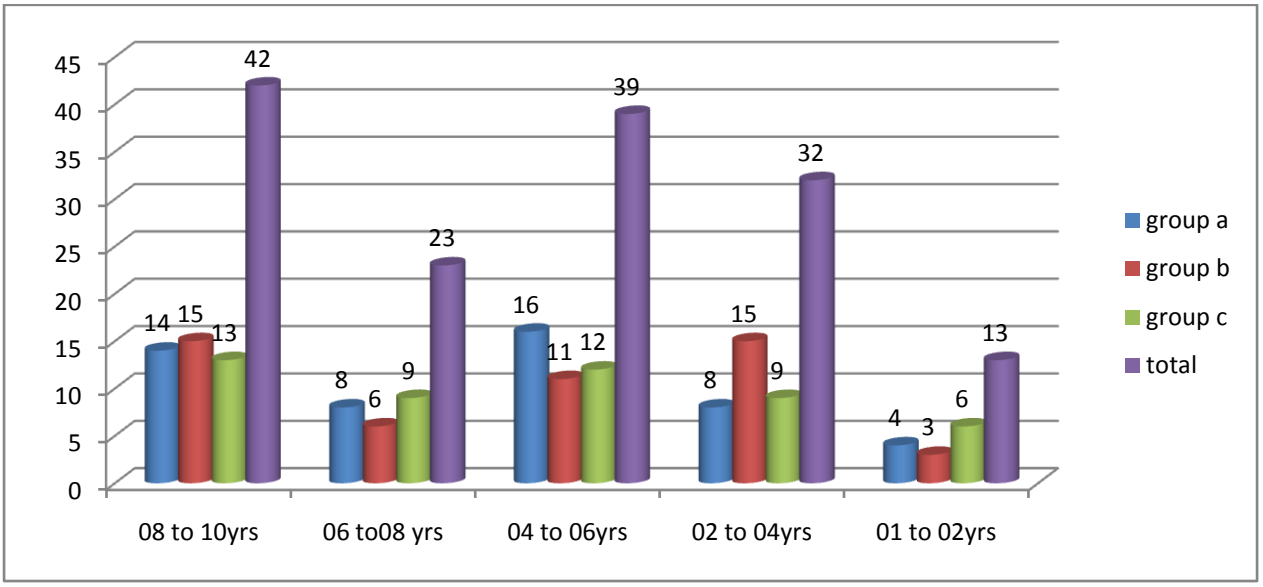


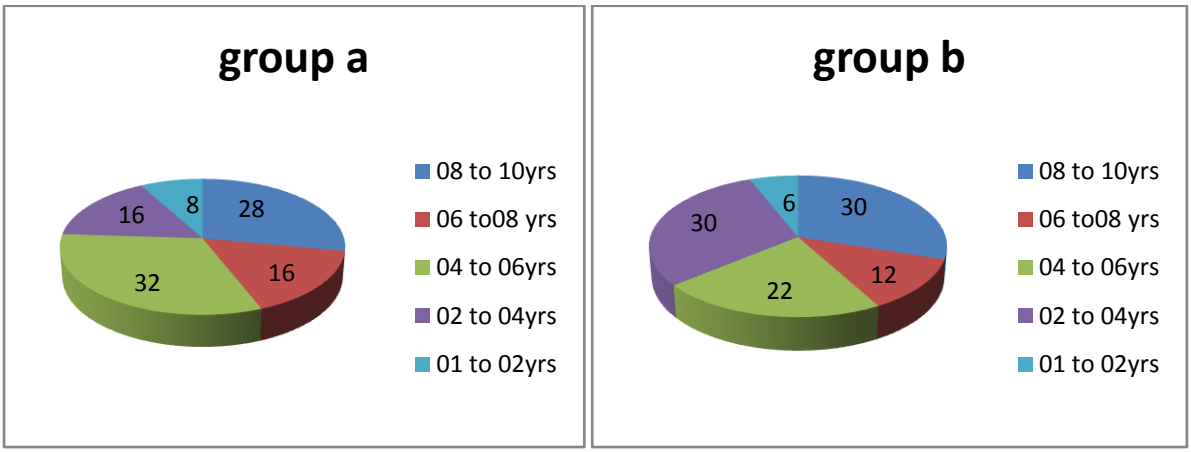


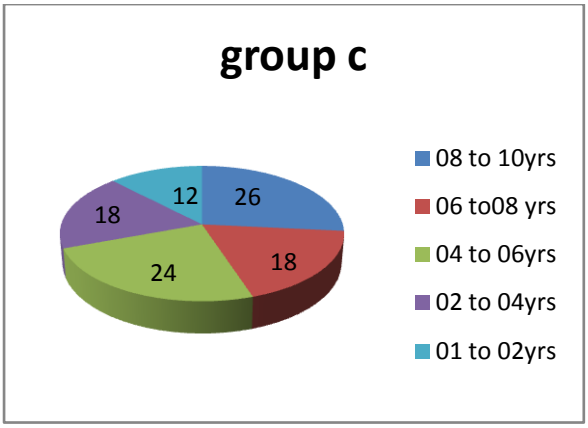




Graph: 23 Nasavarodha(nasal obstruction) duration wise distributions of patients with pulmonary hypersensitivity.







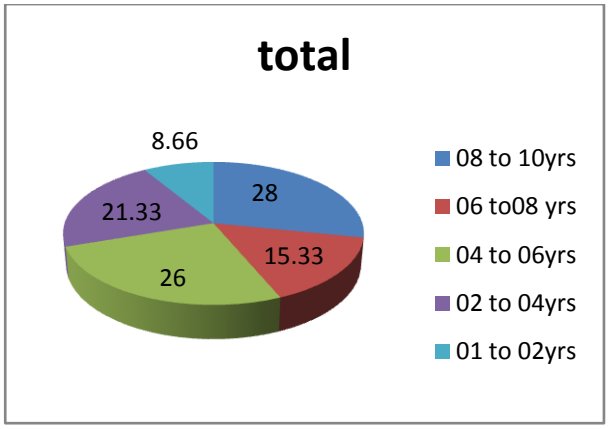


Table No.64

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO MODE OF ONSET OF NASAVARODHA

MODE OF ONSET		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	INSIDIOUS	02	04	08	16	06	12	16	10.66
	GRADUAL	32	64	30	60	31	62	93	62
	ACUTE	06	12	02	04	03	06	11	7.33

Patients had different mode of onset of nasaavarodha,

Among them patients with insidious onset were 16(10.66%)in number,among whom, 2(04%)were in group A, 8(16%)werein group B,and6(12%)werein group C

Patients with gradual onset were 93(62%)in number, among whom, 32 (64%)werein group A,30(60%)werein group B, and 31(62%) were ingroup C

Patients with acuteonset were 11 (7.33%)in number,among whom, 06(12%)were in group A,2(4%)were in group B, and 3(6%)werein group C

Table No.65

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF NASAVARODHA

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	28	56	21	42	12	24	61	40.66
	RECEEDING	03	06	08	16	11	22	22	14.66
	RELAPSING	03	06	11	22	09	18	23	15.33
	STATIONARY	16	32	10	20	18	36	44	29.33

Patients had different pattern of nasaavarodha

Among them patients with progressivenasaavarodha, were 61 (40.66%)in number,among whom, 28(56%)werein group A, 21(42%)were in group B,and12(24%)were in group C.

Patients with precedingnasaavarodha, were 22 (14.66%)in number,among whom, 03(06%)were in group A, 8(16%)were in group B, and 11(22%)were in group C

Patients with relapsingnasaavarodha, were 23 (15.33%)in number,among whom, 03(06%)were in group A,11(22%)were in group B, and 9(18%)were in group C

Patients with stationarynasaavarodha, were 44 (99.33%)in number,among whom, 16(32%)were in group A,10(20%)were in group B,and18(36%)were in group C

Table No.66

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY OF NASAVARODHA

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	08	16	10	20	09	18	27	18
	MODERATE	12	24	09	18	07	14	28	18.66
	SEVERE	31	62	33	66	32	64	95	63.33

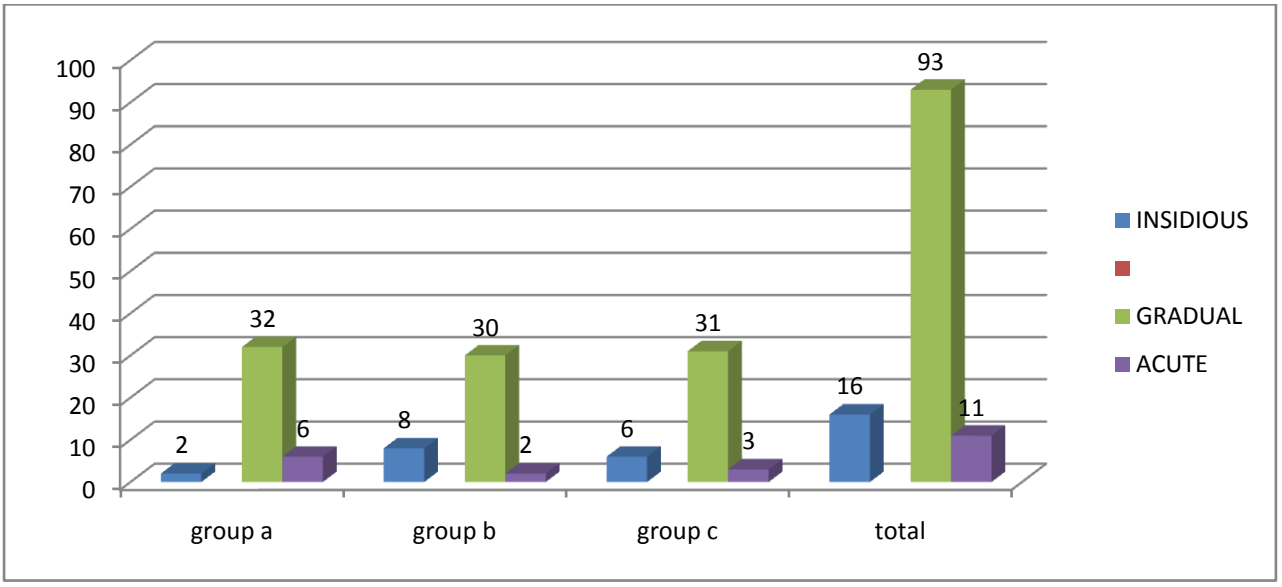
The severity of nasaavarodha was different among the patients,

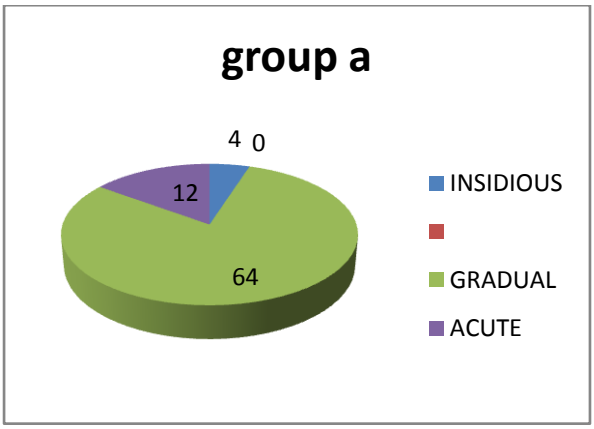
Among them patients mildnasaavarodha were 27 (18%)in number,among whom, 8(16%)were in group A ,10(20%)were in group B,and9(18%)were in group C

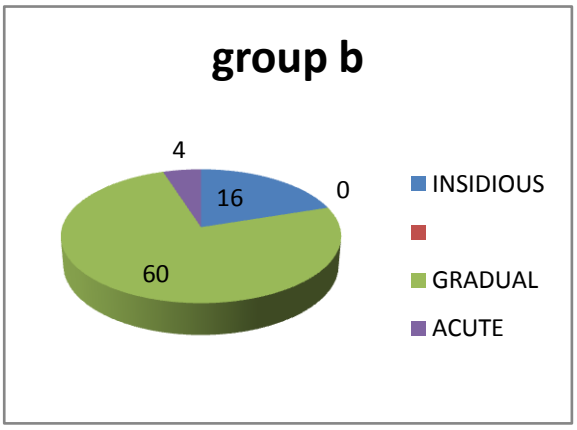
Patients with moderatenasaavarodha were 28 (18.66%)in number,among whom, 12(24%)were in group A, 9(18%)were in group B,and07(14%)were in group C

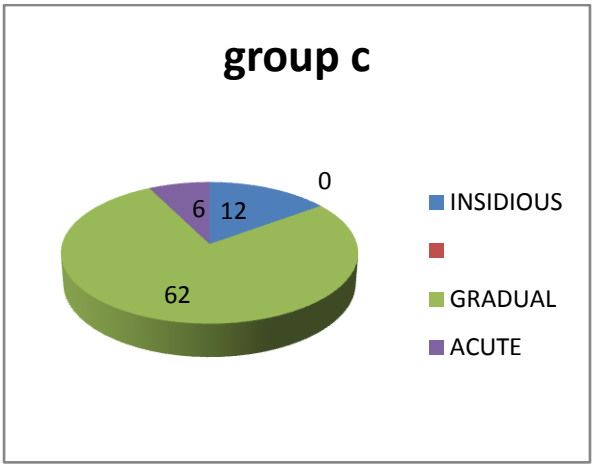
Patients with severenasaavarodha were 95 (63.33%)in number,among whom, 31(62%)were in group A, 33(66%)were in group B,and 32(64%)were in group C

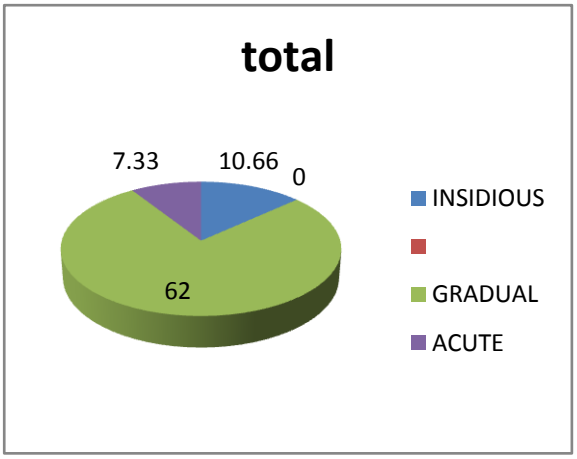
Graph: 24 Nasavarodha modes of onset wise distributions of 150 patients with pulmonary hypersensitivity.



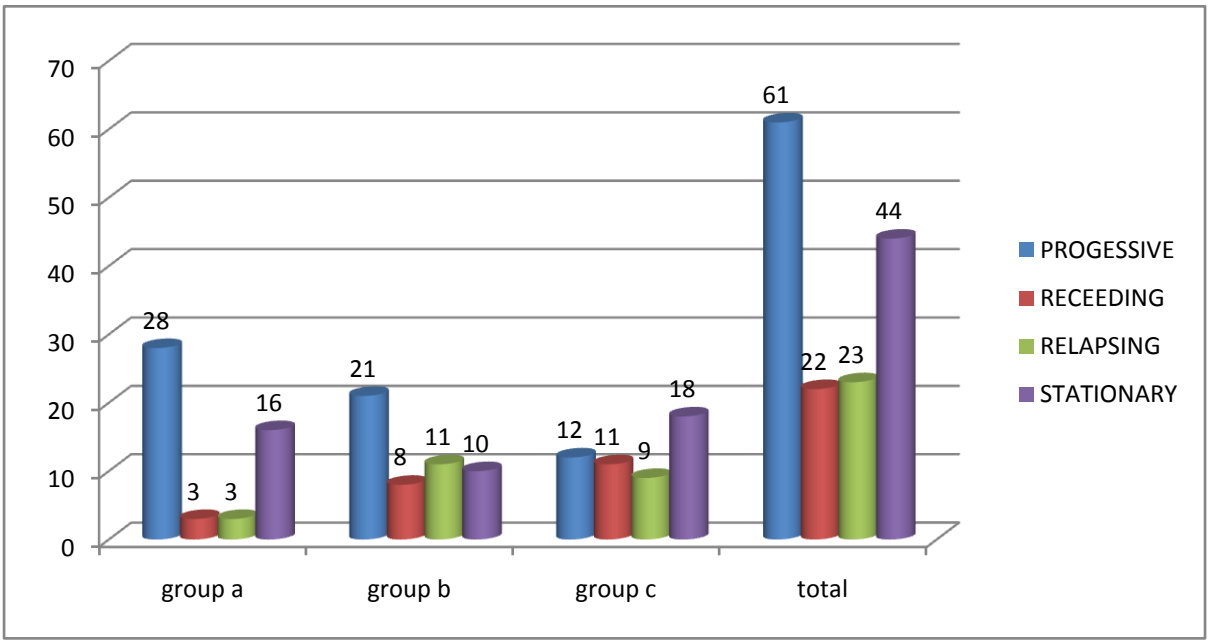


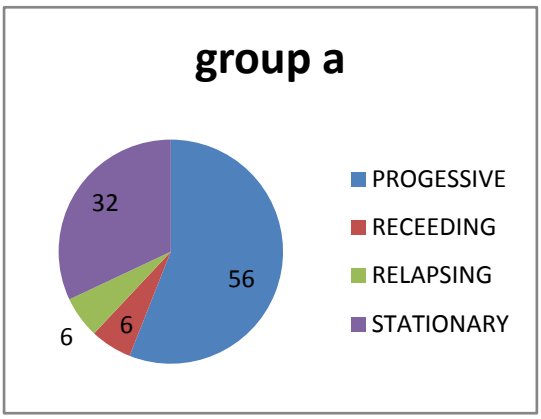


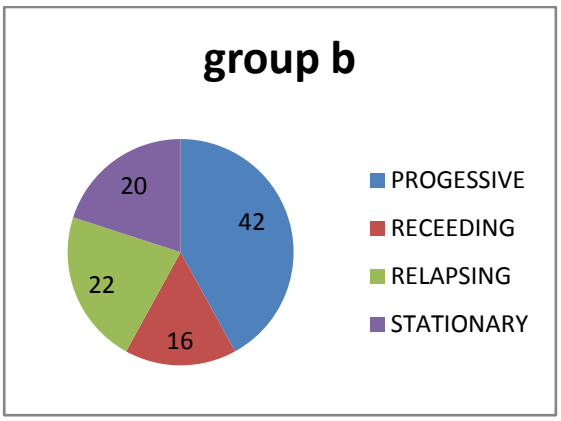


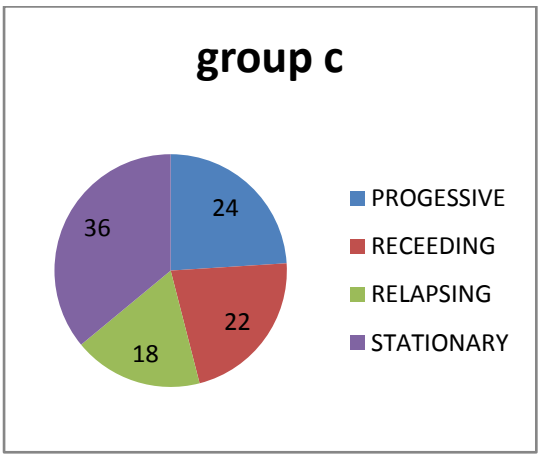


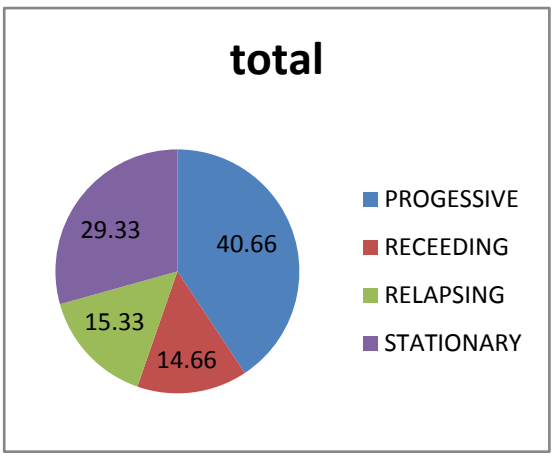
Graph: 25 Nasavarodha pattern wise distributions of patients with pulmonary hypersensitivity.



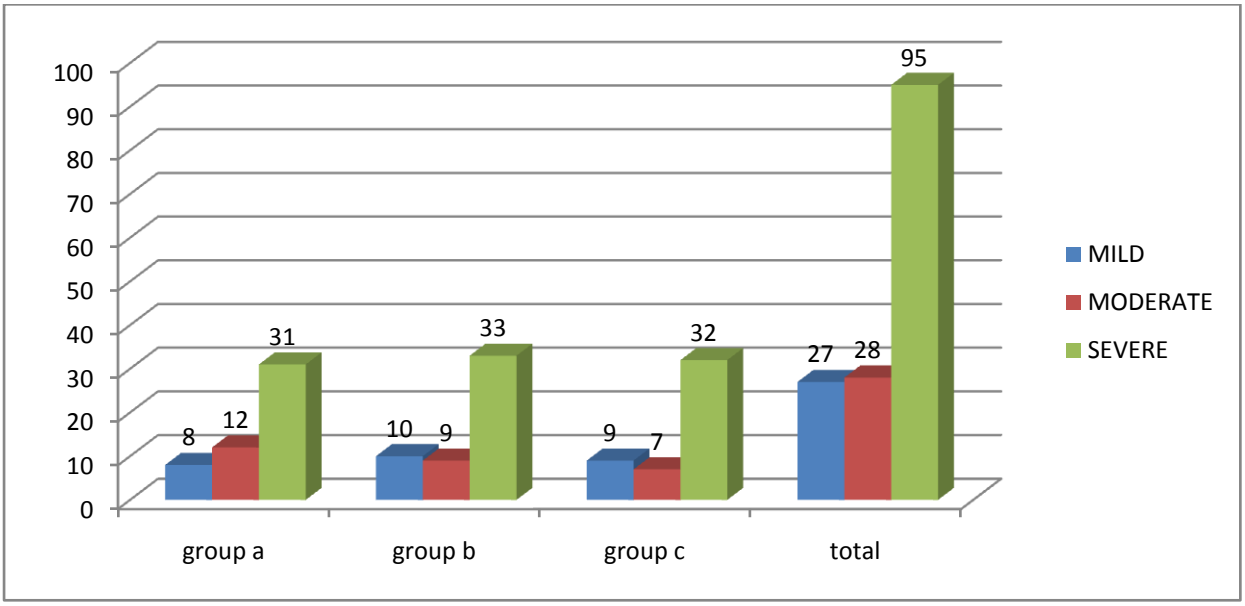


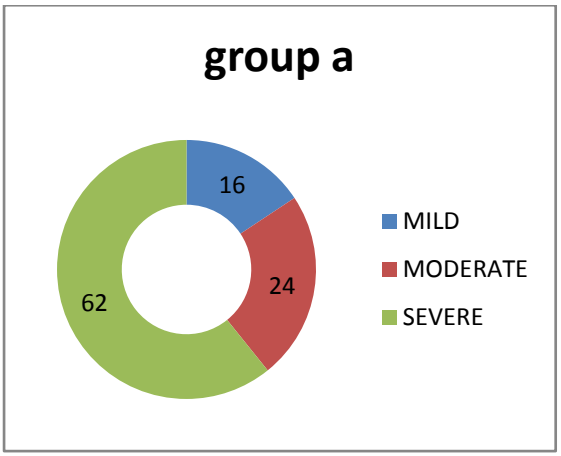


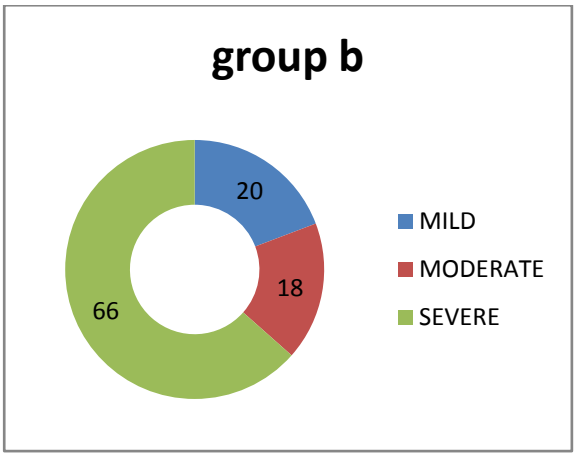


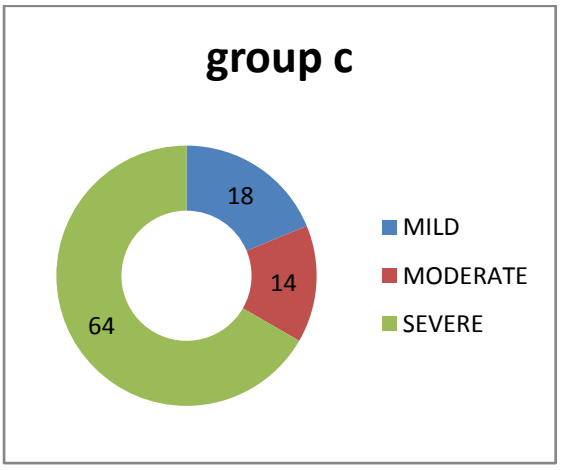


Graph: 26 Nasavarodha severity wise distributions of patients with pulmonary hypersensitivity.









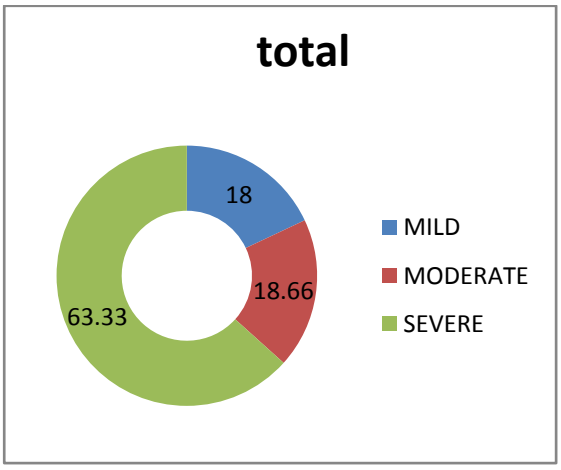


Table No.67

NASASVARODHA KALA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

AVARODHA KALA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	EARLY MORNING	48	96	48	96	49	98	145	96.66
	MORNING	02	04	01	02	01	02	04	2.66
	AFTERNOON	-	-	-	-	-	-	-	-
	EVENING	20	40	15	30	00	00	35	23.33
	NIGHT	44	88	41	82	39	78	124	82.66

The patients had avarodha at different kala

Among all, patients with avarodha early morning were 145 (96.66%), among whom, 48(96%)were in group A, 48(96%)were in group B,and49(98%)were in group C

There Were 04 (2.66%) Patients with avarodhamorning,among whom,02(04%)were in group A,1(2%)were in group B, and 1(2%)were in group C

None of the patients depicted avarodha in afternoon.

There Were 35 (23.33%)Patients with avarodha in evening, among whom, 20(40%) were in group A,15(30%)were in group B, none were in group C

There were 124 (82.66%)Patients with avarodhaat night, among whom44(88%) were in group A,41(82%)were in group B, and 39(78%)were in group C.

KSHAVATHU (SNEEZING)

Table No.68

KSHAVATU(SNEEZING) DURATION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

DURATION		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	08- 10 YRS	14	28	12	24	19	38	45	30
	06-08 YRS	08	16	10	20	09	18	27	18
	04- 06 YRS	16	32	18	36	17	34	51	34
	02-04 YRS	08	16	05	10	02	04	15	10
	01-02 YRS	04	08	05	10	03	06	12	08

Patients had kshavatu of different duration

45(30%)Patients had kshavatuFrom 8- 10 yrs,among whom, 14(28%)were in group A,12(24%)were in group B, and 19(38%)were in group C

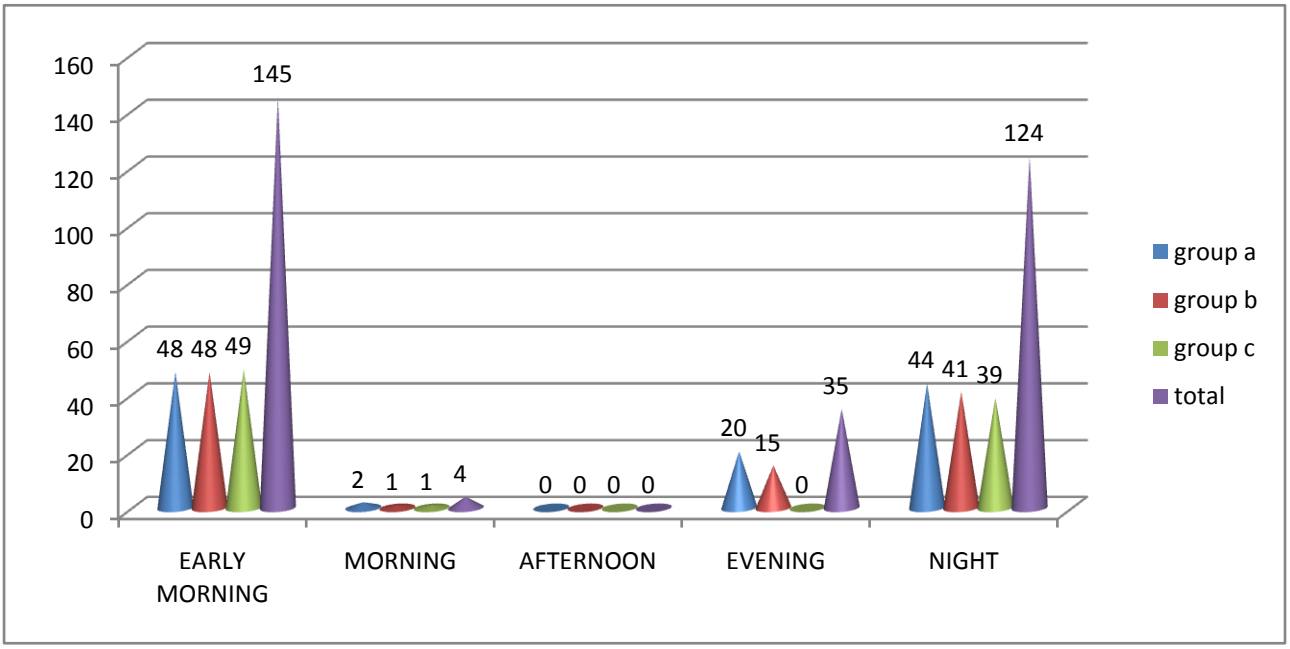
27(18 %)Patients had kshavatu 6-8 yrs,among whom,A 8(16%) were in group, 10(20%)were in group B, and , 9(18%)were in group C

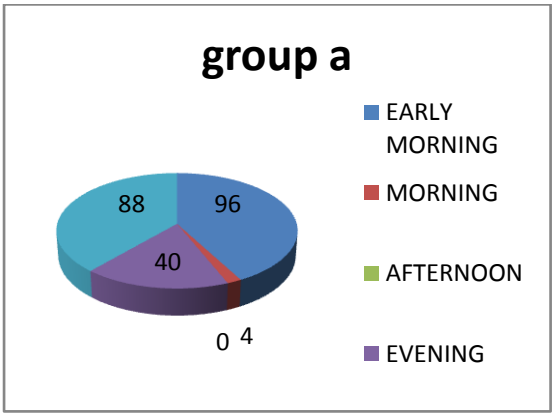
51(34%)Patients had kshavatu 4-6 yrs,among whom, 16(32%)were in group A,18(36%)were in group B, and 17(34%)were in group C

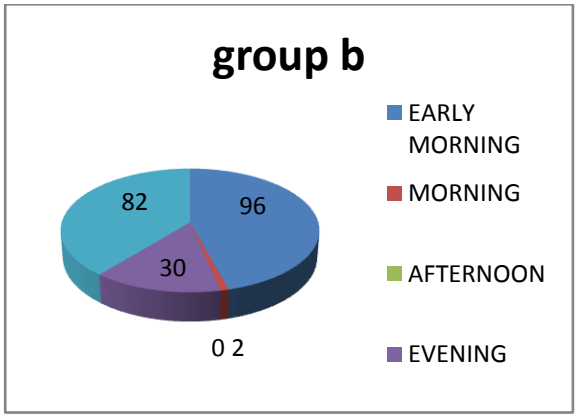
15(10%)Patients had kshavatu 2-4 yrs, among whom, A 8(16%)werein group, 5(10%)were in group B, and 2(4%)were in group C

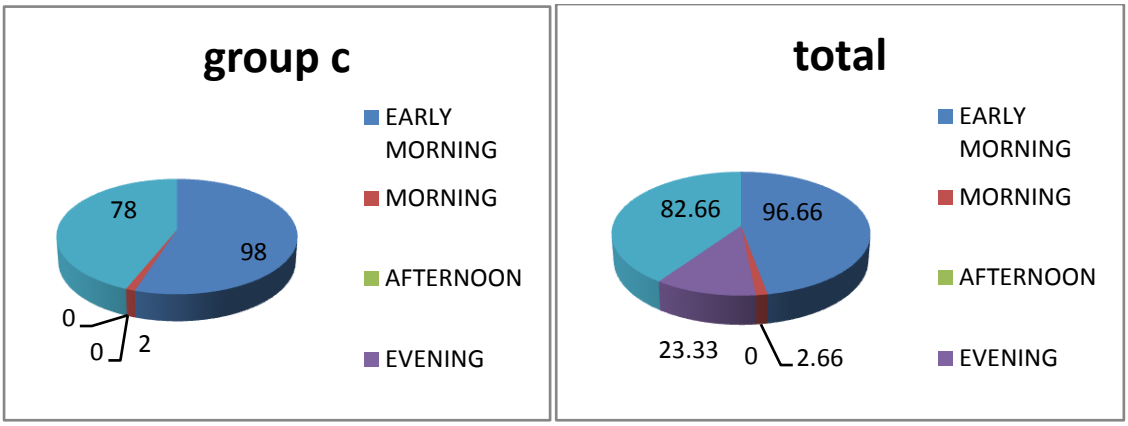
12(8%)Patients had kshavatu 1-2 yrs,among whom,4(08%) were in group A, 5(10%)were in group B, and 3(6%)were in group C

Graph: 27 Nasavarodha kala wise distributions of patients with pulmonary hypersensitivity

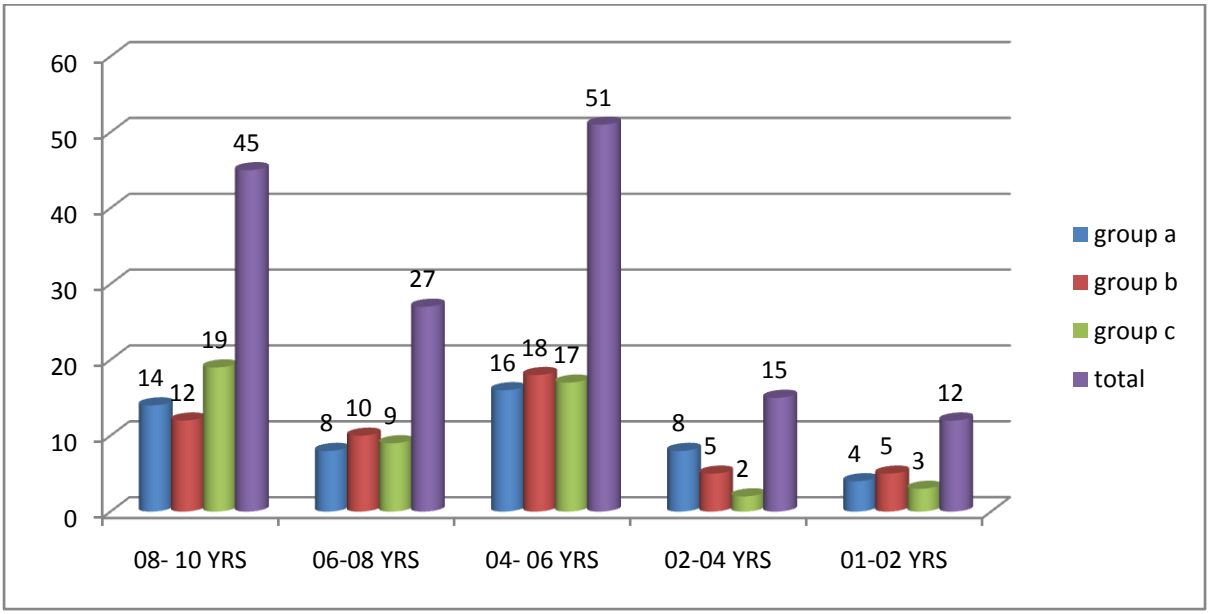


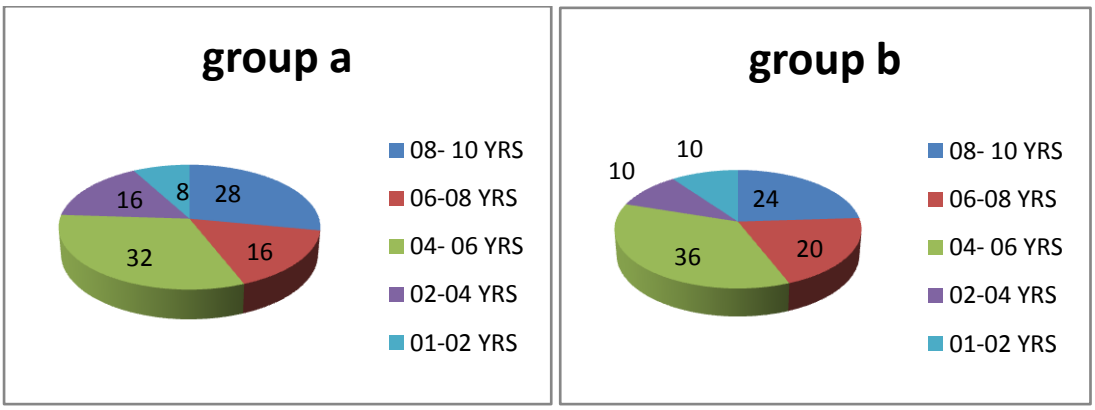






Graph: 28 Kshavatu(sneezing) duration wise distributions of patients with pulmonary hypersensitivity.





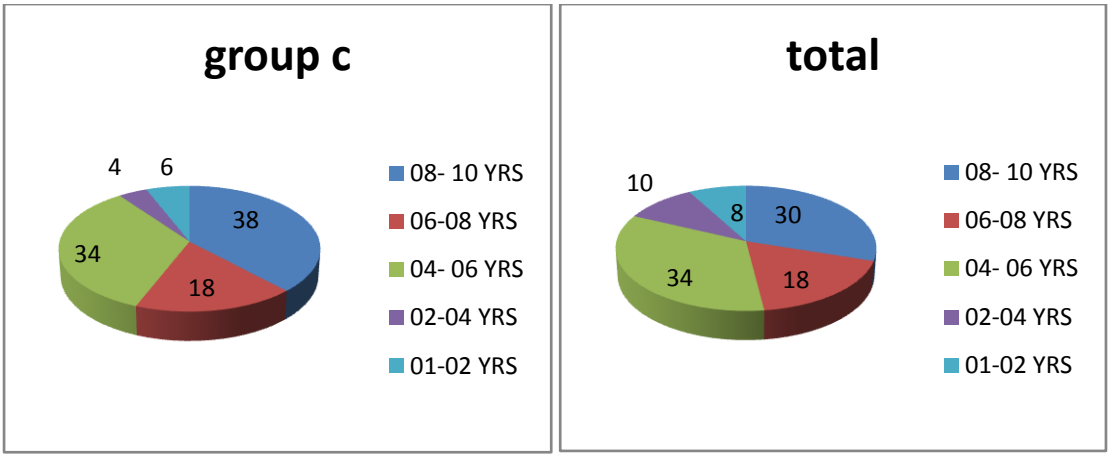


Table No.69

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO MODE OF ONSET OF KSHAVATU

MODE OF ONSET		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	INSIDIOUS	03	06	05	10	04	08	12	08
	GRADUAL	30	60	30	60	25	50	85	56.66
	SUB- ACUTE	15	30	10	20	11	22	36	24
	ACUTE	02	04	05	10	10	20	17	11.33

Patients had different mode of onset of kshavatu,

Among them patients with insidious onset were 12(8%), among whom, 3(6%)werein group A, 5(10%)were in group B,and4(8%)were in group C

85(56.66%)Patientswerewith gradualonset, among whom, 30 (60%)were in group A, 30(60%)were in group B, and 25 (50%)were in group C

36 (24%)Patientswerewithsub-acuteonset,among whom, 15(30%)were in group A, 10(20%)were in group B, and11 (22%)were in group C

17 (11.33%)Patientswere withacuteonset, among whom, 02(04%)were in group A, 05(10%)were in group B,10(20%)were in group C

Table No.70

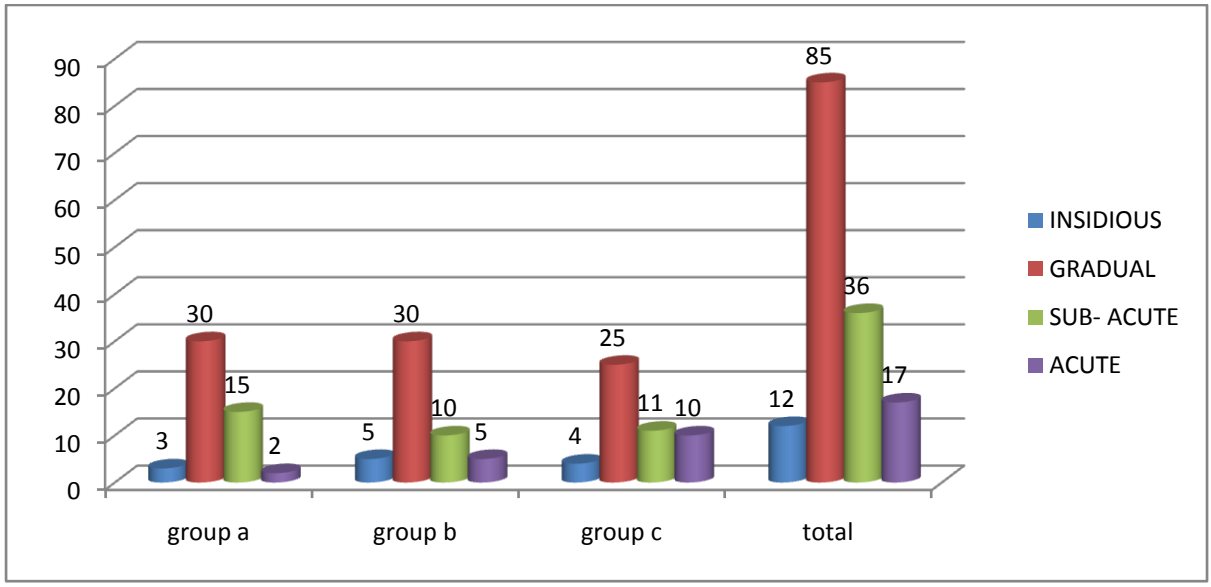
NUMBER OF KSHAVATU WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

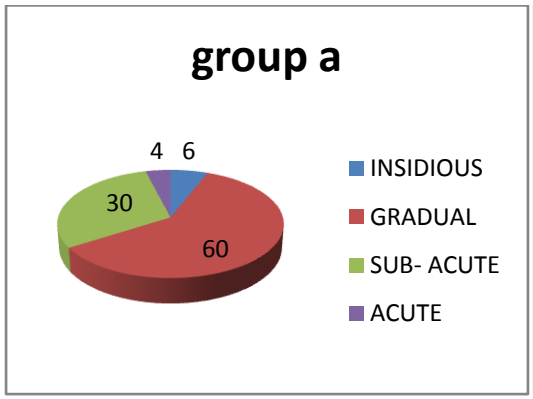
NO. OF KSHAVATHU		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	30- 40	10	20	11	22	16	32	37	24.66
	20- 30	28	56	24	48	21	42	73	48.66

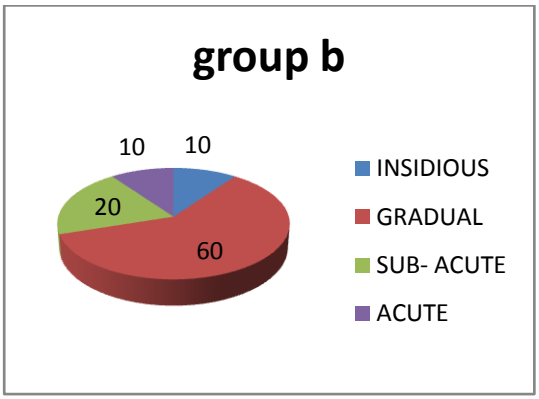
Among 150 patients, 37(24.66%) had number of kshavathus between 30- 40, in whom,10(20%)were in group A, 11(22%)were in group B, and16 (32%)were in group C

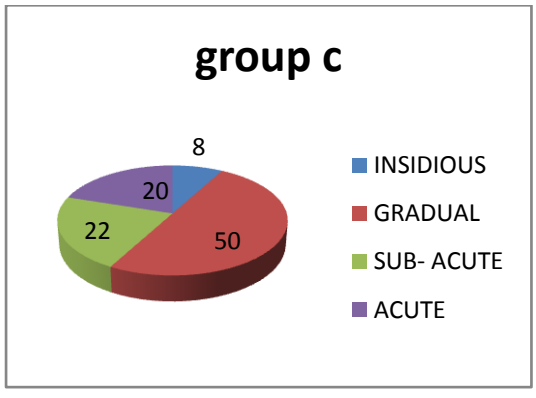
73(48.66%) patients had number of kshavatusBetween 20-30, in whom,28(56%)were in group A, group24(48%)were in B, 21(42%)were in group C

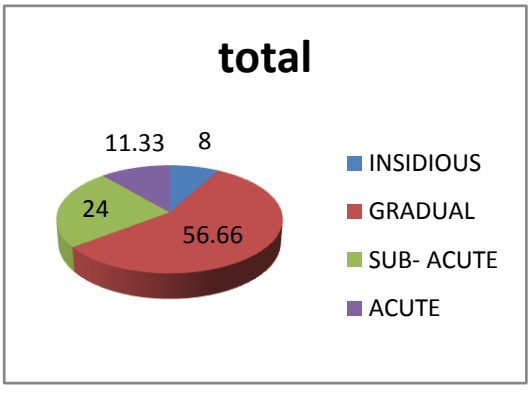
Graph: 29 Kshavatu modes of onset wise distributions of patients with pulmonary hypersensitivity.



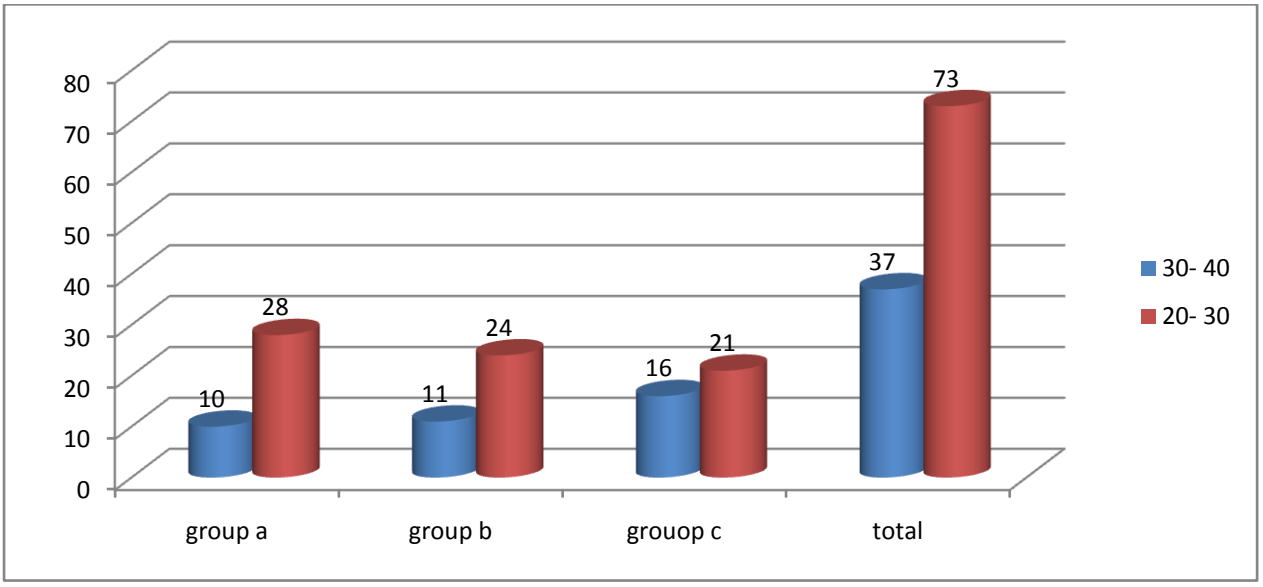


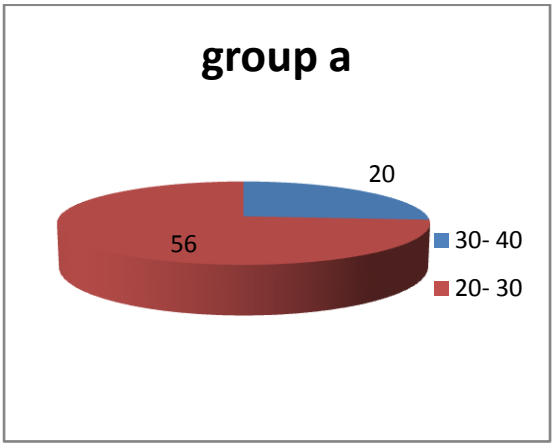


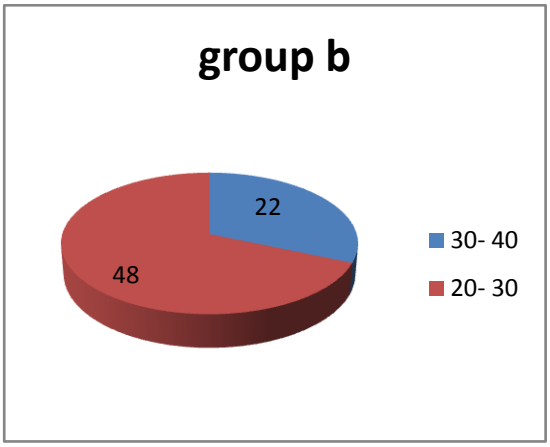


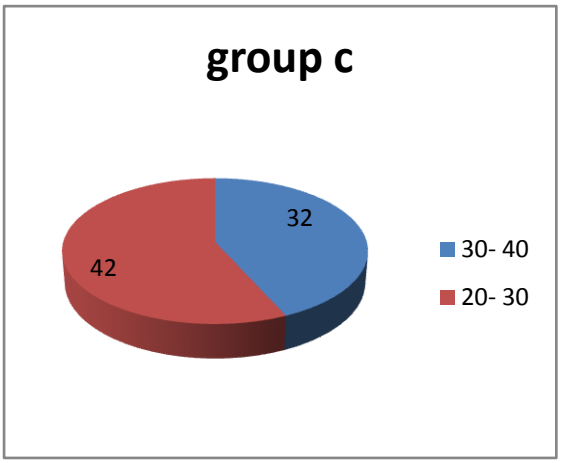


Graph: 30 Number of Kshavatu wise distributions of patients with pulmonary hypersensitivity.









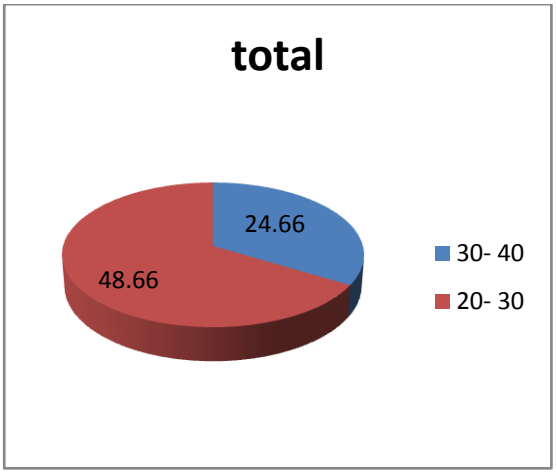


Table No.71

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY OF KSHAVATU

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	07	14	05	10	08	16	20	13.66
	MODERATE	10	20	15	30	11	22	36	24
	SEVERE	33	66	30	60	31	62	94	62.66

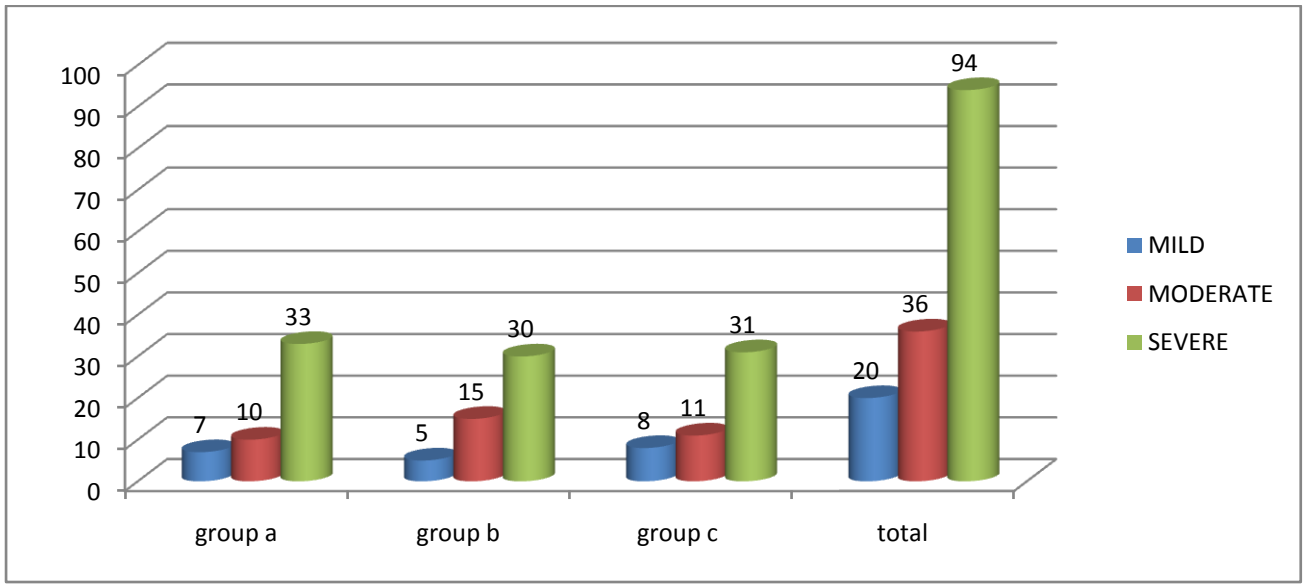
Patients had kshavatu of different severity,

20 (13.66%) patients had mild kshavatu, among whom, 7(14%)werein group A, 5(10%)were in group B, and8(16%)were in group C

36 (24%) patients had moderate kshavatu,among whom, 10(20%)werein group A,15(30%)were in group B,and 11(22%)were in group C

94 (62.66%) patients had sevsrekshavatu, among whom, 33(66%)werein group A, 30(60%)were in group B, and 31(62%)were in group C

Graph: 31 Kshavatu severity wise distributions of patients with pulmonary hypersensitivity.



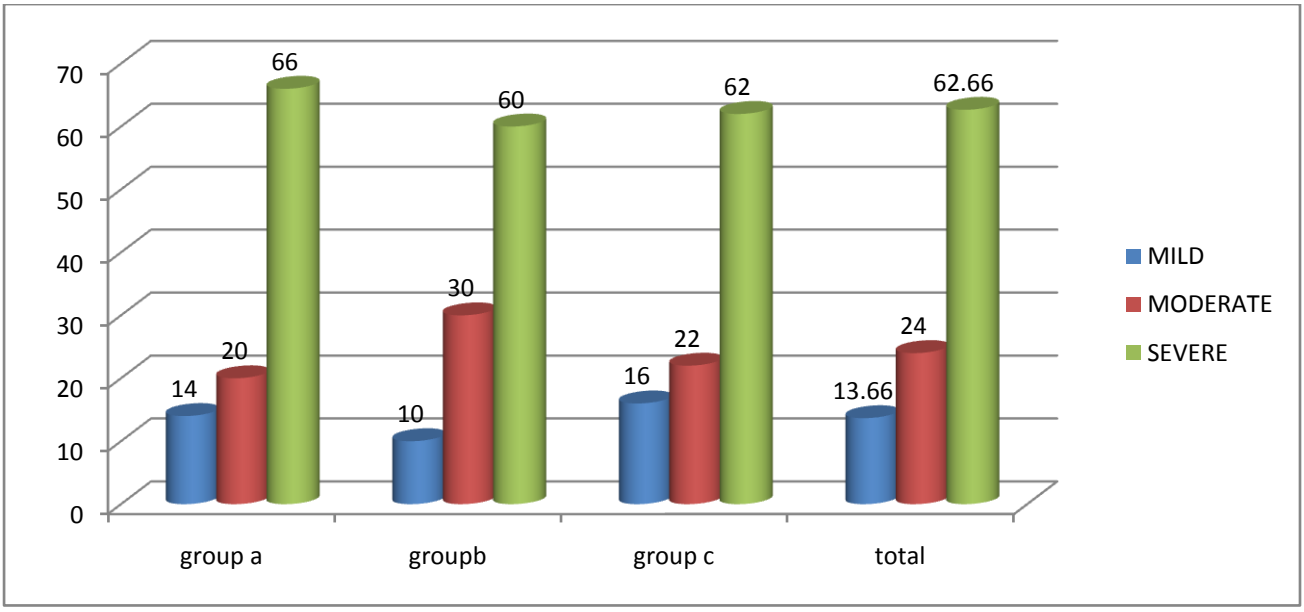


Table No.72

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF KSHAVATU

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	26	52	30	60	35	70	91	60.66
	RECEEDING	02	04	01	02	01	02	04	2.66
	RELAPSING	18	36	14	28	13	26	45	30
	STATIONARY	04	08	05	10	01	02	10	6.66

The patients had kshavatu of different pattern

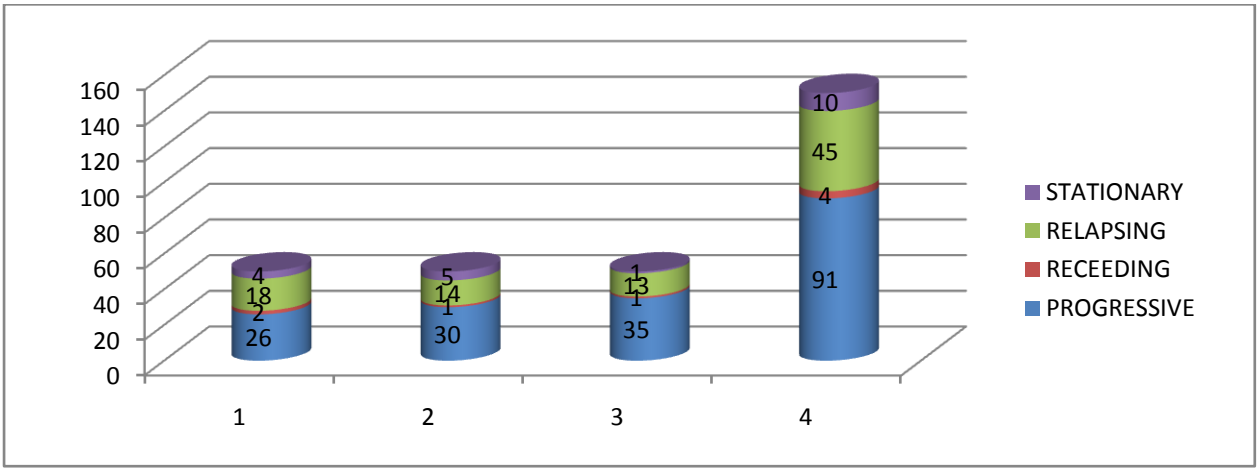
Among them patients with progressivekshavatu were 91 (60.66%) in whom, 26(52%)werein group A, 30(60%)were in group B,and 35(70%)were in group C

Patients with precedingkshavatu were 4 (2.66%),in whom,02(04%) were in group A, 1(2%)wasin group B,and1(2%)was in group C

Patients with relapsing kshavatu 45 (30%), in whom,18(36%) were, in group A,14(28%)werein group B,and13(26%)werein group C

Patients with stationarykshavatu were 10 (6.66%),in whom, 04(08%)were,in group A,05(10%)were in group B,and1 (2%) was in group C

Graph: 32 Kshavatu pattern wise distributions of patients with pulmonary hypersensitivity.



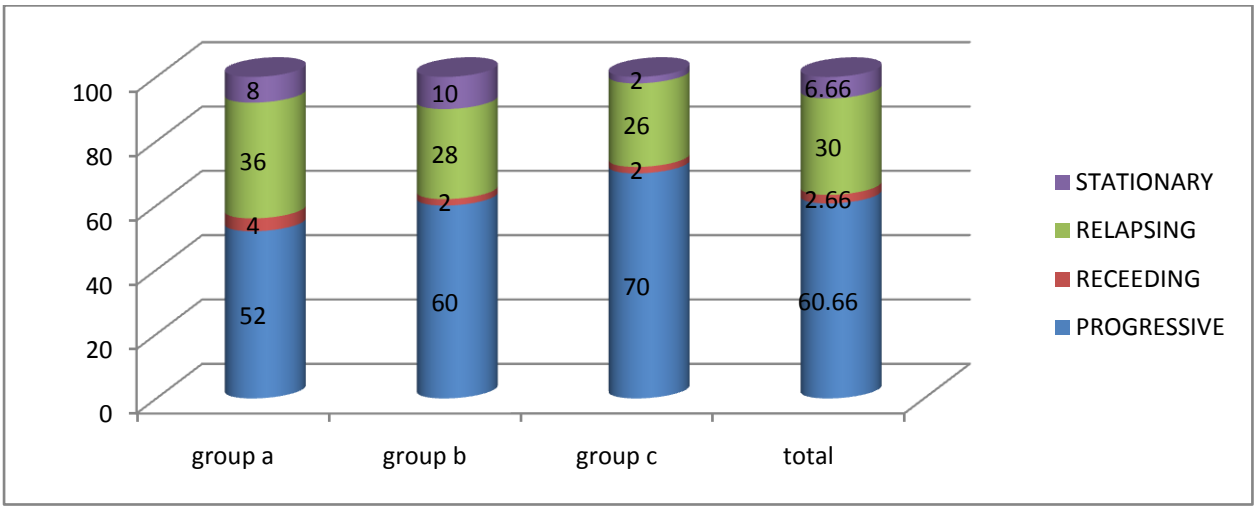


Table No.73

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING SEVERITY OF NASASRAVA AFTER KSHAVATU

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	07	14	05	10	08	16	20	13.66
	MODERATE	10	20	15	30	11	22	36	24
	SEVERE	33	66	30	60	31	62	94	62.66

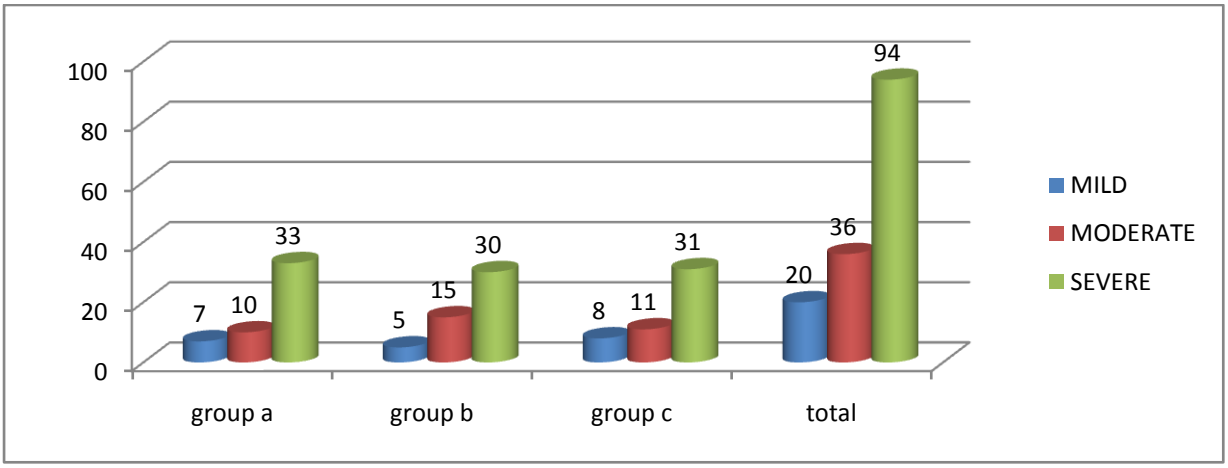
The patients in trial had nasasrava of different severity.

Patients with mild symptoms were 20 (13.66%), among whom, in group A 7(14%)patients,in group B 5(10%) patients, and in group C 8(16%)patients were present.

Patients with moderate symptoms were 36 (24%), among whom, in group A10(20%) patients, in group B 15(30%) patients, and in group C11(22%)patients were present.

Patients with severe symptoms were 94 (62.66%), among whom, in group A 33(66%)patients , group B 30(60%) patients, andin group C31(62%)patients were present.

Graph: 33 Distribution of patients with pulmonary hypersensitivity according to severity of nasasrava after kshavatu



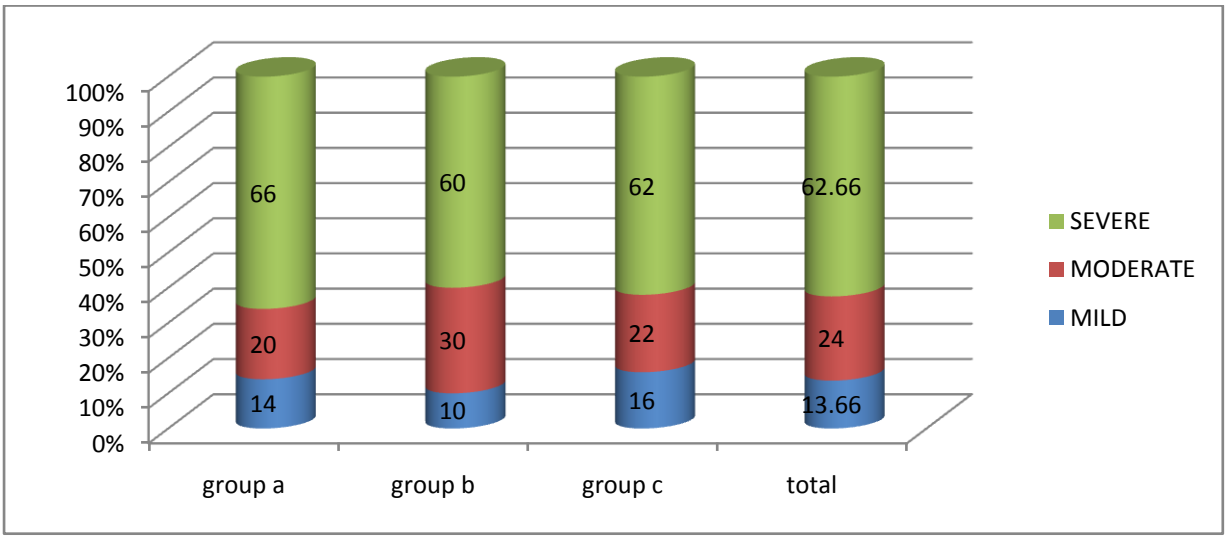


Table No.74

NUMBER OF KSHAVATUS PER DAY WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

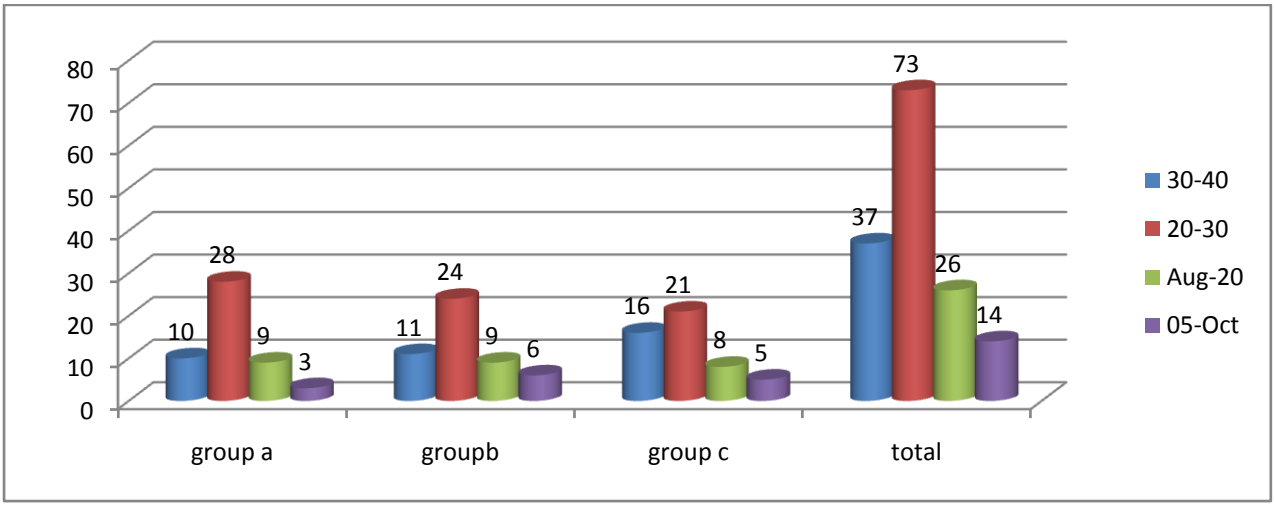
NUMBER OF KSHAVATHU		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	30-40	10	20	11	22	16	32	37	24.66
	20-30	28	56	24	48	21	42	73	48.66
	10-20	09	18	09	18	08	16	26	17.33
	05-10	03	06	06	12	05	10	14	9.33

Patients had different number of kshavathu in an entire day,

Among them number of patients with number of kshavatus between 30- 40 were 37(24.66%), among whom,10(20%) were in group A, 11(22%)werein group B, and16 (32%)were in group C

Patients with number of kshavatus between 20-30 were 73(48.66%),%), among whom, 28(56%)were in group A, 24(48%)were in group B,and21(42%)were in group C

Graph: 34 Kshavatu number per day wise distributions of patients with pulmonary hypersensitivity.



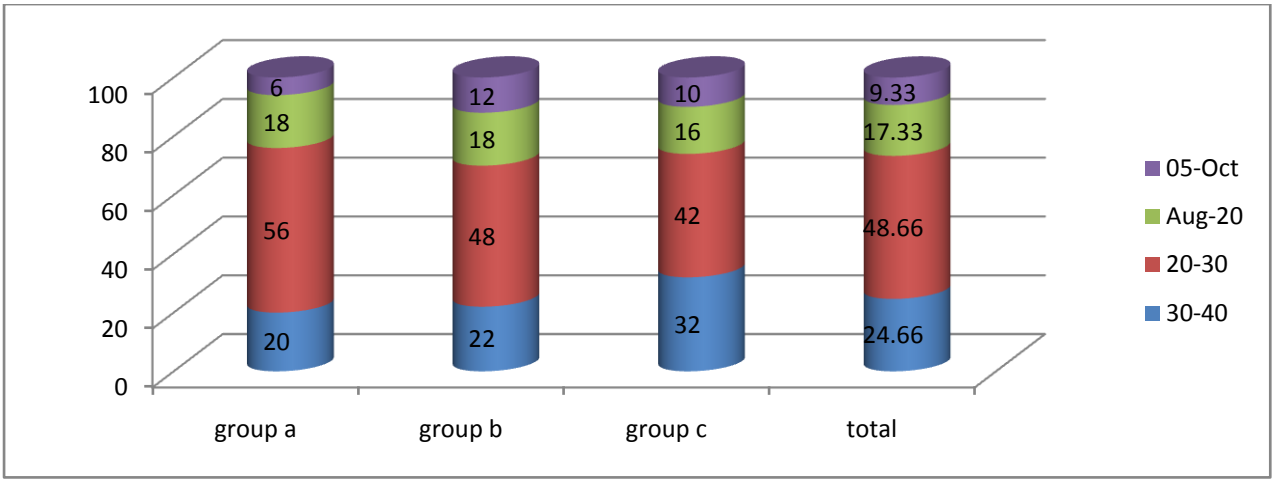


Table No.75

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO NUMBER OF KSHAVATUS AT A TIME CONTINIOUSLY

	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
15 TO 20	09	18	10	20	07	14	26	17.33
10 TO 15	10	20	05	10	13	26	28	18.66
05 TO 10	28	56	29	58	26	52	83	55.33
00 TO 05	03	06	06	12	04	08	13	8.66

Further the patients had different number of kshavathu at a time continuously,

patients with number of kshavatus between 15-20 were 26(17.33%),among whom, 09(18%)were in group A, 1(20%)were in group B,and 7(14%)were in group C

Patients with number of kshavatusbetween 10-15 were 28(18.66%),among whom,10(20%) were in group A,5(10%)were in group B, and 13(26%)were in group C

Patients with number of kshavatusbetween 05-10 were 83(55.33%),among whom, 28(56%)were in group A, 29(58%)were in group B, and 26(52%)were in group C

Patients with number of kshavatus between 0-05 were 13(8.66%),among whom, 3(6%)were in group A, 6(12%)were in group B,and 4(8%)were in group C

Graph: 35 Distribution of patients with pulmonary hypersensitivity according to number of kshavatus at a time.

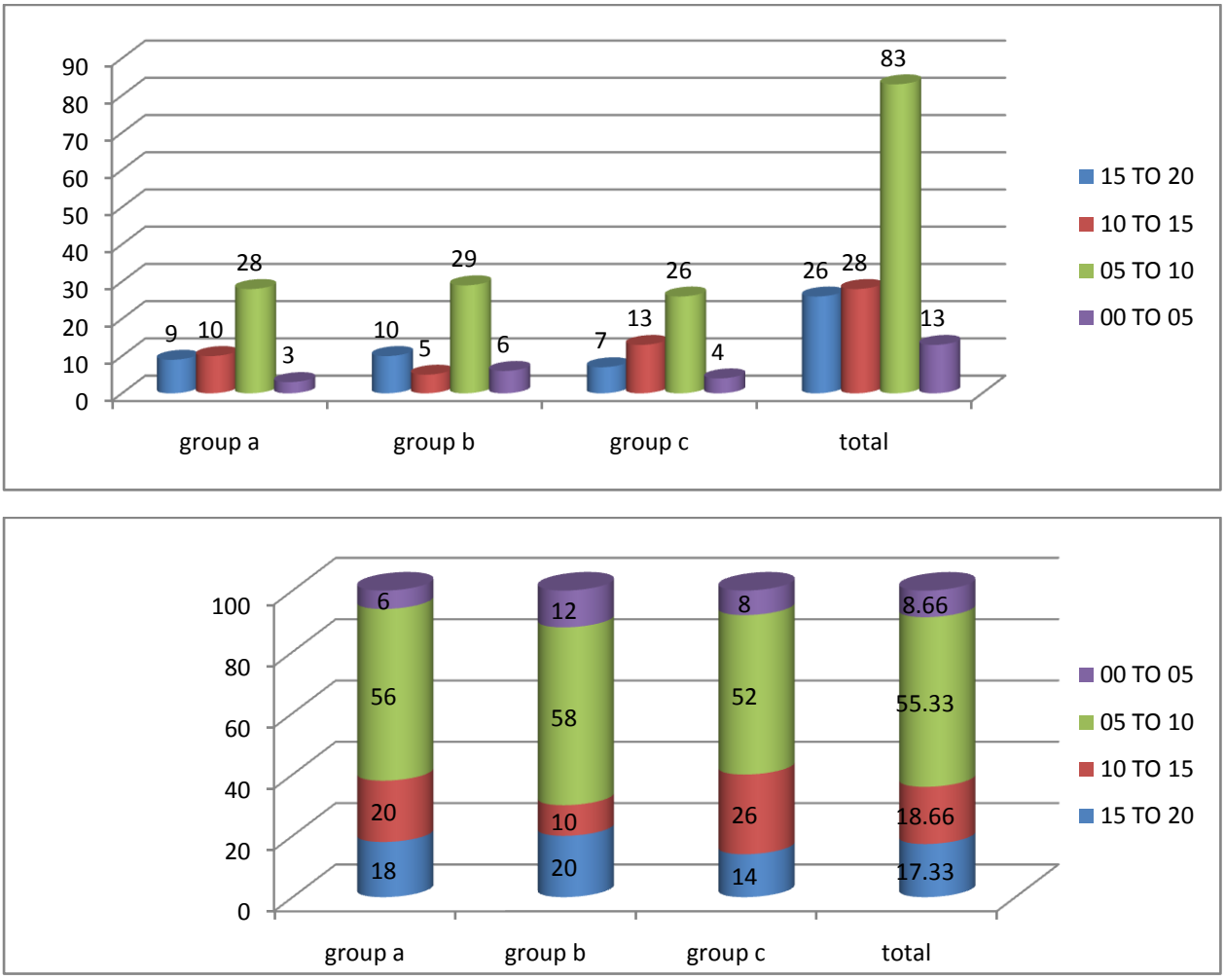


Table No.76

NASA KANDU DURATION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

DURATION		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	08-10Yrs	10	20	13	26	11	22	34	22.66
	06-08Yrs	15	30	03	06	09	18	27	18
	04-06Yrs	14	28	16	32	15	30	45	30
	02-04Yrs	08	16	09	18	10	20	17	11.33

The patients selected for the clinical trial were having nasakanduof various duration.

Among them 34 (22.66%) patients were havingnasakandufrom 08-10 years. In group A, 10(20%) patients, group B, 13(26%) patients and group C, 11 (22%) patients were present.

Among them 27(18%) patients were having nasakandu for 06-08 years . In group A, 15(30%) patients, group B,03(06%) patients and group C, 09 (18%) patients were present.

Among them 45 (30%) patients were having nasakandu for 04-06 years . In group A, 14(28%) patients, group B, 16(32%) patients and group C, 15 (30%) patients were present.

Among them 17(11.33%) patients were having nasakandu for 02-04 years. In group A, 08(16%) patients, group B, 09(18%) patients and group C,15(30%) patients were present.

Table No.77

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO MODE OF ONSET OF NASA KANDU

MODE OF ONSET		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	INCIDIOUS	03	06	06	12	09	18	18	12
	GRADUAL	29	58	23	46	28	56	80	53.33
	SUB-ACUTE	09	18	15	30	09	18	33	32
	ACUTE	09	18	06	12	04	08	19	12.66

The patients selected for the clinical trial were having nasakandu of various modes of onset.

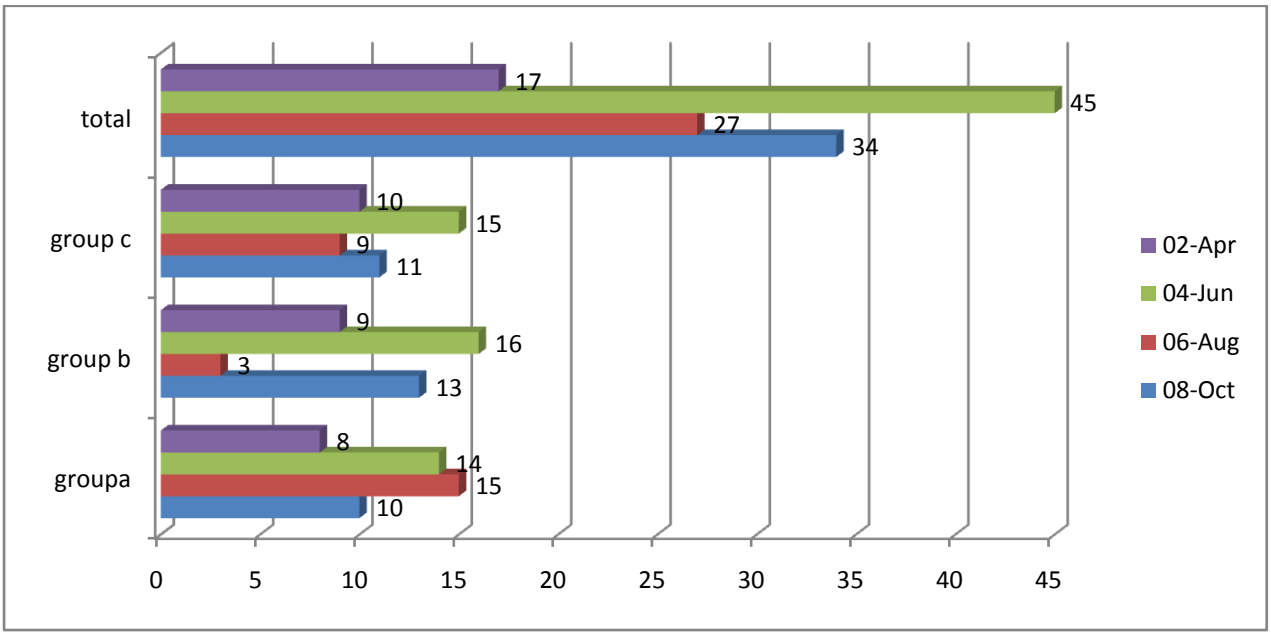
Among them 18 (12%) patients were having insidious onset . In group A 03(06%) patients, group B 06(12%) patients and group C, 09(18%) patients were present.

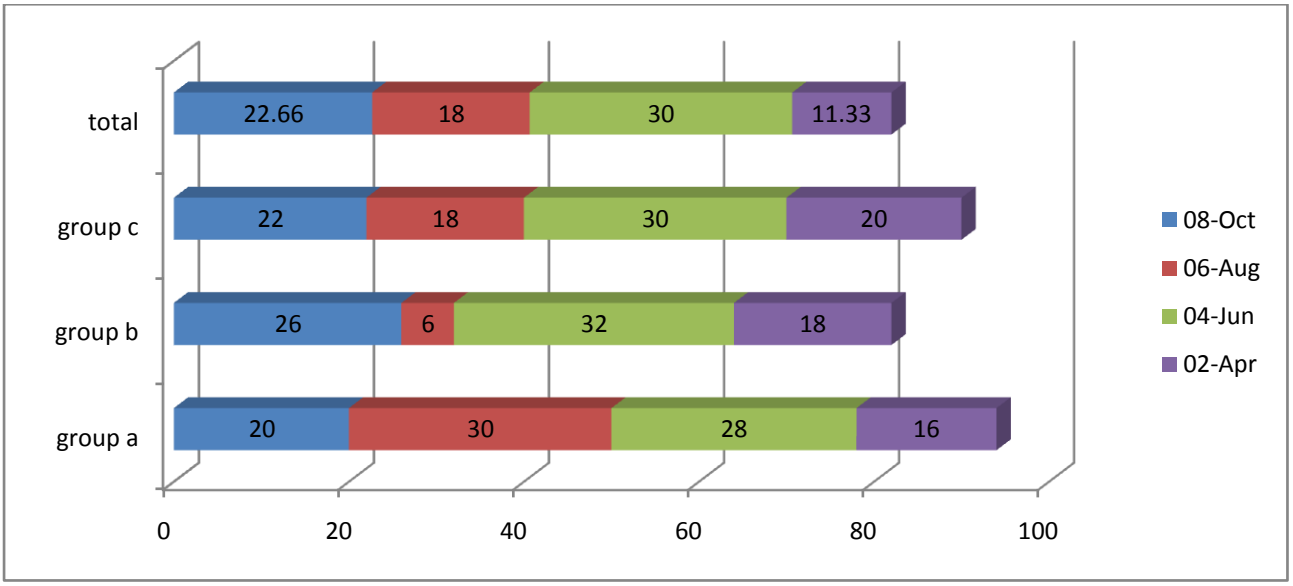
80 (53.33%) patients were having gradual onset . In group A 29(58%) patients, group B 23(46%) patients and group C, 28 (56%) patients were present.

33 (32%) patients were having sub acute onset . In group A 09(18%) patients, group B 15(30%) patients and group C, 09(18%) patients were present.

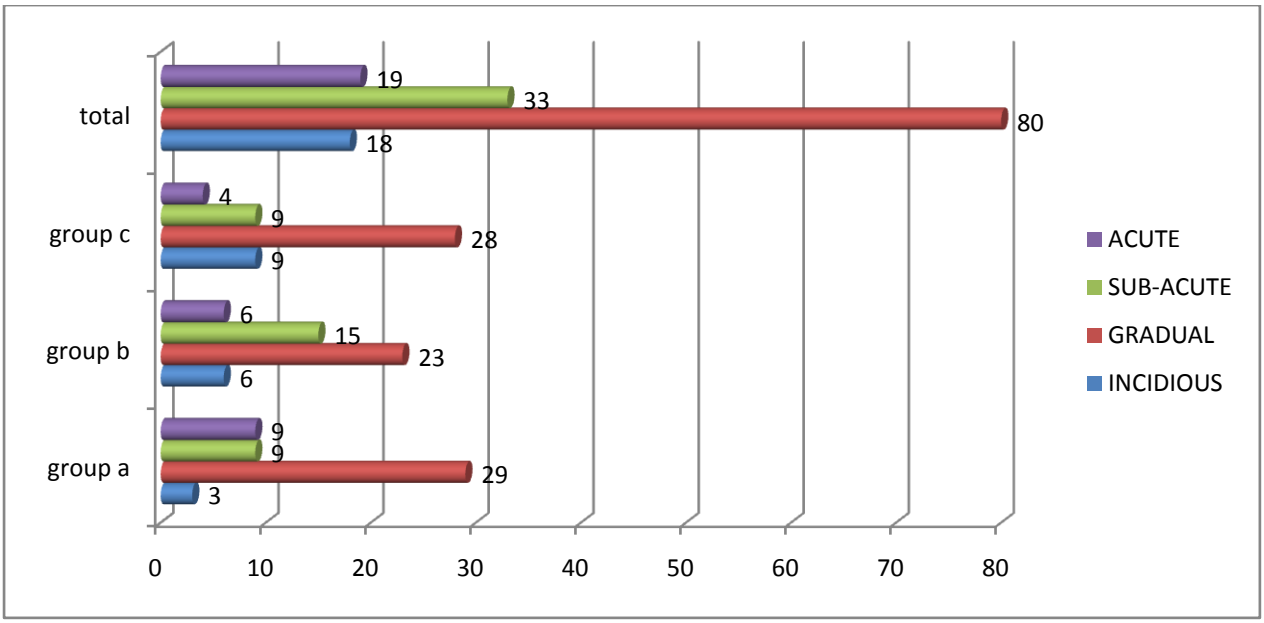
19 (12.66%) patients were having acute onset . In group A 09(18%) patients, group B 06(12%) patients and group C, 04 (08%) patients were present.

Graph: 36 Nasa kandu duration wise distributions of patients with pulmonary hypersensitivity.





Graph: 37 Nasa kandu mode of onset wise distributions of patients with pulmonary hypersensitivity.



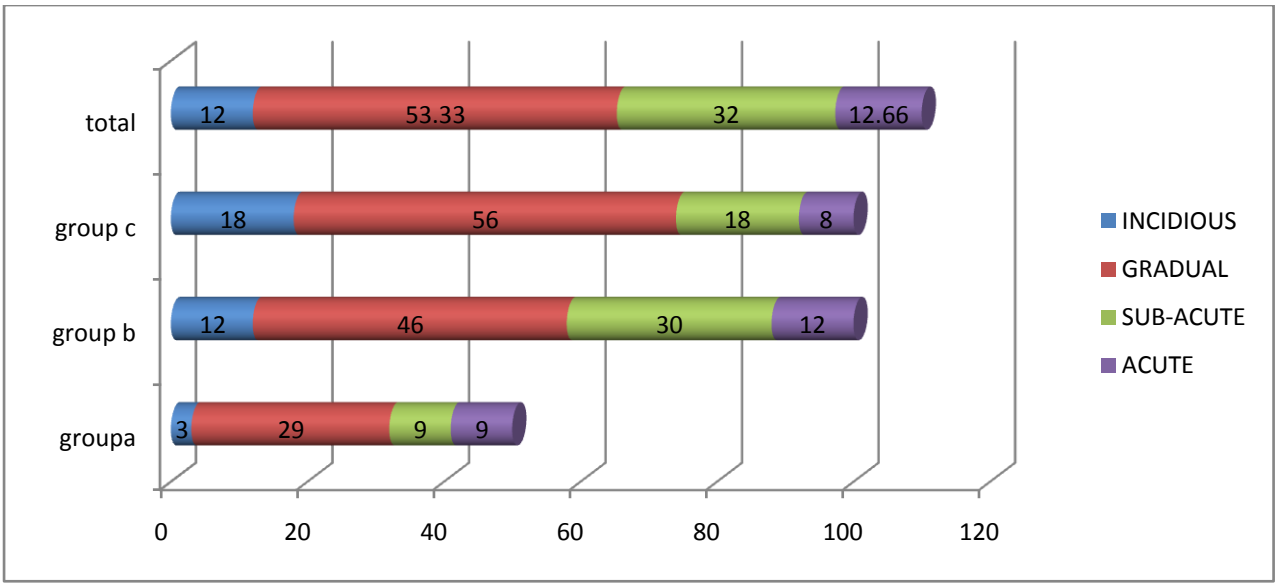


Table No. 78

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF NASA KANDU

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	27	54	24	48	29	58	80	53.33
	RECEEDING	01	02	05	10	06	12	12	08
	RELAPSING	06	12	15	30	10	20	31	20.66
	STATIONARY	16	32	06	12	05	10	27	18

The patients selected for the clinical trial were having kandu of different course.

Among them 80(53.33%) patients were having progressive kandu . In group A, 27(54%) patients, group B, 24(48%) patients and group C ,29 (58%) patients were present.

12 (08%) patients were having receedingkandu . In group A ,01(02%) patients, group B, 05(10%) patients, and group C, 06 (12%) patients were present.

31 (20.66%) patients were having relapsing kandu . In group A, 16(12%) patients, group B, 15(30%) patients, and group C, 10 (20%) patients were present.

27 (18%) patients were having stationary kandu . In group A ,16(32%) patients, group B, 06(12%) patients, and group C, 05 (10%) patients were present.

Table No.79

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY OF NASA KANDU

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	12	24	15	30	15	30	42	28
	MODERATE	28	56	30	60	28	56	86	57.33
	SEVERE	10	20	05	10	07	14	22	29.33

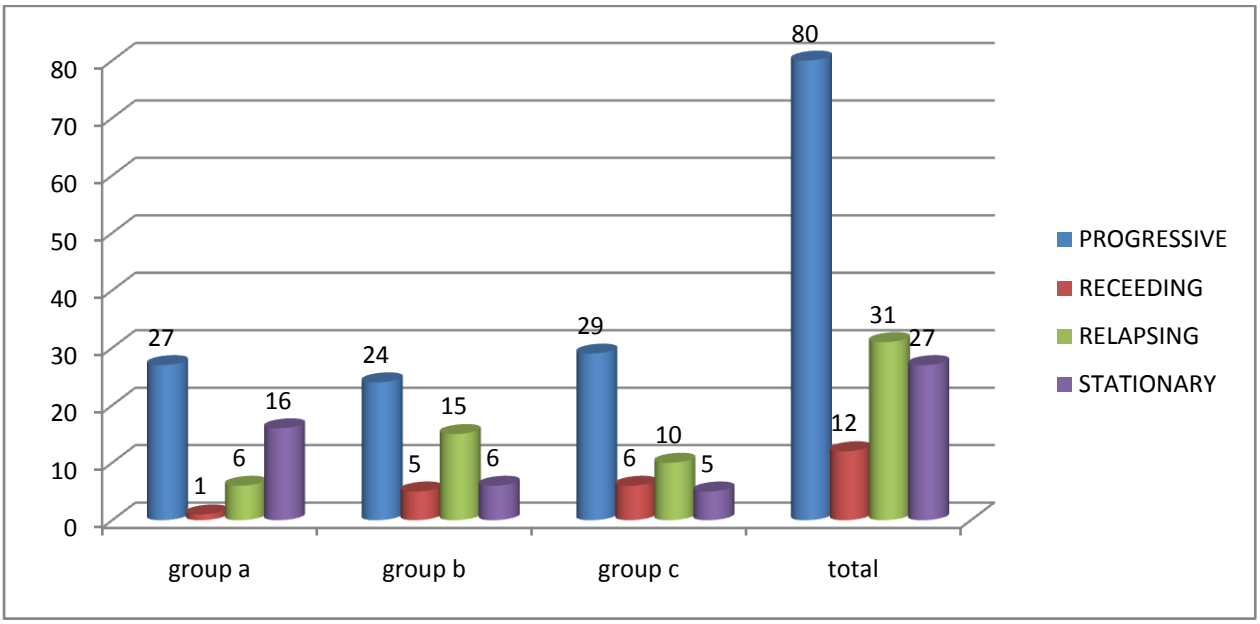
The patients selected for the clinical trial were having nasakandu of different severity.

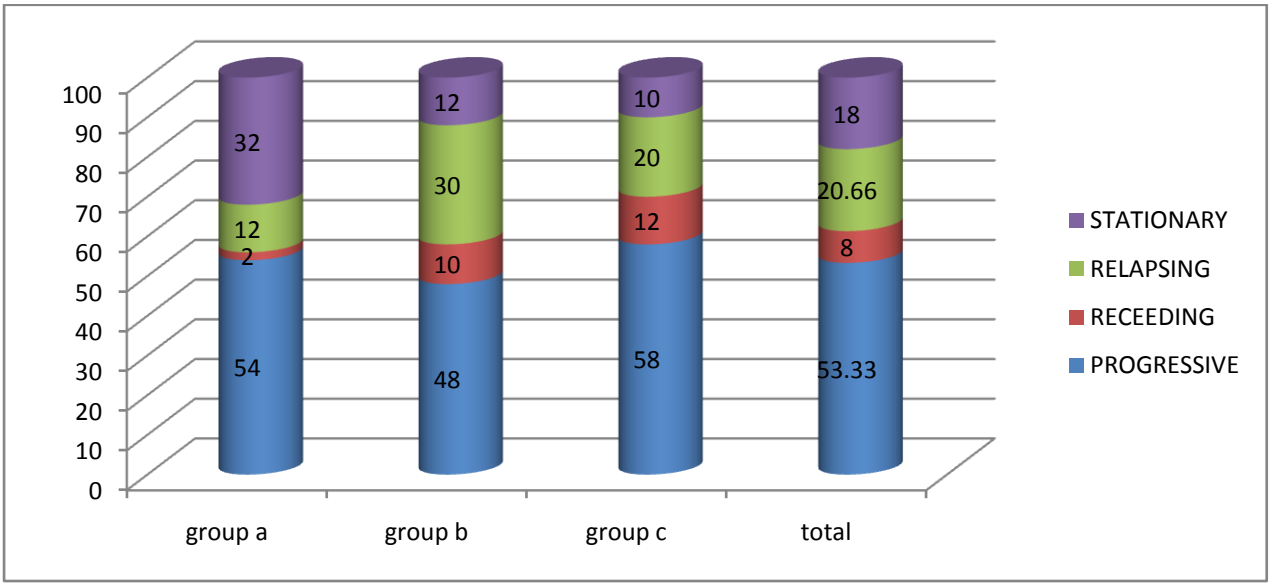
Among them 42 (28%) patients were having mild kandu . In group A, 12(24%) patients,in group B, 15(30%) patients and in group C, 15(30%) patients were present.

86 (57.33%) patients were having moderate kandu . In group A, 28(56%) patients, ingroup B, 30(60%) patients andin group C, 28 (56%) patients were present.

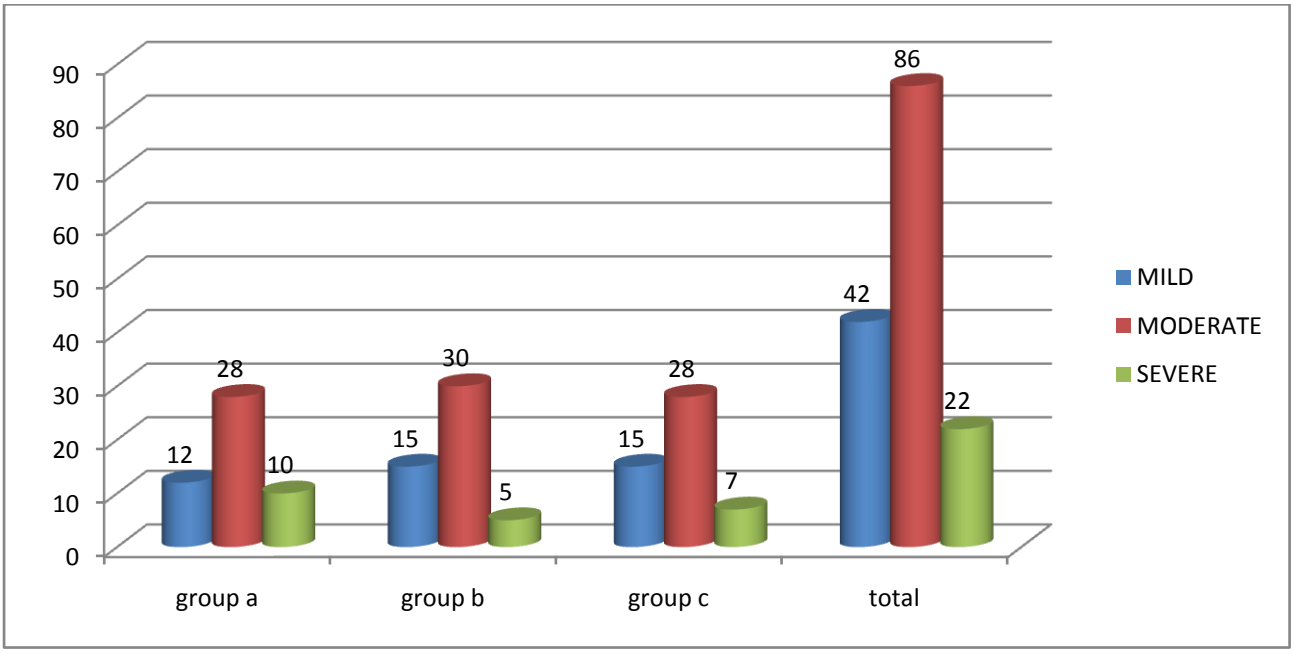
22 (29.33%) patients were having severe kandu . In group A, 10(20%) patients, ingroup B, 05(10%) patients and ingroup C, 07(14%) patients were present.

Graph: 38 Nasa kandu pattern wise distributions of patients with pulmonary hypersensitivity.





Graph: 39 Nasa kandu severity wise distributions of patients with pulmonary hypersensitivity.



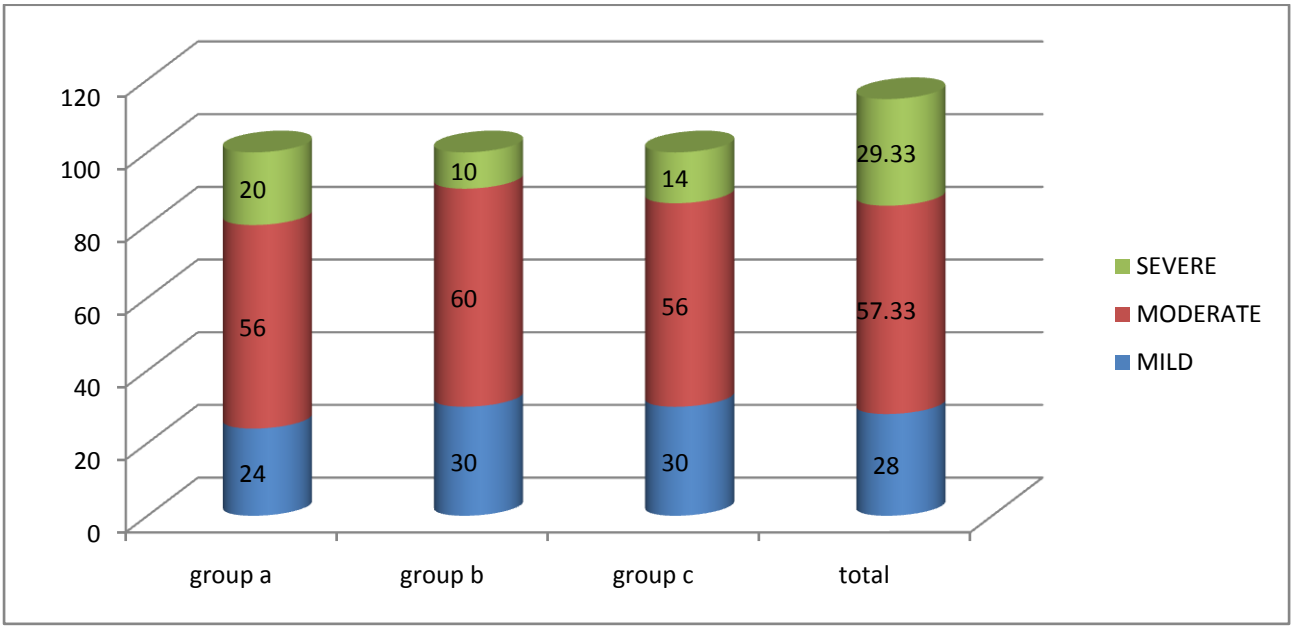


Table No.80

NASA KANDU KALA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

KANDU KALA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	EARLY MORNING	27	54	21	42	31	62	79	52.66
	MORNING	10	20	11	22	15	30	36	24
	AFTERNOON	-	-	-	-	-	-	-	-
	EVENING	16	32	13	26	11	22	40	26.66
	NIGHT	12	24	16	32	20	40	48	32
	MID-NIGHT	38	76	40	80	41	82	119	79.33

The patients selected for the clinical trial were having kandu at different kala.

Among them 79 (52.66%) patients were having kandu in early morning. In group A, 27(54%) patients, ingroup B, 21(42%) patients andin group C, 31 (62%) patientswere present.

36 (24%) patients were having kandu in the morning. In group A, 10(20%) patients, ingroup B, 11(22%) patients andin group C, 15 (30%) patients.were present

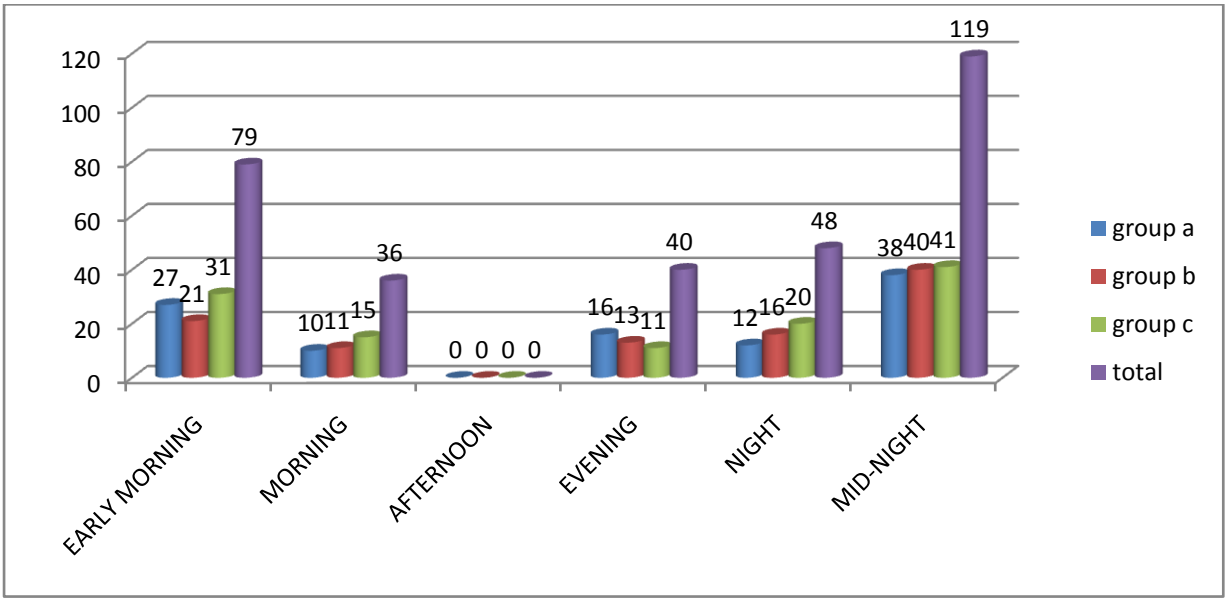
None of them had kandu in the afternoon.

40 (26.66%) patients were having kandu in evening. In group A, 16(32%) patients, ingroup B, 13(26%) patients andin group C, 11 (22%) patientswere present.

48 (32%) patients were having kandu at night. In group A, 12(24%) patients, ingroup B, 16(32%) patients andin group C, 20 (40%) patientswere present.

119 (79.33%) patients were having kandu in early morning. In group A, 38(76%) patients, ingroup B 40(30%) patients andin group C, 41(82%) patients werepresent.

Graph: 40 Nasa kandu kala wise distributions of patients with pulmonary hypersensitivity.



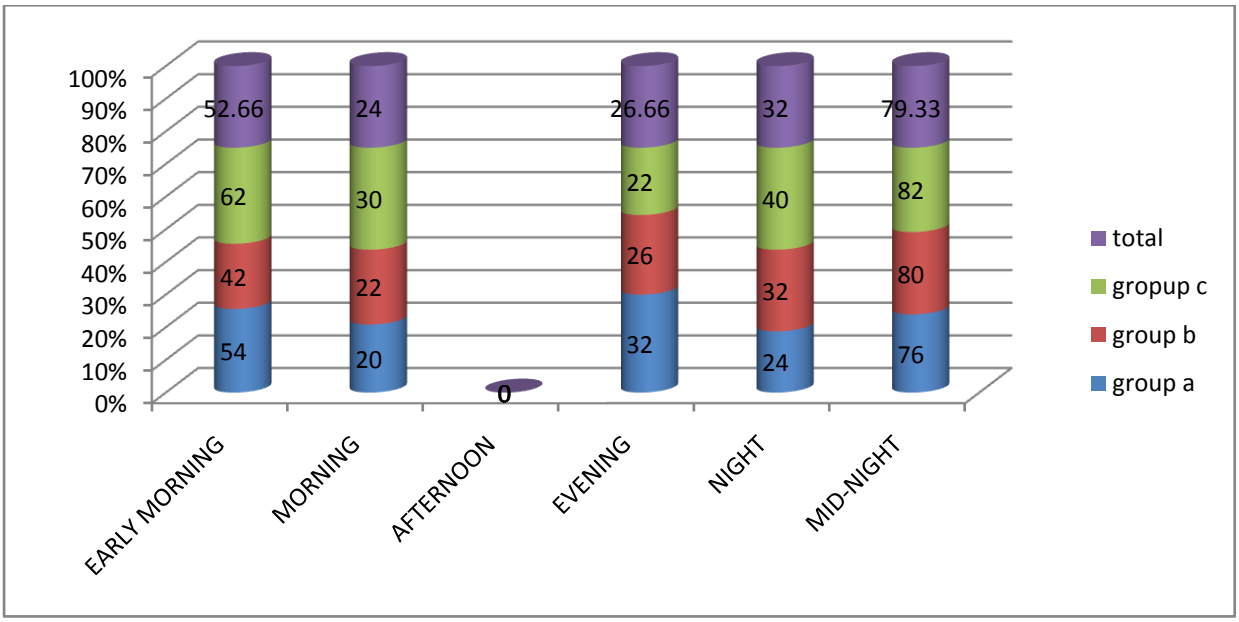


Table No.81 DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING DURATION OF KASA.

DURATION		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	08-10 years	14	28	15	30	16	32	45	30
	06-08 years	08	16	09	18	07	14	24	16
	04-06 years	16	32	19	38	13	26	48	32
	02-04 years	08	16	02	04	10	20	20	13.33
	01-02 years	04	08	05	10	04	08	13	8.66

The patients selected for the clinical trial were having kasa of various duration.

Among them 45 (30%) patients were having kasa from 08-10 years . In group A, 14(28%) patients, In group B 15(30%) patients and In group C, 16 (32%) patientswere present.

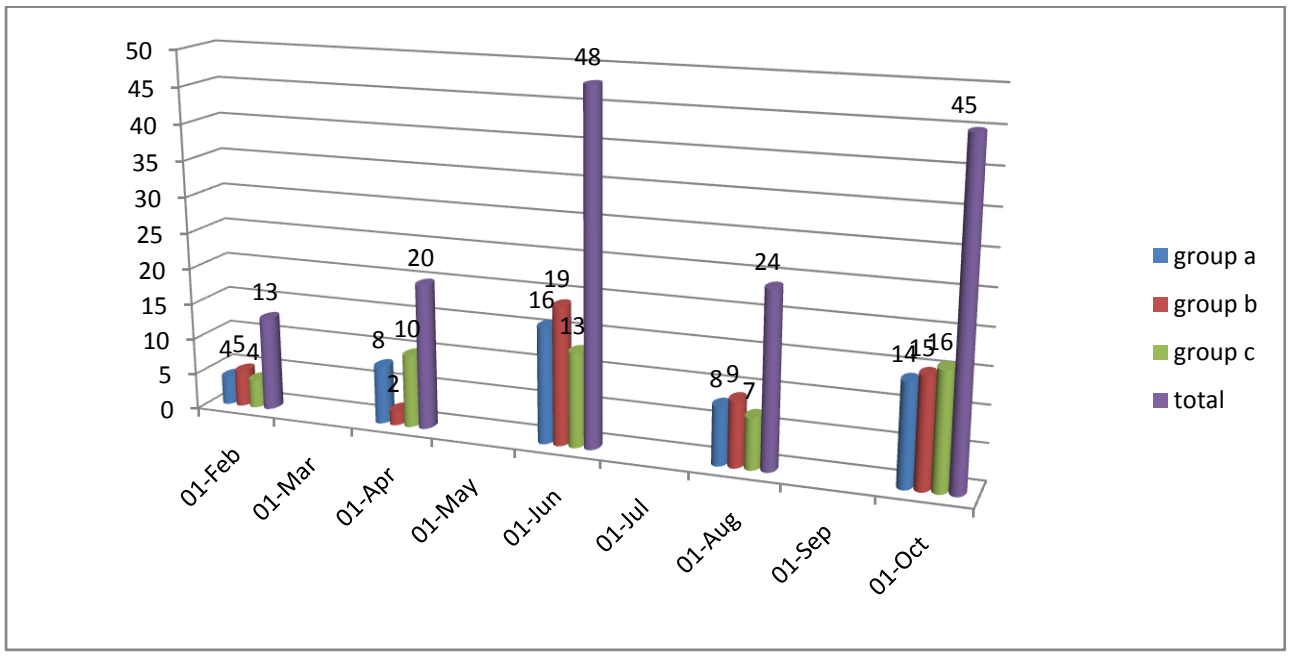
24(16%) patients were having kasa from 06-08 years . In group A, 08(16%) patients, In group B 09(18%) patients and In group C, 07 (14%) patientswere present.

48 (32%) patients were having kasa from 04-06 years . In group A, 16(32%) patients, In group B 19(38%) patients and In group C, 13 (26%) patients werepresent.

20 (13.33%) patients were having kasa from 02-04 years . In group A, 08(16%) patients, In group B 02(04%) patients and In group C, 10 (20%) patients were present.

13 (8.66%) patients were having kasa from 01-02 years . In group A, 04(08%) patients, Ingroup B 05(10%) patients and group In C, 04 (08%) patientswere present.

Graph: 41 Kasa duration wise distributions of patients with pulmonary hypersensitivity.



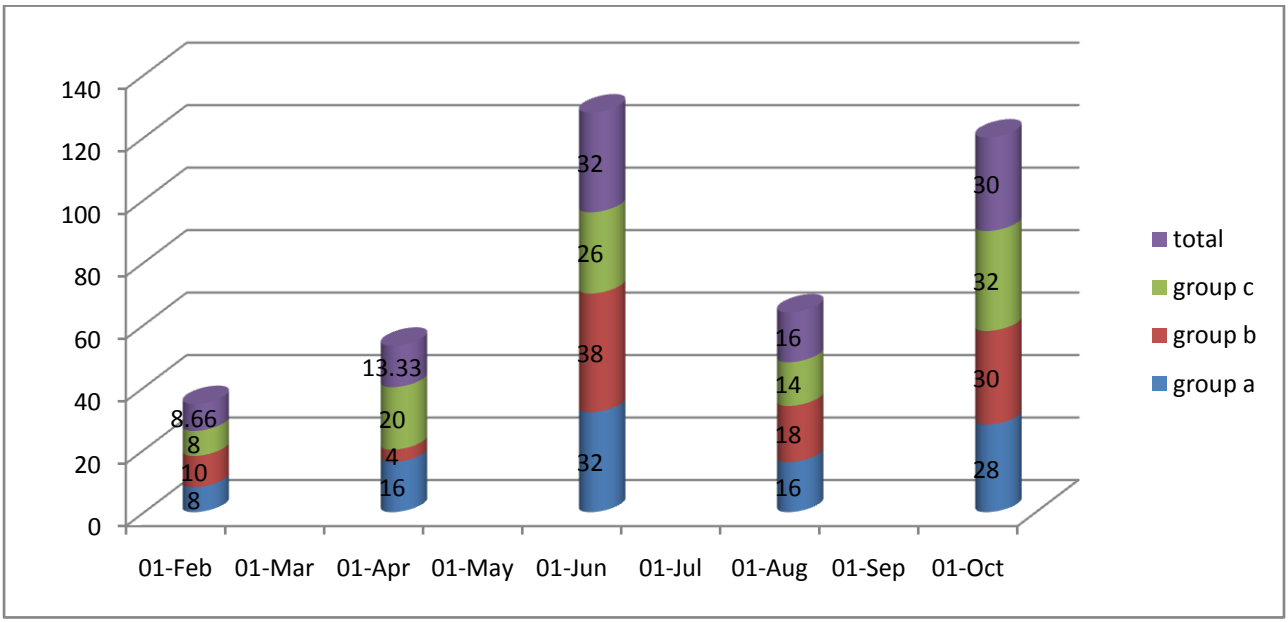


Table No.82

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO MODE OF ONSET OF KASA

MODE OF ONSET		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	INSIDIOUS	02	04	08	16	03	06	13	8.66
	GRADUAL	33	66	33	66	35	70	101	67.33
	SUB-ACUTE	13	26	06	12	07	14	26	17.33
	ACUTE	02	04	03	06	05	10	10	6.66

The patients selected for the clinical trial were having kasa of various modes of onset.

Among them 13 (8.66%) patients were having insidious onset . And in them, In group A, 02(04%) patients, group B, 08(16%) patients and ingroup C, 03 (06%) patients were present.

101 (67.33%) patients were having gradual onset .And in them, In group A, 33(66%) patients, in group B, 33(66%) patients andin group C, 35 (70%) patientswere present.

26 (17.33%) patients were having sub acute onset . And in them,In group A, 13(26%) patients, ingroup B, 06(12%) patients and ingroup C, 07(14%) patientswere present.

10 (6.66%) patients were having acute onset . And in them,In group A, 02(04%) patients,in group B ,03(06%) patients andin group C, 05 (10%) patientswere present

.

Table No.83

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF KASA

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	26	52	25	50	26	52	77	51.33
	RECEEDING	03	06	05	10	08	16	16	10.66
	RELAPSING	05	10	08	16	07	14	20	13.33
	STATIONARY	16	32	12	24	09	18	37	24.66

The patients selected for the clinical trial were having kasa of different course.

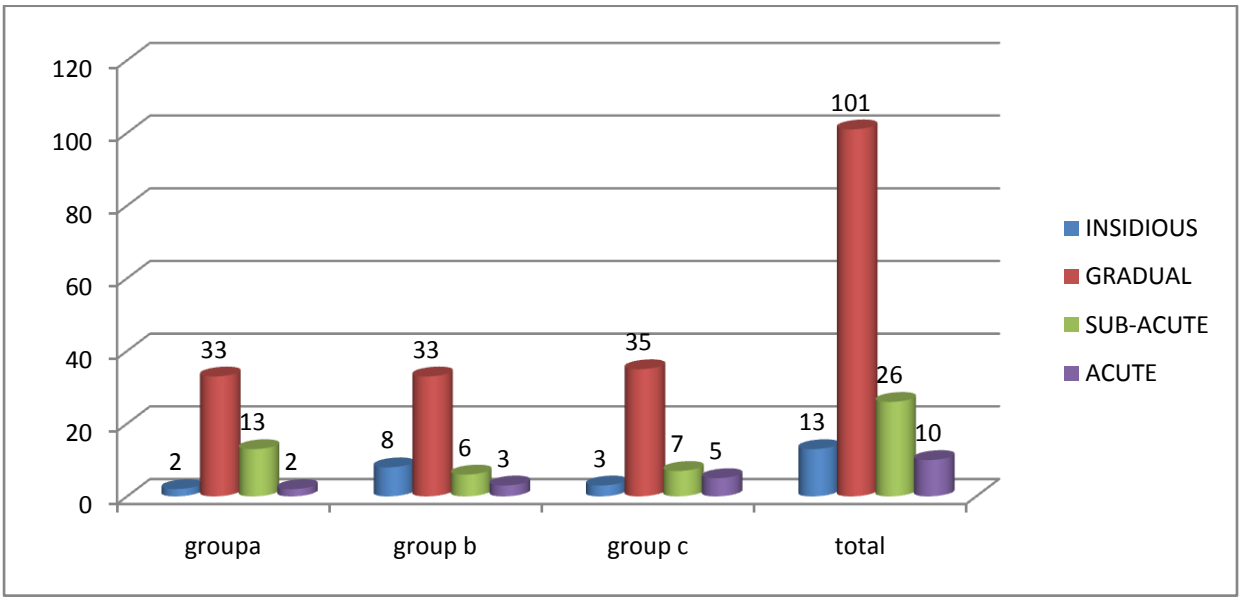
Among them 77 (51.33%) patients were having progressive kasa . In group A, 26(52%) patients, in group ,B 25(50%) patients andin group C, 26 (52%) patientswere present.

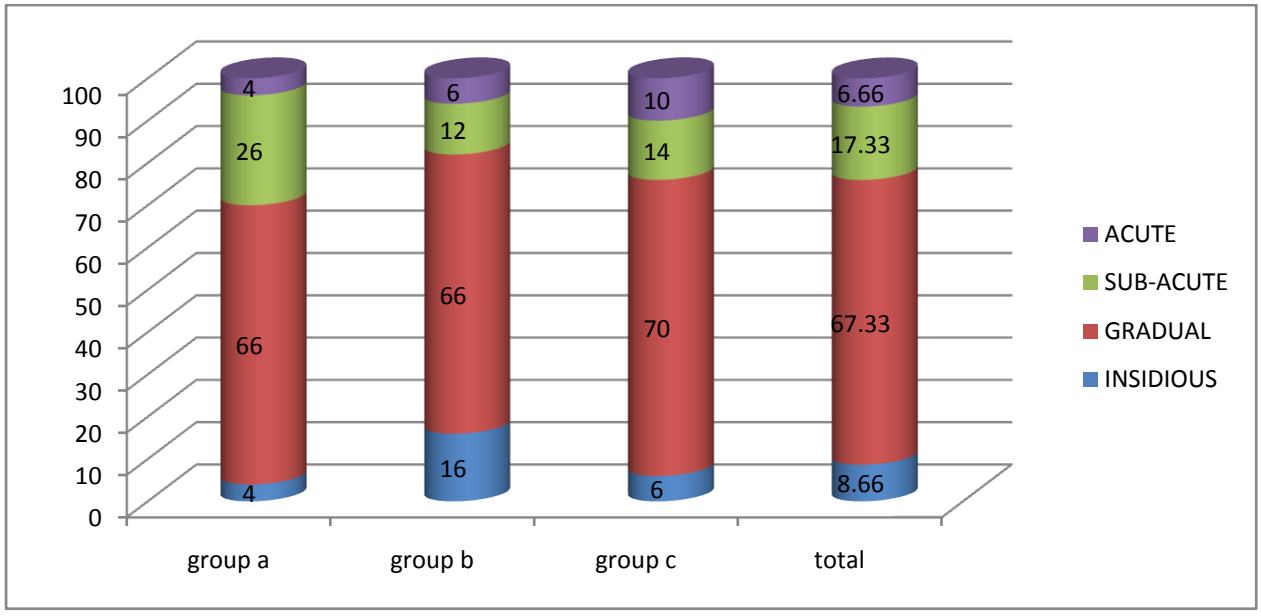
16 (10.66%) patients were having receedingkasa .Among whom, In group A, 03(06%) patients,in group B, 05(10%) patients andin group C, 08 (16%) patients were present.

20(13.33%) patients were having relapsing kasa .Among whom, In group A, 05(10%) patients, in group B, 08(16%) patients and in group C, 07 (14%) patients were present.

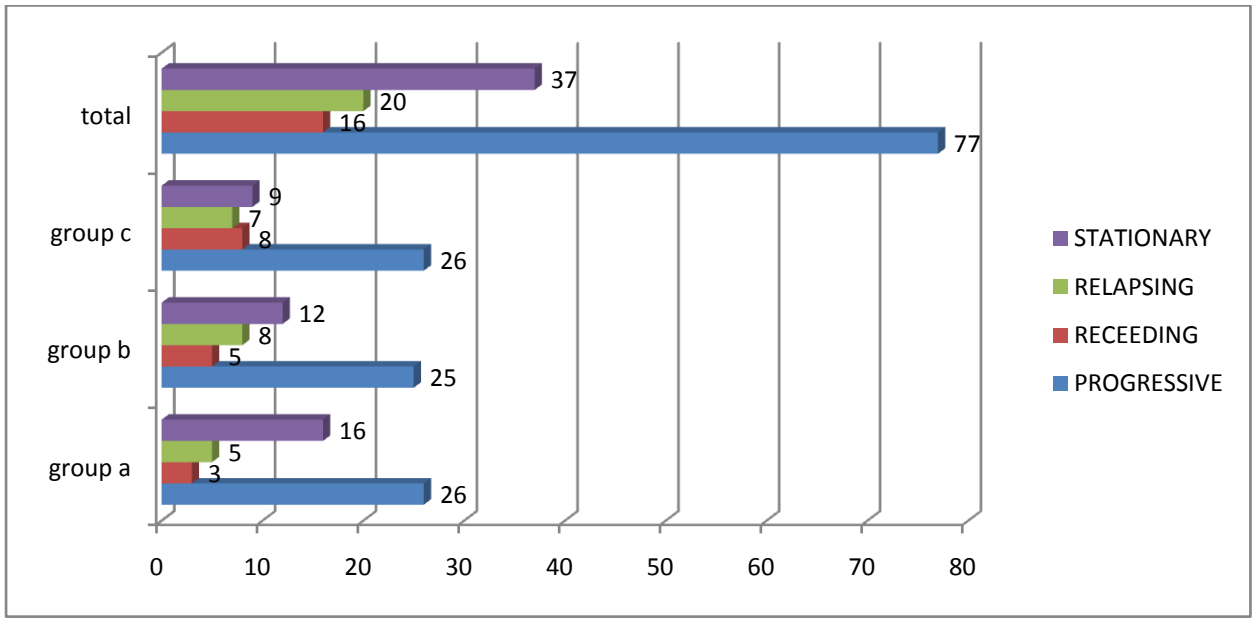
37 (24.66%) patients were having stationary kasa .Among whom, In group A, 16(32%) patients, in group B, 12(24%) patients and in group C, 09 (18%) patientswere present.

Graph: 42 Kasa- modes of onset wise distributions of patients with pulmonary hypersensitivity.





Graph: 43 Kasa- pattern wise distributions of patients with pulmonary hypersensitivity



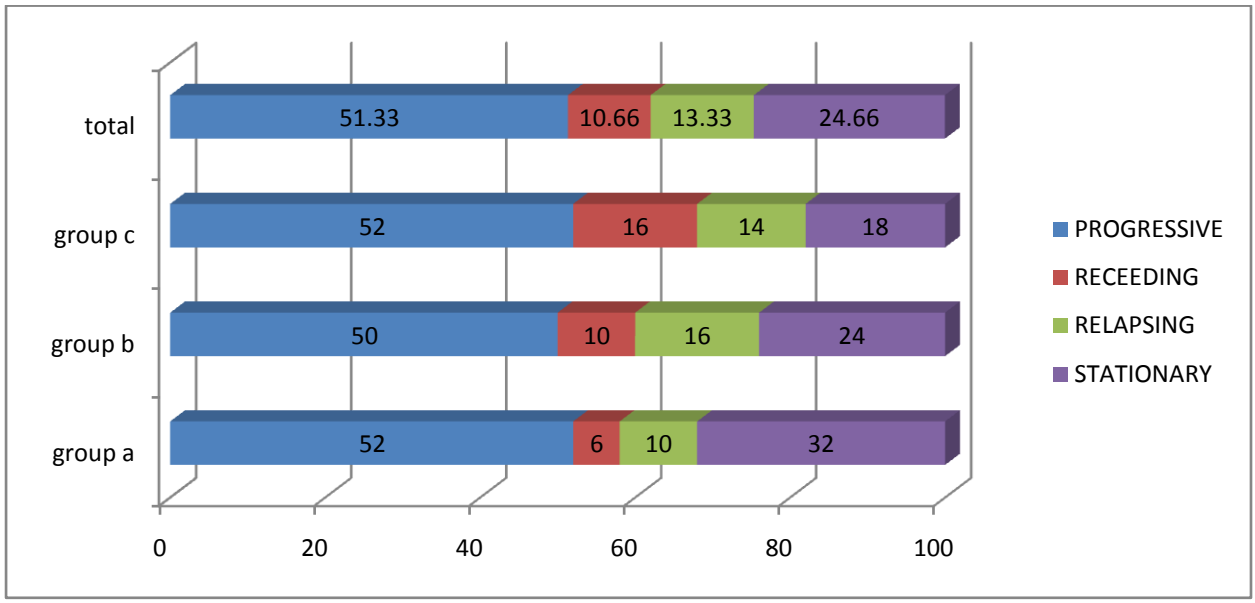


Table No.84

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY OF KASA

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	06	12	09	18	12	24	29	19.33
	MODERATE	14	28	16	32	19	38	49	32.66
	SEVERE	30	60	25	50	29	58	84	56

The patients selected for the clinical trial were having kasa of different severity.

Among them 29 (19.33%) patients were having mild kasa .Among whom, In group A, 06(12%) patients, in group B, 09(18%) patients and in group C, 12 (24%) patientswere present.

49 (32.66%) patients were having moderate kasa .Among whom, In group A, 14(28%) patients, in group B, 16(32%) patients and in group C ,19 (38%) patientswere present.

84 (56%) patients were having severe kasa .Among whom, In group A, 30(60%) patients, in group B, 25(50%) patients and in group C, 29 (58%) patients were were present.

Table No.85

KASA- KALA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

KASA KALA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	EARLY MORNING	30	60	34	68	31	62	95	63.33
	MORNING	04	08	04	08	08	16	16	10.66
	AFTERNOON	01	02	01	02	01	02	03	02
	EVENING	01	02	01	02	00	00	02	1.33
	NIGHT	04	08	06	12	00	00	10	6.66
	MID-NIGHT	10	20	04	08	10	20	24	16

The patients selected for the clinical trial were having kasa at different kala.

Among them 95 (63.33%) patients were having kasa inthe early morning. In group A, 30(60%) patients, ingroup B, 34(68%) patientsand ingroup C, 31 (62%) patients were present.

16 (10.66%) patients were having kasa in the morning. In group A, 04(08%) patients,in group B, 04(08%) patients andin group C, 08 (16%) patientswere present.

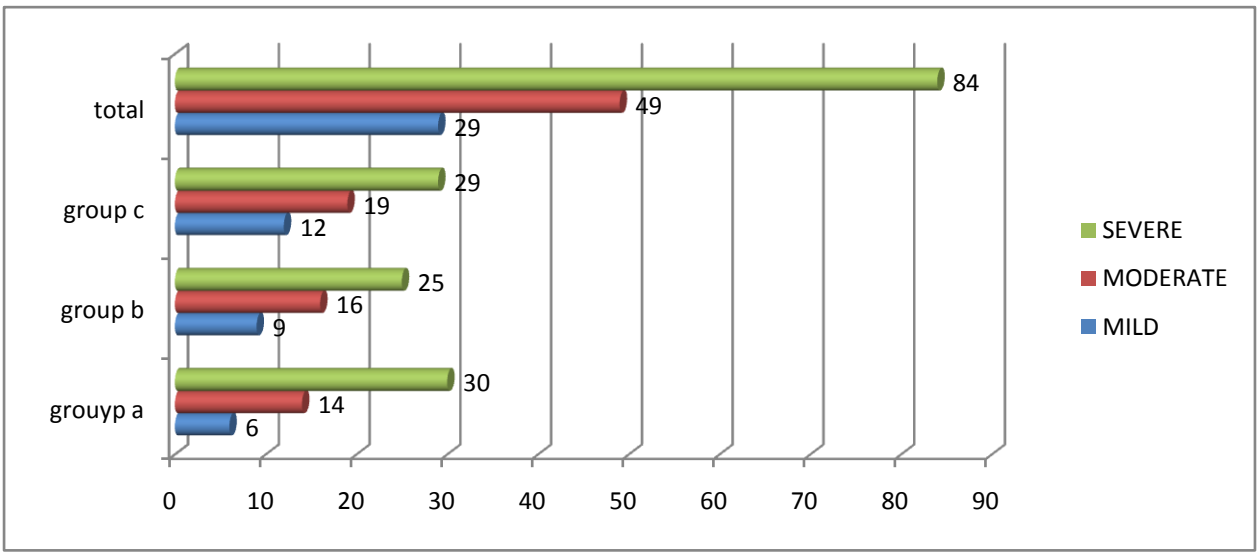
03 (02%) patients were having kasa in afternoon. In group A, 40(80%) patients, ingroup B, 45(90%) patients ,and ingroup ,C 41 (82%) patients were present.

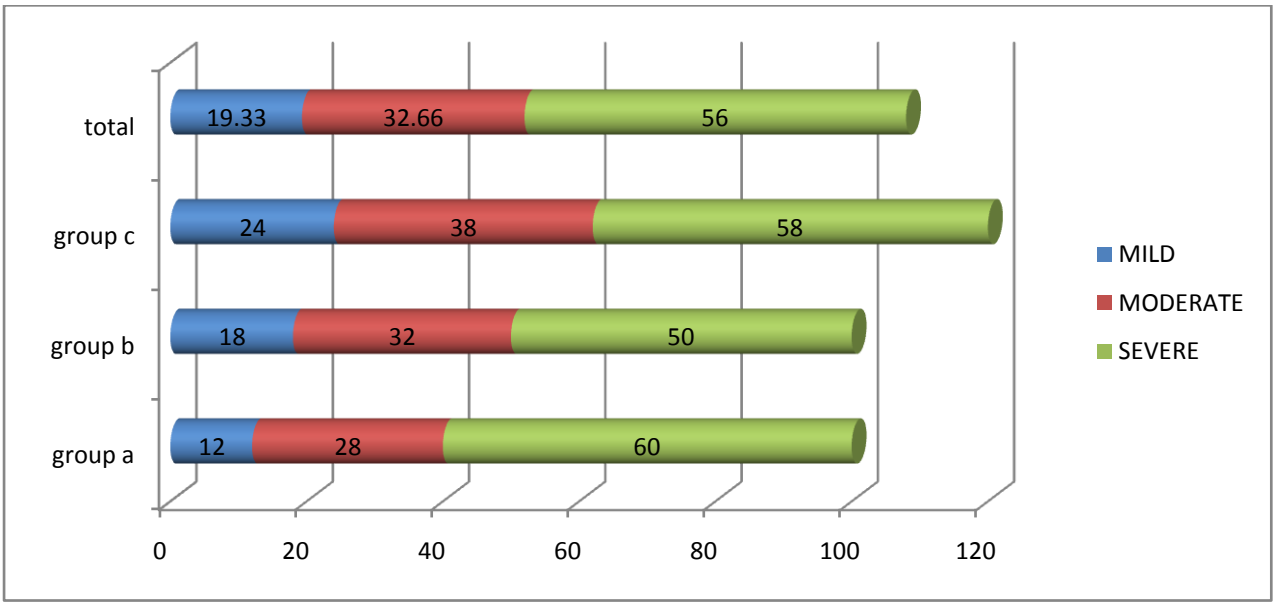
02 (1.33%) patients were having kasa in the evening. In group A, 01(02%) patients, ingroup B, 01(02%) patients andin group C, 00 (00%) patientswere present.

10 (6.66%) patients were having kasa at the night. In group A, 04(08%) patients, ingroup B, 06(12%) patients and ingroup C, 00 (00%) patientswere present.

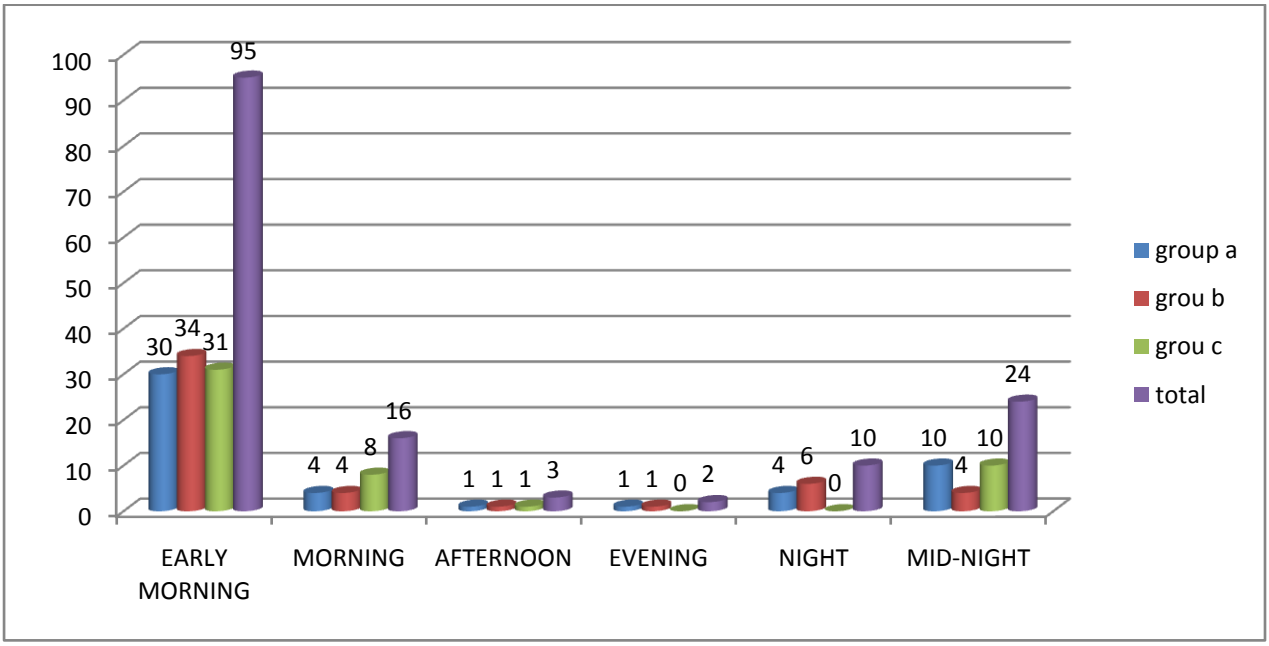
24 (16%) patients were having kasa at mid night. In group A, 10(20%) patients,in group B, 04(08%) patients and ingroup C, 10 (20%) patientswere present.

Graph: 44 Kasa- severity wise distributions of patients with pulmonary hypersensitivity.


total
group c
group b
grouyp a
84
49
29
29
19
12
25
16
9
30
14
6
0
10
20
30
40
50
60
70
80
90
SEVERE
MODERATE
MILD


total
group c
group b
group a
19.33
32.66
56
24
38
58
18
32
50
12
28
60
0
20
40
60
80
100
120
MILD
MODERATE
SEVERE

Graph: 45 Kasa- kala wise distributions of patients with pulmonary hypersensitivity.



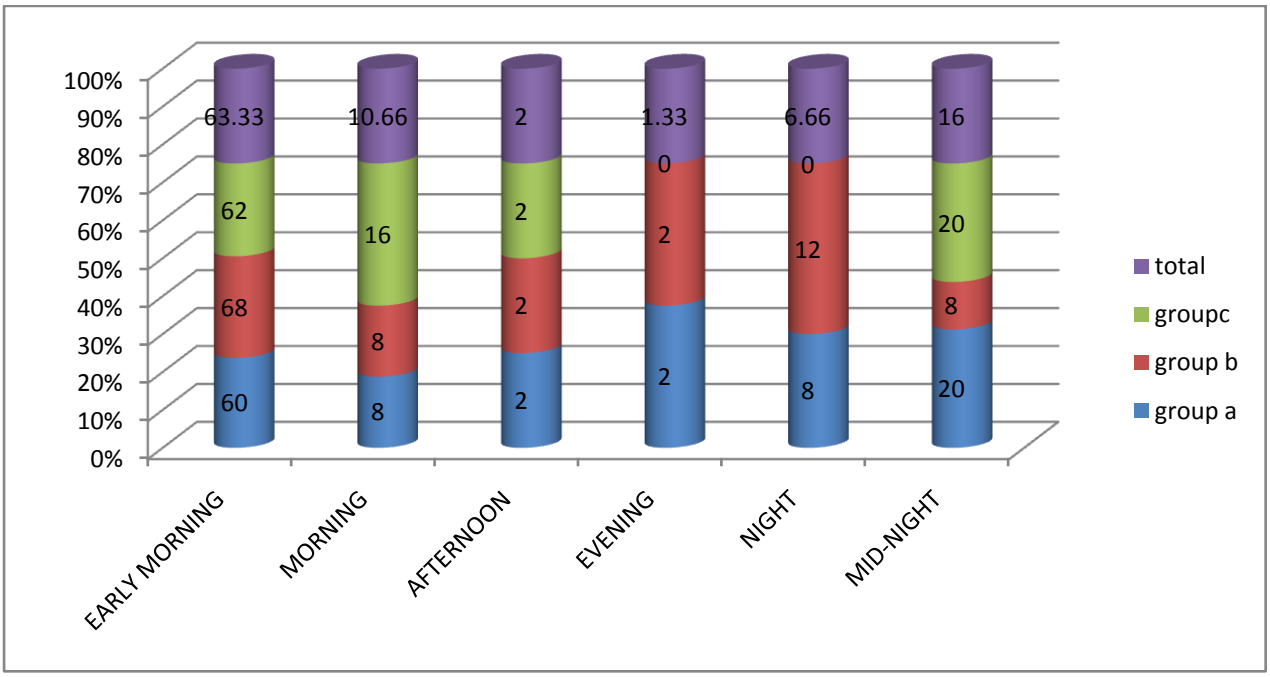


Table No.86

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO TYPES OF KASA

TYPES OF KASA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	RUKSHA	40	80	45	90	41	82	126	84
	SAKAPHA	05	10	01	02	03	06	09	06
	BOTH	05	10	04	08	06	12	15	10

The patients selected for the clinical trial were having different types of kasa.

Among them 126 (84%) patients were having rukshakasa. Among whom,In group A, 40(80%) patients, in group B, 45(90%) patients and,in group C, 41 (82%) patients were present.

09(06%) patients were having sakaphakasa.Among whom, In group A, 05(10%) patients, ingroup B, 01(02%) patients, andin group C ,03 (06%) patients were present.

15(10%) patients were having both the lakshanas.Among whom, In group A, 05(10%) patients,in group B, 04(08%) patient,s and ingroup C, 06 (12%) patientswere present.

Table No.87

SWASHA KRICHRITA-DURATION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

DURATION		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	08-10Yrs	01	02	03	06	00	00	04	2.66
	06-08Yrs	03	06	09	18	11	22	23	15.33
	04-06Yrs	02	04	02	04	04	08	08	5.33
	02-04Yrs	09	18	07	14	02	04	18	12
	01-02Yrs	12	24	15	30	12	24	39	26

The patients selected for the clinical trial were having swasakrichritaof various duration.

Among them 04 (2.66%) patients were havingswasakrichrita from 08-10 years. Among whom,In group A, 01(02%) patients, ingroup B, 03(06%) patients, and ingroup C ,00 (00%) patientswere present.

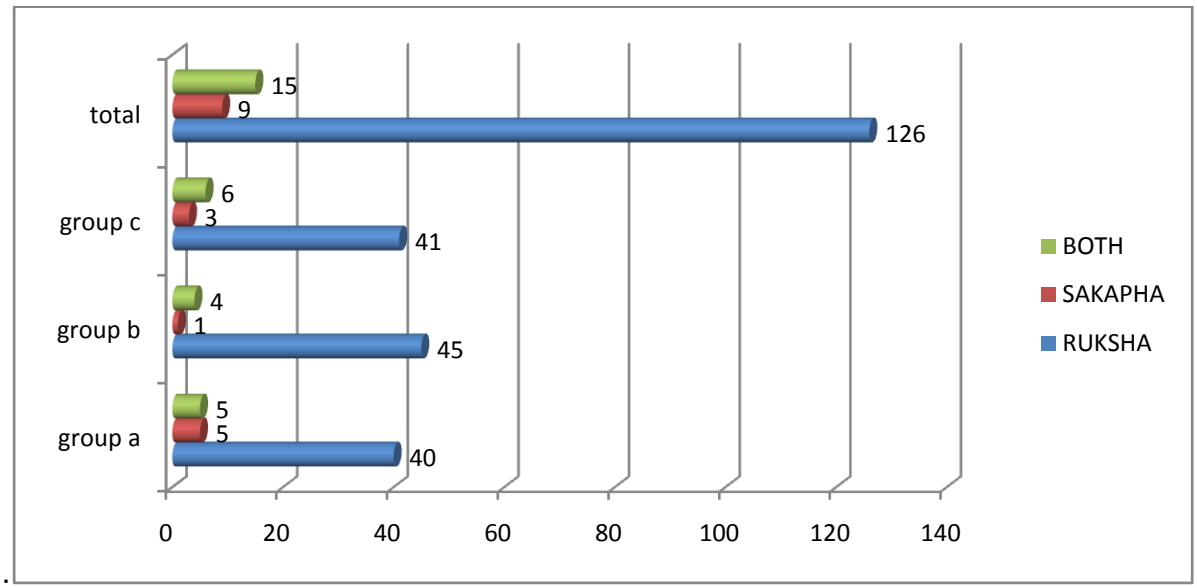
23 (15.33%) patients were havingswasakrichrita from 06-08 years.Among whom,In group A, 03(06%) patients, ingroup B, 09(18%) patient,s and ingroup C, 11 (22%) patientswere present.

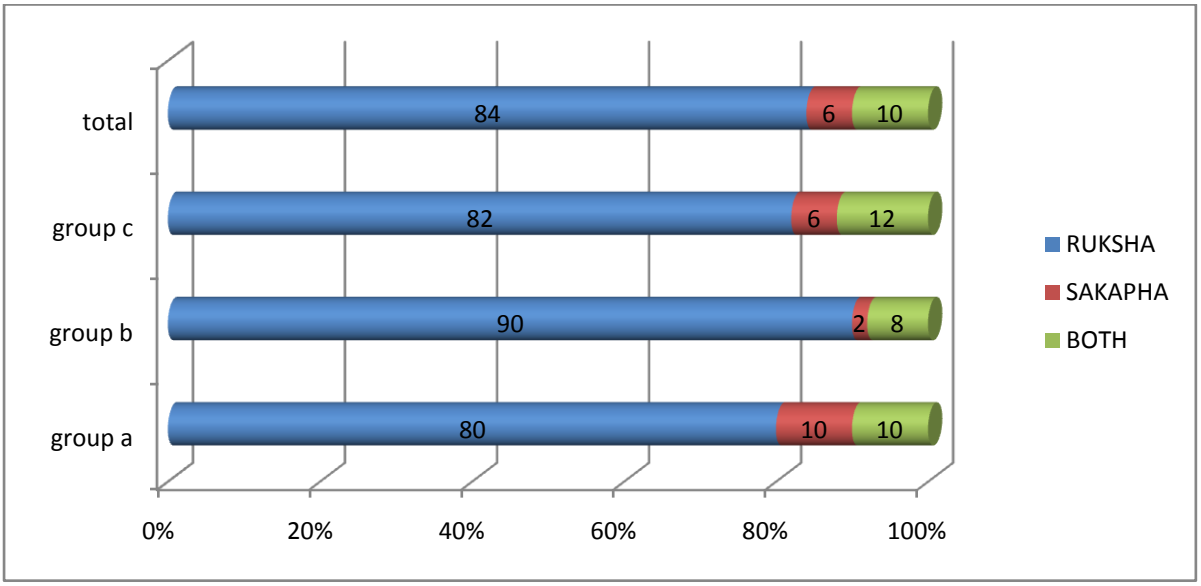
08 (5.33%) patients were havingswasakrichrita from 04-06 years. Among whom,In group A, 02(04%) patients, ingroup B, 02(04%) patients, and ingroup C, 04 (08%) patientswere present.

18 (12%) patients were having swasakrichrita from 02-04 years.Among whom, In group A, 09(18%) patients,in group B, 07(14%) patients ,andin group C ,02 (04%) patientswere present.

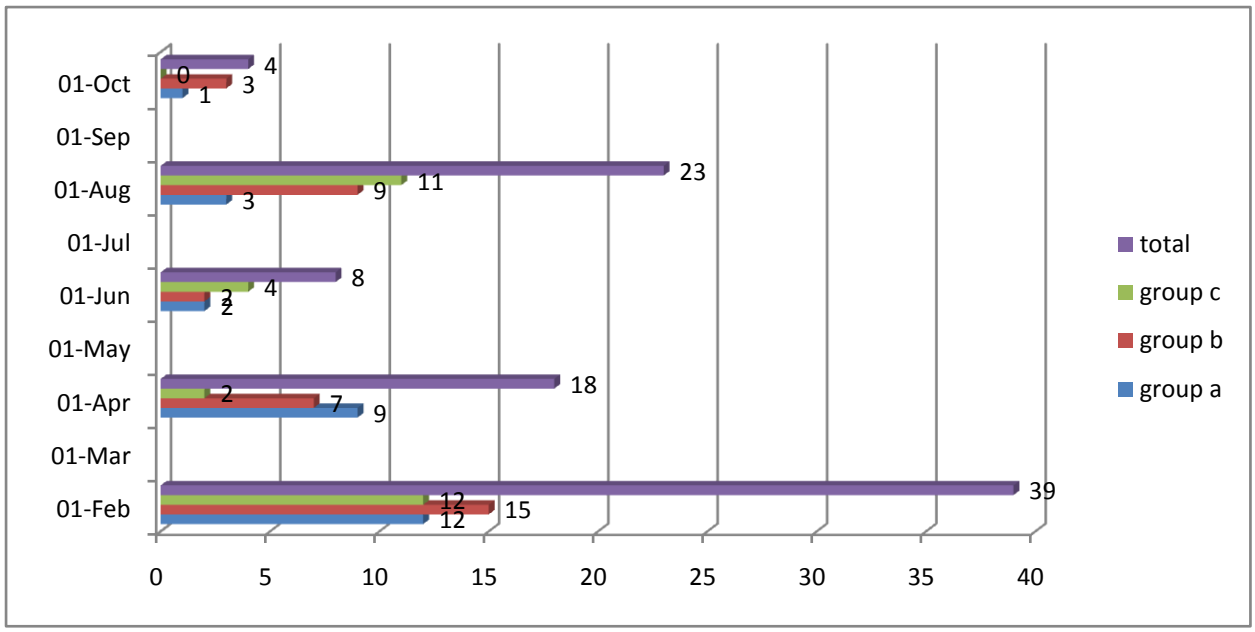
39(26%) patients were having swasakrichrita from 01-02 years.Among whom, In group A, 12(24%) patients,in group B, 15(30%) patients, and ingroup C, 12 (24%) patientswere present.

Graph: 46 Kasa- type wise distributions of patients with pulmonary hypersensitivity.





Graph: 47 Swasha krichrita duration wise distributions of patients with pulmonary hypersensitivity.



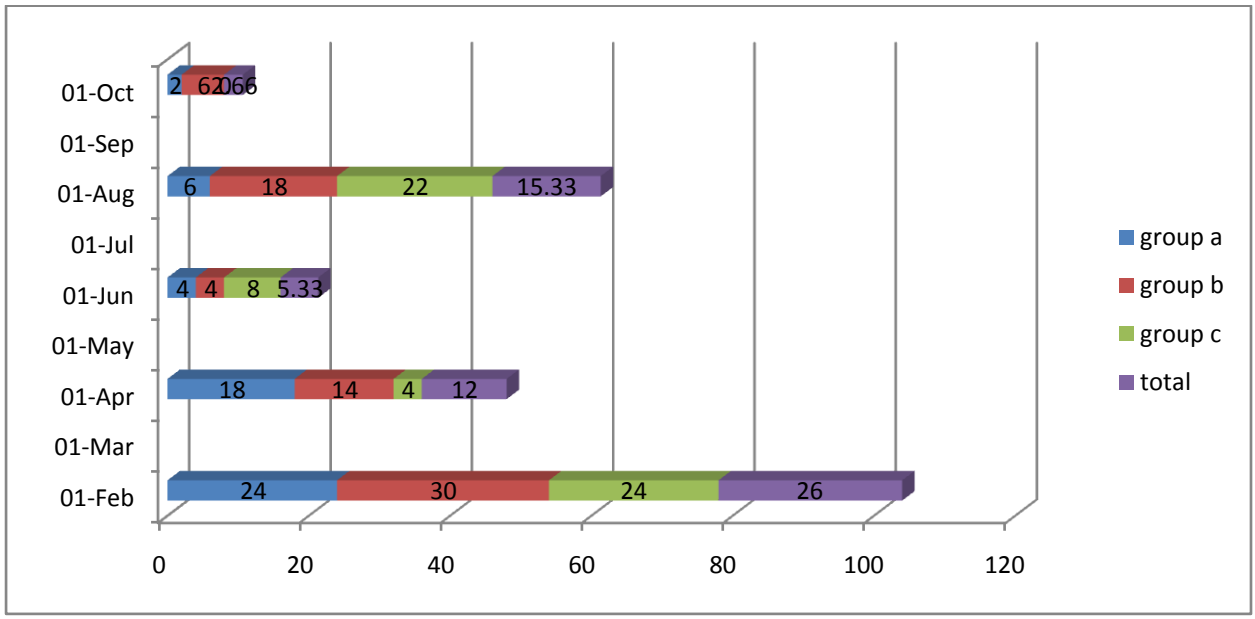


Table No.88

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO MODE OF ONSET OF SWASHA KRICHRITA

MODE OF ONSET		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	INCIDIOUS	-	-	-	-	-	-	-	-
	GRADUAL	19	38	15	30	13	26	47	31.33
	SUB-ACUTE	06	12	02	04	05	10	13	8.66
	ACUTE	-	-	-	-	-	-	-	-

The patients selected for the clinical trial were having swasakrichrita of various modes of onset.

None of them had insidious onset.

47 (31.33%) patients were having gradual mode of onset.Among whom,In group A, 19(38%) patients, in group B, 15(30%) patients, and in group C, 13 (26%) patients were present.

13 (8.66%) patients were having sub acute mode of onset. Among whom,In group A, 06(12%) patients, in group B, 02(04%) patients, and in group C, 05 (10%) patientswere present.

None of them had acute onset.

Table No.89

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF SWASHA KRICHRITA

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	19	38	10	20	09	18	38	25.33
	RECEEDING	02	04	01	02	02	04	05	3.33
	RELAPSING	03	06	02	04	01	02	06	04
	STATIONARY	03	06	01	02	00	00	04	2.66

The patients selected for the clinical trial were having swasakrichrita of different course.

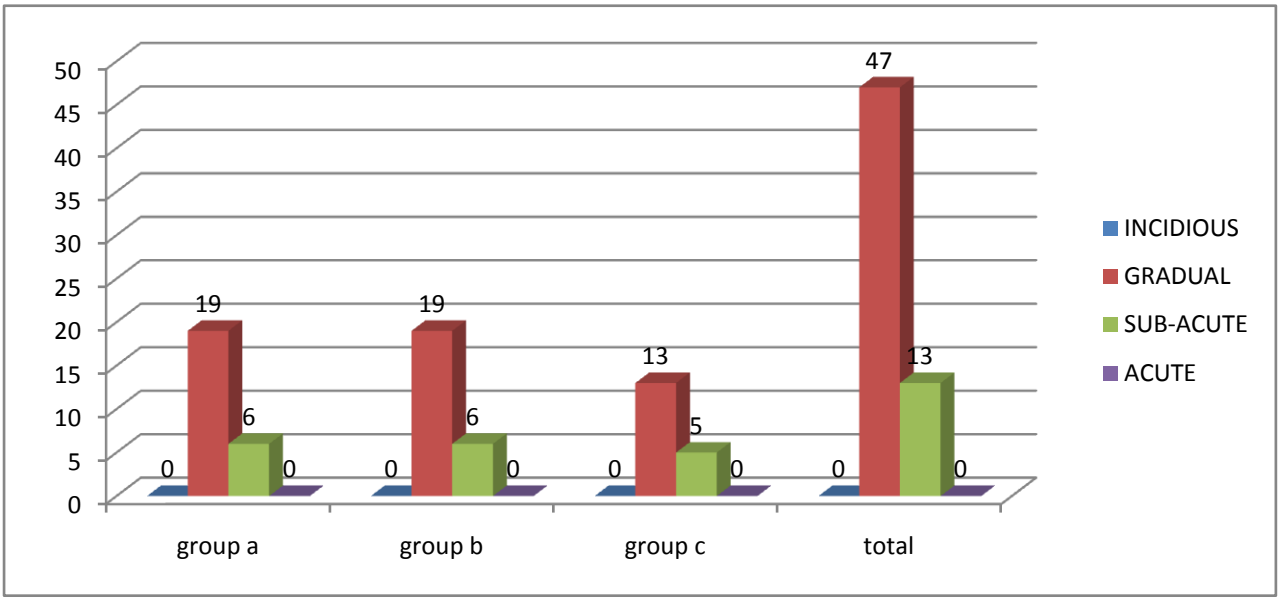
38(25.33%) patients were having progressive course. Among whom,In group A 19(38%) patients, ingroup B, 10(20%) patients, andin group C, 09 (18%) patientswere present.

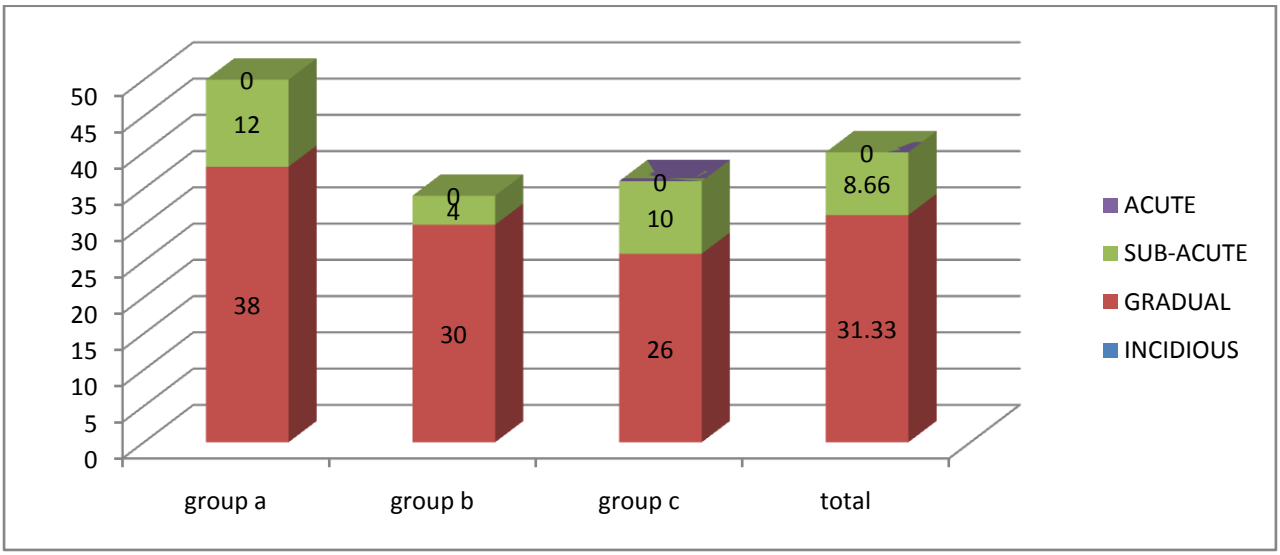
05 (3.33%) patients were having receeding course. Among whom,In group A, 02(04%) patients,in group B, 01(02%) patients, and ingroup C, 02 (04%) patientswere present.

06(04%) patients were having relapsing course.Among whom, In group A, 19(38%) patients,in group B, 10(20%) patients ,andin group C, 02 (04%) patientswere present.

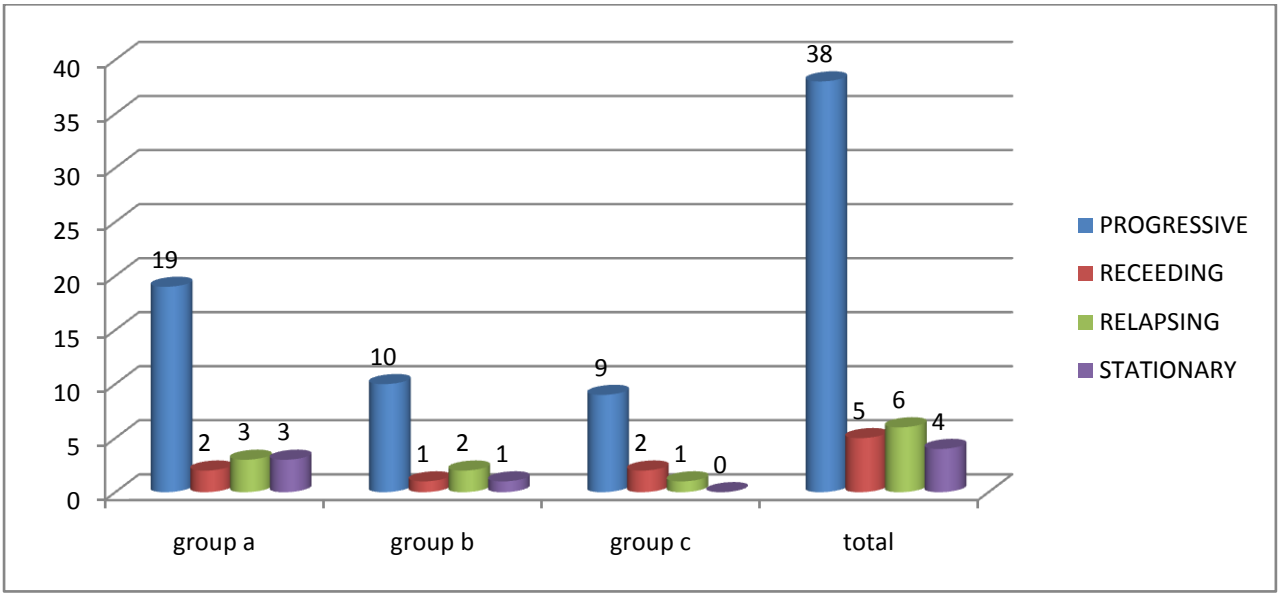
04(2.66%) patients were having stationary course.Among whom, In group A ,03(06%) patients, ingroup B, 01(02%) patients, andin group C, 00 (00%) patientswere present.

Graph: 48 Swasha krichrita- mode of onset wise distributions of patients with pulmonary hypersensitivity.





Graph: 49 Swasha krichrita- pattern wise distributions of patients with pulmonary hypersensitivity.



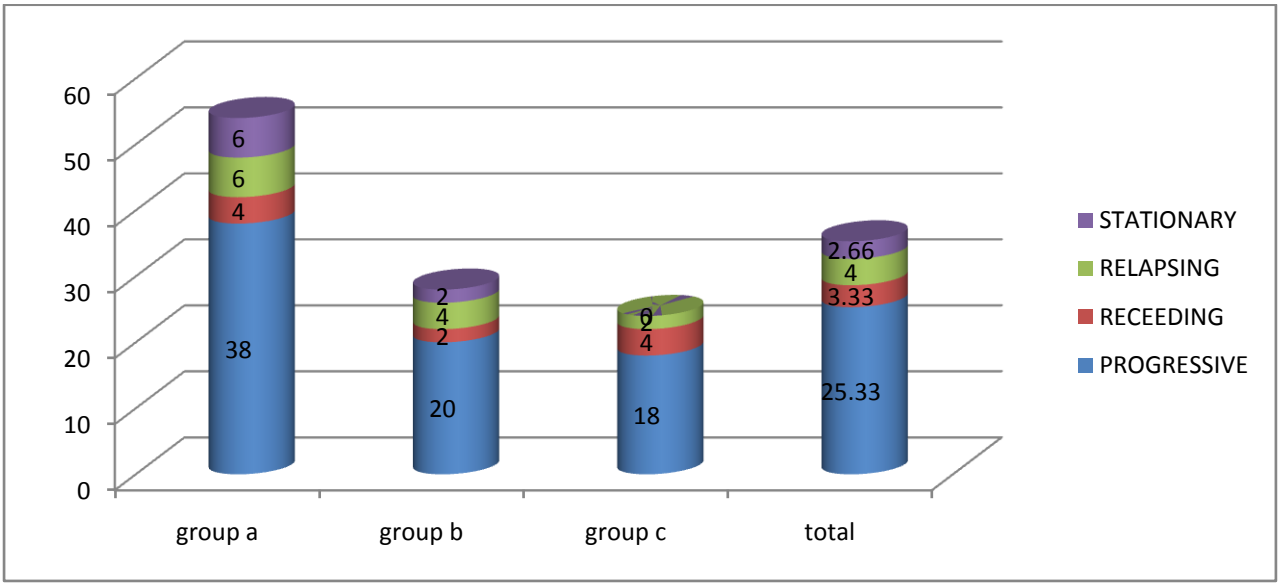


Table No.90

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY

OF SWASHA KRICHRITA

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	24	48	32	64	26	52	82	54.66
	MODERATE	03	06	01	02	00	00	02	1.33
	SEVERE	-	-	-	-	-	-	-	-

The patients selected for the clinical trial were having swasakrichrita of different severity.

Among them 82(54.66%) patients were having mild swasakrichrita . In group A, 24(48%) patients, ingroup B, 32(64%) patients ,andin group C, 26 (52%) patients were present .

02(1.33%) patients were having moderate swasakrichrita . In group A, 03(06%) patients, ingroup B, 01(02%) patients, and ingroup C, 00 (00%) patientswere present.

None of them had severe shwasakrichrita.

Table No.91

SWASHA KRICHRITA KALA WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

SWASHA KRICHRIT KALA		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	EARLY MORNING	13	26	16	32	14	28	43	28.66
	MORNING	01	02	01	02	00	00	02	1.33
	AFTERNOON	-	-	-	-	-	-	-	-
	EVENING	-	-	-	-	-	-	-	-
	NIGHT	02	04	00	00	00	00	02	1.33
	MID-NIGHT	11	22	09	18	18	36	38	25.33

The patients selected for the clinical trial were having swasakrichrita at different kala of the day .

Among them 43(28.66%) patients were having swasakrichrita in the early morning. Among whom, In group A, 13(26%) patients, in group ,B 16(32%) patients, and in group C ,14 (28%) patientswere present.

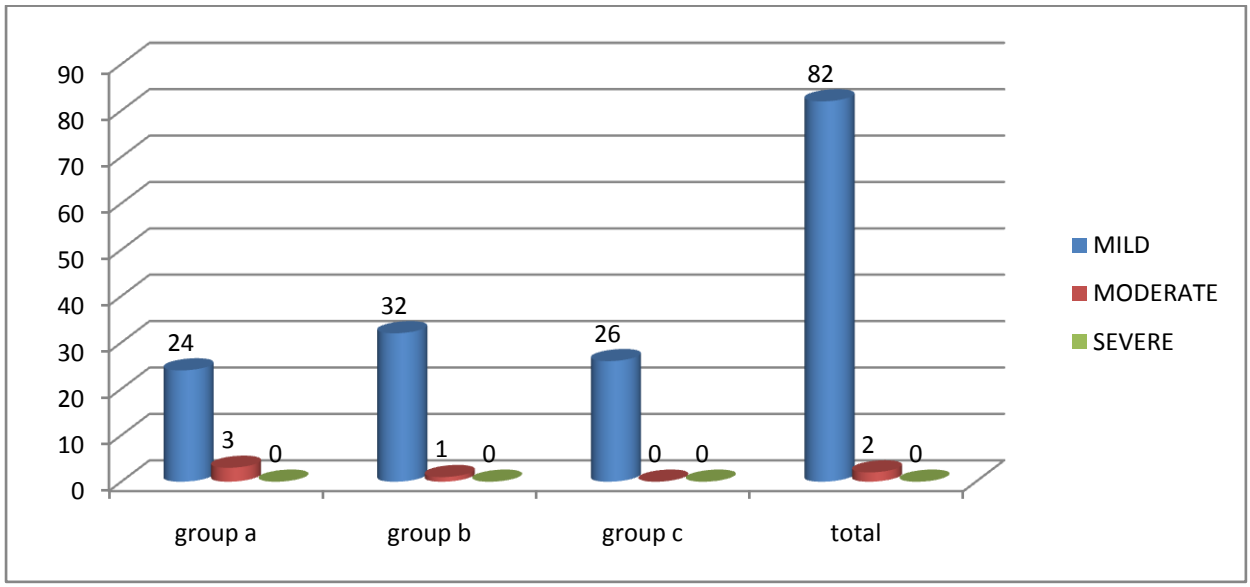
02(1.33%) patients were having swasakrichrita in the morning.Among whom, In group A, 01(02%) patients, in group B, 01(02%) patients, and in group C, 00 (00%) patientswere present.

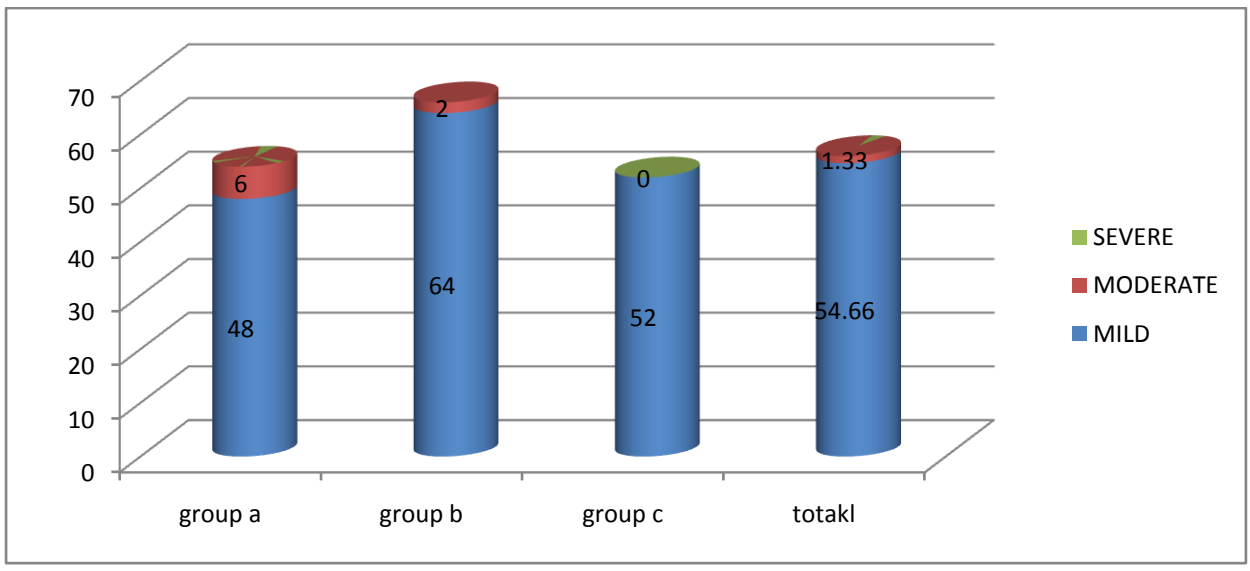
None of them had swasakrichrita in the afternoon and evening.

02(1.33%) patients were having swasakrichrita in the night.Among whom, In group A ,02(04%) patients, in group B ,00(00%) patients ,and in group C, 00 (00%) patientswere present.

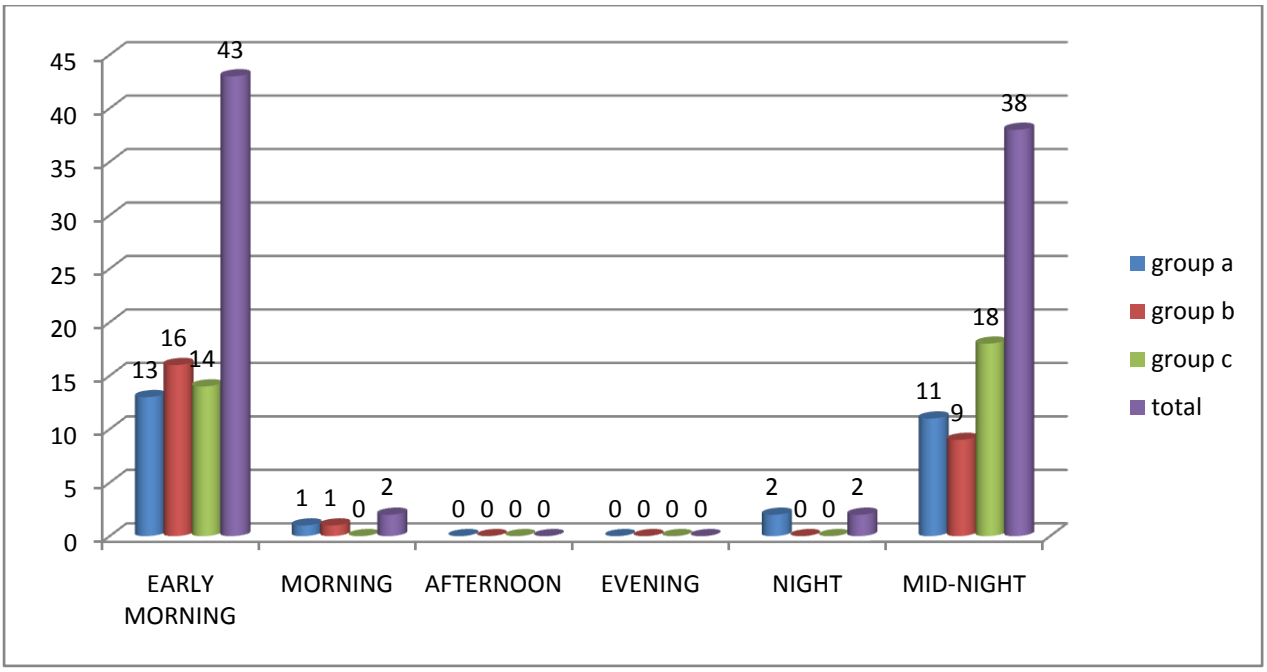
38(25.33%) patients were having swasa in the mid- night. Among whom,In group A, 11(22%) patients, in group B, 09(18%) patients, and in group C, 18 (36%) patientswere present.

Graph: 50 Swasha krichrita- severity wise distributions of patients with pulmonary hypersensitivity.





Graph: 51 Swasha krichrita- kala wise distributions of patients with pulmonary hypersensitivity.



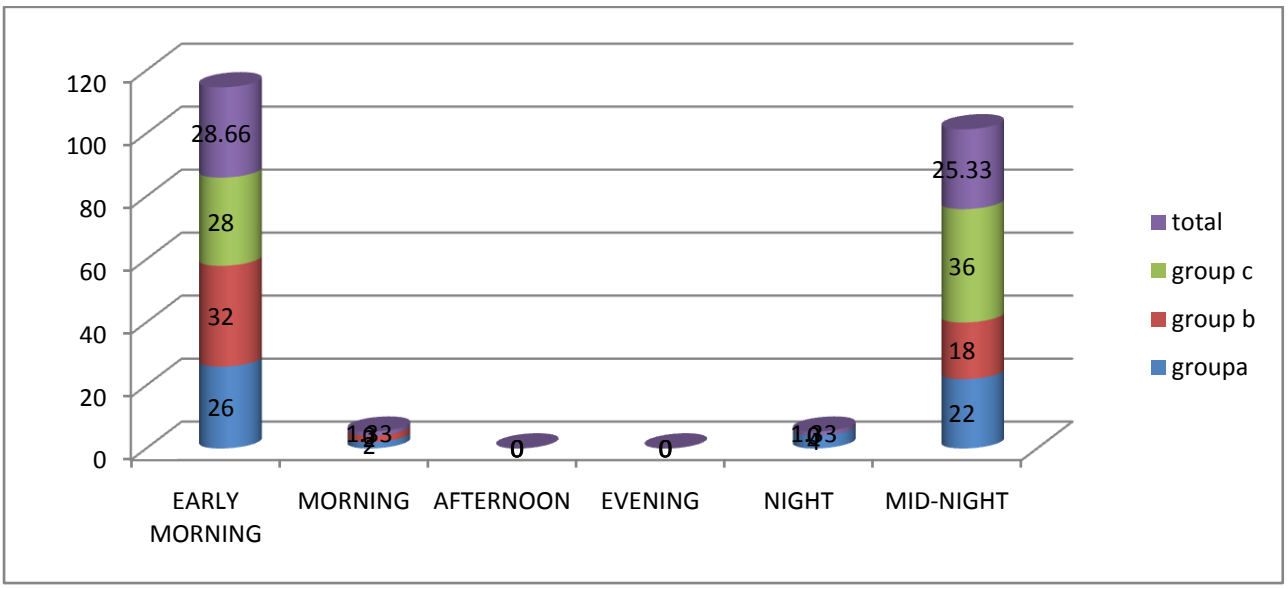


Table No.92

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO NETRAGATA LAKSHANAS

LAKSHANA	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
RAKTA VARNATA	37	74	41	82	32	64	110	73.33
NETRA KANDU	09	18	03	06	12	24	24	16
NETRA SRAVA	24	48	45	90	47	94	116	77.33

The patients selected for the clinical trial were having differentnetragathalakshanas .

Among them 110(73.33%) patients were having raktavarnata in the netra.Among whom, In group A, 37(74%) patients, in group B, 41(82%) patients, and ingroup C, 32 (64%) patients were present.

24(16%) patients were having netakandu . Among whom,In group A, 09(18%) patients, ingroup B, 03(06%) patients, andin group C ,12 (24%) patientswere present.

116(73.33%) patients were having netrastrava. Among whom,In group A ,24(48%) patients, ingroup B, 45(90%) patients, andin group C, 47 (94%) patientswere present.

Table No.93

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO KARNAGATA LAKSHANAS

LAKSHANA	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
VEDANA	02	04	01	02	04	08	07	4.66
BADIRYATA	29	58	23	46	15	30	67	44.66
FEELING OF FULLNESS	23	46	35	70	39	78	97	64.66

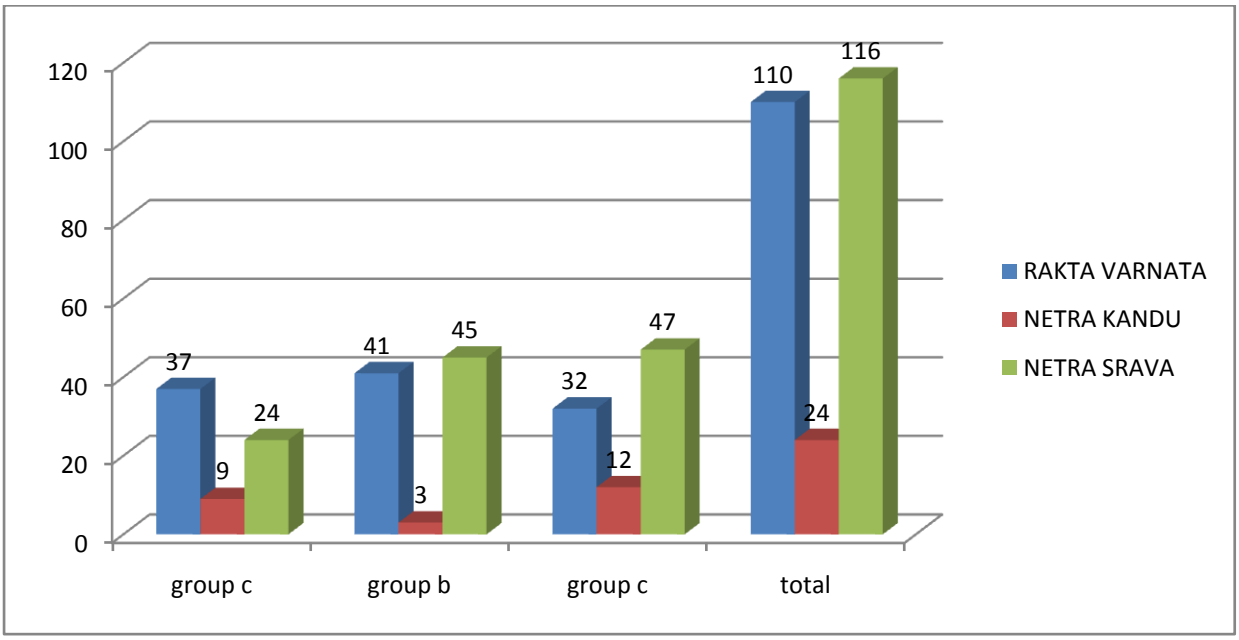
The patients selected for the clinical trial were having different karnagathalakshanas .

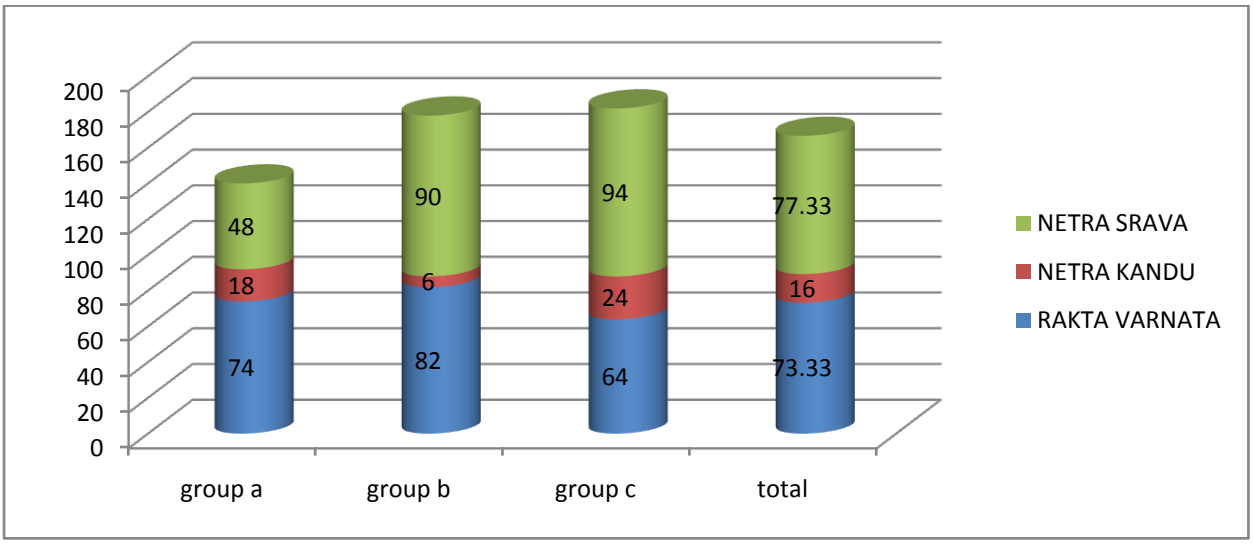
Among them 07(4.66%) patients were having vedana in the karna.Among whom, In group A, 02(04%) patients, ingroup B, 01(02%) patients, and in group C ,04 (08%) patientswere present.

67(44.66%) patients were having badiryata in the karna.Among whom In group A, 29(58%) patients,in group B ,23(46%) patients, and ingroup C, 15 (30%) patientswere present.

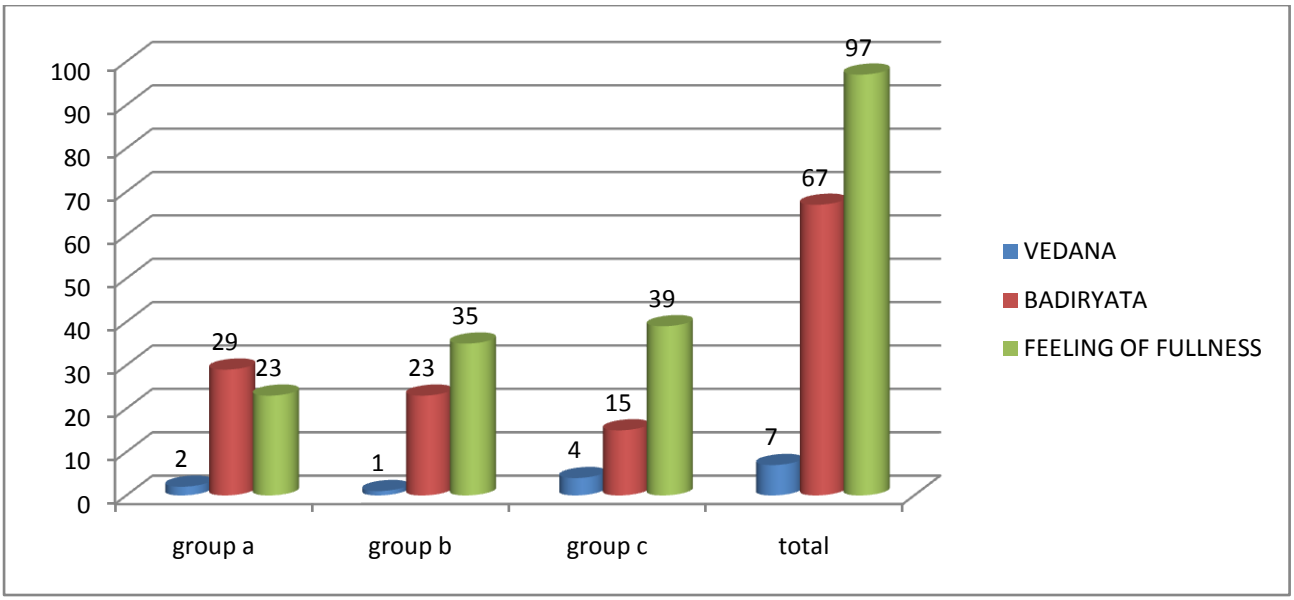
97(64.66%) patients were having feeling of fullnessin the karna.Among whom In group A, 23(46%) patients,in group B, 35(70%) patients, and ingroup C ,39 (78%) patientswere present.

Graph: 52 Netra gata lakshna wise distributions of patients with pulmonary hypersensitivity.





Graph: 53 Karnagata lakshna wise distributions of patients with pulmonary hypersensitivity.



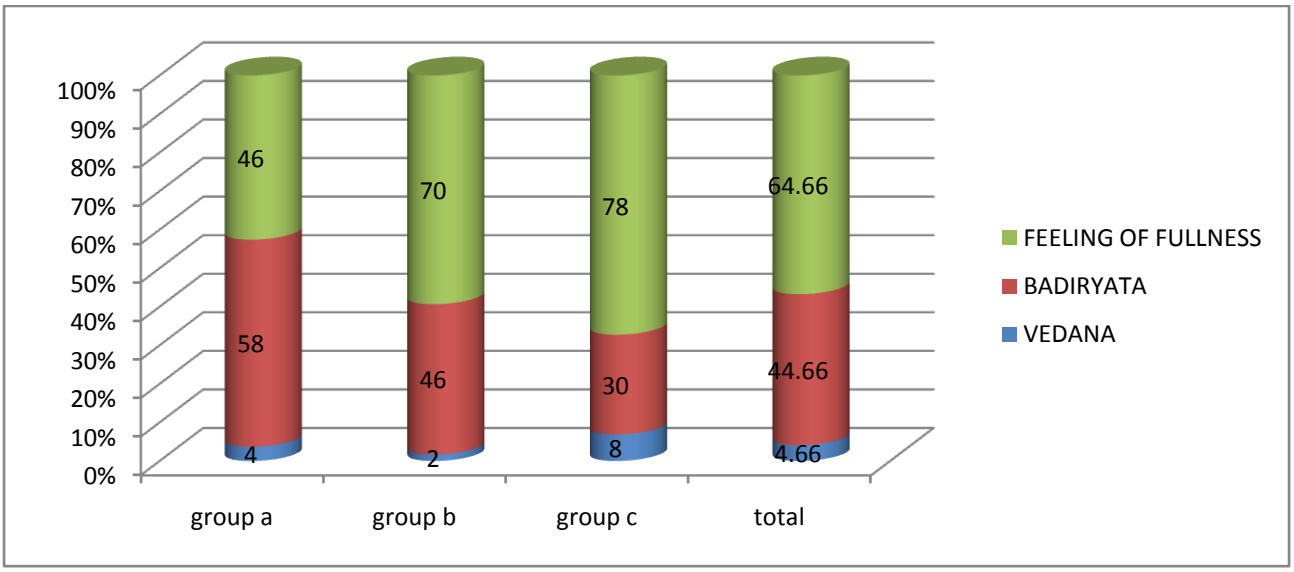


Table No.94

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SHIROGATA LAKSHANAS

LAKSHANA	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
SHOOLA	-	-	-	-	-	-	-	-
GAURAVATA	-	-	-	-	-	-	-	-
BOTH	50	100	50	100	50	100	150	100

The patients selected for the clinical trial had SHIROGATHA LAKSHANAS

No patients had shoola or gauravata individually, but all 150(100%) patients had both. Among whom, in group A, 50(100%) patients, in group B, 50(100%) patients, and in group C, 50(100%) patients were present.

Table No.95

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO GANDHA GNANA

LAKSHANA	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
PRESENT	33	66	36	72	39	78	108	72
PARTIALLY PRESENT	13	26	13	26	10	20	36	24
ABSENT	06	12	01	02	01	02	08	5.33

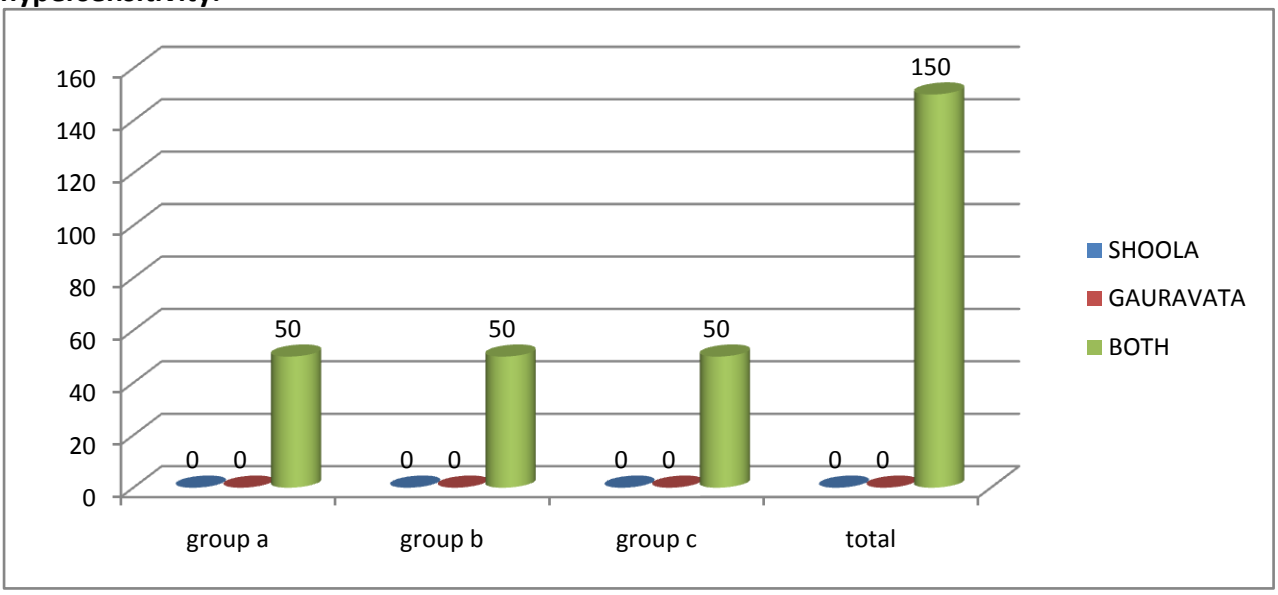
The patients selected for the clinical trial had differentness in GANDHA GNANA

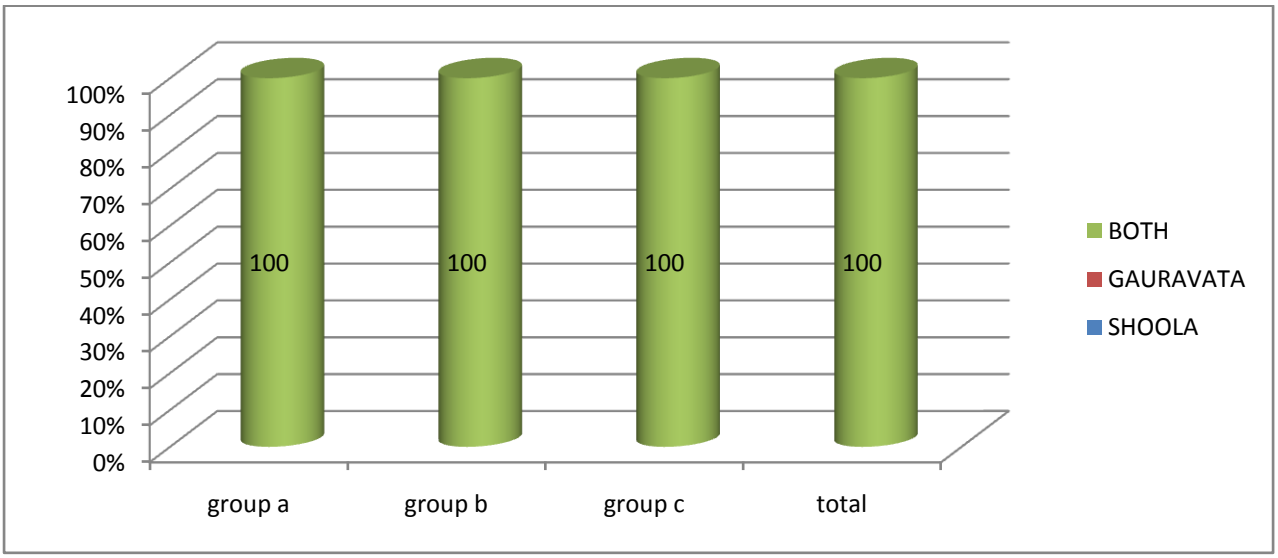
Among them in 108(72%) patientsgandhagnana was present. Among whom, in group A, 33(66%) patients, in group B, 36(72%) patients and,in group C, 39(78%) patientswere present.

In 36(24%) patients gandhagnana was partially present. Among whom, in group A, 13(26%) patients, in group B, 13(26%) patients and,in group C, 10(20%) patientswere present.

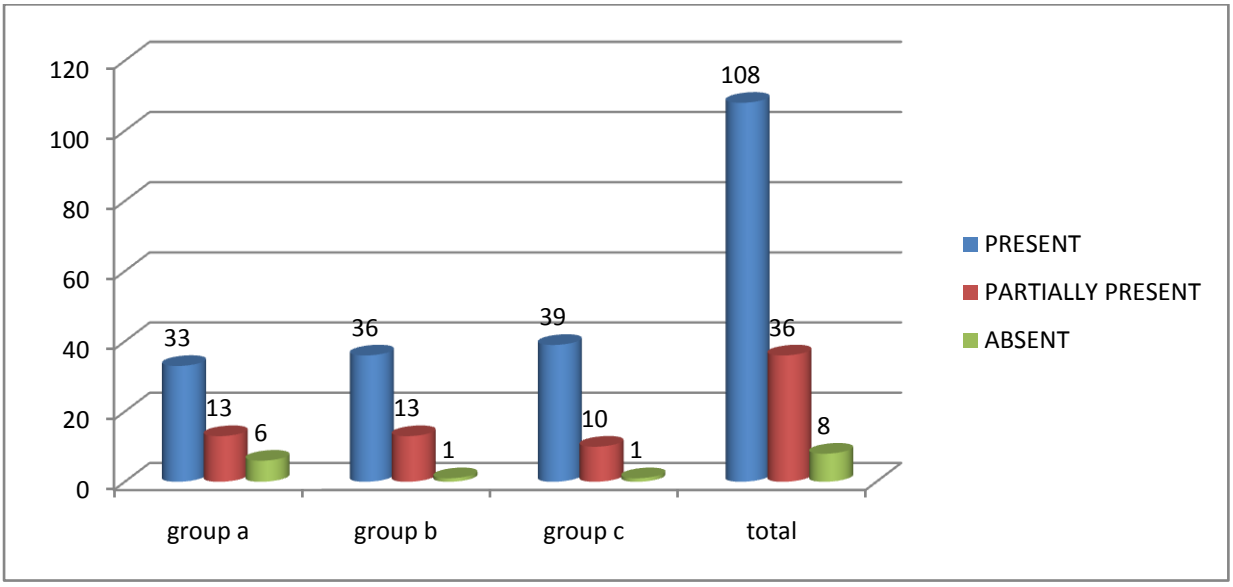
In 08(5.33%) patients gandhagnana was absent. Among whom, in group A, 06(12%) patients,in group B, 01(02%) patients and,in group C, 01(02%) patientswere present.

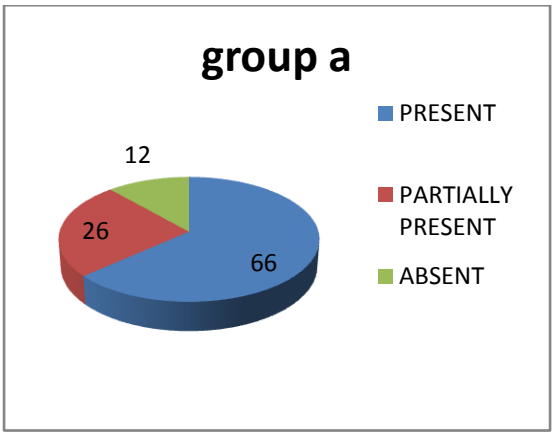
Graph: 54 Shirogata lakshna wise distributions of patients with pulmonary hypersensitivity.

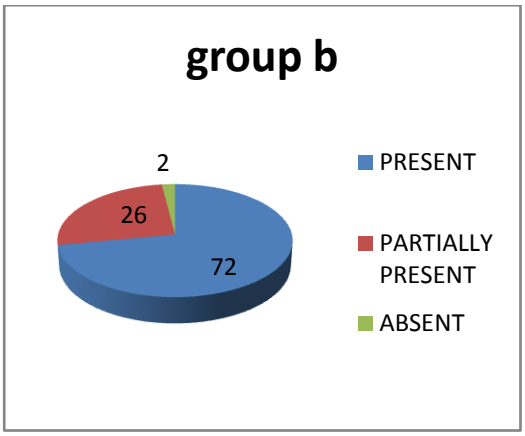


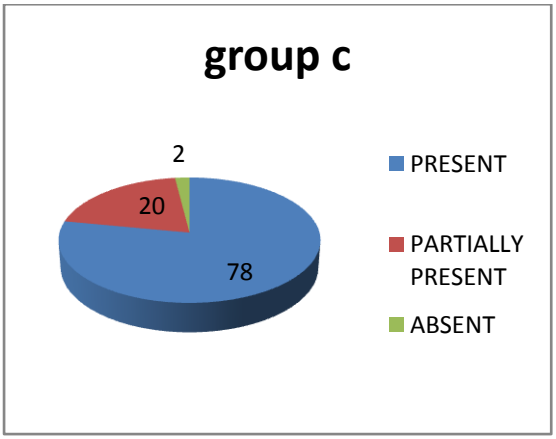


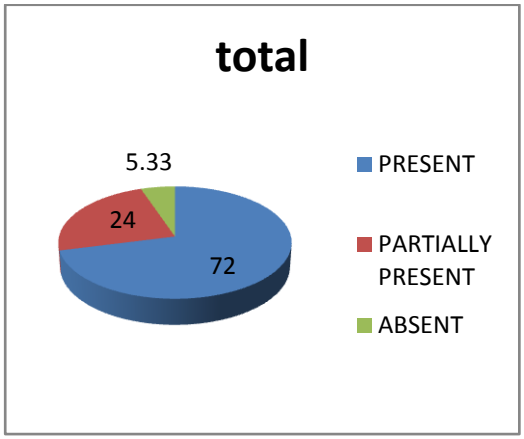
Graph: 55 Distribution of patients with pulmonary hypersensitivity according to Gandha gnana.











URAH VEDANA

Table No.96

URAH VEDANA DURATION WISE DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY.

DURATION	YEARS	GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	08-10 years	01	02	02	04	00	00	03	02
	06-08 years	03	06	00	00	00	00	03	02
	04-06 years	01	02	00	00	07	14	08	5.33
	02-04 years	04	08	00	00	03	06	07	4.66
	01-02 years	09	18	01	02	11	22	21	14

The patients had urahvedana of different duration.

Among them 03(02%) patientshad urahvedana from 08-10 years. Among whom, in group A, 01(02%) patients,in group B, 02(04%) patients and,ingroup C, 00 (00%) patients were present.

03(02%) patientshad urahvedana from 06-08 years. Among whom, in group A, 03(06%) patients, ingroup B, 00(00%) patients and,in group C, 00 (00%) patients were present.

08(5.33%) patientshad urahvedana from 04-06 years. Among whom, in group A, 01(02%) patients,in group B, 00(00%) patients and,in group C, 07 (14%) patients were present.

07(4.66%) patientshad urahvedana from 02-04 years. Among whom, in group A, 04(08%) patients,in group B, 00(00%) patients and,in group C, 03 (06%) patients were present.

21(14%) patientshad urahvedana from 01-02 years. Among whom, in group A, 09(18%) patients, ingroup B, 01(02%) patients , andin group C, 11 (22%) patients were present.

Table No.97

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO PATTERN OF URAH VEDANA

COURSE		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	PROGRESSIVE	01	02	01	02	00	00	02	1.33
	RECEEDING	-	-	-	-	-	-	-	-
	RELAPSING	-	-	-	-	-	-	-	-
	STATIONARY	17	34	00	00	01	02	18	12

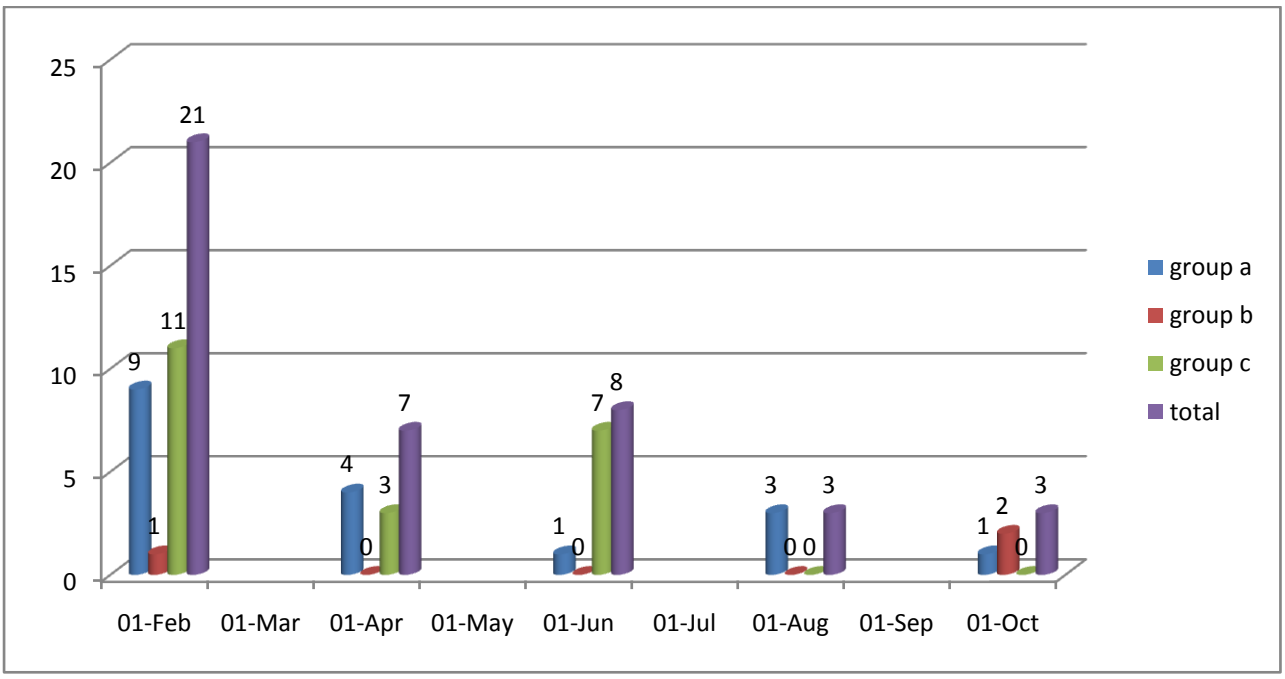
The patients selected for the clinical trial had different course ofurahvedana.

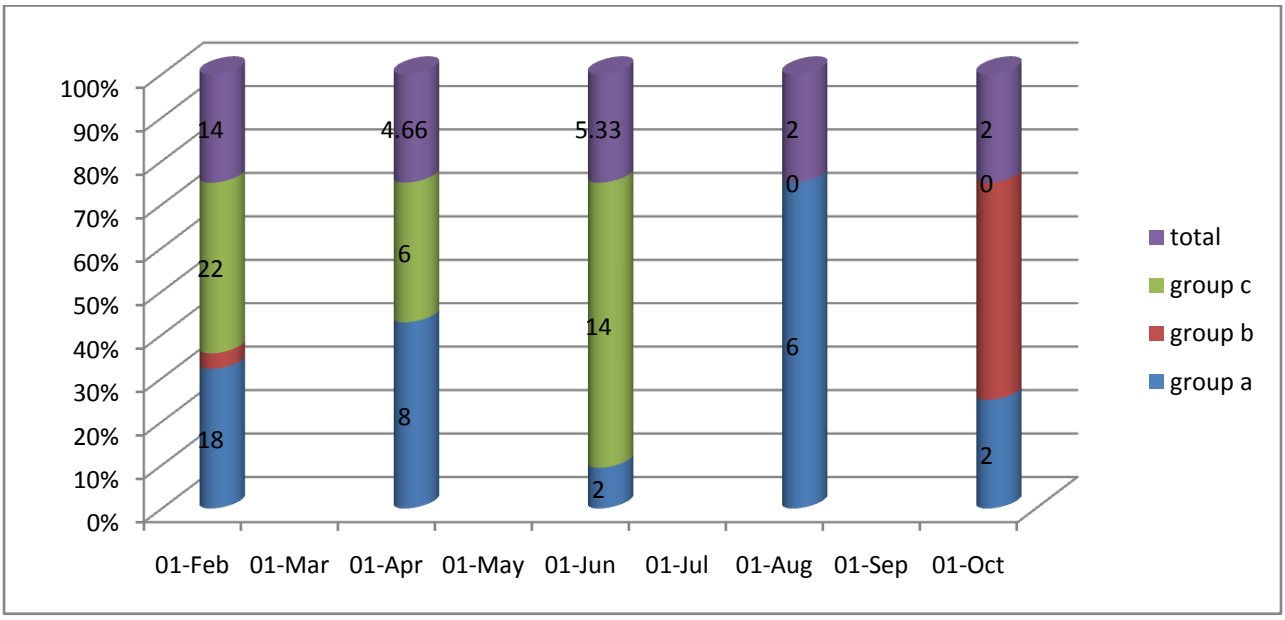
Among them 02(1.33%) patientshad progressiveurahvedana. In group A, 01(02%) patients, in group B, 01(02%) patients, and in group C, 00 (00%) patients were present.

No patients had receeding and relapsing urahvedans.

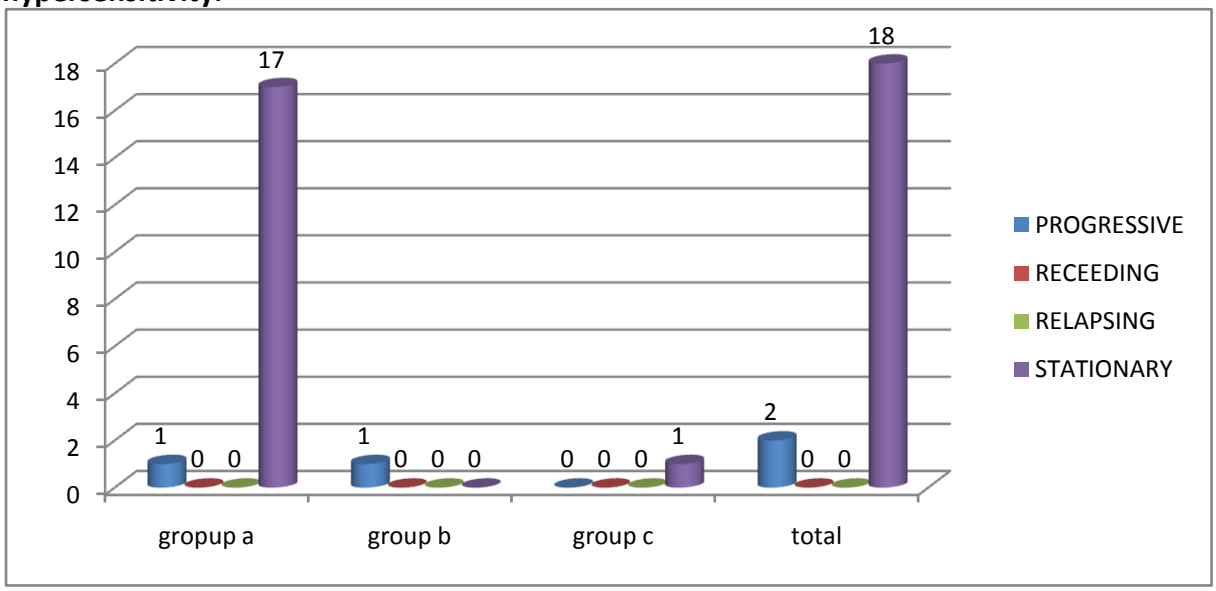
18(12%) patients had stationaryurahvedana. In group A, 17(34%) patients, in group B, 00(00%) patients, andin group C, 01 (02%) patientswere present.

Graph: 56 Uraha vedana duration wise distributions of patients with pulmonary hypersensitivity.





Graph: 57 Uraha vedana- pattern wise distributions of patients with pulmonary hypersensitivity.



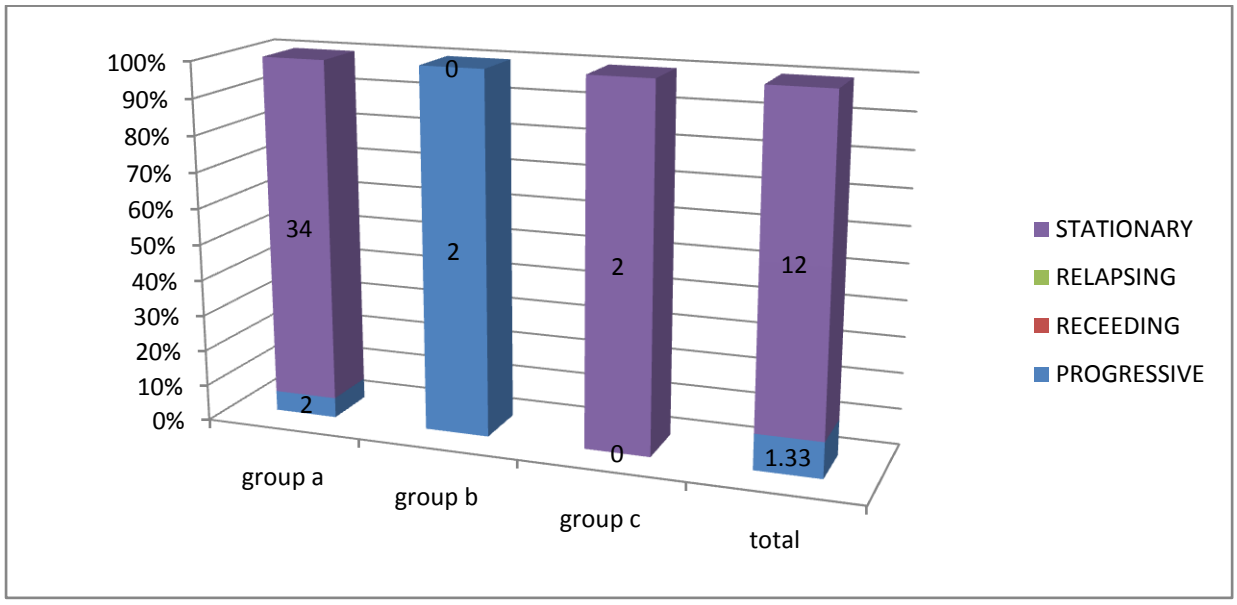


Table No.98

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SEVERITY

OF URAH VEDANA

SEVERITY		GROUP A		GROUP B		GROUP C		TOTAL	%
		N	%	N	%	N	%		
	MILD	16	32	01	02	01	02	18	12
	MODERATE	02	04	00	00	00	00	02	1.33
	SEVERE	-	-	-	-	-	-	-	-

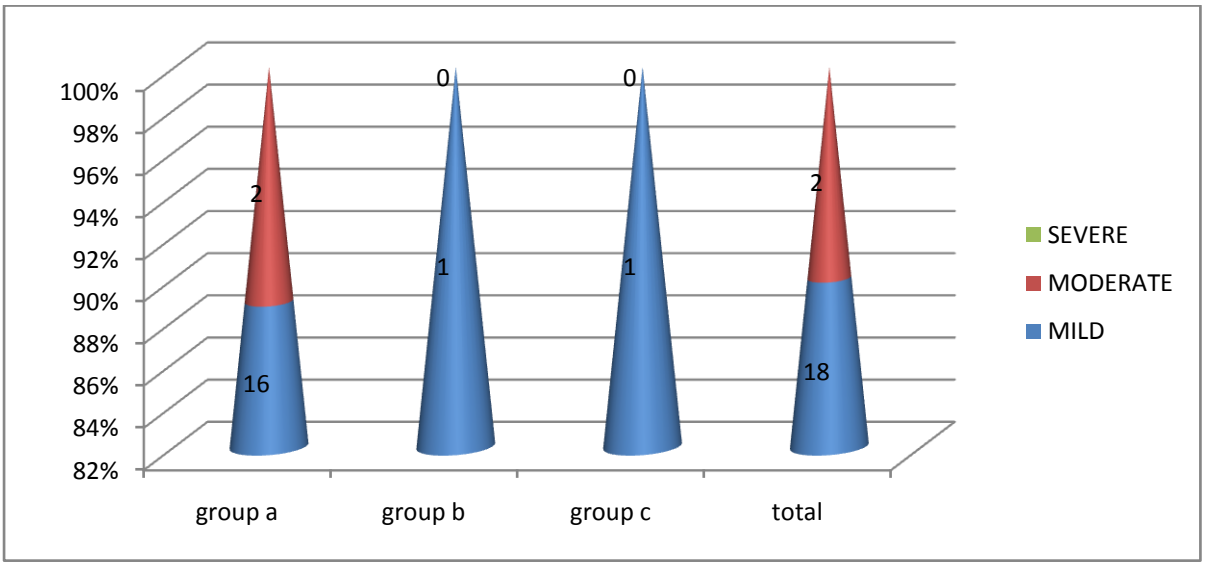
The patients selected for the clinical trial were of urahvedana of different severity.

Among them 18(12%) patients had mildurahvedana .In group A, 16(32%) patients, in group B, 01(02%) patients, and ingroup C, 01 (02%) patientswere present.

Among them 02(1.33%) patients had moderateurahvedana. Among whom, in group A, 02(04%) patients, ingroup B, 00(00%) patients and ingroup C, 0 (00%) patientswere present.

None of them had severeurahvedana.

Graph:58 Urah vedana severity wise distributions of patients with pulmonary hypersensitivity.



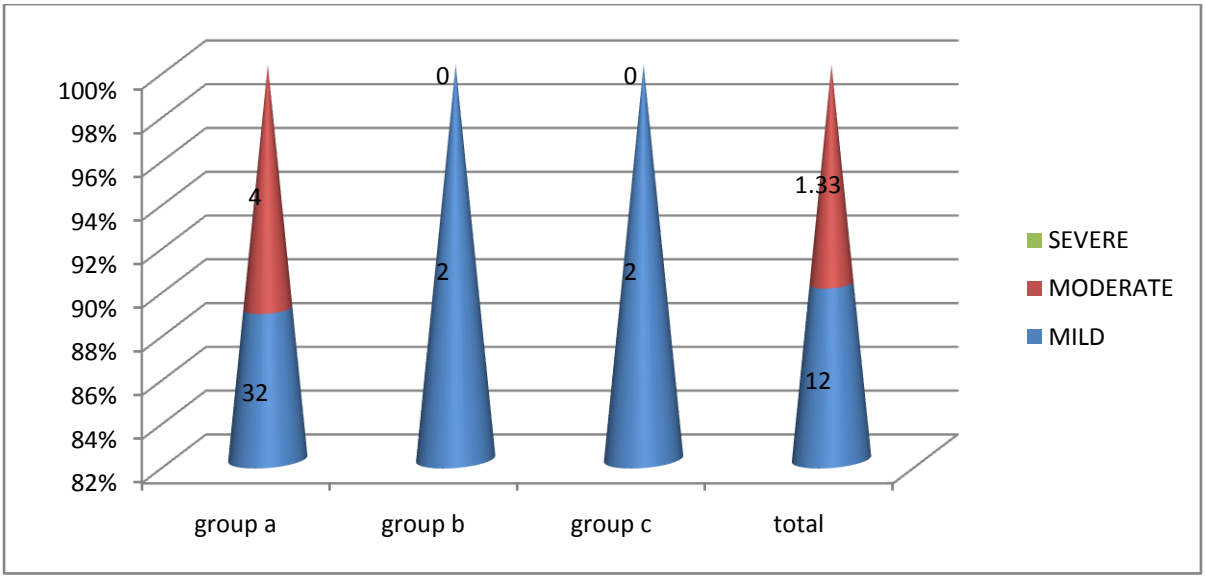


Table No.99

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO AAHARAJA NIDANA

AAHARAJA NIDAANA	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
1. ABHISHYANDI AHARA(DADHI)	50	100	50	100	50	100	150	100
2. SHEETA GUNATMAKA (ICE CREAM & COOL DRINKS)	50	100	50	100	50	100	150	100
3. MADHURA RASA PRADHANA	21	42	32	64	29	58	82	54.66
4. NAVA AHARA SEVANA	09	18	06	12	11	22	26	17.33
5. ANUPA MAMSA SEVANA	24	48	36	72	41	82	101	67.33
6. AUDUKA MAMSA SEVANA	24	48	29	58	35	70	88	58.66
7. AMA KSHEERA SEVANA	-	-	01	02	04	08	05	3.33
8. SHEETA JALA SEVANA	50	100	50	100	50	100	150	100

The patients selected for the clinical trial were exposed to different nidanas.

Among them 150(100%) patientsconsumed ABHISHYANDI AHARA(DADHI). in group A, 50(100%) patients, group B, 50(100%) patients and, group C, 50 (100%) patientswere present.

150(100%) patients consumed SHEETA GUNATMAKA(ICE CREAM & COOL DRINK) . Among whom, in group A, 50(100%) patients,in group B, 50(100%) patients and,in group C, 50 (100%) patientswere present.

82(54.66%) patients consumed MADHURA RASA PRADHANA .Among whom,in group A, 21(42%) patients, in group B ,32(64%) patients, and in group C, 29(58%) patientswere present.

26(17.33%) patients consumed NAVA AHARA .Among whom,in group A, 09(18%) patients, in group B 06(12%) patients ,andin group C, 11 (22%) patientswere present.

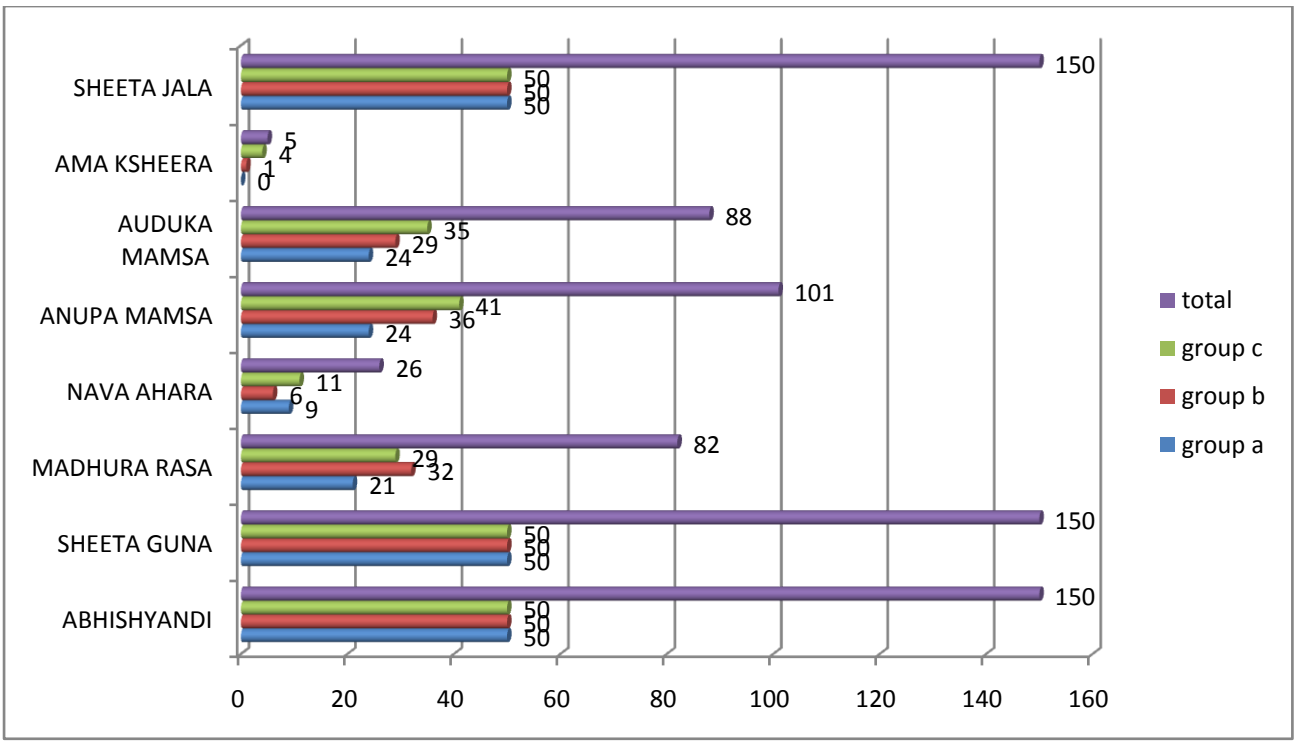
101(67.33%) patients consumed ANUPA MAMSA .Among whom, in group A, 24(48%) patients, in group B, 36(72%) patients, and in group C, 41 (82%) patientswere present.

88(58.66%) patients consumed AUDUKA MAMSA .Among whom,in group A, 24(48%) patients, in group B, 29(58%) patients ,and in group C, 35 (70%) patientswere present.

05(3.33%) patients consumed AMA KSHEERA .Among whom,in group A, 00(00%) patients, in group B, 01(02%) patients ,andin group C, 04 (08%) patientswere present.

150(100%) patients were exposed to SHEETA JALA SEVANA.Among whom,in group A, 50(100%) patients, in group B, 50(100%) patients, and in group C, 50 (100%) patients were present.

Graph: 59 Aaharaja Nidana wise distributions of patients with pulmonary hypersensitivity.



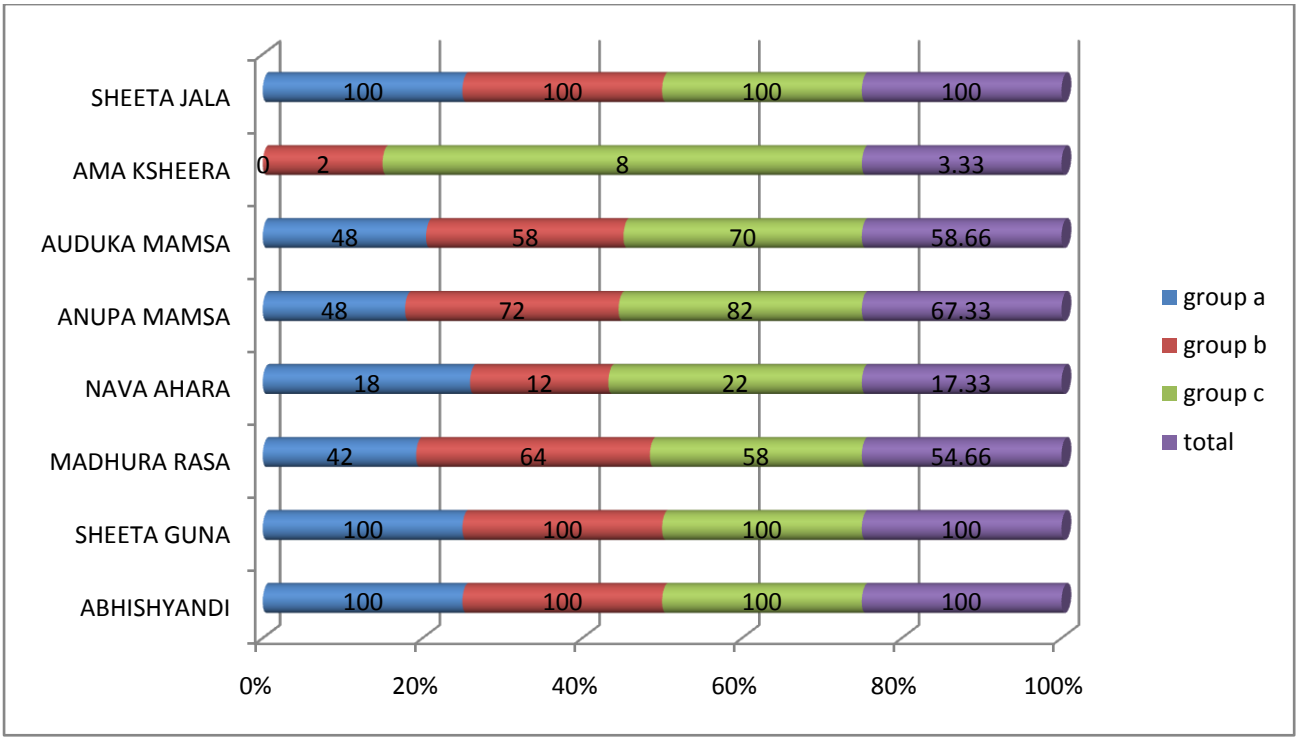


Table No.100

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO VIHARAJA NIDANA

VIHARAJA NIDANA	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
1. DHUMA PANA SEVANA	19	38	31	62	28	56	78	52
2. RAJA (DUST)	21	42	41	82	39	78	102	68
3. STAYING IN SHEETA STHANA(A/C etc)	13	26	21	42	17	34	51	34
4. DIVA SWAPNA	03	06	07	14	05	10	15	10
5. SHEETA VAYU SPARSHANA	50	100	50	100	50	100	150	100

The patients selected for the clinical trial were exposed to different nidanas

Among them 78(52%) patients were exposed to DHUMA PANA SEVANA. Among whom,in group A ,19(38%) patients,in group B, 31(62%) patients ,and group C,28(56%) patientswere present.

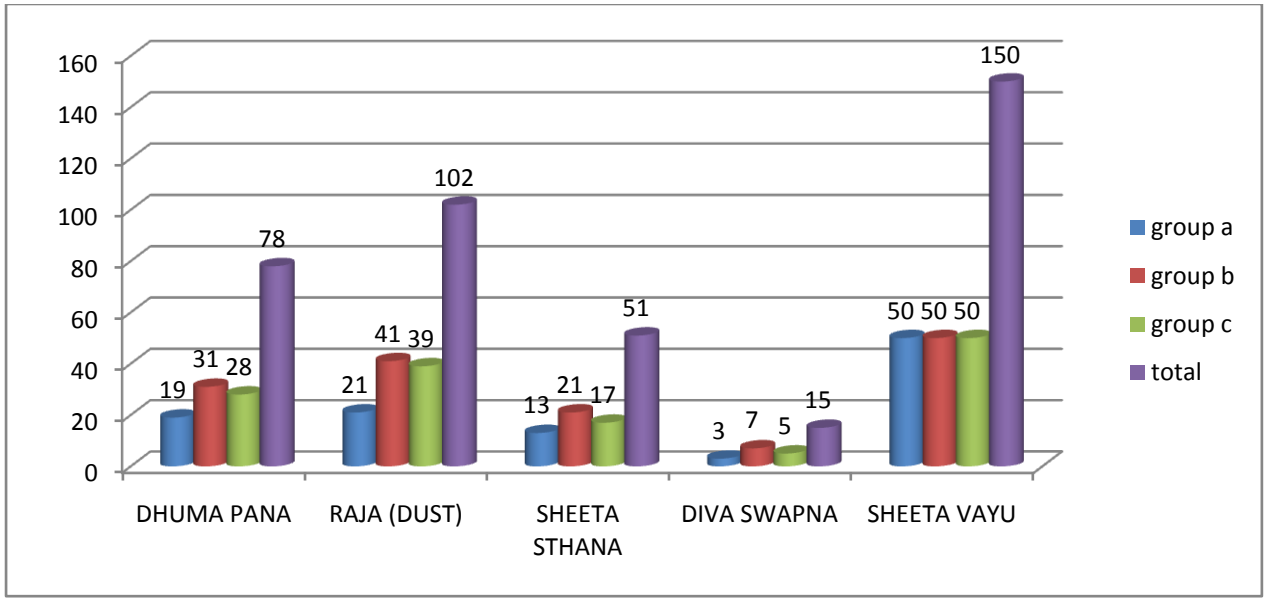
102(68%) patients were exposed to RAJA (DUST). Among whom,in group A, 21(42%) patients, in group B, 41(82%) patients, and in group C, 39 (78%) patientswere present.

51(34%) patients were STAYING IN SHEETA STHANA(A/C etc)). Among whom,in group A, 13(26%) patients, in group B 21(42%) patients, and in group C, 17 (34%) patientswere present.

15(10%) patientspracticed DIVA SWAPNA . Among whom,in group A, 3(6%) patients, in group B, 7(14%) patients ,and in group C,5 (10%) patients were present.

150(100%) patients were exposed to SHEETA VAYU SPARSHA . Among whom,in group A ,50(100%) patients, in group B, 50(100%) patients, andin group C,50(100%) patients were present.

Graph: 60 VihaarajaNidana wise distributions of patients with pulmonary hypersensitivity.



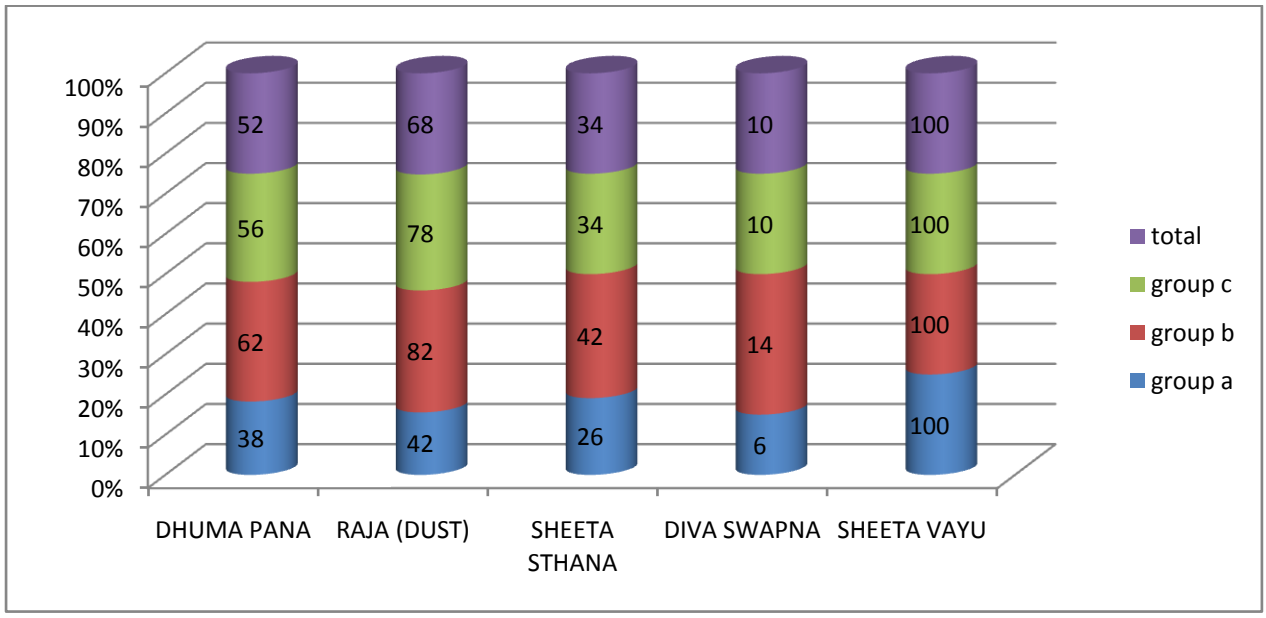


Table No.101

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO ALLERGENS THEY WERE EXPOSED TO

	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
DUST	50	100	50	100	50	100	150	100
POLLEN	18	36	32	64	25	50	75	50
HOUSE MITE	12	24	10	20	26	52	48	32
PET ANIMALS	09	18	09	18	07	14	25	16.66
CHEMICAL FUMES	06	12	15	30	24	48	45	30
COTTON DUST	09	18	04	08	08	16	21	14
PERFUMES	34	68	39	78	22	44	95	63.33

The patients selected for the clinical trial were exposed to various allergens.

Among them 150(100%) patients were exposed to dust. In group A, 50(100%) patients, group B, 50(100%) patients and group C, 50(100%) patientswere present.

75(50%) patients were exposed to pollen. Among whom, in group A, 18(36%) patients,in group B, 32(64%) patients, and ingroup C, 25(50%) patientswere present.,

48(32%) patients were exposed to house mite. Among whom, in group A, 12(24%) patients,in group B, 10(20%) patients ,and ingroup C, 26(52%) patientswere present.

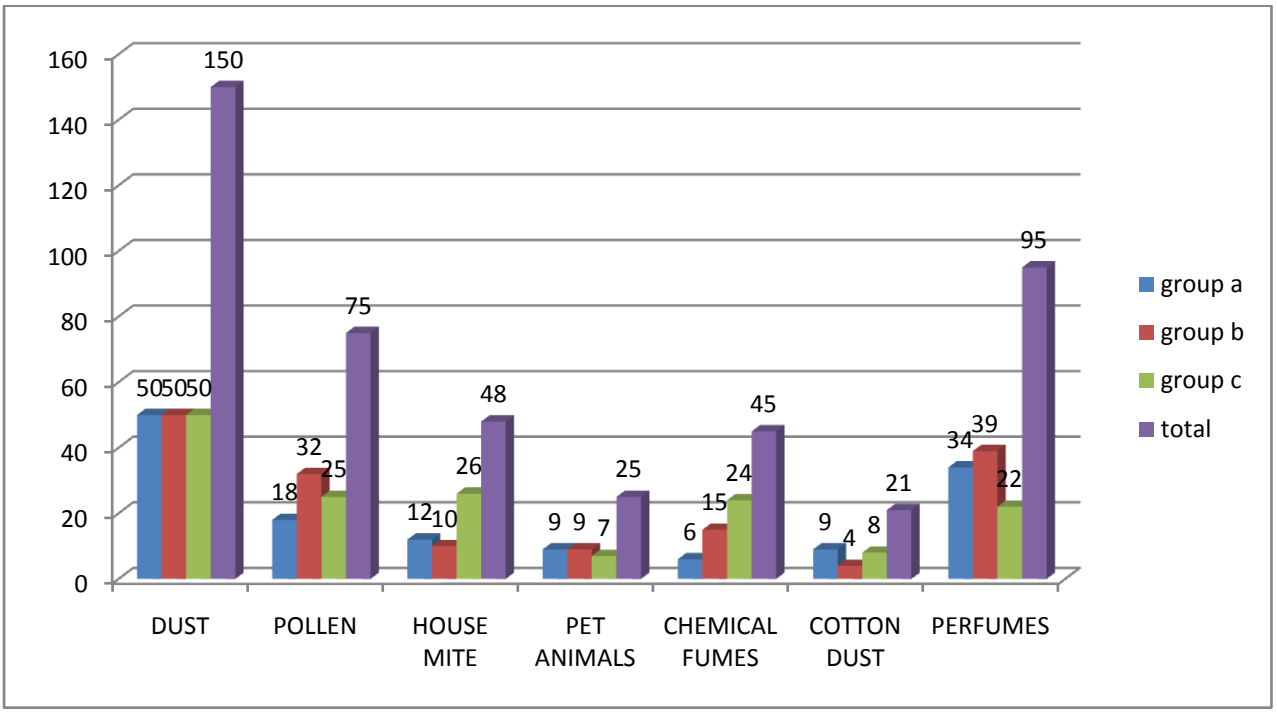
25(16.66%) patients were exposed to pet animals. Among whom, in group A, 09(18%) patients,in group B, 09(18%) patients, andin group C, 07(14%) patientswere present.

45(30%) patients were exposed to chemical fumes. Among whom, in group A, 6(12%) patients,in group B, 15(30%) patients, andin group C, 24(48%) patientswere present.

21(14%) patients were exposed to cotton dust. Among whom ingroup A, 09(18%) patients,in group B 04(08%) patients, andin group C, 08(16%) patientswere present.

95(63.33%) patients were exposed to perfumes . Among whom,in group A 34(68%) patientsin, group B 39(78%) patients andin group C22 (44%) patientswere present.

Graph: 61 Distribution of patients with pulmonary hypersensitivity according to allergens they were exposed to



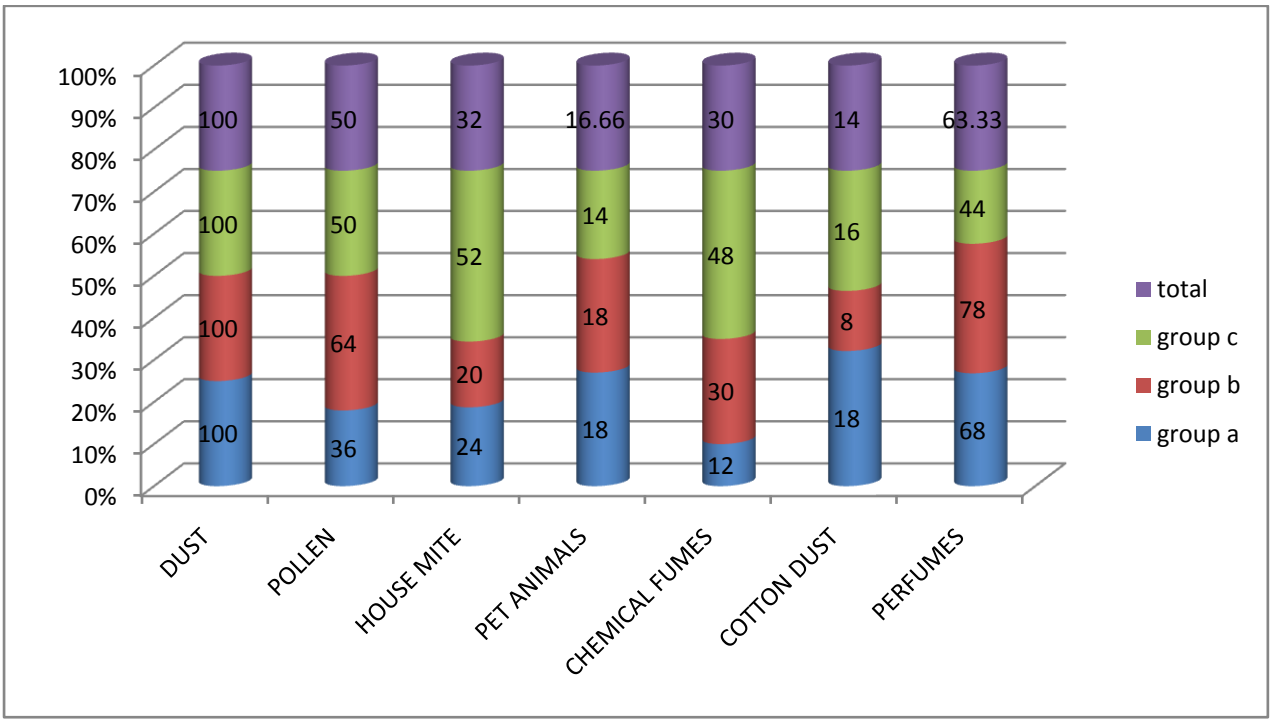


Table No.102

DISTRIBUTION OF 150 PATIENTS WITH PULMONARY HYPERSENSITIVITY ACCORDING TO SMOKES THEY WERE EXPOSED TO

SMOKE	GROUP A		GROUP B		GROUP C		TOTAL	%
	N	%	N	%	N	%		
AGARBATTI	43	86	49	98	42	84	134	89.33
MOSQUITO COIL	39	78	44	88	41	82	124	82.66
CIGARETTE/ BEEDI	14	28	22	44	11	22	47	31.33
ANY SMOKE	50	100	46	92	46	92	142	94.66

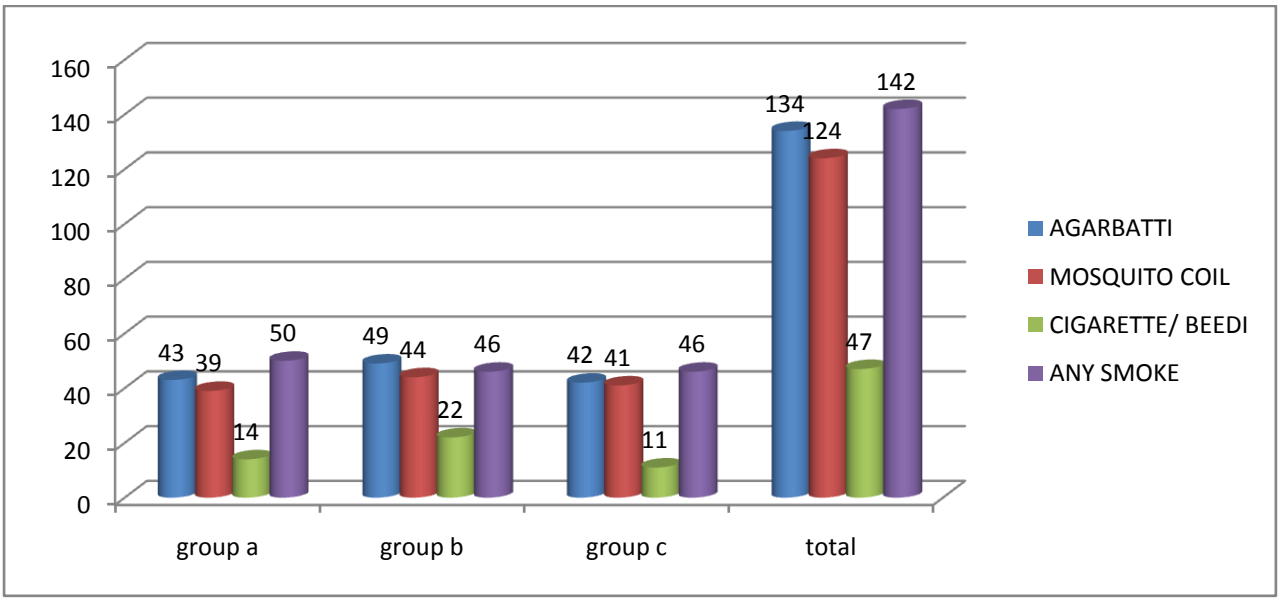
134(89.33%) patients were exposed to agarbatti. Among whom, in group A, 43(86%) patients,in group B, 49(98%) patients, andin group C, 42 (84%) patientswere present.

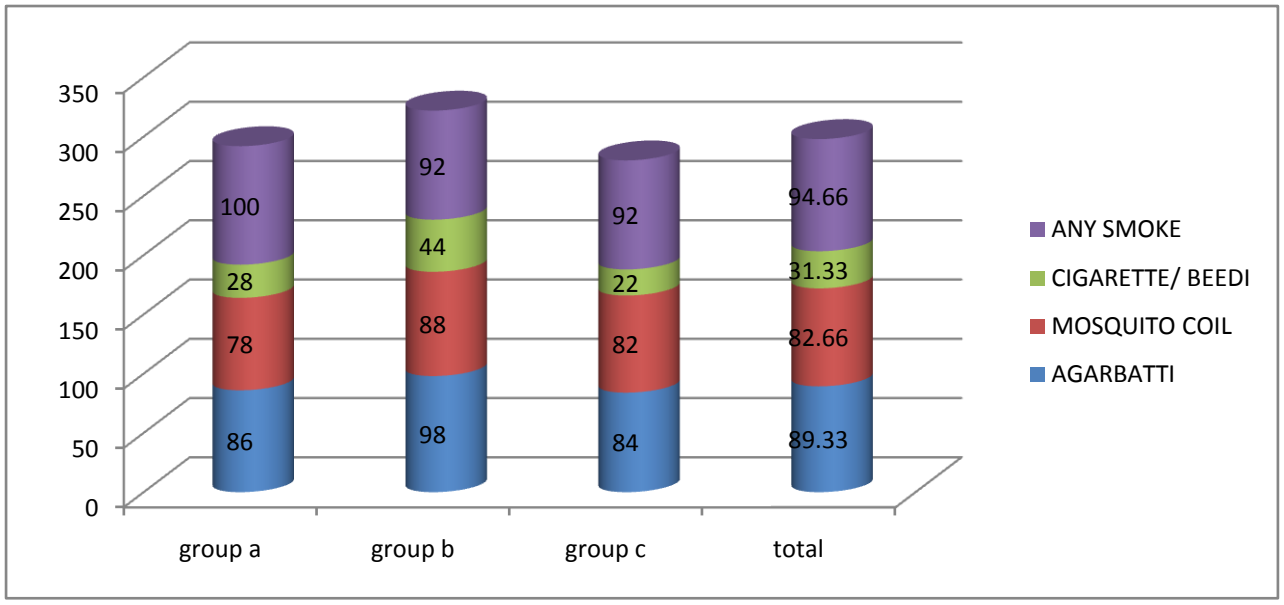
124(82.66%) patients were exposed to mosquito coil . Among whom, in group A, 39(78%) patients,in group B, 44(88%) patients, andin group C,41 (82%) patientwere presents.

47(31.33%) patients were exposed to cigarette/ beedi. Among whom, ingroup A, 14(28%) patients,in group B, 22(44%) patients, andin group C, 11 (22%) patientswere present.

142(94.66%) patients were exposed to some kind of smoke. Among whom, in group A, 50(100%) patients,in group B, 46(92%) patients ,andin group C,46(92%) patientswere present

Graph: 62 Distribution of patients with pulmonary hypersensitivity according to smokes they were exposed to




350
300
250
200
150
100
50
0
100
28
78
86
92
44
88
98
92
22
82
84
94.66
31.33
82.66
89.33
group a
group b
group c
total
ANY SMOKE
CIGARETTE/ BEEDI
MOSQUITO COIL
AGARBATTI

05.2 RESULTS & STATISTICAL ANALYSIS

KASA

Table No.103: Comparison Of Mean Score Of Kasa In Between Groups During The Treatment And Follow Up Period.

GROUP	BT	A	B	C	D	AT	P	Q	R	S
GR A	3.3	3.12	2.4	1.78	1	0.22	0.08	0	0	0
GR B	3.56	3.44	2.5	1.84	1.1	0.16	0.12	0	0	0
GR C	3.56	3.26	2.48	1.52	0.72	0.06	0.02	0	0	0

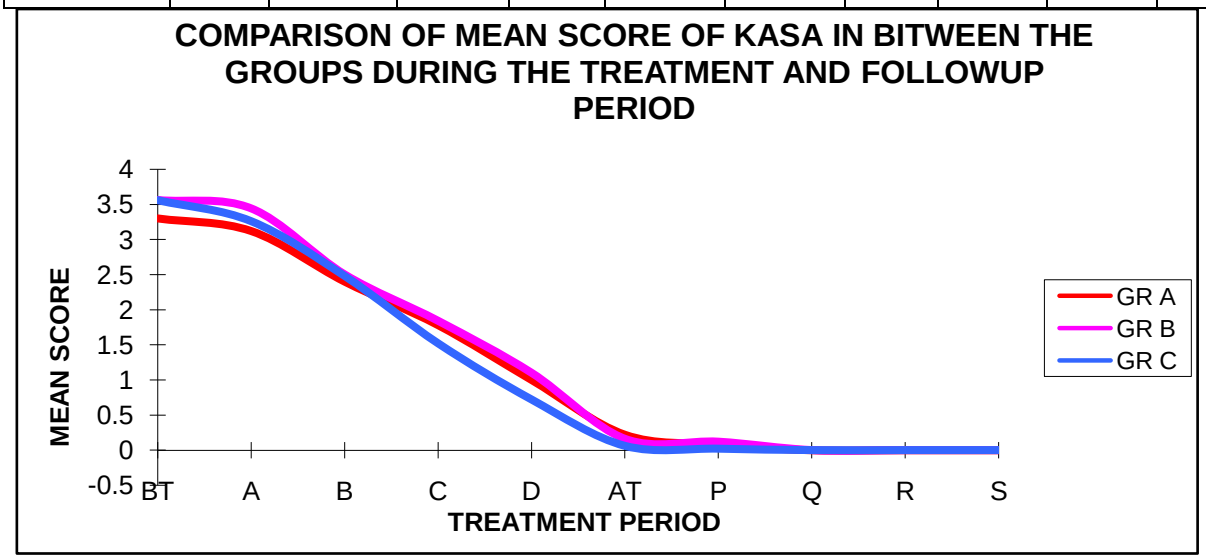


Table no. 104: Statistical analysis of kasa in Group A.

Paired Samples Test

		Paired Differences					t	df	p
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.08000	.77828	.11006	2.85882	3.30118	27.983	49	.000

Table no. 105: Stastitical analysis of kasa in Group B

Paired Samples Test

		Paired Differences					t	df	P
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.40000	.53452	.07559	3.24809	3.55191	44.978	49	.000

Table no. 106: Stastitical analysis of kasa in Group C

Paired Samples Test

	Paired Differences					t	df	p (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	3.50000	.50508	.07143	3.35646	3.64354	49.000	49	.000

From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05

Difference between groups

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other.

ANOVA

Table no. 107: **Stastitical analysis of severity Kasa**

	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	.653	2	.327	2.650	.074
Within Groups	18.120	147	.123		
Total	18.773	149			

As the p value is 0.074 > 0.05 Ho cannot be rejected.

Thus we can say that all the three groups are having statistically significant effect in reducing kasa, but there is no statistically significant difference between the groups.

From which we can say that all the three groups are having similar effect in reducing kasa.

KSHAVATU

Table No. 108 Comparison Of Mean Score Of Kshavathu In Between The Groups During The Treatment And Followup Period

GROUP	BT	A	B	C	D	AT	P	Q	R	S
GR A	3.62	3.48	2.54	1.74	1	0.18	0.08	0	0	0

GR B	3.54	3.48	2.58	1.98	1.16	0.24	0.02	0	0	0
GR C	3.62	3.28	2.32	1.36	0.72	0.06	0	0	0	0

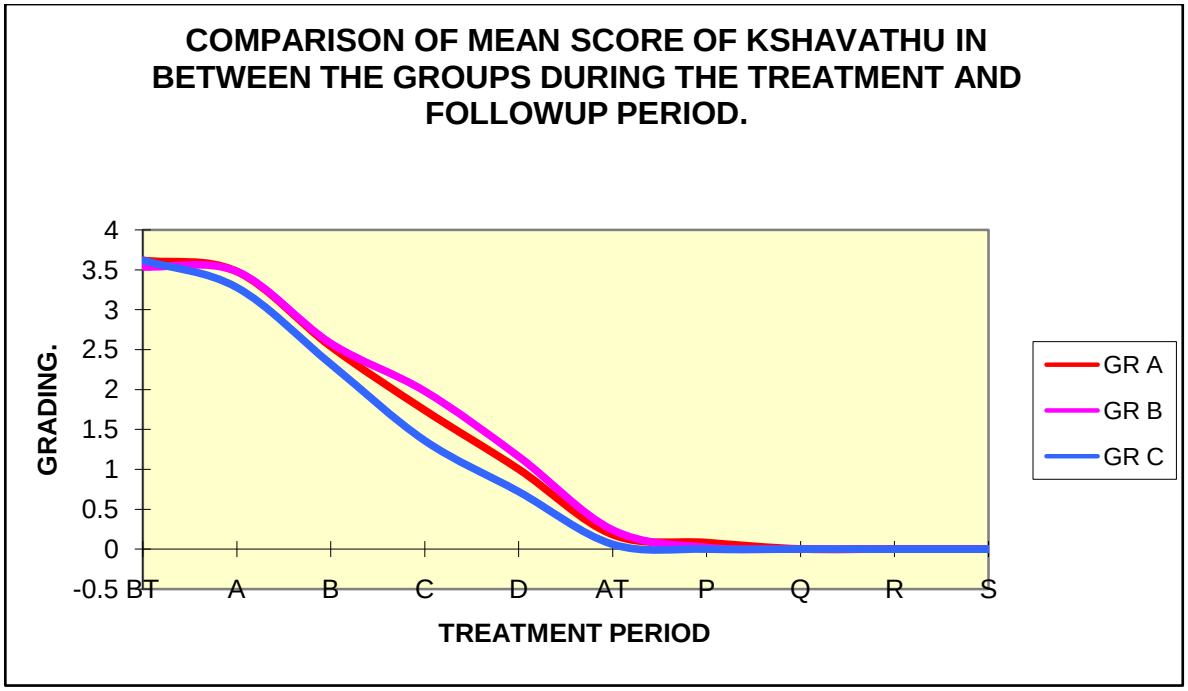


Table no. 109: Stastitical analysis of Kshavatu in Group A

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.08000	.77828	.11006	2.85882	3.30118	27.983	49	.000

Table no. 110: Stastitical analysis of Kshavatu in Group B

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.30000	.54398	.07693	3.14540	3.45460	42.896	49	.000

Table no. 111: Stastitical analysis of Kshavatu in Group C

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	3.56000	.50143	.07091	3.41750	3.70250	50.203	49	.000

From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is <0.05 .

Difference between groups

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other.

Table no. 112: Stastitical analysis of severity Kshavatu.

ANOVA

SEVERITY

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.840	2	.420	3.196	.044
Within Groups	19.320	147	.131		
Total	20.160	149			

As p < 0.05 we can reject Ho at 5 % Los.

Thuds it can be said that there is a statistically significant difference between the readings of the three groups.

Based of 95 % confidence interval of the difference it can be said that the results of group C is superior to others, as 95 % confidence interval of the difference of group C is higher than that of group A and group B .when compared group B is found to be superior to that of group A.

Nasasrava

Table no. 113: Comparison of mean score of nasa srava in between groups during treatment and follow up period.

GROUP	BT	A	B	C	D	AT	P	Q	R	S
GR A	3.58	3.48	2.56	1.78	1.08	0.22	0.12	0	0	0

GR B	3.56	3.44	2.46	1.62	1.1	0.18	0.12	0	0	0
GR C	3.42	3	2.2	1.36	0.72	0.06	0.04	0	0	0

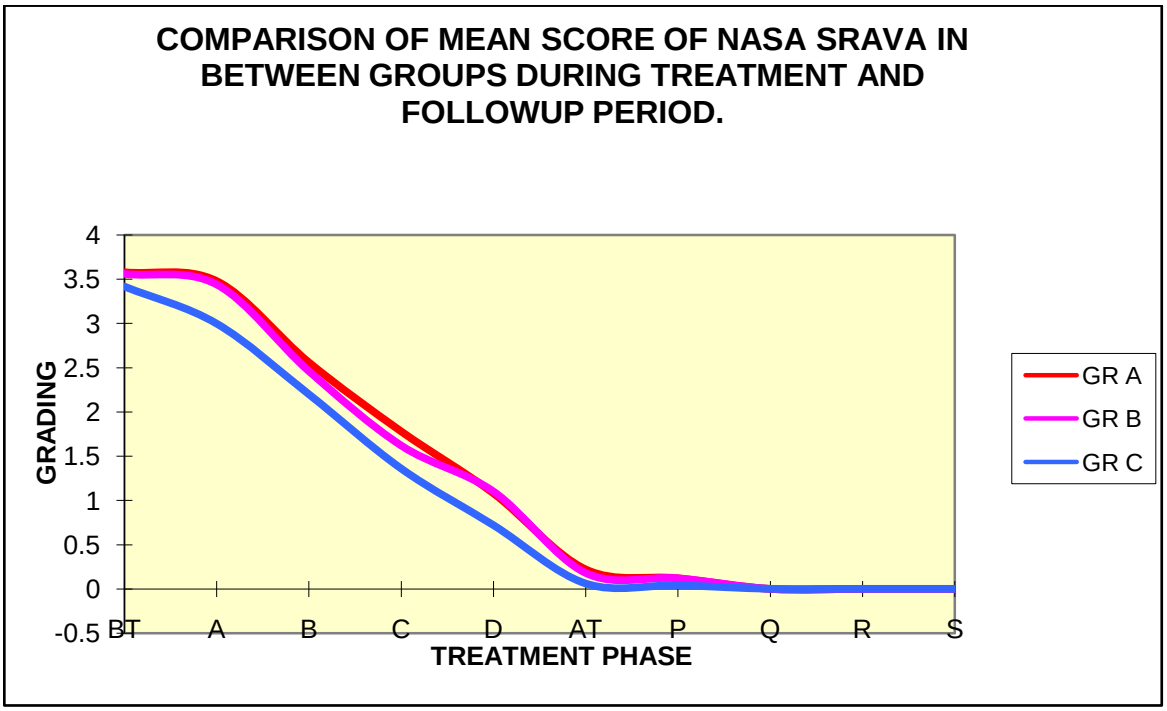


Table no. 114 Stastitical analysis of Nasa Srava in Group A

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	3.36000	.56279	.07959	3.20006	3.51994	42.216	49	.000

Table no. 115 Stastitical analysis of Nasa Srava in Group B.

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	3.38000	.49031	.06934	3.24065	3.51935	48.745	49	.000

Table no. 116 Stastitical analy sis of Nasa Srava in Group C

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	3.36000	.56279	.07959	3.20006	3.51994	42.216	49	.000

From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is <0.05 .

Difference between groups

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other

ANOVA

Table no. 117: Stastitical analysis of severity of Nasa srava.

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.693	2	.347	2.714	.070
Within Groups	18.780	147	.128		
Total	19.473	149			

As p > 0.05 Ho cannot be rejected. Thus we can say that all the three treatments are having highly statistically significant effect in reduction of nasasrava but there is no statistically significant difference in mean of all the groups.

Looking at the values of 95 % CI also it can be said that all the three groups are almost equally effective in reducing nasasrava.

GROUP	BT	A	B	C	D	AT	P	Q	R	S

GR A	3.64	3.52	2.64	1.8	0.96	0.15	0.08	0	0	0
GR B	3.6	3.52	2.58	2.02	1.26	0.28	0	0	0	0
GR C	3.58	2.96	2.3	1.36	0.72	0.04	0	0	0	0

Table no. 118:Comparison of mean score of Nasavarodha between groups during the treatment and follow up

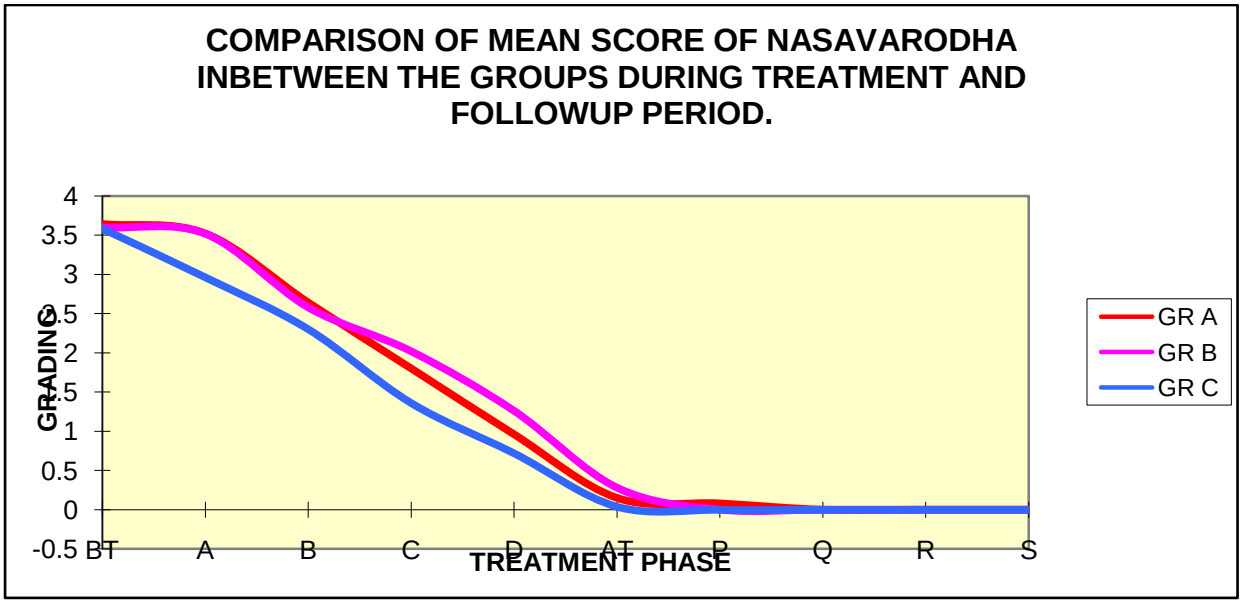


Table no. 119 Stastitical analysis of Nasa Avarodha in Group A.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.48000	.50467	.07137	3.33657	3.62343	48.759	49	.000

Table no. 120 Stastitical analysis of Nasa Avarodha in Group B.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.32000	.47121	.06664	3.18608	3.45392	49.820	49	.000

Table no. 121 Stastitical analysis of Nasa Avarodha in Group C.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	3.54000	.54248	.07672	3.38583	3.69417	46.143	49	.000

From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 .

. Difference between groups

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of atleast one group is different from that of other.

ANOVA

Table no. 122:Stastitical analysis of severity of Nasa Avarodha.

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	1.440	2	.720	5.654	.004
Within Groups	18.720	147	.127		
Total	20.160	149			

As p < 0.05 Ho can be rejected at 5 % LOS.

Thus we can say that there is statistically significant difference in readings of the three groups.

Based of 95 % confidence interval of the difference it can be said that the results of group C is superior to other, as 95 % confidence interval of the difference of group C is higher than that of group A and group B

.when compared group A is found to be superior to that of group B in treating nasaavarodha

Shiroshoola

Table no. 123:Comparison Of Mean Score Of Shirashool In Between Groups Duringtreatment And Followup Period.

GROUP	BT	A	B	C	D	AT	P	Q	R	S
GR A	2.72	2.64	1.76	1.34	0.94	0.06	0.02	0	0	0
GR B	2.54	2.46	1.76	1.28	0.96	0.3	0	0	0	0
GR C	2.66	2.5	2.14	1.32	0.68	0.02	0	0	0	0

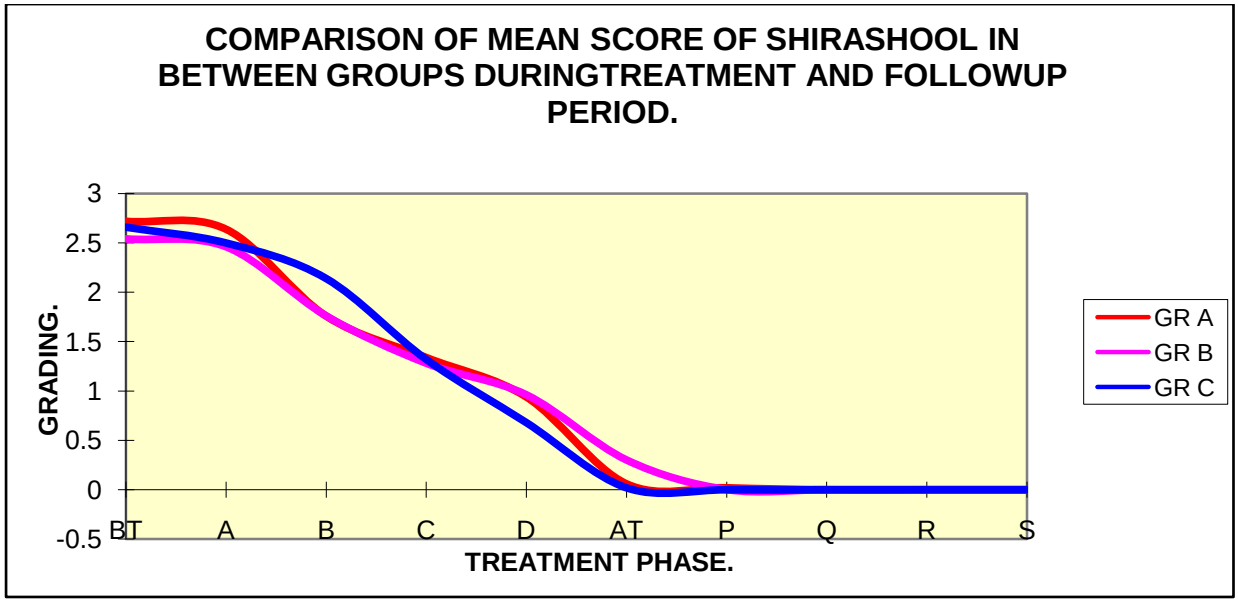


Table no. 124: Stastitical analysis of Shirashoola in Group A.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	2.66000	.59281	.08384	2.49152	2.82848	31.728	49	.000

Table no. 125:Stastitical analysis of Shirashoola in Group B.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	2.24000	.55549	.07856	2.08213	2.39787	28.514	49	.000

Table no. 126: Stastitical analysis of Nasa Avarodha in Group C.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	2.64000	.59796	.08456	2.47006	2.80994	31.219	49	.000

From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 .

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other

ANOVA

Table no. 127: Stastitical analysis of severity of Shirashoola.

SEVERITY	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	2.293	2	1.147	11.787	.000
Within Groups	14.300	147	.097		
Total	16.593	149			

AS P < 0.05 we can reject Ho in this case at 5 % LOS.

Thus we can say that there is a statistically significant difference between the readings of three groups.

Based of 95 % confidence interval of the difference it can be said that the results of group A is superior to other, as 95 % confidence interval of the difference of group A is higher than that of group B and group C.

. When compared group A is found to be superior to that of group B and C in treating shiroshoola

SHIRO GAURAVA

Table No. 128: Comparison Of Mean Score Of Shirogouravata Inbetween The Groups During Treatment And Followup Period.

GROUP	BT	A	B	C	D	AT	P	Q	R	S
GR A	2	2.4	1.52	0.98	0.66	0.02	0	0	0	0
GR B	2.48	2.42	1.6	1.16	1.08	0.28	0	0	0	0
GR C	2.58	2.38	1.9	1.24	0.68	0.02	0	0	0	0

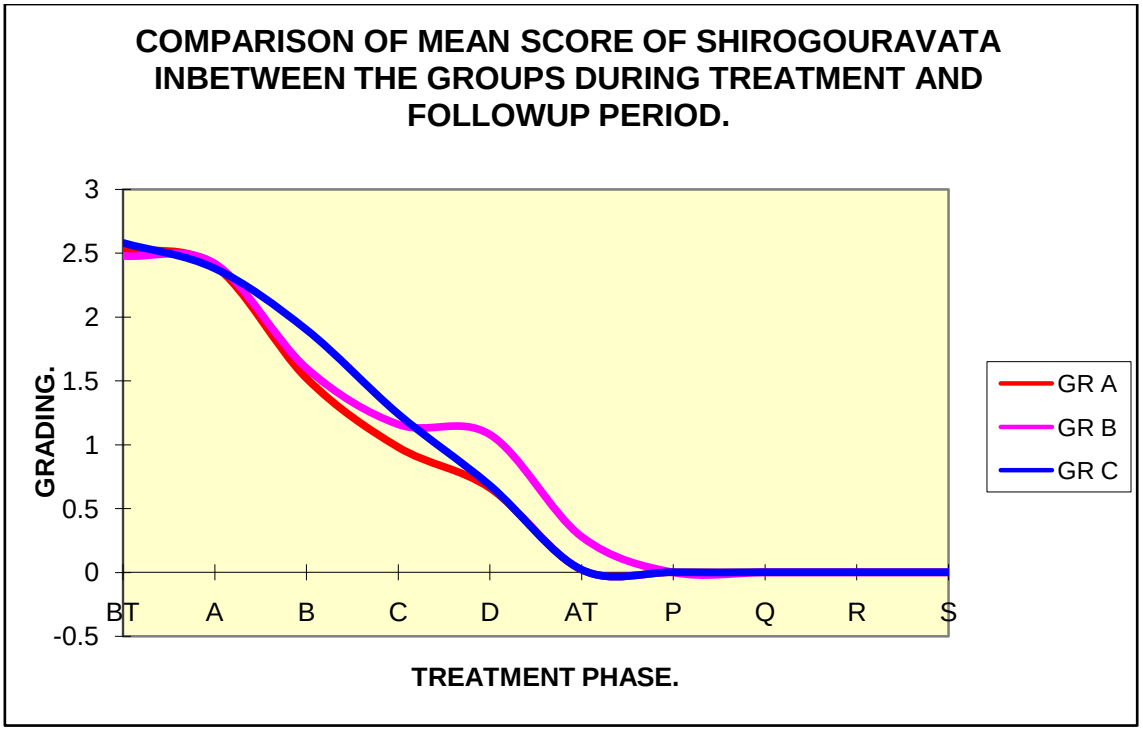


Table no. 129: Stastitical analysis of Shiro Gauravata in group A.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	2.52000	.61412	.08685	2.34547	2.69453	29.016	49	.000

Table no. 13 Stastitical analysis of Shiro Gauravata in group B.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	2.20000	.63888	.09035	2.01843	2.38157	24.350	49	.000

Table no. 131 Stastitical analysis of Shiro Gauravata in group C.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	2.56000	.61146	.08647	2.38623	2.73377	29.605	49	.000

From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 .

Difference between groups

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other

ANOVA

Table no. 132 Stastitical analysis of severity of Shirashoola.

SEVERITY	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	2.253	2	1.127	13.756	.000
Within Groups	12.040	147	.082		
Total	14.293	149			

AS P < 0.05 we can reject Ho in this case at 5 % LOS.

Thus we can say that there is a statistically significant difference between the readings of three groups.

Based of 95 % confidence interval of the difference it can be said that the results of group C is superior to other, as 95 % confidence interval of the difference of group C is higher than that of group A and group B

When compared group A is found to be superior to that of group B in treating shirogaurava.

EIOSINOPHIL

Table no. 133: COMPARISON OFMEAN SCORE OF EIOSINOPHIL COUNT IN BETWEEN GROUPS BEFORE AND AFTER TREATMENT.

GROUP	BT	AT
GR A	8.1	4.18
GR B	8.38	4.28
GR C	8.02	3.58

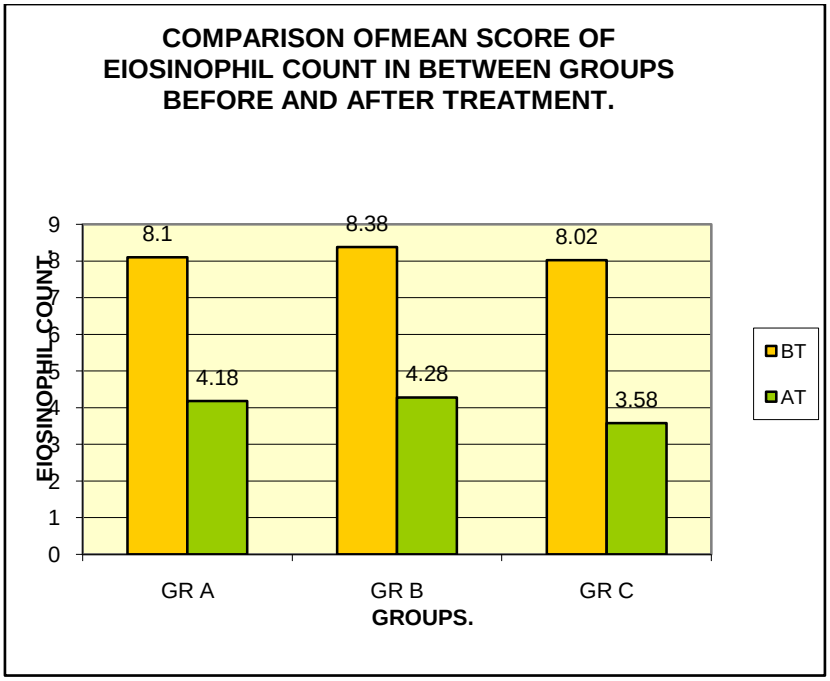


Table no. 134:Stastitical analysis of Eiosinophil counts in group A.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	BT - AT	3.92000	1.61422	.22829	3.46124	4.37876	17.171	49	.000

Table no. 135 Stastitical analysis of Eiosinophil counts in group B.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	BT - AT	4.10000	1.71726	.24286	3.61196	4.58804	16.882	49	.000

Table no. 136: Stastitical analysis of Eiosinophil counts in group C.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	BT - AT	4.44000	1.94999	.27577	3.88582	4.99418	16.100	49	.000

READINGS

The above readings suggest that the difference between pre and post treatment readings is highly significant in all the three groups.

Difference between groups

Ho –there is no difference in means of readings in group A,group B and group C.

H1- the mean of readings of at least one group is different from that of other .

ANOVA

Table no. 137 Stastitical analysis of severity of Eiosinophilia.

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	14.333	2	7.167	10.790	.000
Within Groups	97.640	147	.664		
Total	111.973	149			

As p value is < 0.05, Ho can be rejected at 5 % LOS.

Thus we can say that the there is statistically significant difference in readings of the groups.

Based on the values of 95 % confidence interval we can say that

Treatment in group C is superior than group B and group A, whereas group B is superior than group A in treating eiosinophil

ABSOLUTE EIOSINOPHIL (statistical analysis done using SPSS15.00)

Table No. 138: Comparison Of Absoluteeiosinophil Count Inbetween Groups Before And After Treatment.

GROUP	BT	AT
GR A	821.98	423.12
GR B	871.82	423.4
GR C	857.36	353.98

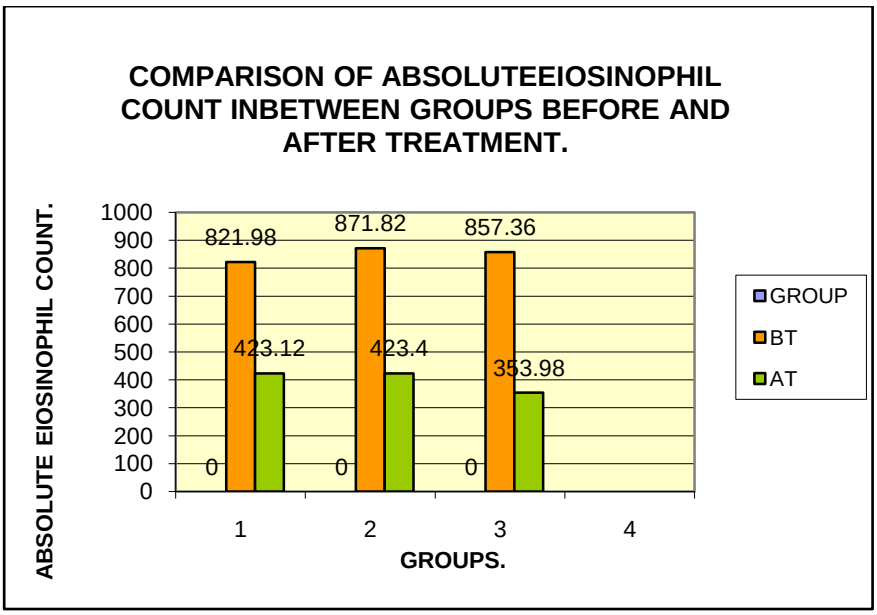


Table no. 139: Stastitical analysis of Absolute Eiosinophil count in group A.

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	398.88000	177.49891	25.10214	348.43537	449.32463	15.890	49	.000

Table no. 140:Stastitical analysis of Absolute Eiosinophil count in group B.

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	448.42000	149.23972	21.10568	406.00654	490.83346	21.246	49	.000

Table no. 141: Stastitical analysis of Absolute Eiosinophil count in group C.

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	503.38000	194.71936	27.53748	448.04137	558.71863	18.280	49	.000

READINGS

The above readings suggests that the difference between pre and post treatment readings are highly significant in all the three groups.

<u>Difference between groups</u>

Ho –there is no difference in means of readings in group A, group B and group C.

H1- the mean of readings of at least one group is different from that of other

ANOVA

Table no. 142: Stastitical analysis of severity of Absolute Eiosinophilia

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	159946.680	2	79973.340	16.511	.000
Within Groups	712011.480	147	4843.616		

Total	871958.160	149			

As p value is < 0.05, Ho can be rejected at 5 % LOS.

Thus we can say that the there is statistically significant difference in readings of the groups.

Based on the values of 95 % confidence interval it can be stated that group C is superior to that of the other two groups in reducing AEC where as group B is found to be superior to group A

SER- IgE

Table no. 143: Comparison Of Ser- Ige Level Inbetween The Groups Before And After Treatment

GROUP	BT	AT
GR A	790.92	128.8
GR B	812.58	151.8
GR C	809.12	76.18

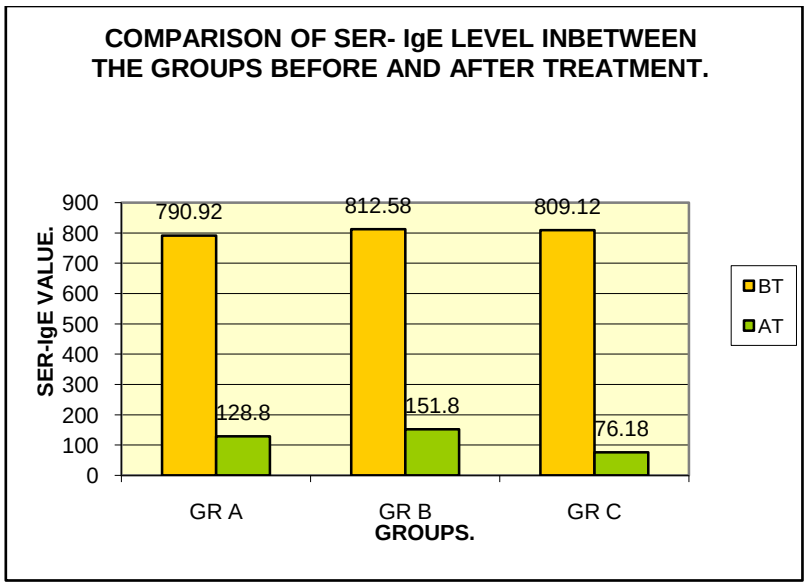


Table no. 144: Stastitical analysis of SER-IgE level in group A.

Paired Samples Test

	Paired Differences					t	df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1 VAR00001 - VAR00002	662.04000	387.70644	54.82997	551.85505	772.22495	12.074	49	.000

Table no. 145: Stastitical analysis of SER-IgE level in group B.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	660.70000	321.18030	45.42175	569.42157	751.97843	14.546	49	.000

Table no. 146: Stastitical analysis of SER-IgE level in group C.

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VAR00001 - VAR00002	732.94000	367.38071	51.95548	628.53156	837.34844	14.107	49	.000

READINGS

The above readings suggest that the difference between pre and post treatment readings is highly significant in all the three groups.

<u>Difference between groups</u>

Ho –there is no difference in means of readings in group A,group B and group C.

H1- the mean of readings of atleast one group is different from that of other.

ANOVA

Table no. 147: Stastitical analysis of severity of SER-IgE level.

	Sum of Squares	Df	Mean Square	F	Sig.
etween Groups	150080.119	2	75040.059	48.699	.000
Within Groups	224968.660	146	1540.881		
Total	375048.779	148			

As p value is <0.05 ,Ho can be rejected at 5 % LOS.

Thus we can say that the there is statistically significant difference in readings of the groups

Group C is found to be superior to other two groups.

Table no. 148

Table showing difference between pre and post treatment readings of Gr A, B, & C

GR A, B, C.	Highly significant	Significant	Not significant
KASA	✓		
NASASRAVA	✓		
NASAVARODHA	✓		
KSHAVATHU	✓		
SHIRASHOOLA	✓		
SHIRO GOURAVATA	✓		

Table no. 149

Table showing difference between pre and post treatment readings of Gr A, B, & C

	Highly significant	Significant	Not significant
Esinophil	✓		
Absoluteesinophil count	✓		
Ser total IgE	✓		

Table no. 150

TABLE SHOWING CAMPARISON OF STATISTICAL SIGNIFICANCE OF LABORATORY VALUES IN BETWEEN Gr A, B, & C.

Esinophil	Treatment in-group C is superior than group B and group A,	Group B is superior than group A in treating eiosinophil
Absoluteesinophil count	Group C is superior to that of the other two groups in reducing AEC	Group B is found to be superior to group A.
Ser total IgE	Group C is found to be superior to other two groups.	Both are almost same.

Table no. 151

TABLE SHOWING CAMPARISON OF STATISTICAL SIGNIFICANCE OF CLINICAL FINDINGS IN BETWEEN Gr A, B, & C.

GR A, B, C.		
KASA	**As the p value is 0.074 > 0.05, All the three groups are having statistically significant effect in reducing kasa, But there is no statistically significant difference between the groups. From which we can say that all the three groups are having similar effect in reducing kasa.**	
NASASRAVA	**As p > 0.05 Thus we can say that all the three treatments are having highly statistically significant effect in reduction of nasastrava But there is no statistically significant difference in mean of all the groups. Looking at the values of 95 % CI also it can be said that all the three groups are almost equally effective in reducing nasastrava.**	
NASAVARODHA	As p < 0.05 Group C is superior to other,	Group A is found to be superior to that of group B.
KSHAVATHU	As p < 0.05 Group C is superior to others,	Group B is found to be superior to that of group A.
SHIRASHOOLA	AS P < 0.05 Group A is superior to other,	Group C is found to be superior to that of group B.
SHIRO GOURAVATA	AS P < 0.05 C is superior to other	Group A is found to be superior to that of group B

Table no. 152: Results

	GR-A	GR-B	GR-C	TOTAL
CURE	40 (80%)	37 (74%)	47 (94%)	124 (82.66%)
SIGNIFICANT IMPROOVEMENT	10 (20%)	13 (26%)	03 (06%)	26 (17,34%)
IMPROOVEMENT.	-	-	-	-
INSIGNIFICANTLY IMPROOVEMEN	-	-	-	-
NO IMPROOVEMENT	-	-	-	-

06 DISCUSSION

06.1 DISCUSSION ON CONCEPTUAL STUDY

The **disease** asatmyajanya pranavahasroto vikara is elaborated in the disease review chapter with respect to its etiology, pathogenesis, clinical features, prognosis and line of management. The allopathic counterpart – pulmonary hypersensitivity is elucidated on similar lines to comprehend the disease complete with latest available authentic reports in order to secure a better understanding of the subject.

06.2 DISCUSSION ON GENERAL OBSERVATIONS

The general observation of 150 patients were as follows -

Total 156 patients were registered in the present study, in which there were 06 dropouts and with remaining 150 patients the clinical trial was completed. Patients selected for the clinical trial were further divided equally into three groups A, B, C (50 in each).

Age –

From the table it is observed that maximum no. of patients i.e. 38% were from the age group of 26-35 years, followed by age group of 36-45 years and 16-25 years, which constituted up to 30 % of the total strength and 20.66% respectively. This is the age group when individual is active & enjoying their life in their own ways. In Ayurveda 'Shira' is

indicated as Marma / uttamang & is advised to protect it. But today people are much conscious about their outlooks, so they are not ready to cover their heads & ears. Similarly intake of cold drinks & ice-cold water is also becoming a fashion. Hence the incidence of above said age group is more susceptible for pulmonary hypersensitivities. (Table 42)

Sex –

Male predominance (57.3%) was evident from the table. This is because males have maximum exposure to dusts & smoke at outdoors. Dust is one of the main allergen responsible for this disease. It can be said that various habits, occupations are responsible for the male patients. Female are lesser affected from these types of precipitating factors. (Table 41)

Religion –

Almost all the patients (90.66%) were Hindu. Majority of the residents of this region belongs to this religion; again the maximum no. to patients visiting the O.P.D. of our hospital are Hindus, Hence the above finding. (Table 43)

Occupation –

The occupation of the patients in the study shows that factory workers (17.33%) and students (15.33%). were in maximum number. Poor hygiene, polluted weather, dust, their fast and changing life style, have an important role on students. This adolescent period is also one, which marks the change in a person from childhood to adulthood. This is the period wherein the hormones are unstable and subtle changes start occurring in the body. Here as a result the immunity also is challenged and the person is exposed to infections and allergies easily. Next in majority are painter 14.66%and the housewives 12.66%.It is because they are exposed to paint an arsenic derivative chemical solution and dust, housewives while cleaning the house and are exposed to smoke in kitchen. They also have erratic eating habits and show more of a tendency to do Vega Vidharana leading to diminished immunity against diseases. (Table 45)

Socio-Economic status –

Maximum no. of patients (45.33%) were from the middle class. Among these people Vegadharana, Atikrodha, indulgence of Vishamashana & Viruddharhara, Ajeerna were common because they were working people. (Table 44)

Habitat –

Maximum no. of patients 60% of total sample size was from urban area and most of them were actually living very close to the dust area. So people residing here have maximum exposure to its dust climate. Again pollution in the cities is much more as compared to the rural settings. Hence maximum patients were suffering from this disease. (Table 46)

Aggravating factor –

Dust was maximum (100%) observed as an aggravating factor. Pollution and exposure to pollens (50%) and perfume 63.33% were another important factor. (Table 9) Climatic change was maximum (92.75%) observed as an aggravating factor. With the change of climate, there is change in % of relative humidity & temperature which is directly related with allergy. They effect in 2 ways –

1) One directly responsible for causing allergic rhinitis humidity <30 can cause drying of nasal mucosa.

2) Secondly they influence the population of house dust mites. (Dermatophagoides farinae & D. pieronyssinus) Researches published on "Seasonal Periodicity of House Dust Mite Population"(in the book "Aspects of Allergy & Applied Immunology"),shows that maximum positivity of house dust was found in the months of August & September & minimum in the months of march, April & May, indicating that the mite population in house dust show a periodic increase & decrease with the change of season. It appears that more rapid growth of these organisms occurs during a period when the temperature is moderate & the relative humidity is high. And a slow growth or no growths when it is relatively dry due to either too hot or too cold. 'Smoke' specially 'bidi smoke' has deleterious effect on humoral antibody production as well as on lymphocyte mediated immunity.

Family history –

It was observed that family history was positive in 86.0% of patients. Since factors affecting IgE production are partly genetic & partly because of their similar food habits & life style. (Table 54).

Food habits –

58.66 % of patients were Niramisha, as most of the patients registered were Hindus. (Table 48)

Koshtha –

Madhyama Koshtha was observed in maximum no. of patients (34.66%) which has also direct relation with Kaphaja Prakriti. (Table 50)

Habit –

95.33% of patients had habit of tea and coffee, 31.33% had habit of smoking and 12.66% tobacco chewing each. These types of addictions are main precipitating factors of local tissue as well as general body system, therefore the addiction have importance in the manifestation of respiratory allergies. (Table 47)

Sharirika Prakriti – Maximum no. of patients i.e. 48.66% had Vata-Kapha Prakriti. This observation clearly indicates the Vata-Kapha dominance in natural body makes patients more susceptible to the diseases occurring due to these doshas predominance. (Table 51)

Sara –

76.66% of patients were of Madhyam Sara and 12.66% had avara sara. 10.66% of the patients had Pravara Sara. This gave an indirect idea of the general body constitution, strength & immunity of the different Dhatus. (Table 53)

Satva –

Madhyama Satva was found in 70% of the patients followed by 12.66% Avara Satva. (Table 52)

Desha –

Maximum no. of patients i.e. 60% were from Anoopa Pradesha. Prevalence Respiratory Allergies maybe maximum in such place. 25% patients had this disease after they came to Anoopa pradesha from Jangal and Sadharana Pradesh. (Table 55)

Aharaja Nidana –

Abhishyandi ahara(dadhi) sevana and sheeta gunatmaka(ice cream & cool drink , sheeta jala sevana was reported in maximum no. of patients i.e. 100% followed by anupa mamsa sevana & ati madhura ahara, auduka mamsa sevana in (67.33%) & (54.66%) % (58.66%) of patients respectively. This indicates that Vishamashana, Ati drava sevana,atisheetaahara & Ati madhura ahara were most important Aharaja nidana of Respiratory Allergies reported by the patients of this study. (Table 47)

Viharaja Nidana – Sheeta Vayu Sparshanasevana was reported in maximum no. of patients i.e. 100% followed by Raja sevana, Dhuma sevana (agarbatti 89.33%, mosquito coil 82.66% and cigarette/ beedi 31.33 %.) & Staying in sheeta sthana (a/c etc) in (34%) of patients

respectively. This indicates that Raja sevana, Vega sandharana, Dhuma sevana & Ritu Vaishamya were most important Viharaja nidana of Respiratory Allergies reported by the patients of this study. (Table 47)

Incidence of allergy Out of total study population (150), all had sensitivity towards dust (100%), 63.33% had perfume allergy, 50% had pollen allergy,32% had house mite allergy, 30% had allergy of chemical fumes, 16.7% had allergy of pet animals and 14% had allergy of cotton dust. Thus it can presume that dust, perfumes and pollen are dominant allergens due to present globalization and day today life style.

Onset - Maximum no. of patients (97.10%) had gradual onset. The disease started with complaint of common cold, which is due to exposure to allergy but due to recurrent exposure to allergens. Only on first exposure symptoms of pulmonary hypersensitivity cannot be obtained.

Course – Maximum no. of patients (63.76%) had progressive course. Due to continuous exposure to allergens, & causative factor disease becomes progressive. Though patients are avoiding cold intake but they are unaware of Viruddhahara & Vishamashana, etc dietary habits. Secondly they are not aware of relative humidity, amount of dust particles in particular area, etc. All these factors lead to a progressive course of disease.

Aggravating factor –

Ajeerna Vishamashana, & sleep after dugdhapana are already indicated in the causative factor of Pratishyaya. This conditions leads to 'Amotpatti' which is root cause of all diseases.

In pulmonary hypersensitivity there is a release of Histamine, which acts on 3 types of receptors, out of which one is H2. Activation of H2 receptors increases the gastric acid secretion in humans (Satoskar). Dry coughing may be due to recurrent exposure to allergens since child childhood causing reactions on H1 receptors leading to muscle contraction. Hence the chances of recurrence of allergic rhinitis are more. Factors affecting IgE production are partly genetic & partly environmental; the later include exposure to house dust mite & passive exposure to parental smoking during childhood. Family history – It was observed that family history was positive in 86% of patients. Since factors affecting IgE production are partly genetic & partly because of their similar food habits & life style.

Nasa srava-

32% of the patients were presenting with 08-10 years of disease history. Followed by 23.33% and 15.33% were of 04-06 years and 02-04 years disease history respectively. The

onset was gradual in 68.66% of patients, progressive in course (40.66%) and severe in nature 63.33% of patients. It was observed more in early morning (96.66%) and immediately after kshavatu in all 150 patients (100%).The srava was watery in consistency in 96.66% of patients and was nirgandhayukta in all 150 patients. (Table 56-62).

Nasavrodha-

28% of the patients were presenting with 08-10 years of disease history. Followed by 15.33% and 21.33% were of 04-06 years and 02-04 years disease history respectively. The onset was gradual in 62% of patients, progressive in course (40.66%) and severe in nature in 63.33% of patients. It was observed more in early morning (96.66%) and mid night in 82.66% patients. (Table 63-67)

Kshavatu-

34% of the patients were presenting with 04-06years of disease history. Followed by 30% and 18% were of 08-10 years and 06-08 years disease history respectively. The onset was gradual in 56.66% of patients, progressive in course (60.66%) and severe in nature 62.66% of total patients. 50% patients had >10 sneezing at a time, 48%patients had >30 sneezing in entire day, 78.12% had sneezing in morning. 75% had sneezing in cool atmosphere while 50% of patients had sneezing in moist atmosphere. (Table 68-75)

Kasa-

Maximum number of patients i.e. 32 % were having kasa for last 04-06 followed by 30% patients were having kasa for 08-10. 67.33%patients were having gradual onset history. In 51.33%patients kasa was progressive and was dry (84 %) in nature. (Table 81-86)

Swasakrichtrata-

Swasakrichtrata was observed in 92 patients (61.33), where in 15% of the patient had 6 to 8yrs chronicity history and was gradual in nature in 31.33%of patients , progressive in course (25.33%), mild in nature (54.66%), and was dominated in early morning(28.66%) and mid night(25.33%). 73.33% of study populations were presenting with Raktavarnata of netra and 77.33% people had Netrasrava. This shows netragatalakshana are quite evident in Chronic Pulmonary Hypertensive conditions. Karnabhadiryata (44.66%) was observed in the present clinical trial .Shirashula and Shirogauravata was present in all study population

(100%). Gandhagnana was preserved in 72% of patients where in 24% of patient had partial Gandhagnana. (Table 87-91)

Examination of nasal mucosa –

Maximum no. of patients i.e. 87.5% had pale nasal mucosa, which is the cardinal sign of allergic rhinitis.

Involvement of Paranasal Sinus –

Frontal & maxillary sinusitis was observed in 40.62% & 31.25% of patients respectively.

Chief complaints –

Kshavatu, nasakandu, kasa, shirashoola and shirogouravata were observed in all the patients, nasavarodha in 99.33% of patients & nasasrava in 93.33% of patients. So it can be concluded that kshavatu, nasakandu, kasa, shirashoolaandshirogouravata, nasavarodha and nasasrava are chief complaints of asaymyajanya pranavahasrotovikara.

Associated complaints –

Shwasakrichrata in 64.7%, raktavarna netra in 73.3%, netra kandu in 16%, netrasrava in 77.3%, karanavedana in 4.7%, karnabadhiryata in 44.7%, feeling of fullness of ears in 64.7%, partial loss of gandhagnana in 24%, and urahavedana in 28% are associated complaints, which were observed in study population.

6.3 DISCUSSION ON RESULTS

Effect of therapies on Cardinal symptoms –

Nasasrava From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 Thus we can say that all the three treatments are having highly statistically significant effect in reduction of nasasrava but there is no statistically significant difference in mean of all the groups. Looking at the values of 95 % CI also it can be said that all the three groups are almost equally effective in reducing nasasrava.

Kshavathu From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 . As p < 0.05 we can reject Ho at 5 % Los. Thuds it can be said that there is a statistically significant difference between the readings of the three groups. Based of 95 % confidence interval of the difference it can be

said that the results of group C is superior to others, as 95 % confidence interval of the difference of group C is higher than that of group A and group B .when compared group B is found to be superior to that of group A.

Nasavarodha From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 . Based of 95 % confidence interval of the difference it can be said that the results of group C is superior to other, as 95 % confidence interval of the difference of group C is higher than that of group A and group B and amongst group A and B, group A is found to be superior to that of group B in treating nasaavarodha

Shiro shoola From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05. Based of 95 % confidence interval of the difference it can be said that the results of group A is superior to other, as 95 % confidence interval of the difference of group A is higher than that of group B and group C and amongst group A and B, group A is found to be superior to that of group B in treating shiro shoola.

Shiro Gaurava From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05. Based of 95 % confidence interval of the difference it can be said that the results of group C is superior to other, as 95 % confidence interval of the difference of group C is higher than that of group A and group B and amongst group A and B, group A is found to be superior to that of group B in treating shiro gaurava.

Kasa From the above it can be said that the difference between the pre and post scores is highly significant in all the three groups as p is < 0.05 As the p value is 0.074 > 0.05 Ho cannot be rejected. Thus we can say that all the three groups are having statistically significant effect in reducing kasa, but there is no statistically significant difference between the groups. From which we can say that all the three groups are having similar effect in reducing kasa.

EIOSINOPHIL The above readings suggest that the difference between pre and post treatment readings is highly significant in all the three groups. As p value is < 0.05, Ho can be

rejected at 5 % LOS. Thus we can say that the there is statistically significant difference in readings of the groups. Based on the values of 95 % confidence interval we can say that Treatment in group C is superior to group B and group A, whereas group B is superior than group A in treating eiosinophil

ABSOLUTE EIOSINOPHIL The above readings suggests that the difference between pre and post treatment readings are highly significant in all the three groups . As p value is < 0.05, Ho can be rejected at 5 % LOS. Thus we can say that the there is statistically significant difference in readings of the groups. Based on the values of 95 % confidence interval it can be stated that group C is superior to that of the other two groups in reducing AEC where as group B is found to be superior to group A.

SER- IgE The above readings suggest that the difference between pre and post treatment readings is highly significant in all the three groups. As p value is < 0.05, Ho can be rejected at 5 % LOS. Thus we can say that the there is statistically significant difference in readings of the groups Group C is found to be superior to other two groups.

Total effect of Therapy –

- ✓ Significant Improvement was found in 47 patients (94%)-group C then in 40 patients (84%) -group A then in 37 patients (74%) in group B.

- ✓ Improvement was observed in 13 patients (26%) group B then in10 patients (20%) in group A and 3 patients in group C (06%).
- ✓ Effect of therapy was statistically highly significant at the level of $p<0.001$ in all cardinal symptoms in all the group (Group A,B&C).
- ✓ Effect of therapy was statistically highly significant at the level of $p<0.001$ in Eiosinphil, Absolute eiosinphil and ser-IgE levels of the patients

- ✓ In follow up study there was not a single incidence of recurrence in patients of any group after treatment. (Table 57)

- ✓ Overall significant improvement was observed in 124 patients 82.66% in total and improvement was observed in 26 patients 17.34%.

When we consider all the above observations it is evident that combined therapy of shirisharishta and Bhriguharitaki rasayana had better results over when used separately.

06.4 DISCUSSION ON PROBABLE MODE OF ACTION

The probable mode of action of drug can be discussed from the results obtained

Pulmonary hypersensitivity involves sensitization of genetically pre-disposed persons to allergens, which interact with dendritic cells and Th-0 helper lymphocytes leading to generation of a clone of Th2 helper cells. The consequence is generation of different types of B-cell activating cytokines leading to IgE production and release. Additionally there is also generation and release of interleukin 5 (IL-5) which is responsible for differentiation and activation of eosinophils; IL-4 induces expression of IgE receptor on mast cells and it also induces endothelium to express eosinophil attracting receptors. After the above basic events subsequent exposure to allergen leads to pulmonary hypersensitivity attack. It has two phases- initial response leading to bronchospasm. In this release of chemical mediators from masts cells play major role. There is a late response which occurs at a variable time after exposure to the allergen. This phase involves progressive inflammation as a sequel to the initial phase. In this reaction there is involvement of the normal inflammatory cells, activated cytokine releasing Th-2 cells and eosinophils causing progressive loss of air way epithelium. In this reaction many factors are involved including nitric oxide and neuropeptides. At present the therapy is directed towards providing relief from the broncho constriction and controlling the inflammation. In the present study attempt was made to assess whether the test drugs posses, immunomodulation property since it is the basic cause of all the pathophysiological changes observed in the disease, the drugs were also evaluated for anti-histamine effect, for effect on bronchial smooth musculature and anti-tussive activity.

06.4.1 Probable mode of action of Shirisharishta:

The disease asatmyajanya pranavaha srotovikara (pulmonary hypersensitivity) characterized by the involvement of *Vata* and *Kapha Dosha* and *Pranavaha, Rasavaha* and *Annavaha Srotas*. The pathology is marked by narrowing, leading to spasm caused by *Vata* by all probabilities and secretions obstructing the channel caused by deranged *Kapha*. Of

course, the vitiation of the *Pitta Sthana* has been described to be the root cause. This indicates towards the derangement of certain enzymatic or hormonal activities which result into further changes, leading to inflamed bronchial mucosa in turn leading to spasm by *Vata* or obstructing secretions by *Kapha*. Hence, logically, the drug administered for the treatment of asatmyajanya pranavaha srotovikara (pulmonary hypersensitivity), should be able to overcome *Vata* and *Kapha* for immediate and symptomatic relief but should also pacify the *Pitta* for a permanent or quasi permanent relief. Keeping this in mind, the review of literature was done to find out a suitable *Kalpana* that would pacify *Vata* as well as *Kapha* but would also mollify *Pitta*. Moreover quick action, patient acceptance with respect to palatability and long shelf life were sought. The *Kalpana* fulfilling the above requirements was found to be *Asava-Arishta Kalpana* processing *Kapha-Vataghna* and *Pitta avirodhi* (S. Su. 45/114-115). More over, *Arishta Kalpana* is one such *Kalpana* can either eliminate the excess *Dosha* or palliate *Dosha* besides improving the *Agni*. Shirisha one amongst the popular compounds amongst the traditional Ayurvedic practitioners was pondered upon. Moreover, it is advocated to be the best *Vishaghna Dravya* and additionally recommended for the use in the disease *Kasa*. Thus, formulating severe *Ama*, as *Ama Visha*, a type of *Visha*, both *Ama* and *Visha* being the causative factors for the disease *Shwasa, Shirisharishta* was administered. *Shirisha* has *Madhura, Tikta, Kashaya Rasa, Anushna Virya* and *Katu Vipaka. Tikta Kashaya Rasa* and *Katu Vipaka* palliates *Prakupita Kapha Dosha, Madhura Rasa* palliates *Prakupita Vata Dosha* and *Madhura Tikta Rasa* palliates *Prakupita Pitta Dosha.* It's potential as best *Vishaghna* helps eliminate/ alleviate the chronic (*lina*) *Ama Visha*, thus contributing to the *Samprapti Vighatana* at multiple levels. The Pitta Avirodhi - Kapha Vataghna property of the Kalpana seems to have contributed to the efficacy of the compound drug Shirisharishta.

Shirisha the main constituent of the formulation is classified as a Vishaghna and Shirovirechaka. Various references are available for the therapeutic use almost all the parts of the drug Shirisha in the major classics like Charaka and Sushruta etc, but latter in present era most of the rsearch works regarding Shirisha gathered arround the bark, seed and leaves only. Various research works have been carried out, where Shirisha bark is reported to have antisthmatic, antiallergic, analgesic, antianaphylactic and bronchodilator actions. The bark is used as one of the ingredient of an Ayurvedic Kadha or decoction used for

treating asthma i.e. Shirisha the Kwath Pharmacologically it was found to show antitussive action and the ability to prevent allergy-induced bronchospasm. Saponin fraction of plant significantly reduced the number of ruptured mast cells in both mesnteric bits and peritoneal fluid obtained from sensitized rats and this effect was identified in both types of systemic anaphylaxis a (active and passive). Various types of saponin and catechins are the main reported constituents in almost all the parts of the drug which have the above reported pharmacological properties. Pippali, Priyangu, Kushtha, Ela, Nilini etc are therapeutic proven flora used for various diseases of allergic and infectious diseases especially related to respiratory disorders. Thus latest research reporting regarding each of the source material contributing in the manufacture of *Shirisharishta* is reviewed with pharmaceutical, pharmacological and therapeutic aspects.

Discussion on Pharmacologic study:

As already mentioned in the introduction *Shirisha* is considered as one of the most useful drugs for the treatment of respiratory disorders in Ayurvedic therapeutic armamentarium. Selection of appropriate drug for treatment is the main objective of any well-meaning physician. However there are many factors, which influence the therapeutic efficacy, like for example site, season and time of collection, processing and manufacturing of the intended preparation. Many a times drug preparations fail to produce expected therapeutic efficacy though they may contain well-known ingredients. In such situation the reason may lie in the above factors. This area remains ignored by mainstream researchers in the field. Taking these factors into consideration a series of studies have been initiated in our Institute. The present study focuses on *Shirisha* considering its therapeutic renown

Respiratory allergy involves sensitization of genetically pre-disposed persons to allergens, which interact with dendritic cells and Th-0 helper lymphocytes leading to generation of a clone of Th2 helper cells. The consequence is generation of different types of B-cell activating cytokines leading to IgE production and release. Additionally there is also generation and release of interleukin 5 (IL5) which is responsible for differentiation and activation of eosinophils; IL-4 induces expression of IgE receptor on mast cells and it also induces endothelium to express eosinophil attracting receptors. After the above basic events subsequent exposure to allergen leads to asthmatic attack. It has two phases- initial response leading to bronchospasm. In this release of chemical mediators from masts cells

play major role. There is a late response which occurs at a variable time after exposure to the allergen. This phase involves progressive inflammation as a sequel to the initial phase. In this reaction there is involvement of the normal inflammatory cells, activated cytokine releasing Th-2 cells and eosinophils causing progressive loss of air way epithelium. In this reaction many factors are involved including nitric oxide and neuropeptides. At present the therapy is directed towards providing relief from the bronchoconstriction and controlling the inflammation. In the present study attempt was made to assess whether the test drugs posses immunomodulation property since it is the basic cause of all the pathophysiological changes observed in the disease, the drugs were also evaluated for anti-histamine effect, for effect on bronchial smooth musculature and anti-tussive activity.

One of the experimental studies conducted at postgraduate institute, GAU, Jamnagar reveals following points on pharmacological action of Shirisharishta.[393]

Effect on antibody formation

A potent immunosuppressant action is expected of a drug combating asthma. Heam agglutinating antibody (HA) titer is a primary method for studying the humoral response. 7 days after the drug schedule, the test samples failed to influence antibody formation against SRBC in a significant manner. The rats were re-sensitized on 7th day, drug administration was continued up to 17 days and once again subjected to heamagglutinating antibody titer. The data suggests an apparent decrease in antibody titer in the drug treated groups in comparison to control group. Statistical highly significant decrease was observed in *Shirisharista*-1 administered group and statistically significant decrease was observed in *Shirisharista*-2 administered group. The results indicate that the test preparations do not modify the primary immune response but markedly suppress the secondary immune response. In this regard *Shirisharista* - 1 was the most potent followed by *Shirisharista-2,* which also produced significant suppression. *Shirisharista* -3 was however produced only a weak effect. This study corroborates presence of immunosuppressant property in Albizzia *lebbeck* earlier reported by Tripathi et al., (1979) by micro agar gel diffusion method in rats. In the primary response the induction phase is important. During this phase T- lymphocytes interact with B-lymphocytes and other cells involved in the immune mechanism in a complex manner. Initially the antigen presenting cells like macrophages process the antigen

and present it to non-differentiated T-cells colony along with histocompatability complex molecules and co-stimulatory factors. This result in the generation of two sub-sets of T-cells called as Th-1 and Th-2 cells. The former controls and modulates cell-mediated immune reaction and the latter is involved in the modulation of antibody formation. Th-2 cells produce cytokines Interleukin-4 (IL-4 and tissue growth factor-b (TGB-b) both of which stimulate B-lymphocytes to proliferate and differentiate in to anti-body producing plasma cells. Majority of the immunosuppressant drugs act by modulating different steps in the induction phase leading to reduced lymphocyte proliferation. It can be suggested that the test drug may not be interfering with the initial stage of induction but may modulate the proliferative stage of this phase. This can be brought about by 1- inhibiting the production of cytokines, 2- modulating cytokine gene expression 3- producing cytotoxicity to the ells involved in the generation and secretion of cytokines and 4- by inhibiting the formation of purine and pyramidine bases in the effector cells (Hardman, JG-2002). The exact elucidation of mechanism of action requires further detailed studies.

Effect on cell mediated immunity

Cell mediated immunity, the second arm of the acquired immunity is responsible for delayed type hypersensitivity and certain T cells suppress antibody production. The test samples were evaluated to assess their effect on cell-mediated immunity against an experimental, which is supposed to represent CMI. It involved injection of the suspension of albumin (25mg/ml) prepared with Aluminium hydroxide (25mg/ml suspension as adjuvant),) in normal saline (G.P.Talwar-1983) – to produce immunological edema. Statistically significant decrease in the paw edema volume was observed in *Shirisharista*-1 and *Shirisharista*-3 at 24 hours in comparison to control group suggesting potent CMI-suppressant activity. In both the groups suppression was evident even after 48 hours though due to variation did not reach statistically significant level. In *Shirisharishta* -2 produced marginal suppression at 24 h and moderate suppression at 48 h. This clearly shows all the three samples contain active principles with immunosuppressant activity. However the difference is in the magnitude of activity expressed. Further the result obtained in this study also corroborates the findings of Tripathi et al., 1979 and Candana Choudhary et al., (1997). Tripathi et al., 1979 who had shown that suppression of cell mediated response in mitogen PHA-induced blastogenic formation of human lymphocytes. Candana Choudhary et al.,

(1997) reported that the potent suppression of aqueous extract and butanolic extract of *Albizzia lebbeck* was comparable to that of di-sodium chromoglycate (DSCG) on most of the parameters. Interestingly in another study by Barua C.C, P.P Gupta et al., (2000) have reported immunopotentiating of *Albizzia lebbeck* effect, contrary to our findings and the findings of above mentioned workers. It is important to examine the reasons for the observation of these contradictory results. The first thing, which should be considered, is the form in which the test plant derived material is administered. For example difference can be expected if the studies are carried out with different extracts especially one study with polar and another with non-polar solvent extracted extract. Further plant extracts being complex entities they may contain both suppressant and stimulant principles whose bioavailability would depend on the physiological conditions prevailing at a given time. Depending on it the nature of activity would be expressed. Other workers have made similar observation with other plants. In the above cited study of Barua C.C, P.P Gupta et al., the authors have evaluated Butanol fraction of hot water extract in the dose of 6.25, 12.5 and 25 mg /kg po. The study shows presence of antibody enhancing effect against SRBC but has reported CMI suppression. It seems the plausible reason for the observed difference may be due to the presence of active principles in non-Butanol fraction with predominant immunosuppressant effect as evident in other studies. If we make attempt to ascertain the mechanism behind the observed cell mediated immunity suppression, the following factors have to be considered and suggestions made accordingly. As mentioned above the Th-1 T-lymphocyte pathway controls cell mediated immunity. The first step in this reaction is the antigen processing and presentation by macrophages and other related antigen presenting cells followed by differentiation of T-cells into different types including Th-1 type. Th-1 cells produce IL-2, tumor necrosis factor-b (TNF-□) and □-interferon (IFN-□). These cytokines activate macrophages enhancing their phagocytosing capacity and stimulate another sub set of T- lymphocytes known as CD8+, which mature into cytotoxic cells, which will neutralize the foreign substance responsible for the initiation of the reaction. Activation of macrophages leads to generation of large amounts of chemical mediators, reactive oxygen metabolites and neutral proteases, which are responsible for the inflammation observed during this reaction. The test drug may produce their effect through the below mentioned probable mechanisms- interference with induction stage, interfering with the activation of Th-1 cells, CD8+ cells, macrophages, inhibition of synthesis and release of cytokines,

inhibition of synthesis and release of phlogistic factors from the activated cells and finally interfering with the activity of the phlogistic mediators. However, the exact mechanism can be elucidated only after detailed studies. Thus suppressed Humoral and CMI found with the test formulations could be the basis of the its efficacy as an anti-allergic, anti-anaphylactic agent and usefulness in asthma. There might be some other mechanisms, which might be operative in the therapeutic efficacy of the preparations and would merit further investigation.

Effect on leukocytes:

The results obtained regarding the effect of test drugs on blood picture in SRBC sensitized rats have been summarized in Table-. Analysis of the data shows that total leukocyte count of the rats apparently decreased in all the three groups in comparison to the control, however, statistically significant decrease was observed only in *Shirisha*rista-1 and *Shirisharista*-2 administered groups. This shows that the test drug may have cytotoxic effect on leukocytes or they may be interfering with their formation and release from the lymphoid organs. Differential count analysis shows that all the three test drugs decrease the neutrophil count. It is a wellknown fact that neutrophils are the first line of inflammatory cells and play important role in the induction and sustenance of the inflammatory reaction. Decrease in their number and magnitude of their activity will lead to attenuation of inflammatory reaction. It is possible that the test drug produces its immunosuppressant effect through decreasing the number of neutrophils. The exact mechanism of this drug induced neutropenia needs to be elucidated. Histopathological studies did not show marked decrease in the cell population in thymus, lymph node and spleen. This indicates that the test drugs do not produce lymphocytopenia. In fact differential count has shown that there is increase in the lymphocyte percentage in test drug administered groups in comparison to control. This apparent increase may be reflective of decrease in neurtrophil count. An apparent increase in the levels of monocytes was observed in all the three test samples which reached significant level in *Shirisharista* -1 and -2 administered groups. However, statistically moderately significant increase in eosinophils levels was observed in *Shirisharista* -1 administered group and this was in comparison to the control group in which eosinophils were not at all spotted during the differential leukocyte count. A statistically significant increase in basophil level was seen in *Shirisharista* -3 administered group. These

changes seem to be due to the preferential decrease in the percentage of Neutrophil. This shows that the leukocytopenia observed with the test preparations is mainly due to the decrease in the Neutrophil count and may be important for the expression of immunosuppression.

The internal organs –Spleen, thymus and lymph nodes were subjected to scrutiny after the sacrifice of the SRBC pre-sensitized rats. An apparent increase in thymus weight was observed in all the test drug administered groups in comparison to control. However it was statistically significant only with respect to the increase observed in *Shirisharista*-1 and *Shirisharishta* -2 administered groups. An apparent increase in spleen weight was observed in all the test drug administered groups in comparison to control. However it was statistically significant only with respect to the increase observed in *Shirisharishta* -2 and *Shirisharishta* -3 administered groups when the data were presented in terms of relative values. Examination of sections of lymph node from drug administered group and their comparison with sections from control group did not reveal any significant change in the cytoarchitecture. Though increase in cellularity of thymus was observed in *Shirisharishta* -1drug administered group it does not seem to indicate immune stimulation rather it may be the tissue response to the CMI suppression observed with the test drug. The rats did not show any remarkable change in the cytoarchitecture of the spleen except for the observation of slight decrease in cellularity in sections from *Shirisharishta* -1 and -2 treated groups. The observed activity profile indicates that the observed effect may not involve lymphoid organs to significant extent.

Anti-oxidant activity:

Much of the pathology observed in air-way inflammation is attributed to the release of reactive oxygen metabolites and neutral proteases by the activated cells mainly macrophages. Hence the test drugs were evaluated for anti-oxidant activity. Evaluation of anti-oxidant activity showed moderate decrease in catalase activity with *Shirisha*rishta -1 the other two samples did not influence the activity. The glutathione content was remarkably elevated in all the three groups but the highest elevation was in *Shirisha*rishta -3 followed by *Shirisha*rishta -2 and *Shirisha*rishta -1 groups. This remarkable increase in glutathione content seems to contribute significantly to the therapeutic benefits observed

with the preparation in airway inflammation. It would be interesting to elucidate the exact mechanism of this elevation especially the activity of the enzyme involved in the key steps. Lipid peroxidation was found to be moderately elevated by *Shirisharista*-1 and the other two samples showed moderate decrease. From the overall consideration of the data generated it can be suggested that glutathione elevating effect may contribute to significantly to the therapeutic effects of the preparations especially for attenuating the air-way inflammation induced degenerative changes.

Anti-histaminic activity:

One of the samples tested for this property was found to be devoid of this effect. However, at very high dose level the tissue response decreased non-specifically. Effect on bronchial musculature: All the three test preparations were evaluated for their effect on bronchial smooth muscle contraction. None of them possess any effect *per se* and they also did not modify the responses to histamine. At higher dose level non-specific decrease in tissue response was observed. The above two tests indicate that the test drugs have no anti-histaminic and bronchodilator activities. The observed therapeutic efficacy do not depend on these properties. However it may produce tissue desensitization, whether this mechanism is operative in vivo remains to be ascertained.

Anti-tussive activity:

Cough is one of the commonest presenting symptoms in Asthma and is one among the symptom triad of asthma. Coughing may be initiated either voluntarily or reflexively. Any disorder resulting in inflammation, constriction, infiltration, or compression of airways can be associated with cough. Sulfiting agents, such as potassium metabisulfite, potassium and sodium bisulfite, sodium sulfite, and sulfur dioxide, which are widely used in the food and pharmaceutical industries as sanitizing and preserving agents, also can produce acute airway obstruction (especially in sensitive individuals) (E.R. Mc-Fadden, Jr, 2001) with cough as a presenting symptom. Hence, evaluation of anti-tussive effect of the test drugs was carried out – using a cough model in which cough is induced by sulphur di-oxide gas in Mice (Tokagi's method) (Ref: PM – 1996 – H 275-278 – Miyagoshi,M.) at 1 hour, 7 days after drug administration. Statistically highly significant reduction in the number of cough reflex was observed in all the 3 test samples. The test samples were administered consecutively for 7

days and the experiment was repeated and statistically highly significant reduction in the cough episodes was observed in all the 3 test samples. For confirmation, the experiment was repeated and statistically highly significant decrease in the cough reflex in comparison to control group was again observed. The observed results clearly indicate all the three test preparation have powerful anti-tussive activity. This activity becomes apparent on both acute single dose and chronic multiple dose administration. Cough is a protective reflex mechanism which helps in the removal of foreign material and air way secretions from both bronchi and bronchioles. However in conditions involving air-way inflammation and neoplasia of the respiratory tract it is inappropriately stimulated. In such situations it is required to administer drugs to suppress the inappropriate and troublesome cough. Clinically effective anti-tussive agents produce their effect by suppressing the cough center present in the brain stem. In asthma the cell factors released from the activated eosinophils act on the epithelium lining the airway leading to its destruction. This exposes the underlying nerve endings, which get easily irritated leading to bouts of cough. The nerve endings are excited by neuropeptides through an action on the □- opioid receptors. In the anti-tussive effect observed with the preparations the following mechanism may be operative. The drugs may be suppressing the cough center in the brain stem. The effect can also be due to immunosuppressant effect leading to decreased cytokine formation and eosinophil activation and migration especially in cough associated with asthma. In the model employed for induction of cough in the present study the cough is caused by the irritation of the air way epithelium. Thus it is likely to be due to non-immunological mechanism- probably through suppression of cough center. Based on the results obtained it can be suggested that all the three preparations are good anti-tussives, they can be employed for suppressing cough due to different causes. In asthma its anti-tussive and immunosuppressant effect would be quite beneficial. It would be pertinent to mention here that the flowers of *Albizzia lebbeck* were found to be anxiogenic and general depressant of central nervous system. These results support the use of the flowers of this plant as a sedative agent. It can be suggested that the presence of some of these principles in the stem bark might have a depressant action on the central nervous system, which pacifies the cough-reflex. This is also contributed by the expectorant action of the *Albizzia lebbeck* as reported by Rayudu & Rajadurai, Leath Sci, 1965, 12, 21; Rayudu, ibid, 1967, 14, 234.

Another study on shirishadi compound revels that,

A randomized double blind placebo control study was performed to assess the efficacy of an Ayurvedic compound “Shirishadi Compound” in Respiratory Allergic Disorders (RADs). The research drug has been found to have anti-allergic, bronchodilator, antistress, adaptogenic, anti-tussive, mucolytic, antioxidant and immunomodulatory effect [32].In a randomized double blind placebo controlled study to asses the effect of an Ayurvedic compound “Shringyadi Yog” in Respiratory Allergic Disorders (RADs) of children, the compound was found effective in alleviating and reducing the morbidity model of RADs].[394]

06.4.2 **Probable mode of action of Bhrigu haritaki rasayana –**

Drug selected for present study is *Bhrigu haritaki* in Gudavaleha form. Drugs are selected on the guidelines given by Aacharya & Research works conducted regarding these drugs in various research institutes. Drugs having Vata Kaphahara, Ushna & Vatanulomana property were selected & Compound preparation in the form of *Gudavaleha was* prepared. Aacharya indicates Avaleha Kalpana in Shamana therapy. Also disease Shvasa results due to vitiation of Prana vayu hence Avaleha Kalpana is useful to act on Prana vayu. Contents of *Bhrigu haritaki* were Kantakari, Haritaki , Shunthi , Maricha, Pippali ,Ela ,Twak, Nagakeshara , Patra,Madhu & Guda. Various research works showed that Shirisha stimulates Adrenal gland & increases glucocorticoid levels whereas Pippali, Kantakari mainly acts as Antihistaminic, . Haritaki does not show any Bronchodilator action but it is mentioned as Srotovibandhanashini.

The causative factors for the production of complete aetiopathogenesis of the Disease asatmyajanya pranavaha srotovikara (pulmonary hypersensitivity) are: the Agni, the Dhatus, the Doshas, Vyadhi kshamatva Shakti etc. So the ultimate aim of the treatment should be correcting in all these involved factors.

The concept of Agni is of paramount interest in Ayurveda. Disturbances of Agni results in Ama formation which by itself may culminate in various ailments or by thwarting absorption and assimilation impede with the efficacy of the drug used in treatment. In Bhrigu haritaki rasayana, most of drug having Agnivardhaka, Deepana, Pachana etc. properties which provoke the Agni. Another important concept forwarded by the Ayurvedic system of medicine is that of Rasayana. Though there are no direct references found in Ayurvedic classics outlining the exact mode of Vyadhikshamatva Shakti. Bhrigu haritaki rasayana

having Rasayana, Jeevaniya, Balya, Brimhaniya, Ojovardhaka, Ayuvardhaka, Dhatuposhaka properties, which indirectly increase the Vyadhikshamatva. On the other hand, scrrening of the available Ayurvedic literature of Rasayana, Jeevaniya, Balya and ojovardhaka drugs reveals that all of these drugs are of Prithvi, Vayu bhuta predominance. Going into the bhautika composition of Bhrigu haritaki rasayana it is seen that the said compound is of Vayu (35%), Prithvi (23%), Agni (18%) predominance as described in the section devoted to drug review. Thus, since the process of Rasayana invariably involves regeneration of the dhatus. Hence Bhrigu haritaki rasayana may undoubtedly augment the process of tissue resistance or repair. To sketch the mode of action of a drug it is also imperative to look into the Rasapanchaka or the properties by which it acts, screening the Rasa of the ingredients of Bhrigu haritaki rasayana that Katu (30%), Tikta (23%), katu Vipaka (44%) subsides the nasa Kandu, Kasa, Ghana nasa srava, Agnimandhya, Jwara etc. The drug on dominence of its Madhura Rasa is found (25%), 56% is Madhura Vipaka. It is Snigdha, Guru and also elevates Vata. Among the functions ascribed to Madhura Rasa are brimhana, Jeevana and Balya. These properties are very much in favour of building up tissues and may increase the Vyadhikshamatva and alleviates Kshavathu, Shirah sholla etc. by its Vatapittahara property. The Gunas present in the ingredients of the selected drug are Laghu (30%), Ruksha (28%), Tikshana (14%) are elevating Nasa srava, Kasa etc. symptoms. Whereas Snigdha (12%), Guru (9%) acts as Balya, Tarpana and Brimhana. Virya is dominated by Ushna (55%), which has been also mentioned to be causing Vatakapha shamaka, Pachana, Deepana etc. actions. Pratishyaya results from the vitiation of vata and kapha. Various ingredients of

Bhrigu haritaki rasayana having Vatakapha shamaka (34%), Tridosha shamaka (33%) properties, which help to bring the affected Doshas in normal level. Details of each constituent drug of Bhrigu haritaki rasayana are as given below-

Bhrigu haritaki rasayana is prescribed as an ideal drug in choice selected for managing allergic condition in oral administration. The main content of this drug is Kantakari (Solanum xanthocarpum) is having Laghu, Ruksha guna, Katu Vipaka, Ushna Virya which helps in digesting the vicious Kapha and thus redusing nasal obstuction. Pippali having Shothahara, Kandughna, Vishaghna, Raktashodhaka etc. karma which, helps in relieving the symptoms of the disease. Goghrita, Guda are Madhura in Rasa, Guru, Snigdha in Guna, Sheeta in Virya and madhura in Vipaka. They also have rasayana, Ojovardhaka, Balya, Brimhana etc. properties that may increase Vyadhikshamatva and

decrease the chance of recurrence. Prakshepa dravyas like Trikatu, Chaturjata , etc. having Deepana, Pachana, vatanulomana, Shothahara, Shleshmahara, Jwaraghna, Kaphanissaraka, Rasayana, Balya etc. properties.

At modern side, most of ingredients of Bhrigu haritaki rasayana are proved as Antiinfammatory, Analgesic, Antipyretic, Antioxident, Immunostimulator, Anti allergic, Anti histaminic pharmacologically. (Database, 2001, 2002)

06.4.3 CUMULATIVE EFFECT OF BHRIGU HARITAKI AND SHIREESHARISHTA.

Most of the drugs of Shireesharishta possess Katu (41.67%) Tikta (33.33%) Rasa. So that it can provide Ama pachana, Kapha shamana and Srotoshuddhi. Laghu (41.17%), Ruksha (23.53%) guna, Katu vipaka (71.43%) are also favourable for above action. On analysis of karma it shows significant Vatakaphahara (57.14%) and Tridoshahara (28.57%) property. Vata and Kapha are the main causative humours but in fact all the three doshas participate in the pathogenesis of the disease. So these properties pacify doshas, especially Vata and Kapha, in Urdhvajatru and alleviate the associated symptoms. Vatanulomaka properties of drugs help retaining its own gati. All these properties are very much effective to reduce the symptoms like heaviness in head, rhinorrhoea, anorexia etc. Due to ingredients Maricha, Shunthi, Pippali, Haridra, which possess excellent Ama and Kaphahara property, it removes obstruction to Vata which is responsible for pathogenesis of the disease. Ushna Tikshna, Shothahara, Vishaghna properties of these drugs and also Kaphaghnata can easily remove the obstruction in Srotasa and facilitate to drainage of collected materials. Specific action on Manadgni and Shirahshula are very much helpful in condition like Jeerna (Dushta) Pratishyaya because these are the two main notorious symptoms which make the patient physically and mentally weak. Organ and system specific rasayanas i.e. Shireesha, pippali, Kantakari, boost up the function as well as immunity of Pranavaha srotasa. Maximum ingredients comprise pharmacological activity as anti-inflammatory antioxidant, anti allergic, wound healing, anti spasmodic, anti bacterial, anti fungal and anti tumor activity, like Haridra, Pippali, Maricha and Shunthi. Haridra has been proved to be the best ant allergic by many research works. Shireesha has been also specifically mentioned for upper respiratory tract infections, being proved an excellent medicine as anti asthmatic, ant allergic, analgesic, antifungal, antibacterial, bronchodilator, anti allergic & anti inflammatory action. Vasa comprises respiratory stimulant, expectorant and mucolytic action. So the drug Shireesharishta possesses Tridoshahara, Rasayana, Amapachana, Srotoshodana and

Agnideepana properties and organ and system specific action on Pranavaha Srotasa. Most of the ingredients also have antipyretic, anti inflammatory, digestive, antiallergic and antibacterial properties. All this description shows that a cumulative effect of Bhrigu haritaki and Shireesharishta, has all the factors to break the pathogenesis of asatmyajanyapranavaha srotovikara/pulmonary hypersensitivity. Also the organ and system specific rasayana property of drug improves local and general immunity, giving a protection against secondary infections and recurrence to the disease

Thus the cobined effect of Shireesharishta and Bhrigu haritaki rasayana can be summerised in the following manner.

Srotorodha in Pranavahasrotas due to Sotha created by Sama Vata.

The main logics behind the actions are:

1. The Dosha-Prashamana effect (Pippali, Talish, Adraka,) acts on the main Doshas which contribute to the Samprapti viz. Vata and Kapha.
2. Deepana-Pachana Karma (Pippali, Haritaki, Adraka,) digest Ama.
3. Vatanulomana property (Pippali, Haritaki, Adraka,) maintains the normal flow of Vata.
4. Shwasa, Kasa, Shothahara Prabhava (Haritaki, Shireesh, Adraka,) act on the symptoms.

The pharmacological studies already reported on the individual drugs also favor its effect in disease Bronchial Asthma-

Anti allergic – Shireesh

Anti inflammatory – Shireesh, Haritaki, Adraka,

Anti tussive - Adraka,

Bronchodilator – Shireesh,

Expectorant - Shireesh, Haritaki, Talish-patra,

Immunomodulator - Pippali

Table No: 153

Probable mode of action of drugs on asatmyajanya pranavaha stroto dushti vikara

Sl no	Name	Karma
1	Pippali, Talish, Adraka,	Dosha-Prashamana

2	Pippali, Haritaki, Adraka,	Deepana-Pachana
3	Pippali, Haritaki, Adraka, Yastimadhu	Vatanulomana
4	Haritaki, Shireesh, Adraka	Shwasa, Kasa, Shothahara

Table No: 154

Probable mode of action of drugs on pulmonary hypersensitivity.

Sl no	Name	Action
1	Shireesh	Anti allergic
2	Shireesh, Haritaki, Adraka,	Anti inflammatory
3	Shireesh,	Bronchodilator
4	Shireesh, Haritaki, Talish-patra,	Expectorant
5	Pippali	Immunomodulator
6	Shirish	Anti tussive
		mucolytic,
	Pippali,	antioxidant
	Pippali, Shirisha & Kantakari	Antihistaminic activity

07 CONCLUSION

The present study was conducted on pulmonary hypersensitivities stressing upon Conceptual, Diagnostic as well as Therapeutic perspectives of pulmonary hypersensitivities, under the lights of the Ayurveda and Modern science. By thorough clinical study, taking in to account both Subjective as well as Objective parameters, the results have been obtained

and are discussed in the previous section. By considering this study as a whole, following conclusions can be drawn.

1. Respiratory Hypersensitivity/ Allergy is nothing but the disease of pranavahasrotus manifested as a sequence of asatmyaavastha produced either due to Sahaja or Viruddha or Visha (Gara/Dooshi) or Satmyasatmya Karana.
2. Family history of hypersensitivity (sahajakarana) was the dominating nidana amongst all nidana (129 patients, 86%).
3. Two main mechanism of the pathogenesis in modern science i.e.Bronco constriction (Bronchospasm) and obstruction due to mucous formation can be correlated asVatikaSamprapti and Kaphaja respectively where Sankochais due Vata andSrotorodha is due to Kapha.
4. Shireesharishta and Bhrigu haritaki showed approximately equal effects on roga bala when used separetly.
5. Where in when used in combination, there was better effect when compaired with when used separetly.
6. The significant reduction in the Eosinophil count showed its Ant allergic Activity. Pippali, Shirisha&Kantakari possess Antihistaminic activity Shirishais having Bronchodilator action which helps to relieve Bronchospasm. Pippali acts as Rasayana to PranavahaSrotasa which prevents further episodes of HYPERSENSITIVITY.

Recommendations for further study

- ✓ Different Shodhana such as Vamana,Nasyaand Shamana Yoga having immuno modulationsproperties, which can eradicateasatmyavastha/hypersensitivity, can be studied in the cases of Asastmyajanyapranavahasrotovikakara / pulmonary hypersensitivities.
- ✓ Further specific studies on Shireesharishtaand Bhriguharitaki to know its action at Tissue level, Biochemical and Hormonal level can be taken up, so that better understanding of karmukata/mode of action is possible.

08 SUMMARY

As We live in a world full of microorganisms. Every facet of our existence exposes us to bacteria, viruses, fungi & numerous parasites. We have a natural micro flora on all over the body, within all orifices & throughout the GI tract. The respiratory tract while performing its physiological function is exposed to a wide variety of air-borne environmental antigens. The lungs are working as filter for the entire circulating blood volume. Thus, these are constantly exposed to various blood & air-borne agents that possess potential to

accelerate inflammation, infection or immune processes. Pulmonary hypersensitivities can be named as Atopic disease. Atopy is defined as familial tendency to sensitization to environmental allergens. Atopic allergy is a type 1 hypersensitivity reaction that produces IgE antibodies to allergens viz. pollen, dust, etc. pulmonary hypersensitivities has always been proved to be a problematic ailment to the doctors. The magnitude of the condition can be understood by the fact that, though it is known from the ancient era & in spite of worldwide efforts to combat this impediment, still there is no definite solution for the problem. There is only symptomatic treatment in modern medicine & so many measures have been adopted to check this disease.

In Ayurvedic classics reference regarding pulmonary hypersensitivities is explained in scattered manner. Samhitas revealed lot of information regarding this troublesome disorder in different contexts.

Hence, in the present study one of the objectives was to develop concept of pulmonary hypersensitivities in Ayurvedi perspective.

Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities nidanas can be broadly classified under two headings,1) Nidanas for asatmyavastha, 2) Nidanas for pranavahasrotodushti.Viruddha (food in compatibility) , vishas-Gara/Dooshi,Beeja dosha,satmyasatmya vikaras plays an important role in manifestation of asatmyavastha/hypersensitivity. Where in Raja,Dhooma,Sheetamaruta sevana,Sheeta abhishyandi ahara svana act as pranavaha sroto dushti karanas which is similar to the modern explanation considering Dust, Smoke ,Food, Pollen, House mite etc as allergens.

Samprapti of Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities reveals the involvement of mainly rakta dhatu and tridoshas. When visha is brought into contact with the body, it will at first cause the dushti of raktha and then the three doshas later it reaches hridaya. Hridaya is the seat of ojas. Visha is having qualities opposite to that of ojas. If the visha gunas predominate, the patient dies on the contrary when ojo gunas are superior to those of visha gunas the patient survives.

Virudhahara is one of the important causes for rakthadushti. It also causes the utklesha of tridoshas Rakta in its dhatu form are responsible for bala, varnam and sukham. It is the basis of life. Thus, raktha is very closely related to ojas in its function. Sufficient

amount of food with appropriate quality will be digested properly and converted into consecutive dhatus. When virudhahara is followed dhatu parinama process will be deranged and affect the ojas. If virudhahara combination is, very toxic death will occur instantly if the derangement is not much stronger, it will lead to decreased bala; vyadhikshamata of the body depends upon bala and the deterioration of it will lead Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities and various diseases.

In the present study of Asatmyajanya pranavahasrotovikara (respiratory hypersensitivity) the purvaroopa mentioned in context of pratishyaya can be considered, as these clinical features are observed almost similar to respiratory hypersensitivity reactions. The purvarupas described by Sushruta includes Sirogurutwa (Heaviness of head) Ksavathu (Sneezing) Angamarda (Body ache) Parihrishtaromata (Generalized horripilation) Stambha (Stiffness)

Nasasrava,Nasavarodha,Nsakandu,Kshavathu,Kasa,Shwasakrichrata,Shirashoola,Shirogourava,Netrasrava,Netraragata and kandu, Karnabadhiryata, Decrease in gandhagnyana are commonly observed in Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities.

Tamaka shwasa(Bronchial asthama),Kshayaja kasa(Chronic obstructive pulmonary disorders) and other irreversible respiratory disorders can be considered as Upadrava/complication of Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities.

Chikitsa is defined as Nidana parivarjana or avoidance of causative factors. Ayurveda being emphatic about swasthya rakshana give priority to prophylactic management. This is very much applicable in the case of asatya janya pranavaha srotodushti vikara. The agantuja hetu, triggering factors have to be avoided in the first place. Charaka has rightly said says, “(Ch.Chi.17/138) primary importance in Shwasa Chikitsa is the avoidance of causative factors. Both Ayurveda and Modern scientist agree to this fact.

There are many instances in the textbooks of Ayurveda, where we may identify the Features, Treatment, and Causes about Allergy. Only thing is that they are described in different parts of the Samhitas. The concept of *Satmya* and *Asatmya* is clearly an indication

of immunity and allergy. Here *Satmya* means Compatibility. The matter of *Asatmya* means Non-compatible to body. I.e. if a medicine or food is not acceptable to the body, body tries to through it away in different fashions. It may be Diarrhea, Vomiting, and Skin manifestations and so on. Hence the treatment runs according to the presenting complaint. Whenever the combination of two drugs or food is going to take place, it acts as either *Samyoga* or *Viruddha.* The *Viruddha* is the causative factor for *Atma/Shareera asathmya* in many instances. Even visha either Gara or Dooshi can manifest such conditions i.e., asatmya avastha.

Hence while planning the treatment of asatmyajanya pranavaha srotovikaras we may require to consider the treatment principles of visha either Gara or Dooshi,viruddha,satmya asatmyavikaras along with pranavahasrotovikara.

Hence it can be said that asatmyajanya pranavaha srotovikara is the condition produced due to tridoshas and rakta dahtu . Vamana and virechana are the choice of shodhana treatment that will help in removing vitiated pitta dosha, the toxins which are responsible for allergy. As the bala is decreased vyadhikshamatva will be less, Rasayana medicines play an important role in increasing the dhtus / oja/bala/ vyadhikshamatva.

The present clinical study on Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities was carried out by selecting 150 patients. After obtaining the written informed consent, the selected patients were assigned into **three groups** i.e. **Group A, GroupB, GroupC o**f **50 patients** each.
All patients of these three groups were given Arohanakrama Snehapana with Kantakarighritha and then Virechana with Trivrit lehya initially.

After Virechana,

For Group A patient – Shirisharishta was given in the dosage of 20ml three times in a day with ushnajala as anupan after food.

For Group B patient – Bhriguharitaki was given in the dosage of 10 gms three times in a day with Sukhoshna dugdha as anupan before food.

For Group C patient- Shirisharishta was given in the dosage of 20ml three times in a day with ushnajala as anupan after food. **Bhriguharitaki** was given in the dosage of 10 gms three times in a day with Sukhoshna dugdha as anupan before food.

This treatment was continued for 30 days. Follow-up was done for six months after the course of treatment.

The Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities patients were included under study according to specific inclusion criteria to assess definite parameters to achieve laid objectives. Patients were followed up for a period of six months. At the end of the study all the observations and Data are statistically analyzed and results derived considering objective and subjective parameters separately. The brief summary of the results can be stated.

Total effect of Therapy –Significant Improvement was found in 47 patients (94%)-group C then in 40 patients (84%) -group A then in 37 patients (74%) in group B.Improvement was observed in 13 patients (26%) group B then in10 patients (20%) in group A and 3 patients in group C (06%).Effect of therapy was statistically highly significant at the level of $p<0.001$ in all cardinal symptoms in all the group (Group A,B&C). Effect of therapy was statistically highly significant at the level of $p<0.001$ in Eiosinphil, Absolute eiosinphil and ser-IgE levels of the patients In follow up study there was not a single incidence of recurrence in patients of any group after treatment. (Table 57) Overall significant improvement was observed in 124 patients 82.66% in total and improvement was observed in 26 patients 17.34%.When we consider all the above observations it is evident that combined therapy of shirisharishta and Bhriguharitaki rasayana had better results over when used separately.

So it can be summarized that Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities is a Episodic and Chirakari vyadhi commonly affecting all the population, if untreated may lead to severe exhibition of complicated pathologies. A multidimensional and holistic Approach is required in managing Asastmyajanya pranavahasroto vikakara / pulmonary hypersensitivities. Combined therapy of Shodhana and Shamana had shown a marked response in trial cases of Asastmyajanya pranavahasroto vikakara / pulmonary

hypersensitivities, suggesting the fact that Cikitsā advised in Āyurveda is more comprehensive, rational and can be adopted in the modern times.

09 APPENDIX

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09.2 Case proforma

DEPARTMENT OF PhD STUDIES IN KAYACHIKITSA, COLLEGE OF AYURVEDA,BHARATI VIDYAPEETH UNIVERSITY, PUNE-43.

Proforma for the studies on,

MANAGEMENT OF PULMONARY HYPERSENSITIVITIES IN AYURVEDIC PERSPECTIVE – A CLINICAL STUDY.

Guide: **Dr. B. K. BHAGWAT.** BAM&S,M.A SC ,Ph.D, F.I.I.M.

Research scholar: **Dr. SAMEER N.NAIK.** BAMS, M.D KAYACHIKITSA.

CLINICAL TRIAL INFORMATION

Group	A	B	C

NAME:

SL NO-

AGE:

OPD\IPD NO-

SEX: M / F

BED NO-

ADDRESS:

DATE OF ADMISSION-

DATE OF DISCHARGE-

OCCUPATION:

MARITAL STATUS:

RELIGION:

SOCIOECONOMIC STATUS:

POSTAL ADDRESS:

DIAGNOSIS-

RESULT-

PRADAN VEDANA –

KALAVADHI

ANUBANDHI VEDANA- **KALAVADHI**

VEDANA VRITTANT-

- NASA SRAVA : YES / NO **If YES ,**

DURATION :

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : **(IF ANY)**

RELEAVING FACTORS: **(IF ANY)**

TYPE OF SRAVA :

SRAVA KALA :

VARNA OF SRAVA :

GANDHA OF SRAVA :

CONSISTANCY OF SRAVA :

- **NASA VARODHA : YES / NO *If YES* ,**

DURATION :

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : (IF ANY)

RELEAVING FACTORS: (IF ANY)

AVARAODHA KALA :

- GANDHA GNANA : YES / NO ***If YES* ,**

- NASA KANDU : YES / NO ***If YES* ,**

DURATION :

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : (IF ANY)

RELEAVING FACTORS: (IF ANY)

NASAKANDU KALA :

- KSHAVATHU : YES / NO ***If YES ,***

DURATION:

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : **(IF ANY)**

RELEAVING FACTORS: **(IF ANY)**

NO. OF KSHAVATHU :

ASSOCIATION OF SRAVA :

- HOARSENESS AND WHEEZE:
- SHWASKRICHRATA: YES / NO ***If YES ,***

DURATION :

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : **(IF ANY)**

RELEAVING FACTORS: **(IF ANY)**

- KASA : YES / NO ***If YES ,***

DURATION :

NATURE OF KASA :

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : **(IF ANY)**

RELEAVING FACTORS: **(IF ANY)**

- URAHAVEDHANA (CHEST PAIN): *YES / NO* ***If YES ,***

DURATION :

MODE OF ONSET : Insidious / Gradual/ Sub acute / Acute

COURSES : Progressive / Receding /Relapsing / Stationary

SEVERITY : Mild / Moderate /Severe

PRECIPITATING FACTORS : **(IF ANY)**

RELEAVING FACTORS: **(IF ANY)**

- NETRAGATA LAKSHANA : **Kandu / Raktavaranata / Srava.**
- KARNAGATA LAKSHANA : **Vedana / Badhiryata / Feeling of Fullness.**
- SHIROGATA LAKSHANA : **Shoola / Gouravata.**

- JWARA : YES / NO ***If YES ,***
  TYPE : Intermittent/Relapsing/Continuous.

ASSOCIATION OF CHILLS & RIGORS : YES / NO

- ANY OTHER ASSOCIATED COMPLAINTS.

- POORVA VYADHI VRITTANT:

- CHIKITSA VRITTANT:

- KULAJA VRITTANT:

- ARTHAVA AND PRASOOTI VRITTANT:

- ATUR CHARYA:

NIDRA : Normal / Disturbed

MALA PARAVRUTTI : Frequency :

Colour :

Consistency :

Odour :

MUTRA PARAVRUTTI : Frequency :

Colour :

Consistency :

Odour :

HABITS

Intake of Alcohol :

Habituated Alcoholic : Yes / No

Duration : Quantity:

Frequency : Variety :

SMOKING :

Habituated Smoker : Yes / No

Duration : Quantity:

Frequency : Variety :

FOOD : Vegetarian/Non-Vegetarian/

Occasional Non-Vegetarian

DIETRIC HABITS : Samashana/Anashana/Adhyashana/Vishamashana

QUALITY OF FOOD : Laghu/Guru/Snigdha/Rooksha/Vishada Sheeta

/Ushna/Abhishyandi/Virudda

MATRA : Guru/Laghu

SWABHAVA : Guru/Laghu

RASA PRADHANYA : Madhur/Amla/Lavana/Katu/Tikta/Kshaya

WATER RECOURSES : (Regularly Used)

OCCUPATIONAL HISTORY : Exposure to industrial dusts-Silica, Asbestos

Exposure to pigeons,parrots,Canaries or parakeets

ROGI PAREEKSHA :

Dashavidha

- Prakrutitaha : Sharirika- Manasika-
- Sarataha : Pravara/Madhyama/Avara
- Satvataha : Pravara/Madhyama/Avara
- Satmyataha : Pravara/Madhyama/Avara
- Samhananatha: Pravara/Madhyama/Avara
- Ahara Shakti : Pravara/Madhyama/Avara
- Vyama Shakti : Pravara/Madhyama/Avara
- Pramanataha : Pravara/Madhyama/Avara
- Deshataha : Jata- Samvruddi-
  Vyadhita-
- Vayataha : Bala/Taruna/Vradha (Age-)

SAMANYA PAREEKSHA :

Pulse- B.P-

R.R- Temperature-

- Built : Slender/Lanky/Muscular/Obese
- Nourishment : Good/Moderate/Poor
- Tongue :
- Nail :
- Icterus : Present / Absent
- Palor : Present / Absent

- Cyanosis : Present / Absent
- Clubbing : Present / Absent
- Lymphadenopathy : Present / Absent
- Odema : Present / Absent
- Jugular vein Prominence (JVP) : Present / Absent
- Weight :
- Height :

SYSTEMIC EXAMINATION

RESPIRATORY SYSTEM

1. Inspection - General
    a. Shape of the chest – normal, barrel shape ,Pigeon chest, Funnel shape
    b. Breathlessness, Wheezing, Sputum, Cough
    c. Skin for cyanosis, pallor, Eruptions, Nodules, Scars.
    d. Neck vein distention - Yes / No
    e. Fingers for tobacco stains and Clubbing of toes and fingers
    f. Position of Trachea - Midline / Deviation interiorly
2. Inspection - Respiratory motion
    a. Bilaterally symmetric – Yes / No
    b. Abnormal breathing – Bradypne /Tachynea / Apnea / Hyperapnea
    Periodic Respiration / Sighing respiration.
    c. Mode of breathing – Thoracic/Abdominal
3. Palpation –
    a. Lymphnodes – Auxiliae/Supercalcular
    b. Trachea - Midline / Deviation interiorly
    c. Vocal fremitus Test -
    d. Movement of the chest

4. Percussion -
    a.
    b.
    c.
    d. Chest expansion test
5. Auscultation -
    a.
    b.
    c.

CARDIO VASCULAR SYSTEM:

CENTRAL NERVOUS SYSTEM:

DIGESTIVE SYSTEM:

LABORATORY DIAGNOSIS:

HEMATOLOGY:

BLOOD:

HB%	ESR	WBC					AEC	IgE
		Total count:						
		N	L	E	B	M		

OTHER INVESTIGATION:

SPUTUM : AFB /CYTOLOGY (if required)

CHEST X-RAY : (if required)

LUNG FUNCTION TEST : (if required)

VIKRUTITAHA PAREEKSHA:

NIDHANAS :

ADIBALA PRAVRUTTA -

AHARAJA	• **Abishyandi ahar** • **Shleshmala ahar** • **Madhur ahar** • **Picchila ahar** • **Guru ahar** • **Sheeta ahar** • **Adhyashana** • **Ati matra ahar** • **Navanna** • **Nava Madhya** • **Sheetal jalapana**

	• **Mamsa- Anupa,Auduka, Gorasa** • **Any others**
VIHARAJA	• **Desha- Jangala,Anupa,Sadharana** • **Avyayama** • **Diwaswapna** • **Sheetagraha(A/C)** • **Sheeta vayu sevan** • **Raja ,Dhooma etc**
MANASHIKA	• **Strees**

POORVA ROOPA :

ROOPA :

SAMPRAPTI GHATAKA:

Dosha- Dushya-

Agni- Aama-

Srotas- Srotodushti lakshana-

Udbhavasthana- Sancharasthana-

Vyaktasthana- Vyadhi Svabhava-

UPASHAYA:

ANUPASHAYA:

SAAPEKSHA NIDANA:

ROGA NIRNAYA:

SADHYA / ASADHYATA:

GRADING BEFORE TREATMENT:

1.Kasa	
2.Nasa srava	
3.Nasavarodha	
4.Kshavath	
5.Shira shoola	
6.Shiro gouravata	
7.Jwara	

0-Absent

+ - Mild

++ - Moderate

++++ - Severe

+++++ - Very severe

Note : Jwara will be indicated in degree F

CHIKITSA:

OBSERVATION DURING TREATMENT :

S / NO	NAME	7th DAY	14th DAY	21th DAY	30th DAY
1	Kasa				
2	Nasa srava				
3	Nasavarodha				
4	Kshavath				
5	Shira shoola				
6	Shiro gouravata				
7	Jwara				

ANALYSIS

Comparative analysis of laboratory findings before and after treatment.

Name of the Investigation		Before Treatment	After Treatment
Hb%			
TC			
DC	N		
	L		
	E		
	B		
	M		
ESR			
AEC			
IgE			

- SPUTUM : **(If required)**
  Sputum AFB/Cytology

- Chest X-ray **(If required)**

- Lung Function Test

COMPARATIVE ANALYSIS OF LAKSHANAS BEFORE AND AFTER TREATMENT

LAKSHANA	BT	AT
1.Kasa		
2.Nasa srava		
3.Nasavarodha		
4.Kshavath		
5.Shira shoola		
6.Shiro gouravata		
7.Jwara		

Fallow-ups : i)

ii)

iii)

iv)

Signature of the Guide

Signature of Researcher

09.3 Incidence of Age, Sex,marital status. Comparison of Treatment before and after.

Table No. 169- showing incidence of age,sex,marital status & camparison of different laboratory investigations before, after &during follow ups in Group A .

Sl N0	AGE	SEX	OCCUPATION	MARITAL STATUS	HAEMMO GLOBIN	EIOSINOPHIL BT	AT	A.E.C BT	AT	IgE LEVEL BT	AT
1	31	M	BANKEMPLOYI	MARRIED	12.8 %	6	4	554	345	225	153
2	24	M	STUDENT	UN MARRIED	13	7	5	567	425	565	135
3	43	M	TEACHER	MARRIED	13.5	8	4	678	450	456	151
4	33	F	TEACHER	MARRIED	11.8	7	4	767	440	765	134
5	26	F	HOUSEWIFE	MARRIED	12.8	9	5	890	425	345	120
6	45	M	MINEWORKER	MARRIED	11	7	3	490	435	876	111
7	33	M	FACTORY JOB	MARRIED	14	8	4	565	330	1123	157
8	37	M	FACTORY JOB	MARRIED	10.6	5	3	475	350	1567	124
9	19	M	STUDENT	UN MARRIED	11.3	10	6	1290	450	1249	170
10	18	M	STUDENT	UN MARRIED	13	9	4	900	445	345	41
11	33	M	PAINTER	MARRIED	12	7	3	870	395	654	51
12	24	M	STUDENT	UN MARRIED	12.4	8	4	980	460	675	105
13	41	F	WEAVER	MARRIED	12.7	10	6	1180	475	357	75
14	36	F	HOUSEWIFE	MARRIED	12.4	9	3	567	330	875	112
15	51	F	HOUSEWIFE	MARRIED	11,9	8	3	875	355	698	135
16	36	M	BANKEMPLOYI	MARRIED	10.8	8	4	675	385	879	128
17	35	F	BANKEMPLOYI	MARRIED	09.8	9	4	760	350	693	106
18	26	M	PAINTER	UN MARRIED	10.8	6	3	610	390	563	145
19	29	F	BANKEMPLOYI	MARRIED	11.5	7	4	690	445	556	135
20	24	M	STUDENT	UN MARRIED	12.5	9	3	895	410	357	85
21	33	M	FACTORY JOB	MARRIED	13.4	6	4	565	325	1680	210
22	26	M	BUISINESS	MARRIED	12.3	8	3	795	440	1980	153
23	44	F	HOUSEWIFE	MARRIED	11.3	10	4	1050	455	540	90
24	47	M	FACTORY JOB	MARRIED	11.4	10	5	1125	465	675	85
25	53	F	HOUSEWIFE	MARRIED	10	9	3	905	425	445	45

Table No 170- Showing Incidence Of Age,Sex,Marital Status & Camparison Of Different Laboratory Investigations Before, After &During Follow Ups In Group A .

Sl	AGE	SEX	OCCUPATION	MARITAL	HAEMMO	EIOSINOPHIL	A.E.C	IgE LEVEL

N0				STATUS	GLOBIN	BT AT		BT AT		BT AT	
26	36	F	BANKEMPLOYI	MARRIED	14.8 %	7	4	456	355	425	123
27	35	M	MINEWORKER	MARRIED	13.8	5	5	656	325	665	35
28	43	F	TEACHER	MARRIED	12.5	8	4	890	350	467	131
19	15	F	STUDENT	UN MARRIED	13.8	11	5	1040	340	965	144
30	26	F	HOUSEWIFE	MARRIED	10.8	9	5	895	435	395	125
31	16	M	STUDENT	UN MARRIED	11.9	7	3	785	335	676	143
32	33	M	FACTORY JOB	MARRIED	12	12	4	1025	430	1883	165
33	56	M	FACTORY JOB	MARRIED	14.6	15	6	1245	450	967	114
34	57	M	STUDENT	UN MARRIED	12.3	7	3	895	350	1049	140
35	19	M	STUDENT	UN MARRIED	11	9	4	765	445	445	61
36	33	F	PAINTER	MARRIED	13	6	3	900	395	554	81
37	31	M	FARMER	UN MARRIED	13.4	9	4	865	440	689	135
38	32	F	WEAVER	MARRIED	11.7	8	6	695	415	457	75
39	36	F	HOUSEWIFE	MARRIED	12.4	6	3	560	330	675	132
40	51	F	HOUSEWIFE	MARRIED	11	7	3	890	375	998	135
41	36	F	BANKEMPLOYI	MARRIED	11.8	8	4	965	380	679	128
42	35	F	BANKEMPLOYI	MARRIED	11.8	10	4	1065	370	493	106
43	26	M	PAINTER	UN MARRIED	10.8	7	3	700	320	663	135
44	29	F	BANKEMPLOYI	MARRIED	11.5	5	3	670	345	1056	112
45	24	M	STUDENT	UN MARRIED	09.5	11	3	1245	440	657	65
46	22	M	FACTORY JOB	MARRIED	12.4	8	4	860	365	1080	150
47	26	M	BUISINESS	MARRIED	12.3	9	3	890	430	1785	153
48	44	F	HOUSEWIFE	MARRIED	12.3	6	4	670	355	840	90
49	47	F	FACTORY JOB	MARRIED	13.4	8	5	984	365	975	85
50	53	F	HOUSEWIFE	MARRIED	12.6	7	3	770	415	865	45

Table No 171- Showing Incidence Of Age,Sex,Marital Status & Camparison Of Different Laboratory Investigations Before, After &During Follow Ups In Group B .

Sl N0	AGE	SEX	OCCUPATION	MARITAL STATUS	HAEMMO GLOBIN	EIOSINOPHIL BT AT		A.E.C BT AT		IgE LEVEL BT AT	
1	41	F	HOUSEWIFE	MARRIED	13.8 %	8	3	356	305	725	61
2	24	M	STUDENT	UN MARRIED	11.8	7	4	956	425	465	35
3	33	M	TEACHER	MARRIED	10.5	6	3	790	450	470	41
4	33	F	TEACHER	MARRIED	11.8	10	4	1040	440	865	144
5	26	F	HOUSEWIFE	MARRIED	10.8	8	4	895	335	395	95
6	45	M	MINEWORKER	MARRIED	13	6	3	1085	435	676	143
7	33	F	FACTORY JOB	MARRIED	13	7	3	1025	415	1083	165

8	37	M	FACTORY JOB	MARRIED	14.9	9	5	1245	450	1167	114
9	19	M	STUDENT	UN MARRIED	10	11	3	895	360	1049	140
10	18	F	STUDENT	UN MARRIED	11.8	11	4	765	425	655	131
11	33	M	PAINTER	MARRIED	13.9	6	3	950	395	754	121
12	21	F	STUDENT	UN MARRIED	12	8	4	865	415	619	115
13	41	M	FACTORY JOB	MARRIED	11.7	9	6	895	315	457	75
14	36	F	HOUSEWIFE	MARRIED	12.4	9	3	830	370	675	132
15	41	F	HOUSEWIFE	MARRIED	11	7	3	790	275	998	145
16	36	M	BANKEMPLOYI	MARRIED	13	8	4	965	280	289	128
17	35	F	BANKEMPLOYI	MARRIED	11.2	10	4	920	270	763	106
18	26	M	PAINTER	UN MARRIED	10.8	7	3	785	360	663	45
19	34	M	FARMER	MARRIED	11.5	9	3	679	345	956	92
20	24	M	STUDENT	UN MARRIED	11.5	11	4	1045	440	457	105
21	33	M	FACTORY JOB	MARRIED	12.4	8	4	660	335	1780	150
22	26	M	BUISINESS	MARRIED	13.3	9	3	890	430	1085	143
23	44	F	HOUSEWIFE	MARRIED	12.3	6	4	670	355	840	130
24	47	M	FACTORY JOB	MARRIED	13.4	11	5	984	365	975	125
25	44	F	HOUSEWIFE	MARRIED	12.6	13	3	1230	325	1365	145

Table No 172- Showing Incidence Of Age,Sex,Marital Status & Camparison Of Different Laboratory Investigations Before, After &During Follow Ups In Group B .

Sl N0	AGE	SEX	OCCUPATION	MARITAL STATUS	HAEMMO GLOBIN	EIOSINOPHIL BT AT		A.E.C BT AT		IgE LEVEL BT AT	
26	22	F	HOUSEWIFE	MARRIED	14.8 %	7	4	456	355	425	123
27	24	M	STUDENT	UN MARRIED	13.8	5	5	656	325	665	35
38	43	M	FARMER	MARRIED	12.5	8	4	890	350	467	131
19	33	F	TEACHER	MARRIED	13.8	11	5	1040	340	965	144
30	26	F	HOUSEWIFE	MARRIED	10.8	9	5	895	435	395	125
31	45	M	MINEWORKER	MARRIED	11.9	7	3	785	335	676	143
32	33	M	FACTORY JOB	MARRIED	12	12	4	1025	430	1883	165
33	37	M	FACTORY JOB	MARRIED	14.6	15	6	1245	450	967	114
34	19	F	STUDENT	UN MARRIED	12.3	7	3	895	350	1049	140
35	18	M	STUDENT	UN MARRIED	11	9	4	765	445	445	61
36	33	M	PAINTER	MARRIED	13	6	3	900	395	554	81
37	24	F	STUDENT	UN MARRIED	13.4	9	4	865	440	689	135
38	37	M	WEAVER	MARRIED	11.7	8	6	695	415	457	75
39	36	F	HOUSEWIFE	MARRIED	12.4	6	3	560	330	675	132
40	51	F	HOUSEWIFE	MARRIED	11	7	3	890	375	998	135

41	36	M	BANKEMPLOYI	MARRIED	11.8	8	4	965	380	679	128
42	35	F	BANKEMPLOYI	MARRIED	11.8	10	4	1065	370	493	106
43	26	M	PAINTER	UN MARRIED	10.8	7	3	700	320	663	135
44	29	F	BANKEMPLOYI	MARRIED	11.5	5	3	670	345	1056	112
45	24	M	STUDENT	UN MARRIED	09.5	11	3	1245	440	657	65
46	33	M	FACTORY JOB	MARRIED	12.4	8	4	860	365	1080	150
47	26	M	BUISINESS	MARRIED	12.3	9	3	890	430	1785	153
48	44	F	HOUSEWIFE	MARRIED	12.3	6	4	670	355	840	90
49	32	F	FACTORY JOB	MARRIED	13.4	8	5	984	365	975	85
50	39	M	FACTORY JOB	MARRIED	12.6	7	3	770	415	865	45

Table No 173- Showing Incidence Of Age,Sex,Marital Status & Camparison Of Different Laboratory Investigations Before, After &During Follow Ups In Group C.

Sl N0	AGE	SEX	OCCUPATION	MARITAL STATUS	HAEMMO GLOBIN	EIOSINOPHIL BT AT		A.E.C BT AT		IgE LEVEL BT AT	
1	44	F	BANKEMPLOYI	MARRIED	14.0 %	5	4	450	345	415	55
2	19	F	STUDENT	UN MARRIED	13.0	5	3	647	315	656	30
3	43	M	TEACHER	MARRIED	12.2	6	4	840	310	458	77
4	33	F	TEACHER	MARRIED	13.	10	3	1140	360	970	134
5	26	F	HOUSEWIFE	MARRIED	11	9	4	900	356	395	111
6	45	M	MINEWORKER	MARRIED	12	8	4	785	320	660	78
7	33	M	FACTORY JOB	MARRIED	12	12	4	1025	430	1883	108
8	37	M	FACTORY JOB	MARRIED	14	13	4	1119	425	925	65
9	19	M	STUDENT	UN MARRIED	12	6	4	886	346	1030	89
10	18	M	STUDENT	UN MARRIED	13	8	4	780	450	430	58
11	33	M	PAINTER	MARRIED	12	5	3	988	375	550	76
12	24	M	STUDENT	UN MARRIED	13	8	4	850	330	679	98
13	41	F	WEAVER	MARRIED	12	6	4	680	405	443	71
14	36	F	HOUSEWIFE	MARRIED	13.4	8	4	560	320	660	122
15	51	F	HOUSEWIFE	MARRIED	12	6	3	880	365	988	125
16	36	M	BANKEMPLOYI	MARRIED	12.8	6	4	955	370	669	118
17	35	F	BANKEMPLOYI	MARRIED	12.8	6	3	1055	365	483	102
18	26	M	PAINTER	UN MARRIED	11	8	4	720	340	670	43
19	33	F	BANKEMPLOYI	MARRIED	12	6	3	663	342	1051	110
20	24	M	STUDENT	UN MARRIED	11	10	3	1251	346	658	68
21	33	M	FACTORY JOB	MARRIED	13	7	4	869	368	1085	65
22	26	M	BUISINESS	MARRIED	12	8	4	880	320	1775	143
23	44	F	HOUSEWIFE	MARRIED	13.3	8	3	660	345	830	80
24	55	M	FACTORY JOB	MARRIED	13	7	4	974	355	965	75

25	32	F	HOUSEWIFE	MARRIED	13.6	8	4	780	325	875	55

Table No 174- Showing Incidence Of Age,Sex,Marital Status & Camparison Of Different Laboratory Investigations Before, After &During Follow Ups In Group C .

Sl N0	AGE	SEX	OCCUPATION	MARITAL STATUS	HAEMMO GLOBIN	EIOSINOPHIL BT AT		A.E.C BT AT		IgE LEVEL BT AT	
26	22	M	BANKEMPLOYI	MARRIED	14 %	8	3	446	345	415	72
27	24	M	STUDENT	UN MARRIED	13	7	4	636	305	645	35
28	43	M	TEACHER	MARRIED	13.5	8	3	870	330	447	44
19	33	F	TEACHER	MARRIED	12.8	11	4	1240	360	985	64
30	34	F	HOUSEWIFE	MARRIED	11	8	4	875	405	375	68
31	38	M	MINEWORKER	MARRIED	12	8	4	765	325	666	96
32	33	M	FACTORY JOB	MARRIED	13	11	4	1125	340	1893	89
33	37	M	FACTORY JOB	MARRIED	14	15	4	1265	366	977	34
34	19	F	STUDENT	UN MARRIED	13	6	3	865	320	1029	20
35	18	M	STUDENT	UN MARRIED	12	9	3	775	335	465	51
36	33	M	PAINTER	MARRIED	14	8	4	920	305	574	91
37	24	M	STUDENT	UN MARRIED	13	8	3	855	350	659	44
38	41	M	WEAVER	MARRIED	12	7	3	675	405	467	85
39	36	F	HOUSEWIFE	MARRIED	12	7	3	580	340	665	67
40	51	F	HOUSEWIFE	MARRIED	13	8	3	870	355	978	105
41	36	M	BANKEMPLOYI	MARRIED	12	9	3	935	350	649	78
42	35	F	BANKEMPLOYI	MARRIED	13	9	3	1055	360	483	96
43	26	M	PAINTER	UN MARRIED	11	8	4	720	340	683	25
44	41	F	BANKEMPLOYI	MARRIED	12.8	6	3	660	325	1036	102
45	24	M	STUDENT	UN MARRIED	10	11	4	1265	320	677	55
46	33	M	FACTORY JOB	MARRIED	13.4	7	3	860	345	1060	99
47	45	M	BUISINESS	MARRIED	13	8	4	880	420	1765	43
48	44	F	HOUSEWIFE	MARRIED	13	8	3	680	365	830	80
49	33	M	FACTORY JOB	MARRIED	13	9	4	964	355	985	75
50	27	F	HOUSEWIFE	MARRIED	12	8	4	750	405	845	35

09.4 FOLLOW UP ASSESSMENTS OF GRADINGS BEFORE, DURING,AND AFTER TREATMENT AND FOLLOW UP.

09.5 BIBILIOGRAPHY

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