INTRODUCTION
CHAPTER - I
INTRODUCTION

In spite of all round developments in the medical research, the problem of Bronchial Asthma still remains a challenge to the medical scientists. Of course this is a chronic disease which is Paroxysmal in nature and highly horrifying to the patients, as well as to the physician. It has a very high incidence i.e. 0.5-2% (Harve) affecting people of every country irrespective of age & sex.

In spite of phenomenal progress in the medical sciences, the treatment of Bronchial Asthma still consists of some palliative symptomatic remedies. Although several powerful drugs have been tried to dilate the bronchials and thereby relieve the symptoms, all these effects are temporary and patient gets attacks quite repeatedly. Several herbal species were tried in different preparations in the treatment of Bronchial Asthma.

Since the time immemorial man has made use of plants in the treatment of disease. The pharmacopias of many countries of the world include even today a large number of drugs of plant origin. The history of medicinal plants dates back to Rigveda, perhaps the oldest repository of human knowledge, which was written in about 4500-1600 B.C. Then there is the Ayurveda (about 2500 B.C.) which contains more detail accounts of many drugs. Following the eight divisions of Ayurveda, there are the comprehensive works of Charaka (1000 B.C.) and Sushruta (800 B.C.) which gave a detailed description of materia medica as it was known to ancients. The work of atreya, Jivaka and Kashyap about (600 B.C.) vyadi
about 500 B.C. Patanjali about (200 B.C.) Nagarjun about (500 B.C.) and the Bower script about (300-400 A.D.) added to the knowledge of herbal medicine. The Mohammedan culture enriched the vegetable materia medica which was more enlightened by coming in touch with Greece Aribia & Persia (Arora 1965).

India is endowed with a very rich flora because of the size of the country with its varieties of climatic and soil conditions prevailing in different geographical regions and as such there is a wonderful opportunity for working on plant products.

Another fortunate factor is that herbal medicaments do not produce any side effects commonly seen after long term administration of synthetic drugs resulting in a revival of interest in their use all over the world in both developing and developed countries. (Jain and Nagara 1990).

In recent surveys it has been seen that 25-30% prescriptions, even in some developed countries contain medicines with plant ingredients. There has been fast growing demand on herbal drugs in the last two decades in every branch of medical care.

Shirisha from the review of Ayurvedic literature it seems, is an important plant of Indian system of medicine and has been mentioned in all the books of Ayurveda of classical medieval and modern age dealing with herbs and medicaments. It is the most popular as an antidote for several types of poison and for the treatment of skin diseases. As for the means of administration it has been prescribed for both external and internal use. As far as its use in Bronchial Asthma is concerned charaka was first
to point the use of A. Lebbeck in the treatment of swasa (Bronchial Asthma).

R. M. Tripathy, P. C. Sen and P. K. Das, Institute of Medical Science studies on the mechanism of action of Albizzia lebbeck in the treatment of Atopic Allergy shows that A. lebbeck has significant crooglycate like action on the mass cell. In addition it appears that it inhibits the early processes of sensitization and synthesis of reaginic type antibodies. If A. lebbeck is given during the first week of sensitization it markedly inhibits the early sensitization processes, while if given during the second week it supresses antibody production during the period of drug administration.

Through the retract of this plant has been tried in different study models in different parts of our country, this has not yet been tried from the North Eastern Region even though this plants are abundant here.

Hence the present study was undertaken with the following aim and objects —

The aim of the present work was to study the effects of the Albizzia lebbeck (Siris) plant in Bronchial Asthma and the objective was as follows—

(1) To see the pharmacological action of the plant extract in Bronchial Asthma.

(2) To evaluate its effect on different respiratory function.

(3) To study the side affects of the plant.

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LITERARY REVIEW

Bronchial Asthma is a condition characterised by recurrent periodic paroxysms of wheezing, frequently associated with dyspnoea, choking and coughing, due to obstruction to expiratory air flow. The patient is symptom free during the period in between attacks. (Cripe, 1962).

Asthma is not a disease entity but one form of clinical presentation of a variety of disorders of the bronchi in which marked changes in bronchial calibre may occur over short periods of time, either spontaneously or in response to treatment. (Cecil and Loeb, 1971).

Asthma is, therefore, the name for symptom produced by widely different conditions and not limited to a particular disease. The term is used for example, in connection with cardiac disease and in advanced renal disease, in which the cause may be partly cardiac and partly metabolic.

The term can also be used in cases of postural dyspnoea produced by the pressure of mediastinal tumor. In bronchial asthma, patient is subjectively and objectively in good health in between the paroxysms, at outset before complications have occured.

The American Society (1962) has mentioned "Asthma as a disease characterised by an increased responsiveness of the trachea
and bronchi to various stimuli and manifested by widespread narrowing of the airway that changes in severity either spontaneously or as a result of Therapy”.

HISTORICAL REVIEW

The word Asthma is derived directly from a Greek word meaning a short drawn breath or panting. Of the eight Egyptian medical papyri only the Ebers Papyrus (about 1550 B.C) throws light on the Egyptian view of respiratory diseases. Ebell (1937) identifies a number of remedies to cure asthma in its fiftyfifth column. Mishnaic text of Talmud contains a phrase which may be translated literally as he was visited by a short spirit of wind. Dandy (1933) claimed that this referred to tetanus whilst Epstein (1935), probably more correctly maintained that Asthma was the condition involved. Hippocrates (Chadwick and Mann, 1950) mentions Asthma and in his essay on the influence of climate on breath regards both convulsions and asthma in children to be due to divine visitations.

The first recognisable clinical description of an asthmatic paroxysm was made by Areteus of Cappadocia (second century, A.D.). Areteus emphasized the plight of asthmatic patient when he wrote "They eagerly go into open air, since no house sufficeth for their respiration" and further on "They breath standing as if desiring to draw in all the air they can possibly inhale.

Celsus about 30 A.D. divided the various conditions presenting
with dyspnoea, into three classes, depending on the degree of difficulty with breathing namely Dyspnoea, Asthma and Orthopnoea. For a long time Asthma was regarded and treated more as a symptom than as a definite condition.

In 1552 Gerolamo Cardano a distinguished physician from Pavia, was called all the way from Italy to Edinburgh to treat John Hamilton, Archbishop of St. Andrews, who for the previous ten years had suffered from Asthma. Cardano enjoyed a good reputation by relieving the Archbishop of his ailment. In the early seventeenth century, Van Halmont himself an asthmatic put forward a rather fanciful theory about the cause of Asthma, blaming it on an imbalance of various "Spirits", an exercise popular at that time. He was the first to point out that Asthma resulted from "a drawing together of the smallest terminal bronchi". The first reference to hypersensitivity being involved in the aetiology of Asthma is also his.

Thomas Willis (1678) said of Asthma "there is scarce anything more sharp or terrible than the fits thereof". He described two forms of Asthma "Pneumonick" and "Convulsive". The former he associated with obstruction of bronchi by thick tumors, swelling of their walls and obstruction from without. He believed convulsive Asthma to be due to "Cramps of the moving fibres of the bronchi" and also of the vessels of the lungs, Diaphragm and muscles of the breast.

In 1968 Sir John Floyer, who like Van Halmont was an asthmatic,
in the first edition of his book "A treatise of Asthma" wrote, "I have assigned the immediate cause of Asthma to straightness, compression or constriction of the bronchi. If the Asthma be put partially described, and false hypothesis build on that description, the practice answering that is very importinent or injurious". He described Asthma as "a laborious respiration, with lifting up the shoulders, and wheezing, from the compression, obstruction and coarctation of some branches of the bronchi and some lobes of the bladders of lungs".

Floyer described how ineffectual the various forms of treatment popular in his day were in relieving him from Asthma. His book was translated into French and German and it was reprinted several times over the next hundred years. Most of his observations are relevant today "The Asthma is a long disease, and it requires a long observation to give a true account of its symptoms, changes and various causes which common patients cannot nicely observe".

Floyer adopted Willis classification, calling convulsive asthma "Periodic" and pneumonick Asthma "continued". This classification was widely accepted until about the middle of eighteenth century. There then developed a tendency to revert back to ancient times and to use word "Asthma" in association with various types of dyspnoea, and spasmodic conditions for instance Angina pectoris, which Hereberdon described in 1768, because known as Asthma, pressorio-dolorficum. There were a few notable exceptions and at the end of eighteenth century
there were physicians who regarded Asthma in the sense of Willis and Floyer. Amongst these were Cullen, a distinguished Edinburgh physician. In 1784 he wrote that "The terms of Asthma has been commonly applied by the vulgar, and even by many writers on the practice of physic to every case of difficult breathing that is to every species of dyspnoea but not distinguishing it with sufficient accuracy from the other cases of dyspnoea, they have introduced a great deal of confusion into their treatises on this subject". Cullen described the condition as an affliction of the nervous system and pointed out that it depended upon the "mobility of the moving fibers of the lungs". He listed as precipitating causes, heat, cold, passion of mind, particular odours and irritations of smoke and dust.

The great French clinician Leaonnec finally actually, described and differentiated Asthma from all the many conditions in which shortness of breath is the outstanding symptom. Leaonnec too attributed Asthma to a spasm of smooth muscle fibres of the bronchi.

For a long time Asthma was thought to cause moderate morbidity and negligible mortality. Indeed, the American physician Oliver Wendell Holmes in nineteenth century went so far as to call it "The slight ailment that promotes longevity". Huber and Koessler's paper in 1922 claimed that death did not occur in asthmatic paroxysm. Physicians totally disregarded what Cullen had written in 1785, "The Asthma though threatening immediate death, seldom occasions it and many patients have lived long under this disease. In many cases however, it does
not prove fatal, sometimes a very quickly and perhaps always at length."

Longet in 1842 first demonstrated that stimulation of the distal end of cut vagus nerve induced contraction of the bronchi. He gave momentum to the theorise of "Nervous Asthma" hinted by Cullen and which were elaborated by Hyde salter in 1868. Salter believed that the lesions of spasmodic Asthma resided in the vagus. He described eosinophil in the sputum of asthmatics before they were termed as such by Ehrlich 1879. William Olser too believed that Asthma was a nervous condition. His concept was prevalent 50 years ago inspite of the fact that Meltzor in 1910 had published a paper in which he suggested that bronchial Asthma could be as a result of an allergic phenomenon. Osier emphasized also the improtance of mucosal oedema, and the viscid mucosal plugs in the pathophysiology of Asthma, an observation which had previously been hinted first by Floyer and later on in 1833 more explicitly by Curshmann.

The condition which Thomas Willis described as "Asthma Masime Terribilis" in 1687 is still one of the most discouraging conditions to treat.

AETIOLOGY

Wiler (1952) has suggested that it would be well to stop searching for the cause of clinical derangement and direct our efforts towards a more complete description of the organism in its environment and the process involved in response to precipitating agents. Such a
point of view is particularly relevant in considering the aetiology and mechanisms of Asthma.

The investigation of bronchial Asthma has been concerned with the role of infections, allergic, psychologic, social, endocrine and hereditary factors. (Stein, 1962).

Citron and pepys (1964) have mentioned that the allergic causes were most common in asthmatics. Early onset Asthma occurs most commonly in atopic individuals, who readily form reaginic antibodies to commonly encountered allergens. These individuals usually give a history of other allergic disorders such as allergic rhinitis and eczema, and a family history of these disorders and early onset Asthma. Generally there are many different allergens implicated in every case.

Late onset Asthma generally occurs in non-atopic individuals, in which external allergens do not play any part in the production of disease to which term "Intrinsic Asthma" is sometimes applied. Ferguson (1973) has reported that possibly the alpha-antitrypsin deficiency and intrinsic Asthma might in some cases stand causal relationship to each other. There is a suspicion, however, that hidden allergens play some role, even in so called intrinsic Asthma. (Robbins, 1974).

A third type "Mixed pattern" may be added where both extrinsic and intrinsic factors operate. The form referred to as mixed Asthma comprises the largest group of patients.

The most common allergens responsible in case of atopic
individuals, are pollen, mite containing house dust, deathers, animal dander and fungal spores which are derived from organic material and enter the bronchi with inhaled air previous exposure to these substances will have stimulated the formation of reaginic antibody (immunoglobulin, IgE), and a Type I antigen antibody reaction in bronchi may follow further exposure to specific allergen. This release pharmacologically active substances, such as histamine, bradykinin, a low reacting substance and serotonin, which promote bronchial constriction, and an inflammatory reaction of allergic type in bronchial mucosa.

The identification of reaginic immunoglobulin IgE by Ishizak and Ishizak (1967a) has unfolded the pathogenesis of atopic skin reactions as well as extrinsic Asthma.

Much less frequently similar effects may be produced by ingested allergens, derived from certain foods, such as fish, egg, milk, yeasts and wheat, which presumably reach the bronchi via the bloodstream. Ingested non-protein substances, particularly drugs, of which aspirin is a notable example, occasionally cause Asthma by forming hapten.

Liehenstein and Osler (1964) have also reported that, when antigen-antibody react (IgE) with each other, they produce a number of pharmacological substances like above.

Augustin (1959) has described different modes of induction of asthmatic response to allergens and recent work on the antigenic study
of grass pollens has shown that a number of different fractions may be operated by electrophoresis, some of which stimulate reaginic antibody formation while others do not.

Ingmar et al. (1965) have reported that in faeces examination perfringens were found elevated in bronchial Asthma as well as in patients of rheumatic arthritis.

Thomas et al. (1974) have reported that 5 patients of children with cystic fibrosis developed allergic bronchospasm after inhaling the pancreatic extracts sprinkled on the food of their children. There was a positive history of allergy but no past history of Asthma, in any of the 5 parents. Asthma responded to bronchodilators, and use of face mask during preparation of meals prevented recurrence. To date Asthma precipitated by inhalation of Pancreatic extract, has not been seen in any of these patients with cystic fibrosis.

Ishizaka and Ishizaka (1967b) showed that IgE was present in minute amount in the normal sera but was markedly elevated in the sera of patients with extrinsic Asthma.

Grieco (1968) reported that out of chemical mediators liberated, bradykinin and SRS, are liberated, perhaps, as a result of histamine induced increased vascular permeability.

2. HEREDITY:

(a) Nature of Inheritance: Individuals who have a hereditary predisposition to anaphylactic reactions are said to be "atopic"
individuals. Heredity is important with positive family history of one or more allergic conditions in 60% cases. One inherits the predisposition to allergy but not necessarily to a particular allergic disease, thus the grand father may have Asthma, the father hayfever, and grandson may have eczema. Nor does one inherit the particular offending allergen e.g. one member of family may be allergic to fish, another to house dust and third to ragweed pollen (Myers, 1959). Cook and Venerveor 91916) often making extensive studies came to the conclusion that genetic influences play a significant role in allergy.

(b) Diagnosis of Inheritance : This type of individual can be detected by positive skin sensitivity test with a wide range of common allergens. They also give a history of other allergic disorders such as allergic rhinitis, Eczema and a family history of these disorders.

(c) Incidence of Heredity : Schwartz calculates that if one parent has asthma and other is healthy, 50% of children will carry the gene, but only 13% will develop Asthma. If both the parents are asthmatics, 70-100% of the children will carry the gene, and 20-25% will develop the disease. If two healthy carriers of gene will marry, 75% will be carriers and 22.5% will become asthmatics from amongst the offsprings.

3. PSYCHOGENIC FACTORS :

(a) Status of Psychological Factors : Until the allergic phenomenon was discovered, Asthma was considered primarily a nervous disease
and is referred to in older medical books as "Asthma Nervosa". With the advent of modern immunology, in which the phenomenon of anaphylaxis was a cornerstone, attention became focused on allergic component and the older view of Asthma as a nervous disease came to be considered obsolete. More recently, in the era of psychosomatic orientation emotional aetiology of asthma has been revived. (Alexander, 1950).

There is a considerable clinical evidence that psychological factors are also of importance in the precipitation and development of some cases of Asthma. The relationship of emotional factors to bronchial Asthma has been reviewed by Wittkower (1935), Dunbar (1938), French and Alexander (1941); Leigh and Knapp (1957). A specific psychological determinant has not been found and it appears that a variable interplay of emotional factors has been mentioned by different observers. They include almost any sudden intensive emotional stimulus, such as sexual excitation, anxiety, jealousy and rage.

Purely psychological stimuli may be the sparks which touches off the explosion. The account emphasises in striking fashion the fact that there may be a summation of the several factors, constitutional, nervous and allergic, in determining the initiation and intensity of the attack. (Coope, 1950)

Ellman (1952) has described a psychogenic type of asthmatics. These patients are usually temperamentally unstable and highly strung,
and emotion commonly sets off an attack, e.g. anger, frustration.

Prigal (1960) has put forward "a unified field theory" to show the inter-relationship of allergy, infection and psyche.

According to Jensen (1953) all diseases can be considered as signifying a disturbance of homoestasis. The concept of homoestasis implies a constant interplay of opposing forces which when balanced permit a state of well being, but when unbalanced by one force or state of forces, produce a state of disease. Asthma may also be considered as being due to a disturbance of the balance of different forces. Laigh and Manley (1956) showed that feelings of inadequacy and tension are extremely common in asthmatics.

b) Experimental and Clinical Evidences: Liddel (1951) has reported that respiratory dysfunction is an invariable manifestation of chronic experimental neurosis in animals and often resembles the laboured breathing of bronchial Asthma. Gnatt (1941) produced in an experimentally neurotic dog 'loud raucous expiration accompanied by loud wheezing'. Maserman and Pechtel (1953) reported that several monkeys exposed to an experimental conflictual situation exhibited "Severe asthmatic attacks lasting for hours". It has been observed by Seitz (1959) that a respiratory wheezing condition develops in cats exposed a feeding frustration test. Stein and Ottenberg studied the affect of emotional situation on several asthmatic patients who were being followed in psychotherapy.
A consideration of the role of CNS is of central importance in the study of psychosomatic illness. There is a growing body of information that indicates, that CNS is related to hypersensitivity and especially to anaphylaxis. Freedman and Fenichel (1958) tested the effect of mid brain lesions on the course of anaphylaxis in guinea pig. Bilateral symmetrical lesions in mid brain reticulum at the level of superior colliculus inhibited anaphylactic death. Szentivanyi (1958); Filipp and Szekely (1958) have demonstrated that lethal anaphylactic shock in guinea pig and rabbit can be prevented by bilateral focal lesions of tuberal region of the hypothalamus when other regions of hypothalamus are injured. The protective effect diminishes or disappears proportionately to the distance of the Tuberal area (Szentivanyi, 1958).

The mechanisms by which CNS lesions inhibit the development of anaphylaxis have not been defined yet. The antianaphylactic effect of Tuberal region may be related to neuroendocrine processes. The area of hypothalamus concerned in prevention of anaphylactic death, is also concerned with the control and release of ACTH (Harris, 1948, Hume, 1953; McCann, 1953). ACTH and cortisons have protective action against anaphylactic shock when guinea pig is used as test species. (Humphray, 1951; Hoene, 1952).

A little more than 2 decades ago Dr. M. Murray Peshkin pointed out that the desperately ill children with interactable Asthma were dramatically relieved by separation from their parents (Parentectomy). This also occurred during the height of the pollen season. He found
that pollen therapy may not be necessary even in specifically pollen sensitive (Abramson, 1961).

Weiss et al. (1976) have reported that in patients who watched a motion picture of other patients having Asthma, significant increases in subjective Asthma were observed, and in two instances, chest sound recording revealed wheezing onset during the viewing. The stimulus was a motion picture of several asthmatic children in severe asthmatic distress, and was derived from patients reports that, thinking about their breathing, either because they became concerned with the possibility of getting an attack or because they have seen somebody in distress, could on occasion Trigger or aggravate their symptoms.

Kagan et al. (1976) studied the relationship between certain psychological and allergic variables in childhood Asthma. The prediction was that across patients of equal illness severity, an inverse relationship would be observed between measures of the involvement of emotional symptom precipitants and allergic factors. This relationship failed to emerge at a statistically significant level when the index of emotional precipitants included only such things as worry, anger, upset, excitement etc. and excluded emotionally toned "respiratory behaviours" such as crying or laughing. When crying was included in the index of emotional precipitants the prediction was clearly supported.

Tal et al. (1976) have reported that vividly remembered incidents of intense anger and similarly recalled fear decreased, 1 sec. forced
expiratory rates (FEV₁) in 30 male and 25 female chronic asthmatic children who had no psychopathology. Forced expiratory volume (FEV₁) increased with relaxation.

In nutshell, emotional factors or psychic stress stimulate the cholinergic activity of the autonomic nervous systems leading to bronchoconstriction.

During the periods of mental strain there is unusual stimulation of the ductless glands especially of adrenals, and when the source of anxiety is removed a reaction occurs as a result of temporary exhaustion of these glands, giving rise to asthmatic attack. Diminished secretion of these glands is (Francis, 1950) associated with increased cholinergic activity as a result of vagal stimulation.

There are so many indications that allergic predisposition and vulnerability in respect of psychological factors are related to each other in some unknown fashion. Kagan et al. (1976) have also supported the relationship between certain psychological and allergic variables in children Asthma.

4. Endocrine Factors

The study of ductless glands and their bearing on Asthma has brought about a great changes of thought on the aetiology of the disease. Crushman believed in endocrine factor as a cause of vagal stimulation. No definite hormone was made responsible, but according to him disturbances in any gland with internal secretion might lead to
vagotonia or sympathicotonia (Banszky, 1959).

**Thyroid**: Recently the part played by thyroid gland in the development of Asthma has been brought to force by Heller (1955). He believes that certain influences (stress) on thyroid can cause an over production of hypothalamus hypophysis-adrenal system, with an adverse effect on hypothalamus-Hypophysis-adrenal mechanism. This leads to dimished secretion of cortisol in the body and consequently creates a condition for antigen-antibody reaction.

**Parathyroid**: Alberg et al. (1972) have reported 9 patients with primary-hyperparathyroidism who also had severe Asthma. This incidence is high compared to over all figure in community of about 2%. In 4 of these patients the Asthma improved dramatically after successful parathyroid surgery. This may explain some relation of parathyroid to Asthma.

**Adrenals**: Sympathetic stimulation from any cause such as stress leads to increased secretion of adrenal glands, vice-versa sympathetic stimulation may be induced by increased adrenal secretion. This increased secretion provided protection against Asthma, as reported by Humphrey (1951), Hoen (1952), that ACTH and cortisone have protective action against anaphylactic shock when guinea pig is used as test species. To diminished secretion, or exhaustion of adrenal secretion especially cortisol give rise to an asthmatic attack. Blumenthol (1965) has reported that during the asymptomatic periods the plasma
cortisol levels obtained were all within normal limits and showed a normal diurnal variation. During the periods of asthmatic attacks induced with Ragweed Pollen extracts under controlled conditions, an increase in plasma cortisol levels for all the subjects was noted along with maintenance of normal patterns of diurnal variation.

**Gonads:** Some patients rotate the onset of their symptoms to puberty, menopause, or menstruation. Unger (Diseases of chest) has described that puberty, menstruation, menopause, pregnancy are important predisposing factors. Asthma may be aggravated by approaching menstruation and lessened when flow starts. Pregnancy usually lessens Asthma, although occasionally a patient will have Asthma only during pregnancy, but there is no proof that any endocrine influence can alone cause Asthma.

**Pancreas:** A pancreatic extract isolated by santenoise was found to increase the excitability of the vagus nerve and to rise the blood sugar level.

5. **Infection:** It is common to find that attacks may develop in relation to bronchial infections, either viral or purulent. In some patients the first attack of Asthma appears to follow an acute bronchial infection, and such patients are often categorized as "Infective." Asthmatics, when no other antigen has been identified. It seems probable that in some instances the infective agent is acting as an antigen, while in others it is merely a non-specific irritant (Howell, 1971).
6. Physical and Chemical Irritants: Some irritants, such as Tobacco, smoke, dust, acrid fumes and cold air, and other atmospheric pollutants are often responsible for Asthma. A number of industrial chemicals, for example aluminium solder flux and Toluene diisocynate (TDI), chemical widely used in the plastics industry may induce Asthma. The mechanism is not known. Vishwanathan (1938) has reported that exposure to cold, activates the enzymes and thus leads to release of histamine and other substances. The second view is that it releases protein metabolites for the tissue to which the organism is hypersensitive.

7. Mechanistic Factor: There are mechanistic theories, such as one which suggests that there are trigger zones in the nasopharynx, which can produce attack of Asthma when stimulated, consequently the patient can be made insensitive by operation, cauterization, or local injection.

8. Other Aetiological Factors:

(a) Incidence: The incidence of Asthma has been estimated from 0.5 - 2% of the population (Harve, 1962). Asthma occurs in all areas and all over the world in approximately 0.5% of the population. (Myers, 1959). Data for incidence of Asthma in India is meagre. A recent morbidity survey conducted among central Government servants and their families in Delhi, showed that the annual incidence of Asthma among government servants is 1.81% (1961). A survey conducted in the community project area in Bihar, showed an incidence of 0.98% for Asthma.
From the data given above, it is reasonable to assume that the incidence of Asthma in India is about 1%. (Vishwanathan, 1967). Smith (1974) has reported that the frequency of Asthma, increased over 20 years period, although the rate of increase has slowed.

(b) Age: Asthma may begin at age, but mostly it starts either in childhood or middle age. Asthma frequently develops in first decade of life in approximately 30% of cases, and most of the other cases are evenly spread over the next three decades. It is rare after 5th decade, but it can occur. According to Rackemann (1931) extrinsic type of asthma frequently develops in thee younger group of patients while the intrinsic type is found more in later age.

(c) Sex: Asthma has nearly the same incidence in both sexes, but some authors have mentioned that males slightly predominate (Beaumont), "Early onset" Asthma is slightly more common in males and "Late onset" Asthma in females (Davidson, 1974). Smith (1974) has also reported that Asthma was commoner in boys than girls.

(d) Race: No race is exempted.

(e) Social Status: Social status has no bearing in the development of bronchial Asthma. It is equally found in Urban and Rural dwellers, amongst the rich and the poor, the intellectual and the simple, the educated and the illiterate, high skilled workers and labourers.

But Vishwanathan (1967) has reported that when incidence has been worked out according to salary group, it is interesting to find
that the rate is higher in higher income group than lower income group. In the group of salary from Rs. 1000-1500, the incidence was 31.7 per thousand, whereas in the group with salary ranging from Rs. 75 -149, the rate was 10.6 per thousand.

(f) Occupation : It is also an important factor in the causation of Asthma. Man working under circumstances, having more dust e.g. stone workers, where the man may remain exposed to organic dust for a long time, and may develop hypersensitivity to that particular type of dust e.g. printers Asthma etc.

(g) Climate : A definite role is played by climate in the occurrence and acuteness of Asthma.

Fein et al. (1965) have reported that a falling barometric pressure and an increasing humidity are often associated with increased frequency and severity of asthmatic attacks. Dry climate is better for asthmatic patients, as the incidence of disease is low there.

(h) Season : A marked increase in the incidence has been noted in the seasons when pollination of plants starts. In India Basant (spring), starting of summer, falling of rainy season or starting of winter season, are the main seasons for asthmatic attacks. Change of temperature also influences the occurrence of attack, as a rising temperature stimulates pollen production, and wheeze, cold weather depress it. (Fein et al., 1965).

(i) Undue Fatigue : It is also a frequent cause of asthmatic
attack or it can evoke the asthmatic attack during the intervening period of the attack.

Jones et al. (1963) have reported that the characteristic asthmatic response to exercise is bronchodilation after a brief exercise, followed by bronchoconstriction after a prolonged exercise. The mechanism of bronchoconstriction is not yet known, but it is prevented by sympathomimetic amines (Jones et al., (1963) and by disodium cromoglycate (Davies, 1968). Tinkelman et al. (1976) have reported that the parasympathetic nervous system has a role in the mediation of exercise induced bronchospasm in children, which can be blocked by atropine.

**PATHOGENESIS**

Variable narrowing of the peripheral airways (bronchoconstriction) is due to one or all the following:

2. Oedema of bronchial mucous membrane.
3. Mucous within the bronchial lumina.

The pathogenesis of bronchoconstriction is not fully understood, while it is known that various substances such as histamine, bradykinin, slow reacting substance (SRS-A) and 5-hydroxytryptamine are liberated, there are other yet unidentified substances which are released in bronchial wall probably from mast cells too. All of these agents both known and unknown cause bronchoconstriction and their relative
importance is uncertain. Many factors appear to be responsible either
directly or indirectly for the release of these mediators, amongst which
are exercise, allergy, infection, but the actual mode of release is
conjectural. Other factors for instance psychological or pharmacological
may potentiate bronchoconstriction. (Price, 1973).

Schild and associates (1951) have shown that the bronchial
muscle excised from the asthmatic patient release histamine and
contracts when exposed to the specific antigen, and this contraction
is not inhibited by atropine. This suggests that histamine is important
in between the immunologic and physiologic reaction.

The identification of reaginic immunoglobulin (IgE) by Ischizeka
(1967a) has unfolded the pathogenesis of atopic skin reactions as well
as extrinsic Asthma.

The airway bronchial tree is under the control of autonomic
nervous system. Cholinergic stimulation causes constriction and adrenergic
stimulation dilation. Cholinergic control of airway is exerted via vagus.
Adrenergic influence is exerted primarily through circulating
Catecholamines. Sympathetic innervation to the bronchi is sparse and
directed chiefly to the cholinergic ganglia, whereas sympathetic
stimulation inhibits ganglionic transmission and relieves and
bronchoconstriction produced by vagal stimulation (Cabezas et al.,
1971)

The exaggerated bronchial activity in asthmatics can be
demonstrated by inhalation of pharmacologic agents, such as histamine or methacholine in doses which will not induce bronchospasm in nonasthmatic individuals. (Parker et al. 1965).

The basic abnormality in bronchial Asthma is thought to be in the adrenergic receptor site within bronchial tree (Szentivanyi, 1968). Considerable evidence of reduced beta receptor response in asthmatic patients has accumulated. The epinephrine induced rise in blood sugar and lactate is reduced (Cookson et al., 1963; Kirk Patrik et al., 1967; Lockey et al., 1967; Middleton et al., 1968) eosinopenia is less (Reed et al., 1970) and urinary excretion of cyclic adenosin monophosphate (C AMP) is less (Bernstein et al., 1972).

The cholinergic and adrenergic abnormalities have a reciprocal relationship, and the degree of severity of the two abnormalities in a series of patients was correlated (Makiine et al., 1970). The bronchoconstriction effect of cholinergic stimulation is prevented by adrenergic agonists. Conversely the vasoconstrictive effect of beta blocker propanolol is prevented by atropine. (Grieco et al., 1971).

Yet another postulation is raised by Grieco (1970), could Asthma represent a disease that involves a lesion at the level of cyclic adenosin monophosphate in bronchial smooth muscle? The ultimate messenger of many harmones in the formation of cyclic AMP from Adenosine Triphosphate (ATP). Defective formation of cyclic AMP might block the normal release of Catecholamines which help to maintain bronchodilation.
Thus the asthmatic patient would be rendered vulnerable to attack of bronchoconstriction.

In nut-shell, allergy, exercise, emotional factors, respiratory infection, aspirine, cold air, dusts and fumes, the precipitating factors of bronchial Asthma, stimulate the cholinergic activity and produce bronchoconstriction.

Fanbury (1973) has reported that it is now known that the lung has ability to synthesize and metabolise prostaglandins. F₂-alpha is the predominant prostaglandin in the lung, and those of E. series have also been indentified. Generally prostaglandin of F series appears to constrict the bronchial muscle and vascular smooth muscles, whereas the dose of E series tends to relax the muscle. It has been demonstrated that airway resistance in man can be influenced markedly by prostaglandin. The potential role of these compounds in the pathogenesis of bronchospasm as well as their possible therapeutic benefit need further evaluation.
REVIEW OF AYURVEDIC LITERATURE

NIRUKTI OF SHVASA

The word SHVASA is derived from the root SHVAS meaning by, to breathe or to respire. Thus it is used to describe the respiration both physiological and pathological. In the later it has been defined in Sanskrit Hindi Shabdkosha as:

"BHOOYO BHOOYO SHVASITI YASMIN ROGE SAH SHVASA" That, a pathological condition having rapid respiration is known as Shvasa. Vijayrakshita has furnished a quotation in the Madhu-kosha commentary on Madhava Nidana, describing the features of Shvasa roga. "SHVASASTU BHASTRIKADHMANASAMVATORDHVA BAMITA". With this description it can be differentiated from increased rate of respiration in physiological states.

Shvasa as a disease entity:

Shvasa or difficult breathing may be an independent disease, or symptom as well, in so many diseases. Charaka has categorically described that, when the symptoms are independent, requiring independent treatment, they should be considered as disease entities. On the other hand, as long as they are dependent on other diseases, and are relieved by the treatment of the original disease, requiring no separate treatment, they should be considered as symptoms (Ch. Nid. 8/40). Hence Shvasa may be a symptom when associated with other diseases, and when it is independent, it is considered as disease entity.
called Shvasa Roga.

Shvasa is found as a symptom in the following disease Jwara, Sannipatajwara, Rajyakshma, Vatik Gulma, Jalodara, Plehodara, Budhodara, Arsha, Pandu, Vatik Grahani, Kshataja, Bisarpa, Madatya, Ashmayaka Drita Nruha, Upātnraka, Hridya-Bighata, Bastikundal, Mutraghata, Gambheera Hikka (Charaka).

Vatika Grahani, Kamala, in terminal stages of oedema, throat, nasal and Eye diseases, Bisha Roga, due to improper use, of Panchkarma, Sternous exercise, in old days due to exertion, Udavarta, Obesity etc. (Sushruta).

SHVASA AS A COMPLICATION

Shvasa has been described as complication in the following disease:

Masurika (small pox), Vataj Prameha, Chhardi, Jwara, Atisara, Yakshma, Gulma, Raktpitta, Bisarpa, Kumbha Kamala, Odema, Bidradhi etc.

CLASSIFICATION OF SHVASA ROGA

Generally the classification of diseases in Ayurveda is proposed on the basis of Doshika involvement, but occasionally we find them on the basis of aetiology, pathology and clinical features, as well. Here is the example where the classification of Shvasa has been proposed on the basis of clinical features, which is as follows.
1. Maha Shvasa

2. Urdhava Shvasa

3. Chhinna Shvasa

4. Tamaka Shvasa

5. Kshudra Shvasa.

1. MAHA SHVASA:

The person in whom the expiratory movement of Vata is aroused, is greatly afflicted, and being obstructed in his respiration, breathes incessantly with a loud and long stertor like an intoxicated bull. He loses all the sense of knowledge and understanding; his eyes are restless; his face gets distorted; his urine and faeces get constipated; his voice is weakened; he gets into moribund state and his intensely hurried breathing is noticeable even from a distance. A person afflicted with such Maha Shvasa, will indeed succumb to it soon.

Sushruta (Su. U. 51/12) says that unconscious patient with pleurodynia, dried throat (dehydrated) and swollen eyes, respires with high pitched stridor folding his body like "V" is a patient of Maha Shvasa. This type of condition is found in various pathological conditions of heart, kidney and brain.

2. URDHAVA SHVASA

That condition is known as expiratory dyspnoea where expiratory
phase is prolonged, while the inspiratory phase is insignificant. The mouth and respiratory tract are obstructed by mucus; the patient is greatly afflicted with his provoked Vata; his eyes are turned upwards; he is oblivious to his surrounding (Coma Vigil), and his gaze is restless, moving hither and thither. Afflicted with pain, he passes into a stupor, his mouth is parched, and he is listless, and in great distress. His expiratory process being excessively provoked and the inspiratory process obstructed, the patient suffers delusion and fainting. This condition of expiratory dyspnoea soon takes away the patient's life. This can be correlated with stertorous breathing or failing respiration, which is found in congestion consolidation pneumonia, abscess, gangrene and infarct of lung, and appoplexy and coma.

3. CHHINNA SHVASA:

In this condition, the patient being afflicted in all his vital breaths, breathes with interruptions, or ceases to breathe altogether (Apnoea), and is in great distress and afflicted with pain as if his vital parts have been undered. He is afflicted with constipation, sweats and fainting, burning and retention of Urine; his eyes are filled with tears; he is greatly emaciated; while struggling for breath his eyes become excessively injected, he is unconscious; his mouth is dry; he becomes delirious; a man who is thus broken down with interrupted breathing (Cheyne stroke's respiration) soon abandons his life. Such condition
is found in uraemic coma, morphine poisoning, and in infants normally.

4. TAMAKA SHVASA

When Vata becomes Reverse in its course, reaches the Srotasa, involves the neck and head, increases the mucous secretion, and produces coryza. Obstructed by coryza, it produce a variety of dyspnœa associated with wheezing sound and painful respiration. Due to acute paroxysmal dyspnœa, patient becomes sad, coughs, and becomes motionless. Due to constant coughing, patient gets fainted frequently. Due to inability to expectorate, patient is greatly distressed, and on the sputum being expectorated he feels comfort for a while. His throat is badly affected, and he can speak hardly. Embarrassed by dyspnœa patient can not be down, nor he is unable to get a sleep, because Vata presses upon both his sides while lying flat on bed. Patient feels ease or comfort in sitting or propped up positions. He likes to take hot things only. His eyes are swollen, forehead is covered with sweat, and he feels a great distress all the times. His mouth becomes dry and respiration like bellows. These paroxysms are intensified by cloudy, humid, and cold weather, easterly wind and by taking Kapha increasing things. The Tamaka Shvasa (Bronchial Asthma) is palliable. It is curable if it be of recent origin.

5. Kshudra Shvasa:

Due to excessive use of dry things, exertion, there takes place
a minor disturbance of Vata in the alimentary tract, which causes a minor dyspnoea. This dyspnoea does not afflict the body like other varieties of dyspnoea. It does not interfere with the normal course of food or drink, does not pain the sense organs and does not give rise to any other disease. It is curable. This may be correlated with physiological dyspnoea.

SANTAMAKA AND PRATAMAKA SHVASA

Two allied conditions Santamaka and Pratamaka have been considered along with Tamaka Shvasa. They are allied because the clinical manifestations are very much common. However, a few differential diagnosis, as follows:

Pratamaka: A patient suffering from Pratamaka Shvasa is overwhelmed by fever and fainting, in addition to other symptoms of Tamaka Shvasa. It is caused by Udhavarta, dust, indigestion, old age and due to suppression of urges. Chakrapani is of the opinion that, though the Kapha and Vata are predominant doshes in Tamaka Shvasa, Pitta is equally vitiated in this allied condition, which is responsible for fever. Acute bronchitis and lung infection as in lung abscess, dyspnoea is a predominant symptom, but it is associated with fever. So such conditions may be diagnosed as pratamaka Shvasa.

Santamaka Shvasa: Santamaka Shvasa is aggravated at the night, and patient feels relief with cold, which is contrary to Tamaka Shvasa,
as the later is aggravated with cold and usually the attacks are precipitated early in the morning. It is known as Santamaka Shvasa because the patient feels to be drowning in the sea of darkness. This clinical feature is very much skin to cardiac Asthma.

On the above clinical basis, the Pratamaka and Santamaka Shvasa could be differentiated from Tamaka Shvasa.

Nidana (Aetiology)

Nidana is a factor which helps in the causation of this disease. It may be specific when one particular factor cause some disease or it may be non specific, when one factor produces several diseases, depending upon other factor, or several factors contribute to the development of one disease. This view of Charaka still holds good today. Many of the bacterial disease have specific cause, on the other hand several metabolic diseases are of multi-aetiological origin. Tamaka Shvasa (Bronchial Asthma) belongs to the later group.

Aetiological factors have been classified by the ancient physicians in several ways and a very good collection has been furnished by Vijayaraksheeta, in the Madhukosha commentary on Madhava Nidana in the chapter dealing with Nidana. Among these classifications, the first one - Asatmendriyartha Samyoga., Pragyapradha, and parinama, have been discussed by Charaka. Accordingly Atiyoga (excessive indulgence), Ayoga (non-indulgence), and Mithya yoga (non-homologous
contacts of five senses), are the most important causes of all the diseases. Through the sensory organs, Shareerika doses - Vata, Pitta, Kapha and Manasa doses - Raja and Tama are affected. As long as our sensory organs are in homologous contact, they remain within normal limits, but when they are exposed to Ayoga, Ati-yoga, Mithya-yoga, Shareerika and Manasika doses are vitiated, producing all the minor and major diseases of physical or mental origin. In addition, the variation in time, both Nityaga and Avasthika, including elimatic changes, have also influence on the doshika level of the body, producing, precipitating or aggravating the lesions. Some of the diseases may be purely of the mental origin due to abnormal functioning of mind, leading to behavioural changes which may ultimately cause physical as well as mental disease.

As far as Tamaka Shvasa is concerned, all the three types of aetiological factors may be responsible in its causation. In nut-shell, Tamaka Shvasa has multi-dimensional aetiology. Further, both the causative factors (Anupashaya) and Therapy (Upashava), have been classified into - Aushada, Anna, and Bihara. And again, Vijayarakshita has furnished a long commentary with examples, on Upashaya in the Madhukosha commentary on Madhava Nidana. Similarly, the classification of Anupashaya can be developed on the same lines. Here an attempt has been done to classify the aetiological factors described in the texts, under several headings, as follows:
Table - 1

NIDANA (AETIOLOGICAL FACTORS) OF SHVASA ROGA

A. AHARA (Dietic Causes)

(i) Dosha Prakopa Shara:

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Ahara</th>
<th>Reference</th>
<th>Dosha Vitiated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Rūksha Anna (Dry and rough diet)</td>
<td>Ch., Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>2.</td>
<td>Jalajapishta (Wet flesh)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>3.</td>
<td>Anupa pishta (Aquatic flesh)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>4.</td>
<td>Dadhi (Excessive use of curd)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>5.</td>
<td>Sheetambu Sevana (use of cold water)</td>
<td>Ch., Su.,</td>
<td>Vata</td>
</tr>
<tr>
<td>6.</td>
<td>Kaphaja ahara (Kapha increasing things)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>7.</td>
<td>Masha (Black gram)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>8.</td>
<td>Nishpava (Bean)</td>
<td>Ch.</td>
<td>Vata</td>
</tr>
<tr>
<td>9.</td>
<td>Pishtanna (Paste Preparations)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>10.</td>
<td>Guru bhojana (Heavy diet)</td>
<td>Ch., Su.</td>
<td>Kapha</td>
</tr>
<tr>
<td>11.</td>
<td>Ama Kshira (Unboiled milk)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>12.</td>
<td>Shaluka (Lotus Rhizome)</td>
<td>Ch.</td>
<td>Vata, Kapha</td>
</tr>
<tr>
<td>13.</td>
<td>Abhishyandhi bhojana (Liquidfacient diet)</td>
<td>Ch., Su.</td>
<td>Kapha</td>
</tr>
</tbody>
</table>
### (ii) Vyadhi Prakopaka Ahara

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Ahara</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Bisha Sovana (Toxin s)</td>
<td>Ch., Ash., Hr.</td>
</tr>
<tr>
<td>2.</td>
<td>Ama dosha (Defective digestion)</td>
<td>Su., Ash., Hr.</td>
</tr>
<tr>
<td>3.</td>
<td>Abhisheyandhi shara (Liquifacient diet)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>4.</td>
<td>Pinyaka (Tila paste)</td>
<td>Ch.</td>
</tr>
<tr>
<td>5.</td>
<td>Tila Taila (Tila oil)</td>
<td>Ch.</td>
</tr>
<tr>
<td>6.</td>
<td>Ama Kshira (unboiled milk)</td>
<td>Ch.</td>
</tr>
<tr>
<td>7.</td>
<td>Kaphaja ahara (Kapha increasing things)</td>
<td>Ch.</td>
</tr>
<tr>
<td>8.</td>
<td>Guru bhojana (Heavy diet)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>12.</td>
<td>Adhyashana (Frequent meals)</td>
<td>Su.</td>
</tr>
<tr>
<td>13.</td>
<td>Dadhi (curd)</td>
<td>Ch.</td>
</tr>
</tbody>
</table>

### (iii) Ubhaya Prakopaka Ahara

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Ahara</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Ama Kshira (unboiled milk)</td>
<td>Ch.</td>
</tr>
<tr>
<td>2.</td>
<td>Kaphaja ahara (Kapha increasing things)</td>
<td>Ch.</td>
</tr>
<tr>
<td>3.</td>
<td>Guru bhojana (Heavy diet)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>4.</td>
<td>Abhisheyandhi ahara (Liquifacient diet)</td>
<td>Ch., Su.</td>
</tr>
</tbody>
</table>
### A. BIHARA

(i) Dosha Prakopaka Bihara.

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Bihara</th>
<th>Reference</th>
<th>Dosha Vitiated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Sheeta Sthana Sevana (To reside in cold climate)</td>
<td>Ch., Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>2.</td>
<td>Sheetasana (cold seat)</td>
<td>Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>3.</td>
<td>Vyayama (Excessive Exercise)</td>
<td>Ch., Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>4.</td>
<td>Gramya Sevana (over indulgence in sex)</td>
<td>Ch., Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>5.</td>
<td>Adhva (Excessive walking)</td>
<td>Ch., Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>6.</td>
<td>Kaphaja bihara (Kapha increasing measures)</td>
<td>Ch.</td>
<td>Kapha</td>
</tr>
<tr>
<td>7.</td>
<td>Apatarpana (malnutrition)</td>
<td>Ch., Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>8.</td>
<td>Mootradivegbidharana (Suppression of urges)</td>
<td>Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>9.</td>
<td>Bhara (Excessive weight lifting)</td>
<td>Su.</td>
<td>Vata</td>
</tr>
<tr>
<td>10.</td>
<td>Shudhatiyoga (Excessive use of emetics and purgatives)</td>
<td>Ch.</td>
<td>Vata</td>
</tr>
</tbody>
</table>
## (ii) Vyadhi Prakopaka Bihara

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Bihara</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Gramy a Sevana (over indulge in sex)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>2.</td>
<td>Adhva (excessive walking)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>3.</td>
<td>Apatarpana (malnutrition)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>5.</td>
<td>Dvandhayoga (mutually antagonist procedures)</td>
<td>Ch.</td>
</tr>
<tr>
<td>6.</td>
<td>Vayu (air)</td>
<td>Ch., Ash., Hr.</td>
</tr>
<tr>
<td>10.</td>
<td>Urasa Pratighata (chest trauma)</td>
<td>Ch.</td>
</tr>
<tr>
<td>11.</td>
<td>Kanta Pratighata (Throat Trauma)</td>
<td>Ch.</td>
</tr>
<tr>
<td>12.</td>
<td>Marmaghata (Trauma of vital organs)</td>
<td>Ch., Ash., Hr.</td>
</tr>
</tbody>
</table>

## (iii) Ubhaya Prakopaka Bihara

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Bihara</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Gramya Sevana (over indulge in sex)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>2.</td>
<td>Adhva (excessive walking)</td>
<td>Ch., Su.</td>
</tr>
<tr>
<td>3.</td>
<td>Apatarpana (malnutrition)</td>
<td>Ch., Su.</td>
</tr>
</tbody>
</table>
### C. NIDANARTHAKARA ROGA (Causative disease)

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Disease</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Anaha (chronic constipation)</td>
<td>Ch.</td>
</tr>
<tr>
<td>2.</td>
<td>Daurbalya (debility)</td>
<td>Ch.</td>
</tr>
<tr>
<td>3.</td>
<td>Atisara (Diarrhoea)</td>
<td>Ch., Ash., Hr.</td>
</tr>
<tr>
<td>4.</td>
<td>Jwara (Fever)</td>
<td>Ch., Ash., Hr.</td>
</tr>
<tr>
<td>5.</td>
<td>Chhardi (Vomiting)</td>
<td>Ch., Ash., Hr.</td>
</tr>
<tr>
<td>6.</td>
<td>Pratishyaya (Coryza)</td>
<td>Ch.</td>
</tr>
<tr>
<td>7.</td>
<td>Ura Kshata (Lung trauma)</td>
<td>Ch.</td>
</tr>
<tr>
<td>8.</td>
<td>Dhatu Kshya (Emaciation)</td>
<td>Ch.</td>
</tr>
<tr>
<td>9.</td>
<td>Rakta Pitta (Bleeding syndrome)</td>
<td>Ch.</td>
</tr>
<tr>
<td>10.</td>
<td>Udavarta (Chronic abdominal distension)</td>
<td>Ch.</td>
</tr>
<tr>
<td>11.</td>
<td>Vishuchika (Acute gastroenteritis)</td>
<td>Ch.</td>
</tr>
<tr>
<td>12.</td>
<td>Atasaka (Intestinal Treper)</td>
<td>Ch.</td>
</tr>
<tr>
<td>13.</td>
<td>Pandu (Anaemia)</td>
<td>Ch.</td>
</tr>
<tr>
<td>14.</td>
<td>Vivandha (Constipation)</td>
<td>Ch.</td>
</tr>
</tbody>
</table>

From the review of the above table, it is evident that it includes several factors which are proposed today to trigger the pathogenesis of bronchial Asthma by including or precipitating antigen-antibody reaction, for which the stage is already ready. However, a great emphasis has been given to different kinds of foods and recipies. This type of description is not available in modern medicine, which deserves consideration for further research. The avoidance of these factors may...
lead to a better management than available today.

Primarily, there are two components of Samprapti, dosha and dushya. The interaction between dosha and dushya in a particular site, produces a group of clinical features recognised as a particular disease. Among the two, doshas are considered as Hetu or cause, and the factors provoking them are known as Hetuprakopaka. On the other hand aetiological factors affecting the dhatus, or particular Adhishthana - Sarotasa or organ, are considered as vyadhi prakopaka, because the provocation of a dosha may lead to a disease of any Srotasa or organ, depending upon the organ sensitivity, but the Bisheshana of organ, tissue or Srotasa; lead to a specific group of symptoms. Hence they are known as vyadhiprakopaka.

Samprapti (Pathogenesis)]

It has been commonly agreed by all the authorities that Shvasa Roga (difficulty in respiration) is caused by the obstruction of Vayu in the Pranavaha Srotasa (lungs) i.e. obstruction in the free air flow within and without. This obstruction is because of vitiation of Pranavayu, as well as by the over secretion of Kapha. As discussed earlier, the precipitation of paroxysmal attack of bronchial Asthma is due to spasm in the bronchi, or due to swelling of the mucous membrane and mucous secretion, or both the factors may be partly responsible. Thus there is a great similarity in the ancient concept and the modern concept,
regarding the pathology. Here the broncho-spasm is contributed by vitiation of prana-vayu, and the swelling of the mucous membrane alongwith secretion of mucous are due to vitiation of Kapha, of course the site of lesion is pranavaha Srotasa.

Samprapti of Shvasa has been furnished by Charaka in three states:

1. First of all the common factors involved in the patho-genesis of Hikka and Shvasa, have been described. In this context, Charaka has emphasized that Kapha and Vata are the chief Doshas involved. Of course, the disease has its root in Pittasthana. Chakrapani is of the opinion that Amashaya (stomach) may be referred as Pittasthana in this context. It is a highly interesting observation. Accordingly, though the Shvasa is a disease of Pranavaha Srotasa, it has much to do with the gastrointestinal tract. It emphasized that whatever attempt is done to relieve the symptoms by the drugs acting on respiratory system, the disease is not likely to be cured, unless and until the gastrointestinal tract is properly treated, from where the disease has beginning.

It may be relevant to record that embryologically, the stomach and lungs, both develop from the same bud and only in the process of growth they are divided into two parts, and the two organs are lodged in two cavities, but still their responses and nature continue to remain identical, for example when an emetic is administered, it irritates the
gastric mucosa and simultaneously stimulates the mucosa of respiratory system causing expectoration, and vice-versa, when an expectorant is administered in large doses, it induces emesis. In pathological conditions as well, they behave in a similar fashion. It is reported that the patients of bronchial Asthma, do suffer from achlorhydria or hypochlorhydria i.e. when the respiratory mucosa is sick, gastric mucosa is also affected.

The idea of derangement of Pittasthana may be further extended to liver as well. It is reported that BSP test is positive in cases of bronchial Asthma, which shows that the liver function does not remain normal in patients of bronchial asthma. It may be a primary cause as well, in the pathogenesis.

Charaka has further mentioned that in Shvasa Roga, the pathogenesis is not limited to the respiratory tract and gastrointestinal tract only. He is of the opinion that the heart is also affected in this disease. At the outset, nutrition of the heart is affected, as Rasa and Rakta are not available at the optimum dose, and at a later stage there may be structural damage when other dhatus (Mamsa) are affected. That is why, even in the natural course, the disease becomes interactable. The effect of bronchial asthma on pulmonary circulation, and development of corpulmonale is well recognised entity in the natural history of the disease. Many of the patients suffering from asthma, succumb due to this complication. Regarding the mechanism of
precipitation of Shvasa and Hikka, Charaka is of the opinion that Vayu situated in the pranavaha Srotasa is vitiated at first, which leads to further vitiation of Kapha in the chest, leading to obstruction, and clinical manifestations. Thus, though the Vata and Kapha are primarily involved in its pathogenesis, the involvement of Vata is prior, and dominant. The role of Vata and Kapha has already been discussed.

2. At the second stage, a common Samprapti for all the types of Shvasa Rogas has been described. Charaka is of the opinion that when the Vata associated with Kapha, obstructs the air passages, the airflow within and without is obstructed, leading to clinical manifestations of asthma. Chakrapani has commented that the obstruction is both at the level of Pranavaha Srotasa and Udakavaha Srotasa. Of course it is to point out that along with the respiratory difficulty. There is also derangement in the circulation of liquids of i.e. Rasa, Udaka being the chief constituent of Rasa.

3. At the third stage, Charaka has offered a special Samprapti in the development of Tamaka Shvasa. At the outset, he has mentioned that Vayu is Pratiloma in Tamaka Shvasa, probably it is to indicate that there is obstruction in the air flow, especially in the expiratory phase. He has also mentioned that in this disease, the head-neck is also involved. It probably refers to the action of accessory muscles of respiration, attached in the region of head-neck. He has further mentioned that there is secretion of Kapha, which interferes in the
free flow of air, giving rise to Ghura Ghyraka Dhwani i.e. wheezing sound, and crepitations heard on auscultation. To combat the situation, the rate of respiration is highly increased, although it is very painful to the patient.

Thus in the nutshell, here Charaka has tried to describe the mechanism of respiration during an asthmatic attack producing cardinal signs and symptoms of the disease.

Poorva Roopa (Prodromal symptoms)

In the sequence of pathogenesis, when the vitiated dohas being to localize, affecting a particular organ or system, certain prodromal symptoms are observed before the full-fledged manifestation of the disease. As mentioned elsewhere, bronchial asthma is paroxysmal in nature. The clinical features of bronchial asthma are the result of broncho-constriction, which is believed to be due to preponderance of cholinergic mechanism over the adrenergic influence. The attempt of the body is to enhance the adrenergic mechanism, as far as possible, but as soon as the cholinergic supervenes the symptoms precipitate. Before this critical point is reached, there is a keen competition in the body humors, which may be considered as doshas or their influence. Their level is above the physiological range, so there are likely to be certain symptoms of course, not of the disease which is produced after that critical juncture. The prodromal symptoms described by authorities are as follows.
By the review of the above table, it is obvious that all the authorities have emphasized a few symptoms in common i.e. constipation, precordial pain, and pain in chest. Of course a few constitutional symptoms have been added by Sushruta. The basic prodromal symptoms constipation, precordial pain, pain in chest, can be very well explained in enhanced adrenergic activity. The relaxation of smooth muscle of intestine, may lead to constipation, and the enhanced cardiac activity may precipitate the precordial/chest pain. The other symptoms - Arati, Vairasya and headache, may be the result of these very basic
changes. A man having constipation, is likely to develop distaste or bad taste of mouth, headache etc.

CLINICAL FEATURES OF TAMAKA SHVASA

A. Features related to Respiratory system :

1. Peenasa (coryza)
2. Kasa (severe cough)
3. Teebra Shvasa (Rapid respiration)
4. Pratiloma vayu (prolonged expiration)
5. Pranaprapidaka Shvasa (severe dyspnoea)
7. Kasa Janya Pramoha (Fainting while coughing)
8. Shleshma amuchya ganya Dukhah (Distress due to inability to expectorate).
12. Muhum Shvasa muhum abdhamyate (status Asthmaticus)
13. Krichha bhashana (Hoarseness of voice)
14. Kanthodhvansa (laryngitis)
15. Shayanaya parshava Avgrahana (pressure on both sides of chest).
17. Aaseeno labhate saukhyama (Orthopnoea)
B. Constitutional Features :
1. Bhrishama arati (severe discomfort)
2. Nidra alabha (Insomnia)
3. Lalata Sveda (Sweating of fore-head)
4. Uchhirta Akshi (wide open eyes)
5. Bishushkasya (dryness of mouth)
6. Pratamyata (Forward bending)

C. Other Features :
1. Ushna upashaya (relief by taking hot things)
2. Meghaich abhivardhate (Precipitated by clouds in sky)
3. Ambubhisch abhivardhate (precipitated by rains)
4. Sheet Vataiha Abhivardhate (Precipitated by cold waves)
5. Pragvataiha abhivardhate (Precipitated by easterly winds)
6. Shleshmalaish abhivardhate (Precipitated by Kapha increasing measures)

It is evident from the above described features that all the cardinal signs and symptoms of bronchial asthma have been nicely included in the description of Tamaka Shvasa. Prolonged expiration, rapid breathing, wheezing, hoarseness of voice, and visible action of the accessory muscles of respiration, are the main signs included. Rest of the symptoms like dyspnoea, orthopnoea, cough, and its precipitation with climatic changes, are the true symptoms of bronchial asthma. With this description there can not be any doubt that the identity of bronchial asthma was not well known to the ancient physicians. They have also
made an attempt to give the pathogenesis of signs and symptoms as far as possible. As far as Doshika relation is concerned, all the signs and symptoms are either due to the Vitiation of Vata or Kapha.

Sadhyasadhyata (Prognosis)

As a whole, Hikka and Shvasa have bad prognosis. Usually they develop in the terminal stage of the disease, so they are considered to be very difficult to be cured. Of course among the five varieties of Shvasa i.e. Maha Shvasa, Urdhava Shvasa, Chhina Shvasa, Tamaka Shvasa, and Kshudra, the former three are incurable and Kshudra Shvasa is easier for the management. As far as Tamaka Shvasa is concerned, in the opinion of all the authorities, it is palliatable only i.e., symptoms can be relieved by proper treatment. Only in the initial stage of the disease and in those who are well build, it can be expected to be cured. With the prolongation of the illness the patient is likely to develop Dhatukshaya, which may be generalized or localized as well in Pranavaha Srotasa, and the heat is also likely to be involved as discussed earlier, then the disease becomes incurable.

CHIKITSA SUTRA (PRINCIPLES OF TREATMENT)

It has been emphasized by Charaka that all the patients of Shvasa should be classified in four groups, for the purpose of management.

1. with good built

2. with poor built
3. Having predominance of Vata

4. Having predominance of Kapha

Among them those having good built and predominance of Kapha, should be treated first with Shodhana therapy i.e., Vamana and Virechana, followed by palliative treatment and dietetic management. On the other hand, the other group with poor built and having predominance of Vata should not be treated with Shodhana therapy. They should be managed with the drugs having alleviative property for Vata. They should be kept on nourishing diet containing soup and fat. The children and old people should also be managed on the same lines.

(Ch. Chik. 17/88-90).

If there is excess of cough with expectoration, and laryngitis, suitable patient may be treated with emetics. On the other hand, the patients of bronchial asthma (probably having dyspnoea as a predominant symptom) should be managed with purgatives.

As far as antidoshika treatment is concerned, all the drugs and diet having alleviative property against Kapha and Vata hot and carminatives, are useful for the patients of bronchial asthma. However, one should not use exclusively drugs that belong to either of the two groups viz., those which alleviate Kapha but aggravate Vata, and those which alleviate Vata but aggravate Kapha. If one has to choose between the two, the drugs alleviative of Vata are to be preferred.
The side effects of nourishing diet and palliative drugs are less and if any, are easily correctable. On the other hand, the ill effects of reduction diet and drugs are many and interactable. (Ch. Chik. 17/149). Hence as a rule the patients of bronchial asthma should be treated with the palliative drugs, and nourishing diet, whether those persons have undergone preliminary purificatory measures or not.
DRUG REVIEW

ALBIZZIA LEBBECK BENTH. (SIRIS)

From the review of the literature, it seems, it is an important plant of the Indian medicine and has been included in all the books of Ayurveda—classical, medieval, and modern age, dealing with herbs and medicaments. It is and for the treatment of skin diseases. Regarding the route of administration, it has been used both for external and internal application. As far as its use in bronchial asthma is concerned, Charaka was first to point out the use of expressed juice of its flowers in the treatment of Shvasa. However, much diversity is observed in the realm of Ayurvedic literature, regarding its properties and uses.

CLASSIFICATION:

Being a tree, it has been classified in Salsaradigana (Sushruta), Prabhadradi gana (Raj Nighantu), Vatadi varga (Shava Prakash Nighantu), Banoushadi Varga (Amarkosha) and Asnadigana (Ashtanga Hridaya).

On the basis of its properties, it has been classified Kashey-Skandha, Vedana Sthapana, and as a best Vishaghna dravya (Charaka), and Shirovirechana dravya (Ashtang Hridaya).

Modern writers on drugs prefer to classify the plants and herbs according to their natural order, hence they have included it in shimbi varga and Babuladi varga, because it belongs to the family of Leguminasae, and sub-family-mimosacoao.

SYNONYMS:

More than 20 Sanskrit synonyms of this plant are available in
ALBIZZIA LEBBECK BENTH. (SIRIS)

BARK
ALBIZZIA LEBBECK BENTH. (SIRIS)
Ayurvedic literature, out of which 17 have been furnished by Raj Nighantu. It is notable that authors of different Nighantus have tried to furnish the number of synonyms, as much as possible, as they provide information regarding the characteristics of the plant. No separate botanical description has been given by them. Available synonyms are as follows :-

1. **Shireesha** : It is called Shireesha as it destroys the diseases, or because the flowers are very fragile (Amarkosha).

2. **Kapitana** : As it is a glabrous tree, or because it is of monkey colour (Amarkosha).

3. **Bhandila**  
4. **Bhandika**  
5. **Bhandira**

6. **Bhandi** : As this plant offers protection against several diseases.

7. **Sheet pushpa**  
8. **Supushpaka**  
9. **Britta pushpa**.

10. **Lomash pushpaka**  
11. **Madhur pushpaka**  
12. **Mridhupushpaka**.

13. **Bahirpushpa** : These synonyms have been used to describe the characteristics of the flowers e.g. size, shape, nature, scent, qualities and taste etc.

14. **Shukeshta**  
15. **Shukpushpa**  
16. **Shukpriya**  
17. **Shuk Taru**  
18. **Shukbriksha** : These synonyms indicate that Shireesha is very much liked by parrots.

19. **Bishhnata** : As it counteracts the several poisons.

20. **Udhyanaka** : Because Shireesha is a garden plant.

21. **Shyamala** : Because there is a variety called Krishan Shireesha.

22. **Shankhini phala** : As the colour of the pod is like conch.
ALBIZIA LEBBECK BENTH. (SIRIS)
23. **Kalinga**: Probably it grows in abundance in Kalinga Desha.

24. **Plavga**: It means a monkey, as it is of monkey colour.

25. **Bipra**: As it looks like a Bipra, when all the leaves are fallen, and pods remain on the tree.

### Name in Different Languages:

<table>
<thead>
<tr>
<th>Language</th>
<th>Name</th>
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<tbody>
<tr>
<td>Sanskrit</td>
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<td>Guna</td>
<td>Laghu, Ruksh and Teekshna.</td>
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<tr>
<td>Veerya</td>
<td>Sheeta (some authors), Anushana (by others).</td>
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<tr>
<td>Vipaka</td>
<td>Katu</td>
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<tr>
<td>Effect on Doshas</td>
<td>Tridosha Shamaka.</td>
</tr>
<tr>
<td>Habit</td>
<td>Found throughout India, upto 1200 m. in Himalayas. The plant is also cultivated.</td>
</tr>
</tbody>
</table>

**Parts Used**: Bark, leaves and seeds have been usually recommended for use. Seeds have been specially recommended for nasal administration.

**Properties and Uses**: It is the best Vishaghna dravya, known in Ayurveda.
(Charaka, Sutra - 25). Hence it has been recommended for the treatment of several types of poisons i.e. Snake bite, rat bite, scorpion sting, wasp bite etc.

In addition it has been recommended for all types of skin diseases e.g. itching, eczema, scabies, erysepels, leucoderma and leprosy. It is supposed to be a blood purifier.

The third group of diseases in which Shireesha is recommended is respiratory e.g. coryza, Chronic Sinusitis, bronchitis, and Asthma. It is known to be anthelmintic as well. It is also recommended for some metabolic disorders i.e. obesity, diabetes and diseases of thyroid, especially by Ashtang Hridaya. It is also recommended for the treatment of eye-diseases and gum lesions.

In Unani system of medicine, bark is used in toothache, deafness, boils, scabies. The flowers are aphrodisiac, emollient, maturant, their smell is useful in hemicrania. The seeds are aphrodisiac, tonic to the brain, used for gonorrhoea and tuberculous glands. The oil is applied topically in leueoderma. (Kirtikar, 1975).

Botanical Description:

An Unarmed deciduous tree 12-21 m. high, bark pale, young shoots glabrous. Leaves abruptly 2 - Pinnate; main rhachis pubescent or glabrous, furnished with a large gland on the petiole above the base and one below the upper most pair of pinnate; petioles swollen and hairy at the base; pinnate 2-3 (rarely 4) pairs, 10-12.5 cm long, their rhachises glabrous or pubescent, swollen and hairy at the base. Leaflets 3-9 pairs, 2.5-4.5 by
1.6-2 cm., with glands between their bases, the lateral leaflets elliptic-oblong, the 2 terminal obovate oblong, all pale green, unequal sided, very obtuse, glabrous above, pubescent and reticulately veined beneath, base obliquely rounded or truncate; petiolules very short, hairy. Flowers white, fragrant, in globose umbellate hands 3.8-7.5 cm. long, more or less pubescent, solitary or 2-4 together from the axils of the upper leaves; pedicels 2.5-3 cm. long, pubecent. Calyx 4 mm. long, pubescent; teeth short, deltoid. Corolla 1 cm. long tube glabrous; lobes 2.5 mm. long, Triangular, acute, pubescent outside. Stamens such longer than the corolla; filaments connate at the base into a short tube. Pods 10-30 by 2-4.5 cm. linear-oblong, bluntly pointed, thin pale yellow, reticulately veined above the seeds, smooth, shining. Seeds 4-12, ellipeoblong, compressed, foveolate on the faces, pale brown.

Pharmacognocy:

Macroscopic and microscopic characters of the young stem and old bark of Albizia Lebbeck have been studied. The bark is dark brown to greenish black, and rough covering being acrid. In young stem the epidermis has a wavycuticle, and unicellular Trichomes are present. Some cortical cells are lignified. Prismatic crystals of calcium oxalate are present. Rhytidome shows alternate layers of cork and cortex. The cork has several rows of radically arranged, suberized are lignified cells. A large number of pitted stone cells containing prismatic crystals of calcium oxalate occur in primary cortex. Phloem is traversed by medullary rays which are 2-4 cells wide and broader. Starch grains are present in cortical and phloem
parenchyma cells, but absent in the medullary ray cells. (Shah and Bhattacharya, 1960)

**Other types of Shireesha**:

1. Albizzia Amara, Bovine and Roxb. (Krishana Shireesha).
2. Albizzia Julibrissin, Durazz (Lal Shireesha).
3. Albizzia Odorotissma - Bonth.
4. Albizzia Procera (Safed Shireesha).
5. Albizzia Stipulata, Bovino.

(Chopra, 1958).

**Chemistry**: Tannins and pseudotannin were found present in the stem bark of A. Lebbeck. The froth number was found to be 417. The uncorrected haemolytic index was 315 and after correlation for digitonin it was 360. (Shah and Bhattacharya, 1960).

From seeds of A. Lebbeck, Saponins based on echynocystic acid have been obtained (Varshney and Geeta, 1970; Varshney et al., 1971, 1973) while friendlin and Y - Sitoslerol were identified in the bark. (Tripathi and Dasgupta, 1974).

**Pharmacology**: The alcoholic extract of root was found to possess anticancer activity against Sarcoma 180 in mice. The stem bark of the plant had hypoglycaemic activity in albino rats. The pods possessed anti-protozoal activity against Entamceba histolytica. It also showed hypoglycaemic activity in albino rats, and anticancer activity in human epidermal carcinoma of the nasopharynx in tissue culture. (Dhar et al., 1968)

R. M. Tripathy, P. C. Sen and and P. K. Das, Institute of Medical
Science studies on the mechanism of action of Albizzia lebbeck in the treatment of Atopic Allergy shows that A. lebbeck has significant crooglycate like action on the mass cell. In addition it appears that it inhibit the early processes of sensitization and synthesis of reagenic type antibodies. If A. lebbeck is given during the first week of sensitization it markedly inhibits the early sensitization processes, while if given during the second week it supresses antibody production during the period of drug administration.