Review of Literature
REVIEW OF LITERATURE

Definition:

Von Pirquet (1906) proposed the term allergy, to described a change of reactivity of the living tissues, with increased or decrease sensitiveness due to the formation of specific antibodies. He stated that the vaccinated person behaves in a different manner from him who has not previously been in contact with such an agent, yet he is not insensitive to it. We can only say of him that his power to react has undergone a change.

Jorden (1955) stated "I do not believe that all allergy is an antigen-antibody reactions by any means and I include autonomic dysfunction, psychic changes, endocrine dysfunction etc., under the broad term allergy.

Shambough (1945) states that an allergic individual is one whose sensitivity threshold is so raised to various materials that exposure to small amounts of such material produces in him an unusual tissue reactions, which is insufficient to cause any such reaction in a normal individual.

Forman sums up the definition very well by stating "Allergy is the sum total of unfavourable stresses and strains which the environment places upon the individual.

Konso et al. 1982 stated that the symptoms of nasal allergy are caused partly by the direct effect of chemical mediators released from mast cells and basophilic cells as result of antigen • antibody reaction and partly by reflex excitation of the efferent nervous pathway resulting from stimulation of sensory nerve endings.
Concept Of Allergy:

a) Allergy as antigen antibody reaction:

Aside from physical allergy, all the conditions cited as examples show the features typical of reaction between antigen and antibody. Prausnitz and Kustner in 1923 have shown that injection of serum from patients with asthma hay fever group into normal human skin induced a passive local sensitization of the skin of the recipient.

Shambough Jr (1945) states that we do not know that a allergic individual makes him develop antibodies against substance which normally do not stimulate antibody production, except that hereditary is an important factor.

b) Hapten Theory of Allergy:

Wolf- Eisner (1906) suggested that antigenic agents such as drugs might combine with the body proteins to form complex antigen. The specificity of which was determined by the non antigenic foreign substances. This hypothesis was the basis for the Hapten theory which was fully confirmed some years later by Landsteiner (1942) and is now accepted explanation of allergy to drugs and other non antigenic substances.

c) Enzymatic Concept of Allergy:

According to this theory, the allergic reaction is produced as a result of abnormal enzyme mechanism in the body (Goldowski, 1958).

d) Autonomic Dysfunction Theory:

Williams (1952) defined allergy as an inherited predisposition to a localised type of autonomic dysfunction mediated by the cholinergic apparatus of the autonomic system.
e) According to White (1992), although the precise offending allergen may be difficult to discover, allergic rhinitis by definition is a disease of known aetiology. Certain people produce an abnormal response to various foreign substances. These can be proteins, or else haptens (e.g. Pollens), which combine with amino acids in the body to from proteins. Whereas in non sensitive subject the reticulo-endothelial system reacts to foreign proteins by producing a specific antibody, susceptible people produce additional reaginic antibodies, associated with the IgE immunoglobulins, and these allergic subjects show a high IgE level in the blood. The Sensitization process is eventually due to the combination of the IgE reaginic antibody with cells such as tissue mast cells. On exposure to the foreign protein the allergen combines with cell-bound reaginic antibodies to release histamine and similar amines and other factors as listed below:

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<th>Mast Cell Mediators</th>
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**Aetiology**

(a) Incidence - Vangham (1933) and Bray (1957) stated that roughly 10% of general population is frankly allergic and 50% gives history of transient episodes.
Shambaugh (1945) stated that at least 90% of chronic nasal infections and 70% of chronic sinus infections can be shown to have an underlying allergic factors responsible for chronicity.

Negus stated that the onset of nasal allergy occurs frequently in first decade of life. In the second and third decades smaller number develop allergic symptoms, and in the fourth, fifth and sixth decades the incidence sharply declines.

The prevalence of allergic rhinitis varies markedly from study to study 0.1% (Schwartz, 1952) to 28% (Malmberg, 1979).

PREDISPOSING FACTORS

1. **Hereditary**: A positive family history is present in 50% of cases (Angel James, 1952).

   Urbach (1946) observed, children with bilateral inheritance develop allergy in 45% of cases, those with unilateral inheritance develop allergy amount to 50% and those without a family history, 7-12%.

2. **Constitutional**: Nasal allergy patients have a poor reactive mechanism to environmental stress which is reflected as a autonomic dysfunction.

3. **Infection**: It leads to allergic sensitization to bacterial products and not to cell (Lucos, 1953).

4. **Endocrine**: Goldowski (1958) quoted, adrenal, ovarian and thyroid hormones are capable of making the nasal mucosa more sensitive.
Symptoms may be precipitated by the influence of pregnancy (Hensel). About one half of patients of rhinitis in pregnancy are found to have sinus infection (Sorri, Hantikainen and Kaija, 1980).

5. **Psychological**

Holmes, Godwell, Wolf, 1950, states many factors of this nature lead to functional disorder of the nose as a stress phenomenon. This type of predisposition appears to be increasing in frequency and importance as a basic factor.

6. **Physical**

Changes in the humidity and content of the inspired air may perhaps render the nasal mucosa liable to allergic disturbances.

A light stimulus of light, cold, heat or mechanical irritant which would not affect that normal nose may cause nasal symptoms in a sensitive subject.

**PRECIPITATING FACTORS**

(a) **Inhalants**

Stafford (1987) states that inhalants form the biggest group and are especially important in adults. A very large number have been recorded and they include dusts, pollens, animal emanations, feathers, orris root used in many cosmetic, fungus fumes, wood etc.

(b) **Ingestants (Foods)**

Are especially important in children. The common food which cause allergy are eggs, meat, rice, curds, wheat, milk, fried fats.

Pinket (1955) classified food allergy into two types.

(i) Fixed (5% cases)  
(ii) cyclic (95% cases)
(c) Infectants:

Stafford 1987, observed liver extracts, insulin and penicillin etc. Can produce allergic reaction.

(d) Chemical Substances:

Drugs like aspirin and potassium iodide can produce hypersensitivity which could be explained by hapten theory of allergy (Wolf, 1906)

(e) Physical Agent:

Physical agents may precipitate allergic reactions. Exposure to strong sunlight, cold heat humidity, barometric pressure changes, wind etc. can precipitate the symptoms in sensitive patients. These probably act as "triggers" and effect the mucosa reflexly as evidence by fits of sneezing which follow stepping out of bed with bare foot on to a cold floor (Hansel, 1930).

(g) Non-Specific Irritants:

Strong smell, tobacco smoke petrol fumes etc. May directly initiate the hypersensitive mucosa and excite the reaction.

PATHOLOGY

Hiranandani, 1964 described die typical changes in nasal mucosa of a case allergic rhinitis. pathologic changes in nasal mucosa in early stages of allergy is reversible one, so that after the hayfever season the "Water logged" nose return to normal. Long standing allergy, especially when complicated by infection results is irreversible hyperplastic and polypoidal changes in the nasal mucosa.
(a) Local Mucosal Changes

Oedema From intracellular and intercellular transudation of tissue fluid. Marked oedema leads to compression of small superficial vessels and produces the characteristic pale swollen mucosa.

(b) Infiltration with eosinophils and plasma cells

Eosinophilic infiltration in tunica propria is a characteristic feature of allergic mucosa. Plasma cells are derived from the reticuloendothelial system.

(c) Epithelial Changes

Individual epithelial cells swell as a result of oedema. In perennial allergy, epithelium shows, more hyperplasia and more degenerations. There is increased number of goblet cells replacing many of the columnar ciliated epithelium.

(d) Excretion of Thin Watery Discharge

This results from increased activity of seromucous glands. Histologically there is hypertrophy of goblet cells and mucosal glands.

(e) Vascular Dilatation

Venous stasis particularly affects the inferior turbinate which become enormously hypertrophied.

(g) Superadded infection

Shambough Jr (1945) stated that at least 70% of chronic sinus infection and 90% of chronic nasal infection have an underlying allergic factor responsible for chronicity. In the mucosa, infiltration by polymorph and lymphocytes predominates. Eosinophils may disappear.
(h) Polyp

Theodore just 1939, reported polypi are more commonly found in conjunction with asthma than with nasal allergy.

SYMPTOMS AND SIGNS

Symptoms

Lindqvist et al 1986, states the most characteristic clinical features are sneezing, nasal discharge and nasal obstruction. These symptoms vary in prominence and degree, in different subjects, with period of remission in between.

(I) Nasal Irritation

Rinkel 1962 observed, tickling or itching sensation in the nose is a common symptoms and is usually followed by sneezing. Itching is pathognomonic symptom of allergy.

(II) Sneezing

It is a reflex action produced by stimulation of hypersensitive mucosa and occurs in paroxysmal sudden attacks in most cases. (Councill, 1984)

(III) Nasal Discharge

Lindqvist 1986, states profuse, clear watery nasal discharge associated with sneezing is a prominent symptom. Post nasal discharge is a frequent complaints.

(IV) Nasal Obstruction

The main feature is its alternating character but it may be persistant due to chronic nasal oedema or polypus. (Dale, 1982)

(V) Anosmia

It is some times complained of intermittently or continuously even in the absence of obstruction.
(VI) Headache

A feeling of heavyness or actual frontal headache is a common symptom. Pinkel (1962) stated that irregular occurrence of headache is characteristic of allergy.

(VII) Frequent "Head Colds"

Increased susceptibility to head colds and the tendency of these to last weeks or month instead of days is characteristic feature.

(VIII) Anosmia

It is due to nasal obstruction preventing odours from reaching olfactory area of the nose. It may be intermittent or constant.

(XI) General Symptoms

These are fatigue, lack of concentration and anorexia (Brown, 1979)

(X) Associated symptoms

According to Mygind & Lewenstein (1982), manifestations of allergic involvement of other systems are frequently found in cases of allergic rhinitis. Cough and hoarseness of voice are the common laryngeal symptoms. Symptoms of bronchial obstruction results from spasm of bronchial muscle or oedema of mucosa.

Eye symptoms like itching, lacrimation, puffiness of eye lids, redness and oedema of conjunctiva are common especially in cases due to inhalant allergen like pollen. Skin manifestations include itching, urticaria, eczema and skin erruptions recurrent attacks of soreness of pharynx, oedema of uvula, soft palate and lips are seen in some cases.

Gastrointestinal symptoms like distress after food, gastric pain, bloating nausea vomiting, and diarrhoea occur in food allergy. Allergy may
affect the ear, producing deafness, tinnitus or chronic discharge, swollen painful joints, bursitis and myalgia me the allergic symptoms of musculoskeletal system.

Albuminuria, heamaturia, bladder initiative and oedema of prostate due to allergy have been reported (Rinkel.1962)

SIGNS

1. **Anterior Rhinosocpy**

Mygind (1979) described the various signs of allergic rhinitis. The pale, boggy, bluish tinged mucosa, is characteristic of the well developed allergic rhinitis. Not all allergic individuals exhibit the classical pale, boggy, blue gray mucosa, it may vary from a normal watermelon red to pathologic pale, pinkish white. In many patients with allergy of short duration there is a cheery red colourations described as a cocks comb type of redness.

During an attack there is swelling of the erectile tissue of the turbinals and increased secretions. The mucous membrane, espeically that over inferior turbinates, is often swollen as completely to occlude the passage. If touched with a probe, it is found to be elastic. The application of cocaine produces some retraction and recieves a more complete view, but the improvement is transitory.

In presence of mucoid or purulent exudate in the vestibule, the atrium, the floor of the nose and the middle meatus is significant. Allergic secretions tend to be more ropy in their consitancy then secretion of inflammatory origin.
2. Posterior Rhinoscopy

According to Mygind (1979), The classical pale, boggy mulberry like posterior tips of the inferior turbinates are significant and should suggest the possibility of an allergy. These however may be physiological for certain individuals.

The occurrence of regenerative lymphoid tissue in the tonsillar fossa is indicative of allergy. The hypertrophy of the lateral bands of the pharynx and the posterior pharyngeal wall are highly suggestive of allergy. Pharyngeal bands frequently thought to be due to infection alone, have also been proven to be due to allergy.

DIAGNOSIS

1. History

A careful and complete history is the first and next important step in diagnosis. It helps to establish the existence of allergic diseases and guides to detect the specific causative agent.

Shambough Jr. (1945) found that it was more valuable than skin test. A positive family history or occurrence of previous allergic manifestations in the patient are important points to be noted. A search should be made to determine such points on the time of the attacks occur, presence of any seasonal variation, effect of change of environment, presence of any trigger mechanism precipitating the attack, the part played by emotional factors and any special intolerance to article of diet. The symptoms are related to season change of climate, a vacation from work or trip away from home, the allergen is most probably an inhalant. Pollen allergy occurs in lowering seasons. If patient is better in summer but worse in cold weather or when inside the home or when dust is stirred up, the offending agent is probably
the house dust. Gas formation, distress or headache occurring after eating point to the possibility of a food allergen (Shambough Jr, (1945).

2. X-ray Pam Nasal Sinuses

Charles et al 1977, states X-ray will give information as to the state of nasal sinuses which is otherwise not easily obtained, as transillumination is not informative and is unreliable. There may be mucosal thickening of the antrum or other para nasal sinuses. Sometime there may be cyst or cysts over the floor of the antrum or fluid level.

3. Antral Puncture

Clear yellow fluid or thick mucus is obtained in uncomplicated cases when there is infection it becomes purulent and bacteriological study is helpful.

4. Nasal Smear

Eosinophilia in nasal mulcous is considered by an impressive assay of authorities to be pathognomic of an allergic condition. Gay (1945), Hanis (1951), Hansel. (1953), Glaser (1958) and Jensen (1956). But Ceolding Wood emphatically states that the functional fate of eosinophil is still unknown and we can accept that eosiniophils occur in nasal tissues after antigen antibody reactions.

Shambough Jr. 1945, stated a Positive Smear may be helpful in diagnosis but repeated examination is necessary to demonstrate them.

A Negative smear does not rule out allergy (Mygind, 1979)

5. Biopsy Of Nasal Mucosa

A typical structures variation is with infiltration eosinophil, (Grist Wood, 1982). Histopathology from the biopsy piece of inferior turbinate shows papillary proliferation of mucous membrane and hyperplasia of
mucous gland, submucusa shows the presence of inflammatory cells which is predominantly eosinophil (Bbargava, 1980), epithelium shows changes for single layered columnar epithelium, broadening of basement membrane, and infiltrilation with mononuclear and plasma cells are the constant features in HPE (Hiranandani, 1964).

**TREATMENT**

Treatment of allergic rhinitis is far from satisfactory. Ideal treatment should be directed towards correction of etiological factor, avoidance of allergens and desensitization. However accurate determination of the cause is often difficult. The complex nature of allergy, endocrine dysfunction, and psychological stress make it difficult in many case to apply adequate treatment even when only one of the factor is present. Various methods of treatment have been advocated, each with its own limitations and degree of success.

1. **Avoidance of Precipitating Factor**

Hagy 1969, quoted this is often successful in case of allergens like articles of food. In others who are sensitive to change of temperature, humidity and direction of wind etc, the precipitation factors cannot be avoided.

2. **Desensitization or hyposensitization**

This is indicated in most of the cases where the allergen is known but cannot be avoided.

2.1 **Specific hyposentization** - The latest theory regarding the mechanism assumes that it stimulates production of an immune or blocking antibody distinct from the reagenic antibody and it blocks the union of allergen and reagin (Harley, 1960).
2.2 Non Specific Hyposensitization - Specific desensitization is successful only with few types of allergens so next attempt was to find out a method which would desensitize or inhibit allergic reactions without the employment of a specific antigen so that it can be applied uniformly to all types of allergy.

3. Antihistamines

Dannenberg & Feinberg, (1951), stated these drugs antagonize the action of histamine by a competitive inhibition at receptor site. A Single dose is effective only for a few hours. Development of tolerance after their prolonged use is common.

4. Corticosteroids

Pipkorn (1983) states, we still do not know how these anti inflammatory steroids are useful in allergic rhinitis. It is not substitute therapy because there is no evidence of any adrenal cortical insufficiency in allergic disease. These drugs are not recommended for regular use. Wihl (1984) observed that steoids exert profound undesired metabolic endocrine, neuro-muscular, immuuoological and cytological effects. The visual contra-indication of cortisone therapy have to be remembered.

5. Sodium Cromoglycate

Wide Et Al (1967), observed its effectively in preventing symptoms, if used before attacks.

6. Histamine Releasing Agents

Drugs like compound 48/80 dextran, peptones and stilnamidine on administration cause depletion of histamine in body and a histamine free period ensures during which no allergic reactions can take place (Halpern 1960).
7. **Gamma Globulin**

Histamine binding power of plasma which has its seat in gamma globulin is found to be reduced in allergic patient. This permits treating the allergic cases with injections of blood serum of gamma globulin of normal person. **Crilland** in 1959 and Bernard Redner in 1963 reported good results with this treatment. The injections probably stimulate synthesis of mobilization of interstitial gamma globulin.

8. **Thyroid Hormone**

In many cases of allergic rhinitis resistant to usual methods of treatment thyroid therapy has been found helpful (Walsh, 1950).

9. **Injections of Sphenopalatine Ganglion With Alcohol**

This reduces sensitivity; of mucosa and relief of symptom have been reported (Hansel, 1943).

10. **Local Treatment**

   (a) **Zink Ionization** - **Proetz (1936)** & **Bhangeeva Et Al (1980)** stated that, this reduces the permeability and sensitivity of mucosa to allergens by altering the reaction of fluid bathing the mucosal cells. In perennial allergy it gives relief in 60-80 % cases.

   (b) **Auto Haemotherapy** - **Rege and Shah**, 1958, tried injections of own blood submucosally in inferior turbinate in cases of allergic rhinitis.

   (c) **Cauterization Submucosal Diathermy and Cryosurgery** - It reduces the bulk of hypertrophied turbinates and relieves nasal obstruction. (Ozenberger 1970 Karjaetal 1975).
(d) Submucosal Injections of Cortico - Steroids - These has been tried in allergic rhinitis and given good results (Gill 1966).

(e) Steroid aerosol - Beclomethasone dipropionate is administered by standard metered aerosol which delivers 50 microgram of drug/puff taken as sniff and has given good results (Brown Storey and Jackson, 1977).

Topical Nasal Drugs

The treatment of allergic rhinitis revolutionized over the last few years by the introduction of various topical preparations of nasal drugs especially topical corticosteroids. Various preparations of nasal drops containing either decongestant and vasoconstriction agent or topical corticosteroids (Beclomethasone, prednisolone) have been used and have shown good results in the treatment of allergic rhinitis.

Xylometazoline Hydrochloride - Zee & Dischoekm, 1962 tested the effect of xylometazoline nose drops in allergic rhinitis in different ways and claimed it to be effective.

Mesek H 1962 treated various cases of allergic rhinitis with xylometazoline nasal drop and described its effectively in allergic rhinitis in his book "clinical experience with xylometazline".

Aschan & Rettner, 1964 explained the various objective, investigations of decongestive effect of xylometazoline in patients of nasal allergy.

Symptomatic relief from nasal congestion associated with allergic rhinitis can be obtained by the use of xylometazoline which exerts its effect by vasoconstriction of mucosal blood vessels which in turn reduces the thickness of nasal mucosa. (Ariens, 1967).
Anggard and Malm, 1983 quoted topical decongestant xylometazoline is more effective than systemic decongestant in the management of allergic rhinitis.

Various' studies have shown in the past that topical nasal drugs preparation containing steroids are the most effective measure in the management of allergic rhinitis.

Proetz (1936) stated that local treatment of allergic rhinitis with zinc ionization gave relief in 60-80% cases of chronic nasal allergy.

Bordley, (1949) reported encouraging results with the hormone used as nose drops in the treatment of chronic nasal allergy.

Goodman Gilman (1950) states cortisone and ACTH provide symptomatic relief in disease of allergy.

Silicox (1958) reported a study of 174 patients suffering from allergic rhinitis. He treated with topical hydrocortisone 4 times a day with decongestant phenylephrine hydrochloride. He says that hydrocortisone solution alone is not useful in case of acute rhinitis, but with decongestant it is more effective.

Leopoldkroman and Green, reported 125 patients on whom prednisolone nasal spray was used, 115 obtained significant relief Simon? (1960) reported on 419 patients with allergic rhinitis who were treated with prednisolone injection locally. Benefit was obtained in 78% of his cases.

Hiranandani, 1964 carried out a clinicopathological study of vidian nerve section in the treatment of allergic rhinitis with satisfying results.
Gill, 1966 carried out a clinical and histopathological study of intraturbinate use of steroids in nasal allergy in 104 patients, with success rate of 81.25% of cases.

Halopainen et al, 1971 recognised that in allergic rhinitis topical application of disodium cromoglycate given good results were short lived.

Mygind, 1973 recognised the application of topically active steroid was very effective in the treatment of allergic rhinitis.

Brown Storey And Jackson, 1977 administered beclomethasone dipropionate by a standard metered aerosol and gave good results in treatment of allergic rhinitis.

Bhargava et al, 1980 Carried out a clinico histopathological study of local application of silver nitrate in the treatment of allergic rhinitis. 51 cases were selected and nasal biopsy was taken before and after treatment. 68.3% of Patients had good relief particularly from sneezing and watering of nose. Potent topical corticosteroids such as intra nasal baclomethasone dipropionate are useful in severe nasal congestion due to allergy.

Cromolyn sodium appears to have some efficacy in suppressing symptoms of allergic rhinitis (Hendels, Weinberg and Wong, 1980).

Gale, Solomon & Tao, 1980 carried out a study comprising of the intranasal topical flunisolide therapy in thirty five children with seasonal allergic rhinitis and concluded 64% of the flunisolide treated group noted substantial or total control.

Bharava, Abhyankar, Shah, 1980 treated the 41 patients of allergic rhinitis with local application of silver nitrate and found 79.4% patients reported relief significantly.
Petrunson, 1981 stated xylometazoline (0.1%) nasal drops are effective in the treatment of allergic rhinitis with fewer side effects. He also stated that nasal drop containing ephedrine and/or naphazoline were commonly used but side effects like reactive congestion, tachyphylaxis and rhinitis medicamentosa were frequently observed.

Malm, Wihl, Lamm and Lindquist, 1981 quoted the value of three objective tests of nasal mucosa in 22 patients with allergic rhinitis treated with a topical corticosteroid.

Empey, Meddler, 1981 stated that in an acute case of allergic rhinitis, topical decongestant may be the most immediately effective remedy. Their study was based on the use of topical decongestant containing pseudoephedrine or phenylepherine in acute cases of allergic rhinitis.

Mygind, 1982 used glucocorticoids topical in allergic rhinitis cases with significant good results.

Swenson, 1982 carried out a study which comprised of topical treatment of allergic rhinitis with a beta adrenocepto-stimulant (KWD 2131).

Interanasal steroid treatment can reduce methacholine induced nasal secretion, reduce the sensitivity of mucosal irritation receptors and lower the number of basophilic as well as eosinophilic cells in the nasal secretion (Wihi, 1982).

Warland, 1982 evaluated the effectiveness of a topical steroid flunisolide in thirty four patients with perennial rhinitis. There was a statistically significant difference in favour of flunisolide.
Balle, Pedersen, 1982 quoted that the treatment of allergic rhinitis in a new, halogenated topical aerosol packed steroid, Budesonide had a significantly better effect.

Hamilton L.H 1982 studied nasal decongestant effect of propylhexidine in patients of allergic rhinitis and found most patients got significant amount of reduction in their symptoms.

Topical cortico steroids administered intranasally are clearly the most effective medications for treatment of chronic allergic rhinitis (Estelle, Simon, 1984).

Asakura Enomoto et al, 1984 examined nasal responsiveness to topical methacholine application in allergic rhinitis and non allergic rhinitis and concluded that methacholine responsiveness was significantly higher in allergic rhinitis.

Wihl 1984 observed the effectiveness of topical intranasal steroid in treatment of allergic rhinitis.

Kwaselow et al, 1985 studied a comparison of intranasal and oral flunisolide in the therapy of allergic rhinitis and evidenced that intra nasal flunisolide is an effective treatment of allergic rhinitis.

Watase, Okuda, 1986 Carried out a study to elucidate the effects of different kinds of autonomotropic drugs in the nasal mucosa as well as on the nasal reaction to specific allergens in patients with nasal allergy.

Lindqvist et al, 1986 Recommended the long term safety and efficacy of budesonide nasal aerosol in perennial rhinitis.
Orgel et al, 1986 Observed clinical rhinomanometric and cytologic evaluation of seasonal allergic rhinitis treated with beclomethasone dipropriouate as aqueous nasal spray. They stated topical BDP was rapidly effective in decreasing mean nasal obstruction, rhino-rhea, sneezing and itching symptoms.

Caorado Olliers, 1987 measured the changes in nasal airway resistance following the topical application of histamine by passive anterior rhinomanometry.

Welsh et al, 1987 Conducted a randomized clinical trial showing the efficacy of beclomethasone nasal solution flunisolide and cromolyn in relieving symptoms of ragweed allergy.

Topical glucocorticosteroids, significantly reduces both the symptoms and level of histamine. TAMP esterase activity and kinin in allergic reactions (pipkorn et al. 1987).

Bende and Pipkorn, 1987 highlighted the efficacy of topical levocabastine, a selective H1 antagonist in seasonal allergic rhinitis.

Pipkorn and Everback 1987, designed a study in order to elucidate the interaction in the treatment of allergic rhinitis and the migration of mast cells.

Togias Proud et al, 1987 Aowed the effect of topical tricyclic antihistamines on the response of the nasal mucosa to challenge with cold, dry air and histamine.

Patients presenting with typical signs and symptoms of allergic rhinitis may respond to avoidance of allergens and to medications for symptomatic relief (Safford 1987).
Topical synthetic corticosteroids and orally administered delta-1 steroids are the commonly prescribed medications used alone or in various combination in allergic rhinitis (Berman, 1988).

Naclerio, 1988 observed that pretreatment the patient of allergic rhinitis with topical steroid flunisolide several days before the pollen season reduces the early response of allergen and inflammation associated with chronic allergic rhinitis.

Meltzer, 1988 documented that topical Corticosteroids including flunisolide therapy decreases the symptoms, improves patency of nasal airways in patients with allergic rhinitis.

Topical nasal decongestants give fast relief from nasal congestion, but their over use may result in rebound congestion (Burse, 1988).

Bunnag et al 1988 suggests that intranasal budesonide is an effective and well tolerated treatment for perennial rhinitis.

Pechler et al, 1988 Carried out a clinical comparison of systemic methyl prednisolone acetate (MPA) versus topical budesonide in patients with seasonal allergic rhinitis and showed the efficacy of topical budesonide over MPA in allergic rhinitis,

Ballas Seltzer et al, 1990 evaluated symptoms relief, nasal airflow, nasal cytology and acceptability of two formulations of flunisolide nasal spray in patients with perennial allergic rhinitis.

The use of decor gestants (symptomimetics) is limited by the so called rhinopathia medicamentosa, when the necessary treatment exceeds 2 or 4 weeks (Albergger, K. 1990).
Klemeaston Lindquist et al, 1990 performed a study showing the effect of single dose of topical glucocorticoid and a cycloxygenase inhibitors on allergen induced changes in nasal reactivity with good results.

Bonsquest and Michel, 1990 reviewed the therapeutic approach to seasonal allergic rhinitis and observed that

1. Topical vasoconstrictors are effective but cause side effects when treatment is prolonged.

2. Topical corticosteroids are highly effective and safe.

Naclerio, 1990 demonstrated that histaminic plays an important role in the mediation of allergic rhinitis.

Darnell, Pecard and Recbard, 1994 carried out a double blind comparison of fluticasone propionate aqueous nasal spiny, Terfenadine tablets and placebo in treatment of patients with seasonal allergic rhinitis to grass pollen and showed higher efficacy of fluticasone topically.

Nonsteroidal anti inflammatory drugs have weak effects in allergic rhinitis as compared with glucocorticoids (Malm, 1994).

Kobayashi, 1994 stated that nasal steroids can be used and are safe and effective as antihistamine in controlling symptoms of allergic rhinitis.

Moper, 1995 Reviewed the therapeutic use of pregnendiones in allergic rhinitis.

Lemanske et al, 1990 observed the significance ana potency of topical fluticasone propionate in allergic rhinitis.

Treatment with mtranasal flimisolide in allergic rhinitis induces marked improvement of clinical symptoms and reduces the total IgE in
nasal secretion. (Pagenelli et al, 1991). topical use of flunisolidc in the
treatment of perennial rhinitis induces marked improvement of clinical
symptoms and that it exerts its effects through its antinflammatory action
on nasal mucosa.

Anrihistamine alone cannot control all of symptoms of allergic
rhinitis. However, the combination of antihistamine with topical
corticosteroids is very effective (White and Kaliner 1992).

Melzer, Orgel et al, 1992 studied the topical acitivity of
i pratropium bromide and anticholinergic agent in perennial allergic rhinitis
and observed its effectiveness in decreasing the rhinorrhea in patients
suffering from allergic rhinitis.

Pedersen, Mygind et al, 1991 observed that budesonide delivered as
pure powder is effective and safe for the treatment of seasonal allergic
rhinitis.

Macrophages are involved in inflammatory processes of allergic

Welch, Garcia et al, 1991 had shown the high effectiveness of
topical triamcinolone in allergic rhinitis cases.

Heyning & Rossel, 1991 stated that topical application of
levocabastine a potent H1 antagonist yielded good clinical results in allergic
rhinitis.

Dechant & Goa, 1991 stated that Levocabastine nasal spray is better
than sodium cromoglycate and placebo in the allergic rhinitis.
Scadding Lund et al., 1991 shown the clinical and physiological effects of fluticasone propionate aqueous nasal spray in the treatment of perennial rhinitis with good results.

Paganeli et al., 1992 stated that

Ratner et al., 1992 stated that fluticasone propionate given once daily is as effective for seasonal allergic rhinitis as beclomethasone dipropionate given twice daily.

Bryson and Faulds; 1992 reviewed the therapeutic potential of intranasal fluticasone propionate in allergic rhinitis.

Sim Hilsmeir et al., 1992 studied the effect of topical corticosteroid in various patients of allergic rhinitis and proved its efficacy.

Swensson & Pipkorn, 1992 stated that topical vasoconstrictor oxymetazolium does not affect hislamine Induced mucosal exudation of plasma in human nasal airway.

Topical glucocorticoids inhibit allergen induced activation of eosinophil in allergic rhinitis (Lozewic et al., 1992).

Krause, 1992 stated that antihistaminics are the mainstay treatment of allergic rhinitis and lopiuii Jccongsuuit should be added for a short time to prevent rebound.

Meltzer, 1992 stated that topical anticholinergic medication ipratropium bromide was safe and effective in reducing the troublesome symptoms of allergic rhinitis.

Bunnag, Jore and Wong, 1992 Proved the efficacy of topical budesonide and oral astemizole in allergic rhinitis.
Long term use of topical corticosteroid in the nose is not harmful to the nasal mucosa. (Bende and Mark, 1992).

Mabsyl, 1993 studied the topical pharmacotherapy for allergic rhinitis and stated that it prevents or ablates both the acute and late phase of allergic response.

Birchall Henderson et al, 1994 studied the effect of topical sodium cromoglycate as intranasal his famine challenge in allergic rhinitis and observed its potent antinflammatory effect.

Thus we can expect better results in allergic rhinitis with topical corticosteroid. Newer topical drugs has proven to be more effective than older topical drugs, in the present study. The topical effect of nasal drugs in allergic reunite, however, need to be studied further.