

**A STUDY ON  
SWASAKASAM**

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## INTRODUCTION

The origin and development of traditional system of medicine date back to many centuries. Since then the phenomenal development of this system was enlivened by the saint siddhars. The guru **AGASTIYA** was the father of this noble system and the treatise comprises of various disciplines in medicine.

The great siddha medicine is illustrious about the spiritual wisdom, scientific knowledge, anatomical structures, religion and philosophy, astrology, breathing exercises and medical science.

Now a day's allergic disease are widely prevalent in people from all the walks of life. This is because of contamination of atmospheric air due to smoke, dust emitted from factories and automobiles. Moreover polluted water also penetrates into the earth and spoils the ground water. The cereals and rice are always treated with pesticides. Thus the air we breathe, the water we drink, the food we eat, instead of nourishing the lives on earth cause havoc to them.

The prime evidence for the selection of this disease and the trial drug was adopted from the '**Gunapadam Mooligai Vaguppu**' and there are so many recipes for '**Swaskasam**' in our text. But the ingredients of these selected drugs suites all the needs for the diseases.

The siddha system is a blend of nature and nurture, the climatic principle, the dietic ethics, habits forming, the daily routine procedures etc. The five primordial principles of earth, water, fire, air and sky have a vital role in causing the disease.

The siddha system gives utmost important to all the above principles, hence the adage says.

“அண்டத்திலுள்ளதே பிண்டம்  
பிண்டத்திலுள்ளதே அண்டம்”

- சட்டமுனி நிகண்டு

“மிகினும் குறையினும் நோய் செய்யும் நூலோர்  
வளி முதலா எண்ணிய மூன்று”

- திருக்குறள்

The changes of the climate have direct impact over the physiology of the body.

The ingestion of the absorbed food substances goes through the seven principles of bodily elements (i.e) Saram, Senneer, Oon, Kozhuppu, Enbu, Moozhai and Sukkilam. In this disease ‘**swasakasam**’ the aggravating factors are the cold climate, allergens, exercises etc.

The pre-clinical survey of these formula shows encouraging results and the patient’s responses results were also good.

The treatment aspects involve the neutralisation of affected humours.

“விரேசனத்தால் வாதம் தாமும்  
சத்தியால் பித்தம் தாமும்  
அஞ்சனத்தால் கபம் தாமும்”

- நோய் நாடல் நோய் முதனாடல்

By giving viresanam (Purgative) Vadha Kutram is neutralized. By giving sathi (emetic) pitha kutram is neutralized. The Kapha Kutram is neutralized by giving anjanam (streaming) and others.

## AIM AND OBJECTIVE

It is the aim of the author to develop siddha system and to select a suitable drug for the incurable diseases like ‘**Swasakasam**’ which is roughly compared to ‘**Bronchial Asthma**’ in modern medicine.

The prime object of the author is to treat the ‘**Swasakasam**’ and paid attention to drugs namely ‘**Mungiluppu Choornam**’ and also the author have done study with the following objects.

- ❖ The detailed study of various siddha literatures dealing with definition, etiology, classification, signs and symptoms, diagnosis of Swasakasam.
- ❖ To know the incidence of the disease with regards to age, sex, socio-economic status, family history and Paruvakalam.
- ❖ To know the derangement of Mukkutram, Poripulungal, Envagai Thervugal, Naadi, Neerkuri and Neikuri in Swasakasam.
- ❖ To do pharmacological studies, Bio-chemical analysis and anti – bacterial studies of the trial drug to establish their efficacy.

A clinical trial was done in 20 patients and the results are discussed. Swasakasam is a distressing chronic respiratory disease with recurring episodes which affects millions of people now a day that causes paroxysms of cough, dyspnoea, tightness of chest and wheezing with severe embarrassment both physically and mentally. The daily increasing sufferers of this disease and the efficacy of siddha system of medicine in curing chronic respiratory disease prompted the author to carryout scientific clinical study on this subject.

## **ABSTRACT**

Since the number of sufferers increasing day by day, the author has chosen the disease, '**Swasakasam**' for her dissertation work. The increasing incidence of the disease is due to changes in life style and environment.

20 patients of either sex were selected as in-patients and twenty out- patients were administered with the trial medicine "**Mungiluppu Choornam**" 4gm BD with honey daily after meals.

The trial medicine was subjected to Biochemical and Pharmacological as well as microbiological analysis.

At the end of trial study, the majority of cases showed good result.

## REVIEW OF LITERATURES

### SIDDHA ASPECT

Our ancient siddhars classified disease into **4448** single disease entities and described each one separately and vastly. They classified the disease on the basis of Vatham, Pitham and Kabam. Swasakasam, which is dealt in this dissertation work, comes under Kaba disease.

“கபத்திணையன்றி காசசுவாசம் காணாது”

- தேரையர்

Siddhars described about ‘**Swasakasam**’ under the classification of ‘**Erumal Noi**’ which is otherwise called ‘**Eelai Noi**’. The major character of the disease is breathlessness, tightness of the chest, sneezing and cough.

Symptoms and sign are slightly differs from author to author.

The human body composed of **72.000** nerves.

Among this, the ten are big nerves (Thasa naadi’s)

“சிறந்த இடைபிங்கலைஞ் சுழ்முனையினோடு

சிறப்பான காந்தாரி யத்தீச் சிங்குவையாய

பிறந்த அலம் புருடனொடு குகுதன்றானும்

பேரான சங்குனியும் வயிரவன்றான்

திறந்த விவை பத்துந்தான் றச நாடியாடும்”.

- யுகிவைத்திய சிந்தாமணி

Yugimuni says that above ten nerves are

“Thasa Naadi’s”

Muklutram relation with elements (i.e) Pancha Bootham

Vali + Ahayam = Vadham

Neruppu = Pitham

Mann + Neer = Kabam



Among five elements Kabam has the qualities of Mann and Neer. This is explained as follows:

“சேத்துமந் தண்ணீர் பித்தந் தீகாற்று வாதமாமே”

- அகத்தியர் நாடி

Muklutram relation with tastes and elements

Earth + Water	=	sweet
Earth + Fire	=	Sour
Water + Fire	=	Salt
Air + Sky	=	Bitter
Air + Fire	=	Pungent
Air + Earth	=	Astringent
Air + Sky	=	Vadham
Fire	=	Pitham
Water + Earth	=	Kabam

“வழங்கிய வாதம் மாத்திரை யொன்றாகில்  
தழங்கிய பித்தந் தன்னிலரைவாசி  
அழகுங்கபந் தாடைங்கிய காலோடில்  
பிறங்கிய சீவர்க்குப் பிசுகொன்று மில்லையே”

- குணவாகடம்

When Vatha, Pitha, Kaba are in the ratio of  $1:1/2:1/4$  in the body, it indicates that the man is physiologically normal in health according to **Gunavagadam.**

“வாத பித்த மைய மூன்றும்  
வன் பலத்துடனே தத்தம்  
பேத மொன்றில்லா வண்ணம்  
பேசிய தானந் தன்னில்

நீதியாய் நிலைத்து நிற்கில்  
நெடும்பிணி சிக்கவில்லை  
தாதுவுமொன்றோ டொன்று  
தாவிடில் பிணிகள் தானே”

- நோய் நாடல், நோய் முதனாடல் திரட்டு

So the alteration of Kaba Thathu, altered the functions of the Ezhu Udar Kattukal and other Thathus indicated the disease ‘**Swasakasam**’.

**REVIEW OF LITERATURE**  
**SIDDHA ASPECTS**

**Veru Peyargal (Synonyms)**

Swasa Irumal  
Isivu Irumal  
Izhuppu Irumal

**Definition:**

Swasakasam is characterised by cough with expectoration, breath sound like hissing of snake, throat irritation, indigestion, flatulence, redness of the nose, emaciation, low pitched voice, excessive salivation.

**Noi Varum Vazhi:**

**Yugi Vaidhya Chinthamani Says,**

§Å, çýÈ Å¼ç, Á;õ Ò", Âç É;Öö  
Á£Û, çýÈ À;Éð¼;ø ÁçÕììó ¼;§É  
- 690

À;Éð¼;ø ÀÃÁ;ì, çÉç ÁçÕì", Â;Öö  
À;ÃÁ; Á;Áç°í, û Ò°çì", Â;Öö  
¼;Éð¼;ü °ï°;Ãó ¼Åç÷ì", Â;Öö  
°ÃçÀ¼;ô À¼;÷ð¼í, û Ò°çð¼ Ä;Öö  
¼£Éð¼;ü |À;°çÂ;Á ÄçÕì", Â;Öö  
§°Âç"Æ Â;÷§ÁÄçýÀì °ç"¼Å ¼;Öö  
Á;Éð¼;ü Á;Ðì, Á"¼¼ Ä;Öö  
Á¼ð¼;Öï ÍÅ;°çôÀÐ ÁÕ×í, ;§½  
- 691

**Diet and habits:**

- ❖ Excessive smoking
- ❖ Excessive intake of cold water

- ❖ Increased body heat
- ❖ Taking non-vegetarian diet
- ❖ Immobilisation
- ❖ Taking improperly cooked food.
- ❖ Starving on hunger

" , i½SÅ S¾Å"¾îî ÀçÃçð¾ Àñ¾õ  
 , ÇÅ;Ê ¾çýÈ;Öí ,¾Åý Èý"É  
 S¾;½SÅ çç÷¾;ð"° |°;ýÉ ¾;Öõ  
 Ö°çÅ;É À¾;÷ð¾|Áî°çø Àñ¾ç É;Öõ  
 SÅ½SÅ ´ÖÅ÷ |°ö¾ çýÈç ¾ý"É  
 Áç ,ÁÈóð | , î"Á ,¾;ý ÁçÇöð SÅ;÷îîõ  
 SÀ½SÅ °"À¾ÉçSÄ |°;ýÉ SÀîí  
 ÀçÃñS¾;÷îîí , ;°ö Åóð ÀçÈîîó ¾;SÉ"

- 692

**Character and Behaviours:**

- ❖ Excessive Coitus
- ❖ Over Stress
- ❖ Stealing foods which were prepared for god
- ❖ Cursing life partner
- ❖ Tasting other's foods
- ❖ Forgetting one's help
- ❖ Those who didn't keep his words



## Roga Nirnaya Saram Says,

«î, çÉç «¼ç, ãçòð |çîí òñ½i, ç ÅiÔ  
«§¼iÔ, Ái, §Áø|°ýÚ Ái÷Àçø ¼í, ç ñ¼iîõ.

Due to excessive body heat, gas ascends to the lung field, thus causes the disease.

## Murkurigal (Preliminary Signs)

|¼iñ"¼ òñÀð¼ð§ÀiÄ §çi¼ø, Àçý |¼iñ"¼ °çÅð¼ø,  
|¼iñ"¼Äçø ÓûÇiø ÌððÁð §ÀiýÈ ñ÷î°ç ²üÀ¼ø, ÌÄ§Äi"°  
Ì"È¼ø, ãîî ç£÷ Àiö¼ø, Ái÷Ò §çi¼ø, ÝîûÇ |ÀiÔÇçø ÅçÔðÀõ  
Ó¼ÄçÄÉ ñ¼iîõ.

- ❖ Sore throat
- ❖ Pricking sensation in the throat
- ❖ Decreasing the pitch of voice
- ❖ Running nose
- ❖ Pain in the chest
- ❖ Desire towards hot foods

## Noi Enn (Classification)

### Yugi Vaidhya Chindamani says,

Swasakasam is described as one of the twelve types of Kasam.

The twelve types are,

1. Mandhara kasam
2. Pakka Mandharakasam
3. Sudar kasam
4. Vadha Kasam
5. Pitha Kasam
6. Swasa Kasam
7. Ratha Kasam
8. Silethuma Kasam
9. Peenisa Kasam

10. Vadhapitha Kasam
11. Pitha silethuma Kasam
12. Thontha Kasam

**Sikitcha Rathna Dheepam @ Vaidhya Chinthamani Says**

There are twelve types of Kasam. They are

1. Mandhara Kasam
2. Patcha Mandhara Kasam
3. Sudar Kasam
4. Vadha Kasam
5. Pitha Kasam
6. Swasa Kasam
7. Ratha Kasam
8. Silethuma Kasam
9. Peenisa Kasam
10. Vadha pitha Kasam
11. Pitha Silethuma Kasam
12. Thontha Kasam

**Raja Vaidhya Bodhini - Part I says**

There are twelve types. Those are

1. Vadha Kasam
2. Pitha Kasam
3. Sethuma Kasam
4. Vadha Pitha Kasam
5. Pitha Sethuma Kasam
6. Mandhara Kasam
7. Swasa Kasam
8. Shaya Kasam
9. Sudar Kasam
10. Peenisa Kasam

11.Naadha Kasam

12.Thontha Kasam

**Anubava Vaidhya Deva Ragasyam Says,**

There are five types. Those are,

1. Vadha Kasam
2. Pitha Kasam
3. Silethuma Kasam
4. Ratha Kasam
5. Kshaya Kasam

**Ashtanga Hrudhayam says,**

There are five types. These are,

1. Vadha Kasam
2. Pitha Kasam
3. Kaba Kasam
4. Shadha Kasam
5. Kshaya Kasam

**Jeeva Rakshamirtham Says,**

There are 5 types of Kasam. These are,

1. Vadha Kasam
2. Pitha Kasam
3. Sileshma Kasam
4. Ratha Kasam
5. Kshaya Kasam



## Madhava Nidhanam @ Roga Vrichayam says

There are 5 types of Kasam, Those are,

1. Vadha Kasam
2. Pitha Kasam
3. Kaba Kasam
4. Shadha Kasam
5. Kshaya Kasam

## Roga Nirnaya Saram says

There are 5 types of Kasam. They are

1. Vadha Kasam
2. Pitha Kasam
3. Sileshma Kasam
4. Ratha Kasam
5. Kshaya Kasam

## SWASA KASAM:

### Kuri Gunangal:

The signs and symptoms are described in many siddha literatures.

They are described as follows,

### Yugi Vaidhya Chintamani Says,

"Áñ"ÁÂ;ö s,i"Æ,ðÊ pÕÁç Å£ðö  
Á;ç; ,ö sÀ;ÄSÁÂ;ö ìíí Á;°ö  
¼çñ"ÁÂ;ö |°ÖÁÖñ¼; ÁÊì, Êììì  
°£Ä½Áçø Ä;ÁSÄÄ ÂçÚ °Ðö  
çÝ"ÁÂ;ö ç;°çÂÐ ¼ÉøSÄ; Ä;ìö  
çÇçóÐ ¼öÒÄüÈçÁÖí ÌÄÖí ,öÓö  
-ñ"ÁÂ; Öñ½;ì, ç æÚí s,½ç  
-ÆÚSÁ ÍÁ;° ,i °ð¼ç |É;ôSÄ"



**Sikitcharathna Deepam @ Vaidhya Chinthamani** says,

«¼ç, Á;É §, i"ÆÔ¼ý þÕÃø, ç; , ôÀ;õÀçý °εÈ"Ä ´ò¼  
ÍÁ; °ò¼çý °ò¼õ, °εÃ½Áçý"Á, ÅÂçÚÔÀç°õ, ç; °çÂçø «É§Ä;Î  
ÅÈø°ç, -¼ø ÅüÈø, ÌÃü , õÃø, -ñ½;î , çø ÅÆÅøÀ;É çεåÈø

Cough with expectoration of large quantity of sputum, breath sound like hissing of snake, indigestion, flatulence, dryness and heat in the nose, emaciation, low pitched voice, viscous salivation.

**Raja Vaidhya Bodhini - Part I** says

Å;¼ ç;ÉÔõ, Àçò¼ ç;ÉÔõ, ´ÕíÎ §°÷óÐ -"Á"Âô §Ä;ø  
|ÁøÄ °Èç ç¼î , çø, ÅÂçÚ þ"Ãî°ø, «ýÉî |°ÃçÂ; "Á, §, i"Æ  
, õ¼ø, þÕÃø, ç; °ç ÅÃø¼ø, ÌÃü , õÃø, ÓÐ| , Öõò ±Ãçî°ø, §Äü  
ÍÁ; °õ.

While the vadha pulsation and pitha pulsation together is felt like the movement of tortoise, Swasakasam characters are flatulence, indigestion, mucoid sputum, cough, dryness of nose, low pitched voice, burning sensation along the vertebral column, dyspnea.

**Mukutra Verupadugal (Pathology)**

In siddha system of medicine, the manifestation of all the diseases are the result of derangement of Doshas ie. Vadha, Pitha, Kaba. The prime factor which is involved in Swasakasam is Kaba, which is accompanied with vitiated Vadha or Pitha and produce clinical symptoms of Swasakasam. This is clearly indicated by Theraiyar as,

" , Àò¼ç"É ÂýÈç , i° ÍÁ; °õ , i½;Ð "  
- §¼"ÃÄ÷

1. Excess of Kaba in the respiratory organs affect the Melnokku kal and Uyir kal and so the vayu is not able to reach the terminal points of respiration which producing gasping and laboured breathing.

2. Some authors say that the disease is caused by deranged Vadha. This thought is also acceptable because the destruction of Vayu in the respiratory tract is abnormally present.
3. Excessive intake of Vadha prompting diet induces Pitha Kutram. This type of Pitha produce more heat and this heat goes to head resulting in running nose, heaviness of head and neck, sneezing and also induces formation of water vapours in the lungs and causing narrowing of air passage, which leads to the onset of the disease. This is indicated as,

"Àçò¼§Á ÁçÏó¼i Ä£"Ç  
 ÂçÕÁÖö |ÄÄòÐ ççüÏö"  
 - §çïö ç¼¼ø §çïö Ó¼ø ç¼¼ø

So the changes in the diet and habits which increase Vadha and Kaba produce the clinical symptoms of Swasa Kasam.

In Uyir Nilaigal, Anagatham (chest) which is the residence of Udhanan (Melnokku kal) and Pranan (Uyir kal) is deranged.

When Pranan, the primary vayu is affected it leads to difficulty in breathing and involvement of Udhanan leads to cough and sneezing. Involvement of Kirugaran leads to running nose, cough, sneezing. Involvement of Devathathan leads to tiredness. Involvement of Samanan causes inability to control the other Vayus and causes loss of appetite. Involvement of Sadhaga pitham leads to sluggishness. In Kaba, the derangement of Avalambagam leads to dyspnea, cough, wheezing. In the seven Udal Thathus, Saaram, Senneer are affected which leads to lethargy and depression. In severe cases Oon and Kozhuppu are also affected leads to symptoms of emaciation and body pain.

## Piniyari Muraimai (Diagnosis)

The way of diagnosis is very important by which a physician can deal the disease, then only he will rule out the cause of the disease which is the main thing to be treated. Thiruvalluvar said,

"ṣḷḷḷḷḷḷ ṣḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ  
Ḗḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ Ḗḷḷḷḷḷḷ"

The diagnosis is based on four criterias

1. Poriyal arithal
2. Pulanal arithal
3. Vinathal
4. En Vagai Thervugal

### 1. Poriyal arithal:

Porigal are the five organs of perception. They are Eyes, Ears, Nose, Tongue and Skin. Poriyal Arithal is examining the Pori of the patient by the Pori of the physician. In swasa Kasam, it is as follows,

Mei (Skin)	:	Normal
Vai (Tongue)	:	Excessive salivation
Kann (Eye)	:	Some times affected (redness)
Mookku (Nose)	:	Running nose
Sevi (Ear)	:	Normal

## **2. Pulanal Arithal:**

Pulanal are the five objects of senses.

Ooru (Sensation)	:	Warmth
Oosai (Sound)	:	Normal
Ozhi (Vision)	:	Normal
Suvai (Taste)	:	Normal
Natram (Smell)	:	Altered or absent due to running nose and inflammation of nasal mucosa.

## **3. Vinadhah:**

By Vinadhah, the physician knows about the patient's Name, Age, Occupation, Native place(Thinai), Family history, Socio - economic status, Diet habits, Prone to any allergens (eg: dust, smoke, pollens), His complaints, History of previous episodes, Frequency of attacks by changes in season, Relevant history of treatment and Habits etc.,

### **Kaalam (Age Distribution)**

The period of human life is totally 100 years. This is divided into three stages, according to the domination of three humours. As per this

1. Vadha Kaalam - 1 to 33 years
2. Pitha Kaalam - 34 to 66 years
3. Kaba Kaalam - 67 to 100 years

Even though in each of these stages, the other humours are also involved a particular humour is dominating more. According to this, Kaba types of diseases are more prone in the later stage (Kaba Kaalam).

### **Ivagai Nilangal:**

Study of Ivagai Nilangal is very important and useful because there may be possibility of the disease in some area. Ivagai Nilangal are,

- |             |   |                                |
|-------------|---|--------------------------------|
| A. Kurinchi | - | Mountains and its surroundings |
| B. Mullai   | - | Forests and its surroundings   |
| C. Marutham | - | Plains and its surroundings    |
| D. Neithal  | - | Seas and its surroundings      |
| E. Palai    | - | Deserts and its surroundings   |

### **Kurinchi:**

“குறிஞ்சி வருநிலத்திற் கொற்றமுண்டி ரத்தம்  
உறிஞ்சி வரு சுரமுண்டாம்- அறிஞருரைக்  
கையமே தங்குதரத் தாமைவல்லை யுங்கதிக்கும்  
ஐயமே தங்கும் அறி”

- பதார்த்த குண சிந்தாமணி

In Kurinchi Nilam, people are affected by fever that reduces blood level in the body, diseases related to spleen and liver and mainly by Kaba diseases.

### **Mullai:**

“முல்லை நிலத்தயமே ரிநிரை மேவினுமவ்  
வெல்லை நிலைத்தபித்த மெற்குறுங்காண்- வல்லையெவனின்  
வாதமொழி யாதத னுண்மன்று மவை வழிபோய்ப்  
பேதமொழி யாதறையப் பின்பு”

- பதார்த்த குண சிந்தாமணி

Though Mullai Nilam is the place of cattles, it is the place of increasing Pitha, Vadha also joined to that Pitha due to these Kutrams many diseases occur.

### **Marutham:**

“மருதநில நன்னீர் வளமொன்றைக் கொண்டே  
பொருதனில மாதியநோய் போக்கும் - கருதநிலத்  
தாநிரதஞ்சூழ அருந்துவரென் றாற்பிணியெல்  
லேறிரதஞ் சூழ்புவிக்கு மில்”

- பதார்த்த குண சிந்தாமணி

Marutha Nilam, due to its water sources, cures all the three Vatha, Pitha and Kaba diseases.

**Neithal:**

“நெய்தனில் மேலுப்பை நீங்கா தூறினுமது  
வெய்தனில் மேதங்கு வீடாகும் - நெய்தல்  
மருங்குடலை மிக்காக்கும் வல்லுறுப்பை வீக்கும்  
கருங்குடலைக் கீழிறக்குங் காண்”

- பதார்த்த குண சிந்தாமணி

Through Neithal Nilam has the dominant taste of Uvarppu (Salty), it is the place of Pitha Vayu. The people who dwell here are susceptible to odema due to Kaba, Silipatha Rogam (Filariasis), Kudalanda Viruthi (Hernia).

**Palai:**

“பாலை நிலம்போற் படரைப் பிறப்பிக்க  
மேலைநில மியாது விரித்தற்கு - வேலைநில  
முப்பிணிக்கு மில்லாம் முறையே யவற்றகலாம்  
எப்பிணிக்கு மில்லா ம.:தெண்”

- பதார்த்த குண சிந்தாமணி

The Palai Nilam is the place for grief and place of deadly Vadha, Pitha and Kaba diseases.

**PARUVA KAALAM (Season)**

As the earth revolves around the sun once in a year, the earth gets sunlight at various positions. With reference to the position of the earth towards the sun, year is divided into 6 seasons.

They are,

1. Karkaalam (Avani & Purattasi) : August & September
2. Koothirkaalam (Iyppasi & Karthigai) : October & November



3. Munpanikaalam (Margazhi & Thai) : December & January
4. Pinpanikaalam (Masi & Panguni) : February & March
5. Elavenilkaalam ( Chithirai & Vaigasi) : April & May
6. Mudhuvnilkaalam ( Aani & Aadi ): June & July

According to literature, Swasa kasam comes during rainy season (Karkaalam). In Koothirkalam due to cold wind, is also prone to the disease.

Swasa kasam mainly occurs due to vitiation of Kaba. Kabam thannilai sirappurum Kaalam - Karthigai to masi.

முவுரு மீறி முனிவு கொளாமல்  
 தத்தம் நிலையில் தன்னரசியலும்  
 காலவரைதனை கிளரக் கேண்மின்  
 ஆடியாதியாய் ஐப்பசி ஈராய்  
 ஆனிலமதற்கோ ரரசியல் காலம்  
 மீன் முதலாளி வீறுகொள் மந்திரி  
 தேள் முதன் மாசி சேனாபதிக்கே  
 - நோய் நாடல் நோய் முதனாடல்

Hence the disease can occur in the later part of Koothir Kaalam to early part of Pinpani Kaalam, (i.e.) from the last two weeks of October to the first two weeks of February.

Totally the prevalence of disease is from August to February.

## **7. MUKKUTRA NILAIGAL:**

### **Vadham**

#### **Pranan:**

It is responsible for respiration.

In Swasa kasam, Vayu is affected leading to difficulty in breathing.

#### **Abanan:**

It helps in excretion of urine and motion.

In Swasa kasam, some patients had constipation.

#### **Viyanan:**

Its main function is distribution of saaram.

In Swasa kasam, this distribution is affected, thus emaciation occurs.

#### **Samanan:**

Samanan is the vayu that controls other vayus and digestion.

In Swasa kasam, this vayu is affected since it cannot control the other vayus.

#### **Udhanan:**

Its main function is inspiration and expiration and distributes the saaram equally to all tissues.

In Swasa kasam, this vayu is affected due to difficulty in breathing.

#### **Nagan:**

This vayu maintains opening and closure of eye lids and is not affected in Swasa kasam.

#### **Koorman:**

This vayu is responsible for vision and yawning and is not affected in Swasa kasam.

**Kirugaran:**

It causes salivation, running nose, sneeze, and cough and maintains appetite. In Swasa kasam, this vayu is deranged causing running nose, sneeze, cough and loss of appetite.

**Devathathan:**

It is responsible for tiredness, anger and emotional expression. In Swasa kasam, this vayu is deranged causing emotional stress and insomnia.

**Dhananjeyan:**

It produces swelling of the body after death and escapes through the scalp after the third day of death.

**PITHAM****Anal pitham:**

This lives in the stomach and helps in digestion. In Swasa kasam, most of the patients complained loss of appetite & indigestion.

**Ranjagam:**

This is residing in stomach and gives colour to the blood. In Swasa kasam, some patients are anaemic.

**Sadhagam:**

It resides in the heart and executes the day to day activities with the help of mind and brain. In this disease, restlessness, breathlessness present.

**Aalosagam:**

It resides in both eyes and is responsible for clear vision.

**Prasagam:**

It resides in skin and gives complexion.

In Swasa kasam, some patients have itching in any part of the body.

## **KABAM**

### **Avalambagam:**

It is residing in lungs and helps other four types of Kaba to function and also helps in the function of heart.

It is deranged in Swasa kasam patients, since the presence of tightness of chest, cough, wheezing, and dyspnea.

### **Kilethagam:**

It is present in the stomach and gives moisture to the food materials and also helps in digestion.

In this disease, some patients have indigestion.

### **Pothagam:**

Living in the tongue and responsible for taste sensation, is not affected in Swasa kasam patients.

### **Tharpagam:**

Living in the head and keep the eyes cooling.

In Swasa kasam, there may be redness of eyes.

### **Sandhigam:**

It resides in the joint and helps for free movements.

## **UDAL KATTUGAL:**

### **Saaram:**

It is the energy part of end product of digestion.

It strengthens the body and mind. It is deranged in Swasakasam due to loss of appetite causing tiredness in the body and mind and causes emaciation

**Senneer:**

It is responsible for knowledge, strength, boldness and healthy complexion. This is deranged in some patients with anaemia.

**Oon:**

It gives the structure to the body and is responsible for the movement of the body and is not affected in Swasa kasam.

**Enbu:**

It gives the shape to the body and is responsible for motion of the body is not affected in Swasa kasam.

**Kozhuppu:**

When the organs are doing their work this gives lubrication and facilitates their work, is not affected in Swasa kasam.

**Moolai:**

It is present in the core of the bone which strengthens and maintains the normal condition of the bone, is not affected in Swasakasam.

**Sukkilam / Suronitham:**

It is responsible for reproduction.

When the seven Udal Katukal increase or decrease from the normal level, the normal functioning of the body is affected.

**4. EN VAGAI THERVUGAL:**

It is the basic diagnostic principle and the uniqueness of the Siddha system of Medicine. The following lines reveal this as follows.

“நாடிப்பரிசம் நாநிறம் மொழிவிழி

மலம் முத்திரமிவை மருத்துவ ராயுதம்”

- நோய் நாடல் நோய் முதனாடல்

And,

“மெய்க்குறி நிறந்தொனி விழி நாவிருமலம் கைக்குறி”

- நோய் நாடல் நோய் முதனாடல்

The diagnostic value of EN VAGAI THERVUGAL is specific to Siddha system of Medicine and presumes the vitiated doshas in the patients.

En Vagai Thervugal are

- a. Naa (Tongue)
- b. Niram ( Colour of the skin)
- c. Mozhi ( Speech)
- d. Vizhi ( Eye)
- e. Malam ( Motion)
- f. Moothiram (Urine)
- g. Sparisam ( Palpation)
- h. Naadi ( Pulse)

**a. Naa**

It is noted for its colour, ulcer, growth, coating, colour and consistency of the sputum that is spitted from mouth, mode of speech.

In Swasakasam, patients have the sputum scanty and mucoid.

**b. Niram**

Colour of skin, conjunctiva, and teeth.

In Swasakasam, the colour of the skin, conjunctiva, may be pale. In some patients, the conjunctiva may be red due to conjunctivitis.

**c. Mozhi**

Generally, speech is generated from the voice box. Abnormalities are low pitched speech, lalling, diplegia, monotonous speech, jerky, scanning, indistinct, lisping.

In Swasa kasam mode of speech may be emotional or difficulty in speech, low pitched voice, wheezing sound is heard.

**d. Vizhi**

Type of eye - redness, ulcer, pallor, protrusion, tears, shedding of eye lashes, excreta of eye, diseases of eyes are noted.

In Swasa kasam, the eyes may be red.

**e. Malam**

Consistency hard or semisolid or diarrhoea, undigested food, fluid resembling the water used to clean meat, colour, frothy, dysentery, bloody, pus, mucous, smell, frequency of defaecation, constipation, reduced or increased stool content, lower abdominal pain during defaecation are noted.

In Swasa kasam, the patients may be constipated.

**f. Neer @ Moothiram**

Colour - yellow, black, white copper coloured, mixed colour, colour of fumes, smell-smell of fire, honey, sweet odours, fragrance of flowers, fruity odour, odour of deer flesh, frothy or not, frequency and quantity are noted.

In Swasa kasam it may be transparent and frothy.

**g. Sparisam**

Heat or coldness of the body.

It may be cold due to sweating in this disease.

**h. Naadi**

Naadi is the very important helpful observation for diagnosis and prognosis.

In **Noi Naadal, Noi Mudhal Naadal** Text, Naadi is defined as,

“ உடலில் உயிர் தரித்திருப்பதற்குக் காரணமான சீவசக்தி  
எதுவோ அதுவே தாது அல்லது நாடி எனப்படும்.”

## Genesis of Naadi:

The three Uyir Thathukkal are formed by the combination of three Naadis of Dhosha and three Vayus of Dhosha.

Idakalai + Abanan = Vatha

Pinkalai + Pranan = Pitha

Suzhumunai + Samanan = Kaba

This can be felt one inch below the wrist on the radial side by means of palpation by the three fingers - index, middle and ring fingers corresponding to vadha, pitha and kaba respectively.

“கரிமுகனடியை வாழ்த்திக் கைதனில் நாடிபார்க்கில்  
பெருவிரலங்குலத்தில் பிடித்தடி நடுவே தொட்டால்  
ஒரு விரலோடில் வாதமுயர் நடுவிரலிற் பித்தம்  
திருவிரல் மூன்றிலோடில் சேத்தும நாடிதானே”

- அகத்தியர் நாடி

## Naadi Nadai in Swasa Kasam:

### Vatha Kaba Naadi:

“பாங்கான வாதத்தில் சேத்தும நாடிப்  
பரிசித்தால் திமிர்மேவு முளைச்ச லாகும்  
தீங்கான இருமலுடன் சந்தி தோடம்  
சேர்ந்த விடம் வெடிகூலை யிருத்ரோகம்  
வாங்காத ஈளை மந்தார காசம்  
வலியுடனே புறவீச்சுயள் வீச்சு வீக்கம்  
ஓங்காணுஞ்சுர முடனே **கவாச காசம்**  
உண்டாகும் வெகு நோய்க்கு முறுதிதானே”

- சதக நாடி

### Iya Naadi:

“தானமுள்ள சேத்து மந்தானிளகில் வெப்பு,  
சயமீளை யிருமல் மந்தார காசம்  
ஈனமுறுஞ் சந்நிவிட தோடம் விக்கல்  
யிருத்ரோகங் கரப்பான் விரண தோடம்



மானனையீர் சூலைதிரள் வியாதி வீக்கம்  
வருஞ்சக்தி **சுவாசம்** நெஞ்சடைப்பு தூக்கம்  
ஏனமுறுங் காமாலை பாண்டு சோபை  
ஏழுசுரங்கள் பலதுக்கம் விட முண்டாமே”

- சதக நாடி

### **Kaba Pitha Naadi:**

“இடமான சேத்துமத்தில் பித்த நாடி  
எழுந்தணுகில் விடமுடனே வீக்கமுண்டாம்  
திடமான குளிர் காய்ச்சல் மஞ்சள் நோவுந்  
தேகத்தி லுளைச்சலிளைப் பிருமல் வாந்தி  
விடமான நெஞ்சடைப்பு **சுவாசம்** விக்கல்  
வெகு சுரமும் நாவறட்சி பாண்டுரோகம்  
அடமான குவளைரத்த மதிசாரந்தான்  
அணுகி வெகுபல நோய்க்குந் தடங்கண்டாயே”

### **Iya Ushnam:**

“கதிப்பான சேத்துமத்திலுட்டிணங் கூடல்  
கலந்த குணஞ்சயமிருமல் **சுவாசகாசம்**  
மதிப்பான கோழைரத்தம் விப்புருதியுடனே  
வளர்நாசிகா பீடமிருத் ரோகம்  
கொதிப்பான சிங்ஙவையாக் கிராணவாயு  
கொட்டாவி விக்கல் மந்தாரகாசம்  
துதிப்பான வீரலத்திக் காய்வுரத்தம்  
தோன்றுமிகு பிணிபலவுந் தொந்திப்பாமே”

- சதக நாடி

### **Iya Vayu:**

“தொந்தித்த சேத்துமத்தில் வாயுகூடித் தொடர்ந்த  
குன்மம் நெஞ்சடைப்பு **சுவாசகாகம்**  
வந்தித்த குரல்தனிலே உறுத்த லீளை  
வழுவழுப்பு நீருறல் மலத்தில் சீதம்  
வெந்தித்தல் கொழுத்தல் குத்துந் திமிர்வியாதி  
வீச்சுடனே வலியெட்டுந் திரட்சி பாண்ட  
அந்தித்த கிறுகிறுப்பு மயக்கம் விக்கல்  
ஆனபல பிணிகளுமே வந்தட ருந்தானே”

- சதக நாடி

Hence the Naadi Nadai in Swasa kasam is Kaba, Vadhakaba, Kaba Pitha, Iya Ushna, and Iya Vayu Naadis.

### Nei Kuri:

This urine examination is unique in Siddha system of Medicine. For this examination urine is collected in the early morning in a pure glass vessel. Patient is advised to avoid excessive diet, and in take of diet during irregular timings on the previous day of examination.

“அருந்துமாறிரதமும் அவிரோதமதாய்

அ.கல் அலர்தல் அகாலவூன் தவிர்ந்தழற்

குற்றளவருந்தி உறங்கி வைகறை

ஆடிக்கலசத் தாவியே காது பெய்

தொருமுகூர்த்தக் கலைக்குட்படு நீரின்

நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே”

- சித்தமருந்துவாங்கச்சுருக்கம்

“நிறக்குறிக் குரைத்த நிருமாண நீரிற்

சிறக்க வெண்ணையோர் சிறுதுளி நடுவிடுத்

தென்றுத் திறந்தொலி யேகாதமைத்ததி

னின்றதிவலை போம் நெறிவிழியறிவும்

சென்றத புகலுஞ் செய்தியை யுணரே”

- நோய் நாடல் நோய் முதனாடல்

A drop of gingelly oil is dropped on a wide glass vessel containing the urine to be tested which is kept under sunlight in a calm place. The derangement of the three dhoshas can be diagnosed by the mode of spread of gingelly oil on the surface of urine.

“அரவென நீண்டின் அ.தே வாதம்”

“ஆழிபோற் பரவின் அ.தே பித்தம்”

“முத்தொத்து நிற்கின் மொழிவதென் கபமே”

- நோய் நாடல் நோய் முதனாடல்

- ❖ Oil spreading like a snake indicates Vatha.
- ❖ Oil spreading like a ring indicates Pitha
- ❖ Oil spreading like a pearl indicates Kaba

“அரவிலாழியும் ஆழியில் அரவும்  
அரவில் முத்தும் ஆழியில் முத்தும்  
தோற்றில் தொந்த தோடங்களாமே”

- நோய் நாடல் நோய் முதனாடல்

Oil spreading like snake and ring, ring and snake, snake and pearl, ring and pearl all comes under Dhondha Dhosham.

In Swasa kasam, most of the Nei Kuri findings result- pearl like oil floating on the urine.

**DISEASES SIMILAR TO SWASAKASAM are,**

**Mandara Kasam**

“தானான தூயதோர் நாசி தன்னில்  
சலநோய் நீர்தான் விழுந்த தும்மலுண்டாம்  
மானான மார்புநெஞ் சடைத்து முச்சு  
வலுவாக பாம்புபோல் சீற லாகும்  
கானான கண்டமோடு முகமுங் காதும்  
காயமதுங் கசிவாகி வியர்வை யாகும்  
ஏனான இருமலோடு கோழை கம்மல்  
இரைப் பாகு மந்தாரகாச மாமே”

- யூகி வைத்திய சிந்தாமணி

In Mandara kasam there is running nose, sneezing, tightness of chest, breath sound like hissing of snake, sweating all over the body, cough, expectoration, dyspnea, etc.,

In Swasa kasam there is no sweating all over the body.

## Kandakiragam:

“வகையான குரலதனைப் பற்றி நொந்து  
மார்போடு பிடரியினில் வலி யுண்டாகி  
நுகரான சரீரமெல்லாம் நொந்த ழாற்றி  
நுணுக்கமாய்ச் சுவாசமது புறப் படாமல்  
முகையான நாவாவே மூச்சு மாறி  
முகத்திலே வியர்வாகி விலாநோ வுண்டாம்  
புகையான வன்னத்தைப் பருகொட் டாது  
பரியகண்ட கிரகத்தின் பண்பு தானே”  
- யூகி வைத்திய சிந்தாமணி

In Kandakiragam, there is difficulty in speech, pain in the chest and occipital region, pain all over the body, breathlessness, mouth respiration, sweating in face, pain in the ribs, anorexia.

In Swasa kasam, there is no pain in the occipital region.

## Silethuma Vadha Suronitham:

“பண்பாக வுடல்குளிர்ந்து வயிறு வீங்கிப்  
பதைப்பான விடந்தொட்டாற் போல் நோவாந்  
திண்பான சிரசு நெற்றி நோக்கா டுண்டாஞ்  
சிலேட்டுமமாய்க் கோழையோடு சுவாசமாகும்  
மண்பாக மயக்கமொடு கனவு முண்டாம்  
வாய்வறண்ட ருசியில்லா வருத்த மாகும்  
நண்பாக நாடியுமே படப படக்கும்  
நற்சிலேட்ம கரோணிதமாம் நாடுங் காலே”  
- யூகி வைத்திய சிந்தாமணி

In Silethuma Vadha Suronitham, there is chillness of body, abdominal distension tenderness in abdomen, headache, expectoration, dyspnea, fainting, dreaming, decreased salivation, loss of taste, rapid pulse etc.,

In Swasakasam, there is no abdominal distention and decreased salivation.

## Swasa Pitham:

“கருத்தாகச் சுவாசமது மிகவுண் டாகுங்  
கனமாக வயிறுமே ஊதிக் காணும்  
உருத்தாக உடலதுதான் மிகவ லிக்கு  
முறுமே கேணி போல் வாய்நீர் தானும்  
மருத்தாக மயங்கியே கண்ம றைக்கும்  
மார்பிலே வலியோடு இரும லுண்டாந்  
துருத்தாக வயிறதனின் பசியோ வில்லை  
சுவாசமாம் பித்தத்தின் சூட்சந் தானே”  
- யுகி வைத்திய சிந்தாமணி

In Swasa Pitham, there is increased respiration, flatulence, pain all over the body, brashing, loss of consciousness, pain in the chest followed by cough, loss of appetite etc.,

In Swasa kasam there is no loss of consciousness.

## Swasa Silethumam:

“திறமையாய் நெஞ்சுதனிற் கோழை கட்டுஞ்  
சிக்கென்று தானிருமி மூக்க டைக்குங்  
குறுமையாய் குற்ட்டென்று சுவாசங் காணுங்  
குளிரோடு சுரமுண்டாய் மயக்க மாகும்  
மறமையாய் மார்போடு நெஞ்ச டைக்கும்  
வாய்வறண்டு மூக்கதனில் நீரே பாயும்  
வெறுமையாய் மிகத்தண்ணீர் தாப முண்டாய்  
விடுசுவாச சிலேட்டுமத்தின் விவரந் தானே”  
- யுகி வைத்திய சிந்தாமணி

In Swasa Silethumam, there is congestion in lungs, nasal block, cough, dyspnea, fever with rigor, syncope, tightness of chest, dryness of mouth, running nose, excessive thirst etc.,

In Swasa kasam, there is no fever with rigor, excessive thirst etc.,

## LINE OF TREATMENT:

The line of treatment of **Swasa Kasam** consists of the following.

1. Kalichal Maruthuvam - to bring the dhoshas in equilibrium
2. Internal Medicine - Mainly anti-spasmodic, expectorant, to relieve the spasm and to expel the sputum.
3. Diet - to maintain tridhoshas and energy.
4. Prevention methods - to strengthen the muscles of respiration (Prahnayama)
5. Yoga therapy - to maintain dhasa vayukkal and to improve mental and physical health.

### 1. Kalichal Maruthuvam (Purgation)

Patients have to be given laxative Nilavagai Chooranam 5 gm with hot water at the bed time of the previous day, before starting the internal medicine.

### 2. Administration of internal medicine

For the treatment of the disease Swasakasam, Mungiluppu choornam 4gm twice daily with honey after meals.

### 3. Diet

Siddhars advice the diet regimens for Kaba patients and they are explained below.

“கத்தரி பேய்ப்புடல வரை யிருபாகல் பருங்காளா கண்டகாரி  
அத்திக் காய்களும் வருக்கைமாயற்றை கரையால் பீர்க்கரும்  
பிஞ்சுவேர்  
மொய்த்த குரணங் கதலித் தண்டுகளைப் பூமுளங்கி  
முருக்கரும்பும்  
அத்தி பூசணிக் காயருள்ளி வள்ளியுங் கபத்தோர்க் காணாமே”

- பதார்த்த குண சிந்தாமணி

“வேளை மணத்தக்காளி மென்சீதை சக்கரவர்த்தி  
பீளை வசலை சுக்கு பெண்கணங்கள் - வேளையில்  
செந்தளிர் களைக்கீரை செய்வர் கபதேகர் நிதம்  
வந்தளியுணத்தான் மகிழ்ந்து”

- பதார்த்த குண சிந்தாமணி

### Vegetables to be added

- ❖ கத்தரி (Solanum melongena)
- ❖ பேய்புடல் (Trichosanthes cucumerina)
- ❖ அவரை (Dolichos lab-lab)
- ❖ கண்டங்கத்தரி (Solanum xanthocarpum)
- ❖ வாழைக்காய் (Musa paradisiaca)
- ❖ முருங்கை (Moringa tinctoria)
- ❖ சுண்டை (Solanum torvum)

### Tubers to be added

- ❖ இஞ்சி (Zingiber officinale)
- ❖ முள்ளங்கி (Raphanus sativus)
- ❖ கருணைத்தண்டு (Amorphophallus companulatus)

### Greens to be added

- ❖ மணத்தக்காளி (Solanum Nigrum)
- ❖ கரிசாலை (Eclipta alba)
- ❖ பீளை (Aerva lanata)
- ❖ வசலை (Basella alba)
- ❖ சிறுக்கீரை (Amaranthus gangeticus)
- ❖ மணலிக்கீரை (Gisekia pharmacoides)
- ❖ புளியாரைக்கீரை (Oxalis corniculata)

## Diet Restriction

Siddhars advised to avoid certain food items during diseased conditions.

They are,

“கடுகு நற்றிலத் தெண்ணெய் கூழ்பாண்டங் கடலை  
வடுவ தாகிய தெங்குமா வருக்கை நற்காய  
மழவி லாதவெள் ளுள்ளிகொள் புகையிலை மதுபெண்  
இடறு பாகவோ டகத்தி நீக்கிடலிச் சாபத்தியம்”

- தேரையர் வெண்பா

- ❖ Mustard
- ❖ Gingelly oil
- ❖ Bengal gram
- ❖ Coconut
- ❖ Mango
- ❖ Asafoetida
- ❖ Garlic
- ❖ Horse gram
- ❖ Tobacco
- ❖ Alcohol
- ❖ Bitter guard
- ❖ Sesban
- ❖ And also, they are advised to avoid coitus

These are general diet and habitual restrictions for all diseases.

## Prevention

1. Avoid chill and cold weather
2. Avoid working in dust, cement, cotton mills and in husks.
3. Avoid smoking
4. To sleep in phoenix mat, prevent Kaba diseases.



“சிற்றீச்சுப் பாயிற் றினமும் படுப்பவருக்  
குற்றிடுமே காந்த லுடம்புவருஞ்-சுற்றியதோர்  
வாயுவுறும் பித்தமறு மற்றுங் கபந்தீருந்  
தாயகமா மிக்குணத்தைச் சாற்று”

- அகத்தியர் குணவாகடம்

## 5. Advice to practice Prahnyamam

### **PRAHNAYAMAM (Breathing Exercise)**

Prahnyamam or breathing exercise mainly consists of Pooragam (inhalation of air by deep inspiration), Kumbagam (holding the breath as far as possible) and Resagam (exhalation of air by expiration)

By this exercise, the duration of Kumbagam is increased. This results in proper gaseous exchange which provides increased oxygen supply to the cells.

By the regular practice of Prahnyamam, one can get a feeling of reduced both mental and physical stress and enjoy pleasure. It provides good concentration and meditation. This practice also gives good appetite, strength, enthusiasm, rigor and vitality.

During breathing exercise, the lungs fill with fresh air in its anatomical dead space also and expand well and get proper supply of oxygen by proper expansion of chest. So, Prahnyama practice is one of the prevention for Swasa Kasam.

“நாளொன்றுக்கு இருபத்தோராயிரத்து அறுநூறு  
நலமான சுவாசந்தானெ முந்திருக்கும்  
கோளொன்றிப் பதினாலாயிரத்து நானூறு  
குவிந்த மூலாதாரத்துள் ளொடுங்கும்  
பாளொன்றி யேழாயிரத்திருநூறு சுவாசம்  
பாழினிற் பாய்ந்திடுமென் றறிகப்பின்னை  
ஏளொன்றி யிதனையே யுட்சாதித்தால்  
எப்பொழுதும் பாலரா யிருக்கலாமே”

- யூகி வைத்திய சிந்தாமணி

## **YOGA THERAPY**

Yogasana is one of the most spiritual legacies gifted by our ancient sages. The practice of asanas strengthens the body and mind.

Asanas strengthen the muscles of respiration and diaphragm as well as regulate respiration. So, practising asanas is more helpful in asthmatic patients as supportive therapies. The following asanas are helpful in Asthma.

- ❖ Bujankasanam
- ❖ Chakrasanam
- ❖ Machasanam
- ❖ Mayurasanam
- ❖ Patha hasthasanam
- ❖ Arai machayendhirasanam
- ❖ Trikonasanam
- ❖ Savasanam

## MODERN ASPECTS

### Anatomy and physiology of respiratory system

#### **Introduction:**

It is by means of breathing that every cell in the body receives its supply of oxygen and at the same time gets rid of  $\text{CO}_2$  as products of oxidation. Respiration is a two – fold process where by the interchange of gases takes place in tissues called **internal respiration** and in the lungs known as **external respiration**.

#### **Nose:**

The nose is divided into 2 nostrils by a nasal septum. The external opening of the nose (anterior nares) opens into the vestibule of the nose. The inner lining of vestibule contains a number of coarse hairs which project downwards. These nasal cavities are lined with highly vascular mucous membrane that continuous with that of pharynx and with that of the paranasal sinuses.

The filaments of the olfactory nerve arise from small receptors scattered in the mucous membrane of the nasal cavity. Apart from these special sensory nerve endings, there are touch and pain sensitive nerve endings distributed in the nasal lining that help in the protective reflex of sneezing.

#### **Pharynx:**

This is common muscular tube that lies behind the nose and the mouth. This pharynx is guarded by tonsils at the sides and adenoids at the top. Tonsils and adenoids are collections of white blood cells which act as a defense army - guards by fighting away any dangerous germs that may contaminate the inhaled air. These wbc in fact surround the bacteria to digest them away and themselves die in the process to be replenished

soon. The muscles of pharynx are chiefly useful in swallowing. The pharynx opens in to the larynx in front and esophagus behind.

### **Larynx:**

Larynx is composed of pieces of cartilages to maintain its shape and patency. Attached on the top is a small piece of cartilage called epiglottis that closes the larynx like a lid during swallowing so that the food glides over it in to the esophagus and not into the trachea. The vocal cords are thin flaps that project from the side wall of the larynx. The movements of these flaps are controlled by highly efficient groups of delicate laryngeal muscles. The vibration of these cords due to the air passing through the glottis cause 'voice' production.

### **Trachea:**

The trachea (wind pipe) about 10cms long extends from the larynx to divide in to right and left bronchi. These are incomplete rings of cartilages in the wall to maintain the patency under varying intra thoracic pressure conditions occurring during breathing. The trachea divides in to right and left bronchi which enter the corresponding lungs.

### **The Lungs:**

The lungs, two in number are the principal organs of respiration. They fill the chest cavities one on each side separated in the middle by the heart, its blood vessels and other structures. They are divided in to lobes by fissures, the right has 3 lobes and the left has 2 lobes.

### **Bronchi:**

The right and left bronchi are formed by the bifurcation of the trachea. The right bronchi is shorter and divides in to 3 branches to enter the 3 lobes of the right lung and the left is longer and divides into 2

branches to enter the 2 left lobes of the left lung. These further divide and re divide into a number of bronchi like the branches of tree to ramify into smaller bronchi and finally end as bronchioles that enter the air sacs called alveoli.

The larger bronchi have incomplete rings of cartilages in their wall and they disappear in smaller bronchi. The inner lining of the bronchus has ciliated layer of cells, the movement of the cilia being upwards to remove the finer pollutants that would have escaped the nose. The inner lining also contains mucus secreting cells that secrete thin mucus that provides lubricating layer of moisture and also helps to humidify the air. The most important in the bronchus is the thin circular layer of smooth muscle that runs all the way along the bronchial tree.

The contraction of this thin circular bronchial muscle leads to narrowing and its relaxation to dilatation of the air passages.

### **The Alveoli:**

Alveolus is lined by a single layer of flat cells. It is here, that the blood comes into almost direct contact with the air, being separated only by exceedingly thin permeable membrane consisting of 2 layers of flat cells. Oxygen diffuses from the inhaled air in the air sac to the blood moving very slowly in the capillaries and carbon dioxides moves from the blood to the air sac due to differences in partial pressure of the 2 gases.

Pulmonary artery and veins carry deoxygenated and oxygenated blood from and to the heart respectively. Bronchial arteries supply nourishment to the lungs.

**The pleura:**

Each lung is surrounded by a double layer of membrane called the pleura. The inner layer of the pleura closely surrounds the lung and the outer layer covers the interior of the chest wall. Between the pleural layers there is a thin layer of lubricating fluid to prevent friction.

**Physiological aspects:**

**Mechanics of Breathing:** During normal quiet breathing, inspiration is the active process and expiration is the passive process. During inspiration there is enlargement of thoracic cage and expansion of lungs. During expiration the thoracic cage and lungs decrease in size and attain the pre-inspiratory position.

**Muscles of Respiration:**

Respiratory muscles are of 2 types. Inspiratory muscles and expiratory muscles.

**Primary inspiratory muscles**

Diaphragm

External intercostal muscles

**Primary expiratory muscles**

Internal Inter costal muscles

**Accessory inspiratory muscles**

Stenomastoid, Scaleni, Anterior Serrati, Elevators of scapulae and pectorals.

**Accessory expiratory muscles**

Abdominal muscles.

**Movements of thoracic cage:**

Inspiration cause enlargement of thoracic cage. The size of thoracic cage increased in all diameters. Increase in antero posterior and transverse diameters occur due to elevation of ribs. The vertical diameter of thoracic cage is increased by the descent of the diaphragm.

**Movements of lungs:**

During inspiration, due to enlargement of thoracic cage, the negative pressure is increased in the thoracic cavity this cause expansion of lungs. During expiration the thoracic cavity decreases in size to the pre-inspiratory position. The back pressure in the thoracic cavity also comes back to the pre-inspiratory level. This compresses the lung tissues so that the air is expelled out of the lungs. The lungs also have some – tendency of recoiling, still the lungs donot collapse.

**Pulmonary function test:**

Most of the pulmonary function tests are carried out by using spiormeter.

The air in the lung is classified in to two divisions.

1. Lung volumes which can be breathed by the subjects, the volumes being dynamic.
2. Lung capacities which include two or more primary volumes.

**Lung volumes:**

There are 4 lung volumes, namely

- 1) Tidal volume
- 2) Inspiratory reserve volume
- 3) Expiratory reserve volume
- 4) Residual volume.

**Tidal volume (TV):**

The volume of air breathed in and out of the lungs in a single normal quiet respiration. Tidal volume signifies the normal depth of breathing.

Normal value : 500 ml

**Inspiratory Reserve volume (IRV)**

The additional amount of air which can be inspired forcefully beyond normal tidal volume.

Normal value : 3300ml

**Expiratory Reserve volume (ERV)**

The additional amount of air that can be expired out forcefully after normal expiration.

Normal value : 1000ml

**Residual volume (RV)**

The amount of air remaining in the lungs even after forced expiration.

Normal Value : 1200ml.

**Lung capacities:** There are 4 lung capacities namely.

1. Inspiratory capacity
2. vital capacity
3. Functional residual capacity.
4. Total lung capacity.

**Inspiratory capacity:**

The maximum volume of air that can be inspired starting from the end expiratory position.



$$\begin{aligned} \text{IC} &= \text{TV} + \text{IRV} \\ &= 500 + 3300 = 3800\text{ml} \end{aligned}$$

**Vital capacity:** The maximum amount of air that can be expelled out forcefully after a maximal (deep) inspiration.

$$\begin{aligned} \text{Vc} &= \text{IRV} + \text{TV} + \text{ERV} \\ &= 3300 + 500 + 1000 \text{ ml.} \\ &= 4800 \text{ ml} \end{aligned}$$

**Functional residual capacity (FRC).**

The volume of the air remaining in the lung after normal respiration.

$$\begin{aligned} \text{FRC} &= \text{ERV} + \text{RV} \\ &= 1000 + 1200 \\ &= 2200 \text{ ml} \end{aligned}$$

**Total lung capacity**

The amount of air present in the lungs after a maximum deep inspiration

$$\begin{aligned} \text{TLC} &= \text{IRV} + \text{TV} + \text{ERV} + \text{RV} \\ &= 3300 + 500 + 1000 + 1200 \text{ ml} \\ &= 6000 \text{ ml.} \end{aligned}$$

**Regulation of respiration:**

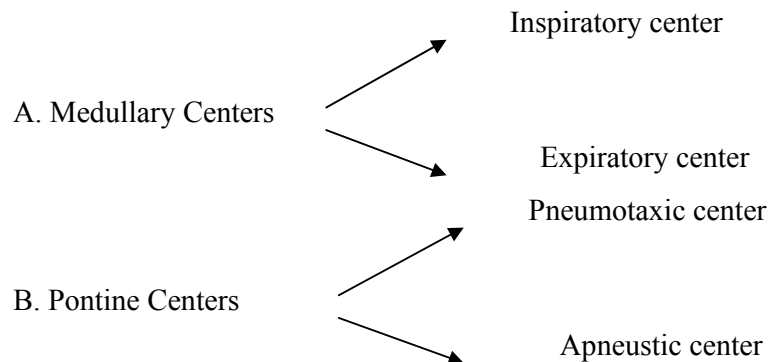
Respiration is a reflex process. Emotion and exercise increase the rate and force of respiration but the altered pattern of respiration is brought back to normal within a short time by some regulatory mechanism in the body.

The pattern of respiration is regulated by two mechanisms namely.

1. Nervous Mechanism
2. Chemical Mechanism

## **Nervous Mechanism :**

Involves the respiratory centre afferent nerves and efferent nerves. Respiratory centers situated in the reticular formation of the brain stem and classified in to 2 groups namely.



## **Nervous connection of respiratory centers:**

### **Efferent pathway:**

The nerve fibers from the respiratory centers leave the brain stem and descend in to the anterior part of the lateral columns of the spinal cord.

These nerve fibers terminate in the motor neurons in the anterior horn cells of cervical and thoracic segments of spinal cord. From the motor neurons of spinal cord, two sets of nerve fibers arise which are.

1. Phrenic nerve fibers supplying diaphragm
2. Intercostal nerve fibers supplying the intercostal muscles

Vagus nerve also contains some efferent fibers from the respiratory centers.

### **Afferent pathways:**

Impulse from peripheral chemoreceptor and Baroreceptors are carried to the respiratory centers by the fibers of glossopharyngeal and vagusnerves.

## **BRONCHIAL ASTHMA**

‘**Asthma**’ is a disease whose presence dates back to at least the time of Hippocrates, who noted a condition of deep and heavy breathing.

The Greeks had labeled this condition as “**asthma**” the meaning of which was ‘**panting**’.

Bronchial asthma is a chronic inflammatory disorder of the airways in susceptible, inflammatory symptoms are usually associated with widespread but variable air flow obstruction and an increase in airway response to a variety of stimuli. Obstruction is often reversible, either spontaneously or with treatment.

### **Aetiology**

A number of factors are responsible either in the causation or exacerbation of bronchial asthma. A brief account of each of these factors will be discussed.

### **Atopy and allergy:**

House dust mite allergens appear to be the most common one associated with asthma.

Occupational asthma occurs due to allergens and sensitisers.

It has been shown that subjects with apparently intrinsic asthma (Normal skin tests) have higher levels of circulating IgE than the non-asthmatic population.

Some studies however challenged the assumption that childhood asthma is largely of allergic etiology.

Both indoor and outdoor allergen exposures have increased asthma morbidity.

Some plants which produce allergen containing particles that are less than 10 micron. Rag weed and grass pollination are definitely associated with asthma. Pollen allergy is usually season – related and is more closely linked to hay fever and allergic conjunctivitis.

**Mould:** Mould spores are generally smaller than pollen grains and are more likely to penetrate the lower respiratory tract.

House dust mite plays a major role in causation of asthma. The commonest mite is '**Dermatophagoides pteronyssinus**'. High levels are found in mattresses, pillows, carpets, upholstered furniture's, bed covers, clothes and soft toys.

**Animal allergens-** Dogs, cats and other pet animals including rodent are commonly kept in homes. All breeds of cats produce common allergens and cat's saliva and cat danders are potent allergens. The major cat allergen is **Feld I**, which is a protein secreted by cats salivary, sebaceous and lacrimal glands.

**Cockroach allergen** – “The cockroach asthma” is a more severe form of the disease having perennial symptoms and high levels of IgE.

### **Food Allergen and Breast Feedings:**

With breast feeding there is a decreased risk (about 20%) for development of asthma. There are some reports that regular consumption of oily fish is associated with a reduced risk for asthma in children. It has been hypothesised that decreased dietary antioxidant vitamin intake is associated with a decreased serum levels, IgE and a significant decrease of atopy. Recent experimental data showed a reduced risk with intake of lecithins (Wheat germ agglutinin from whole wheat products).

**Infection:**

Viral respiratory infections provoke and alter asthmatic responses. The susceptibility of asthmatic airway to viral inflammation is due to persistent allergic mast cell and eosinophil derived inflammation, stimulates the release of cytokines such as tumour necrosis factor – alpha, which cause an increase in the expression of receptors for human respiratory viruses on the airway lining epithelium once the virus enters the epithelial cells, it replicates and generates a wide variety of proinflammatory cytokines which further enhance eosinophil and mast cell inflammation.

**Provocateurs of asthma:**

The principal infection provocateurs of asthma in childhood during the first 2 years of life are respiratory Syncytial virus (RSV), parainfluenza virus and rhinovirus. Mycoplasma pneumoniae, H. influenzae and gram negative bacteria can synthesize histamine both in vivo and in vitro. The presence of the mediator may contribute to the broncho constriction and other effects of histamine that can accompany bronchial infection.

**Drug:**

About 5-20 % of adults with asthma will experience severe and even fatal exacerbations of broncho constriction after ingestion of aspirin or certain non steroidal anti inflammatory drugs (NSAIDS). These drugs are as follows.

Aspirin, Ibuprofen, Indometacin, Piroxicam, Sulindac, Tolmetin, Naproxen and Diclofenac sodium.

Although the exact mechanism not known, it is non immunologic and probably depends on inhibition of cyclo-oxygenase. Other drugs

that are known to exacerbate asthma include beta – blocker drugs. Eye drop preparations of this class of drugs can induce asthma.

Recently, inhaled verapamil, a calcium channel blocker, has been reported to induce severe bronchospasm in mild asthma.

### **Exercise induced asthma:**

Exercise–induced broncho constriction is one of the manifestations of the asthmatic diathesis.

Airway narrowing develops within 2-3 minutes after cessation of exercise. Exercise – induced asthma is due to mainly of smooth muscle contraction. It has been suggested that heat and water loss leads to changes in mediator releases that cause constriction of smooth muscles. Swimming is the preferred exercise for person with asthma.

### **Occupation and Asthma:**

Occupational asthma is the commonest industrial lung disease in the developed world particularly in grain workers, bakers, millers, sulphur workers and other occupations.

Industries in which asthma occurs include plastics and paint manufacturing, electronics, welding, metal refining, photography, health related industries, antibiotics and cosmetic manufacturing, dyeing, forestry and food processing.

Agents capable of inducing occupational asthma can be vapours, gases, aerosols, or particulate matters and can range from very low molecular weight inorganic chemical to complex organic macromolecules.

Some of these agents are shown.

<b>Agents</b>	<b>Occupation</b>
Animal proteins (Urine, Danders)	Laboratory workers, veterinarians.
Shellfish, egg proteins, pancreatic enzyme, papain, amylase	Food processing
B. subtilis enzyme	Detergent factory
Poultry mites droppings, feathers	Poultry farmers.
Flour grain	Bakers
Storage mites, Soyabean wheat	Farmers
Midges	Fish food manufacturing
Silk worm moths and larvae	Silk workers
Wood dust	Carpenters and saw mill workers.
Platinum salts	Refining
Nickel salts	Plating
Cobalt salts	Diamond polishing

Occupational asthma includes reflex vagal broncho constriction in response to an irritant effect on specific receptors, inflammatory broncho constriction secondary to toxic concentration of gases, direct pharmacological reaction by agents such as organic insecticides and beta – adrenergic blocking agents or by immunologic mechanism.

#### **Tartrazine and sulphite sensitivity:**

Tartrazine is a yellow dye commonly employed in food and medication. Sulphiting agents used to preserve foods and beverages. Sulphur dioxide released in mouth and stomach from sulphites has been incriminated as the cause of precipitation of asthma.

**Rhinitis and sinusitis:**

Nasal and sinus pathology can aggravate asthma, particularly if there is uncontrolled drainage of mucoid or mucopurulent material down the nasopharynx, where it can contribute to cough and irritability of larynx.

It is also possible, but not proven that sinus infection may aggravate asthma through reflex mechanism.

It is now being appreciated that allergic rhinitis and bronchial asthma are considered as 'one airway, and one disease'. Recently there has been a renewed interest in the role that 'histamine' plays in the lower air way disease, and 'leukotrienes' which are more potent inflammatory mediators than histamine play a role in upper airway disease.

**Gastro – Esophageal reflux (GER)**

1. Reflex vagal broncho constriction occurs secondary to stimulation of sensory nerve fibers in the lower esophagus. This mechanism is supported by the finding that acid infusion of the esophagus in asthmatic patients leads to increased airway resistance that rapidly reverses with antacids and infusion of acid in to lower esophagus of asthma children during sleep induces broncho constriction.
2. The second proposed Mechanism is micro-aspiration particularly during sleep. The partial narrowing or occlusion of upper airway during sleep, followed by an increase in intra thoracic pressure might predispose the patient to nocturnal GER and consequently to respiratory symptoms.



**Psychological factors:**

Depression most often associated with asthma may be secondary to a chronic disease. Occasionally psychological illness, family disputes or marital harmony may be major factors in the aetiology of intractable asthma.

**Pollution:**

Passive smoking is known to be a risk factor and there is evidence that diesel fumes are associated with increased allergic responses. Ozone and other oxidants contained in photochemical smog which occurs in areas of high traffic density, high sunlight and temperatures, act as respiratory irritants and can exacerbate asthma.

Indoor air pollution due to cooking fuels, such as gas, biomass and kerosene contain oxides of nitrogen and are responsible for increased respiratory symptoms. Environmental tobacco smoke is important in the development of childhood asthma and in adults. Asthmatic smokers have increased hyper responsiveness to metacholine. Asthmatic smokers have higher sputum total cell and neutrophil numbers and 1 L- 8 concentrations compared to asthmatic non smokers.

**Endocrinal Factors:**

Oestrogen plays a role in the pathophysiology of asthma and long term use and for high doses of post menopausal hormone therapy increase subsequent risk of asthma.

**Genetics and asthma:**

Allergic rhinitis and atopic dermatitis are common among familiar family members of the asthmatic patients. Molecular genetic linkage studies indicate that the 'atopy' gene locus is on chromosome 11.

Cytokines are important components in the pathogenesis of asthma.

The chromosome 5 contains an **IL 4** gene cluster which encodes the allergic cytokines **IL3,4,5,9,13** and **GM – CSF** (Granulocyte macrophage colony stimulating factor). This gene is closely linked to inheritance of an increased IgE response and to increased bronchial hyperresponsiveness.

The other genetic component operates through the human lymphocytes antigen (**HLA or MHC class II**) molecules **HLA - DR, HLA-DP, HLA-DQ** which provide the mechanism for antigen recognition and presentation to and by T and B lymphocytes.

## PATHOLOGY

The histological changes in Bronchial asthma relied on Postmortem specimens taken from People dying in status asthmatics. Since the 1960s, epithelial shedding and influx of eosinophils into the airway mucosa have been associated with Bronchial Asthma large segments of the airway from the major bronchi to the peripherals are occluded with a mixture of tenacious secretions containing serum protein mixed with mucus and cellular debris. Crystalline material consisting largely of major basic protein derived from eosinophil granules (**Charcot – Leyden crystals**) may be present. There is oedema dense eosinophilic infiltration and epithelial denudation in the bronchial wall. Airway samples obtained at open lung biopsy show goblet cell hyperplasia peri-bronchial smooth muscle hypertrophy and apparent basement membrane thickening. The strips of epithelial cells are called **curschmann's** spirals, clumps of cells (creola-bodies) or isolate metaplastic cells are common. Eosinophils, Neutrophils and mono nuclear cells were present in increased numbers in the post capillary venules and were frequently in close contact with the vascular endothelium.

Another important observation was the presence of apparent thickening of the subepithelial basement membrane. Monoclonal antibody studies suggest that the sub- basement membrane band consists of types III and V collagen together with fibronectin. This suggests the fibroblastic origin of the band. Recent data further revealed an expanded network of subepithelial myofibroblasts correlates with the degree of sub epithelial thickening suggesting a repair response secondary to chronic inflammation. Extensive collagen deposition within the bronchial mucosa might influence the mechanical properties of the airways and contribute towards bronchial hyper responsiveness.

**Airway remodeling:**

Chronic inflammation in the airway leads to structural changes including hypertrophy and hyperplasia of airway smooth muscle and thickening of the reticular layer of the basement membrane. This later thickening is due to deposition of collagen from activated myofibroblasts in response to cytokines and growth factors released during the inflammatory response.

The remodeling of airways in bronchial asthma involves structural changes in the epithelium, the myo-firboplast and extra-cellular matrix including the basement membrane and smooth muscle.

This remodeling process is mainly caused by a complex interaction of inflammatory cells that are central to pathogenesis of asthma with structural tissue cells. The inflammatory cells such as eosinophils, T cells, mastcell, and macrophages together with structural tissue cells play important effector role through the release of a number of cytokines, mediators and chemokines.

Airway inflammation and remodeling contribute significantly to the decline in lung function in bronchial asthma.

## **PATHOGENESIS**

Chronic airway inflammation involving many cell types and inflammatory mediation accompanies the bronchial hyperresponsiveness asthma. Nevertheless, the precise relationship of the inflammatory cells and their mediators to airway hyper reactivity is not fully understood.

The disease is triggered by environmental antigens such as dust, pollens, animal danders and foods. A positive family history of atrophy is common and asthmatic attacks are often preceded by allergic rhinitis, urticaria or eczema. Serum IgE levels are usually elevated. A skin test with the attending antigen results in an immediate wheal and flare reaction, a classic example of type I IgE mediated sensitivity elicits an acute immediate response and a late phase reaction.

Recall that exposure of presensitized IgE coated mast cells to the same or a cross reacting antigen stimulates the release of chemical mediators from these cells. In the case of air borne, the reaction occurs first on sensitized mast cells on the mucosal surface the resultant mediator release opens the mucosal inter cellular tight junctions and enhance penetration of antigen to the more numerous sub mucosal mast cells. In addition, direct stimulation of sub epithelial vagal receptors (Parasympathetic) provokes broncho constriction through central and local reflexes. This occurs within minutes after stimulation and is called the acute or immediate response. The mediators of IgE triggered reactions include both primary and secondary mediators.

The primary mediators include

1. Histamine which causes broncho constriction by direct and cholinergic reflex actions, increased vascular permeability, and increased bronchial secretions.
2. Eosinophilic and Neutrophilic chemotactic factors (eg) – leukotriene B<sub>4</sub> which selectively attract eosinophils and Neutrophils. Histamine is probably important in the first few minutes of an asthmatic attack.

The secondary mediators include:

1. Leukotrienes C<sub>4</sub>D<sub>4</sub> and E<sub>4</sub> extremely potent mediators that cause prolonged broncho constriction as well as an increased vascular permeability and increased mucus secretion.
2. Prostaglandins D<sub>2</sub> (PG D<sub>2</sub>) which elicits broncho constriction and vasodilatation.
3. Platelet activating factor (PAF) which causes aggregation of platelets and release of histamine and serotonin from their granules.
4. Cytokines, such as **IL**<sub>1</sub> tumors necrosis factor (**TNF**) and **IL**<sub>6</sub> some of which are found to exist in the preformed state with in the mast cell granules. The acute reaction is thus associated with broncho constriction, edema, mucus secretion, flushing and in certain instances hypotension.

This is followed by the late phase reaction which starts 4 to 8 hours later and persists for 12 – 24 hours.

The late phase reaction is mediated in part by a swarm of leukocytes, neutrophils, eosinophils and lymphocytes recruited by the chemotactic factors and cytokines derived from mast cells during the acute phase response or by other mediators produced by the chronic inflammatory

cells already present in asthmatic, suffering a recurrent attack. The leukocytes release a second wave of mediators that stimulate the late reaction. Histamine releasing factors produced by various cell types, induce release of histamine from basophils cause broncho – constriction and edema. In addition neutrophils cause further inflammatory injury and the major basic protein of eosinophils cause epithelial damage and airway constriction. The presence of both immediate and delayed reactions in IgE mediated events helps explain the prolonged manifestation of asthma.

### **CLINICAL PRESENTATION OF BRONCHIAL ASTHMA:**

The usual symptoms include cough, wheezing, shortness of breath, chest tightness and modest degree of sputum production.

Most patients complain of the onset of an attack of bronchial asthma following allergic pharyngitis in the form of sore throat, pain in the throat, itching, sneezing, running nose or a blocked nose.

The incidence of IgE mediated allergy (allergic rhinitis, atopic dermatitis, hay fever) and bronchial asthma in close relatives is very high.

In children there will be evidence of hyperinflation of the lungs with use of accessory muscles and appearance of hunched shoulders and “Pigeon chest”.

The intensity of breath sounds in symptomatic asthma will be reduced and expiratory phase is prolonged. Presence of rhonchi is characteristic finding in asthma and will be present in most patients. Rhonchi may be heard in many other conditions including chronic bronchitis, pulmonary oedema, bronchial stenosis, foreign body aspiration, upper airway obstruction, aspiration pneumonia and pulmonary embolism etc.

It is often said that “all that wheezing is not asthma”. Crepitations are not the findings of asthma unless there are secondary infections or a complication like allergic broncho pulmonary mycosis.

## **CLASSIFICATION**

### **Intrinsic and Extrinsic asthma:**

The intrinsic asthma usually has late onset with no history of atopy or allergy and is non-seasonal. The skin test for allergens is usually negative and the serum IgE level is often normal. Asthma associated with polyarteritis nodosa and aspirin sensitive bronchial asthma is usually of intrinsic type.

### **Late onset asthma:**

Definition: “asthma with onset of symptoms in adult life in a patient with no pre-existing, persistent respiratory symptoms”.

Asthma induced by drugs and occupational asthma may belong to this category. Asthma of adult onset may be the first sign of the development of polyarteritis nodosa.

### **Occupational asthma:**

There can be two categories of asthma related to work place. They are ‘occupational asthma’ and ‘work-aggravated asthma’. Occupational asthma is characterized by variable airflow limitation, bronchial hyperresponsiveness or both due to conditions in a particular work environment not to stimuli outside the work place. Work – aggravated asthma is aggravated by irritants or physical stimuli in the work place.

History of atopy and smoking are important determinants to induce occupational asthma that occurs through an IgE – dependent mechanism.

### **Nocturnal asthma:**

A number of mechanisms have been hypothesized to explain the phenomenon of nocturnal asthma including exposure to dust, mite allergen, late-phase allergic reactions, effects of posture and sleep stages



on airway tone, gastro-oesophageal reflux, impaired mucociliary clearance, airway cooling and changes in circadian rhythms of circulating hormones (adrenaline and steroids). Bronchial responsiveness to histamine and allergen challenge increase during sleep and mast cell mediator release is enhanced. Circulating eosinophils increase which may allow their ingress into pulmonary tissue together with decreased plasma catecholamine and cortisol levels. All these factors may influence airway tone, inflammation and responsiveness during sleep and produce the observed clinical picture.

## **DIAGNOSIS OF BRONCHIAL ASTHMA**

The diagnosis of asthma is a clinical one; there is no confirmatory diagnostic blood test, radiographic or histopathological investigation.

### **Signs of asthma:**

During exacerbations the patient will often have wheeze and reduced lung function, either reduced peak flow or an obstructive pattern on spirometry.

Additional information which may contribute towards a clinical suspicion of asthma includes – personal or family history of asthma or other atopic condition, worsening of symptoms after exposure to recognized triggers such as pollens, dust, feathered or furry animals, exercise, Viral infections, chemicals and environmental tobacco smoke, and worsening of symptoms after taking aspirin NSAIDS and  $\beta$  blockers.

### **Laboratory studies:**

Spirometry should be undertaken to document severity of airflow obstruction and to establish acute bronchodilator responsiveness for all patients in whom the diagnosis of asthma is being considered. Usually

there will be a normal vital capacity with either impaired FEVI or impaired MMEF.

The low FEVI in bronchial asthma is due to increased resistance because of broncho constriction and remodelled airway walls.

Three readings should be taken and the best recorded graphically for easy inspection.

**Other laboratory investigation for bronchial asthma includes:**

- ❖ Complete and differential blood counts
- ❖ X-ray
- ❖ Sputum examination and stain for eosinophil is (sputum eosinophils are highly characteristic of asthma and neutrophils predominate in bronchitis sputum.
- ❖ Nasal secretions and stain for eosinophils (neutrophilic nasal discharge indicates sinusitis)
- ❖ Sputum culture.
- ❖ Sputum differential eosinophil count is one of the most objective test in patients with bronchial asthma
- ❖ Complete pulmonary function studies including flow – volume loops which may reveal the presence of upper airway obstruction.
- ❖ Determination of specific IgE antibodies to common inhalant allergens with skin tests or with in vitro tests is useful to find out the role of allergy in the asthma patients.
- ❖ Incorporating a skin prick test using commonly inhaled allergens is a simple, safe, inexpensive, rapid, and most common way of assessing the contribution of atopy.

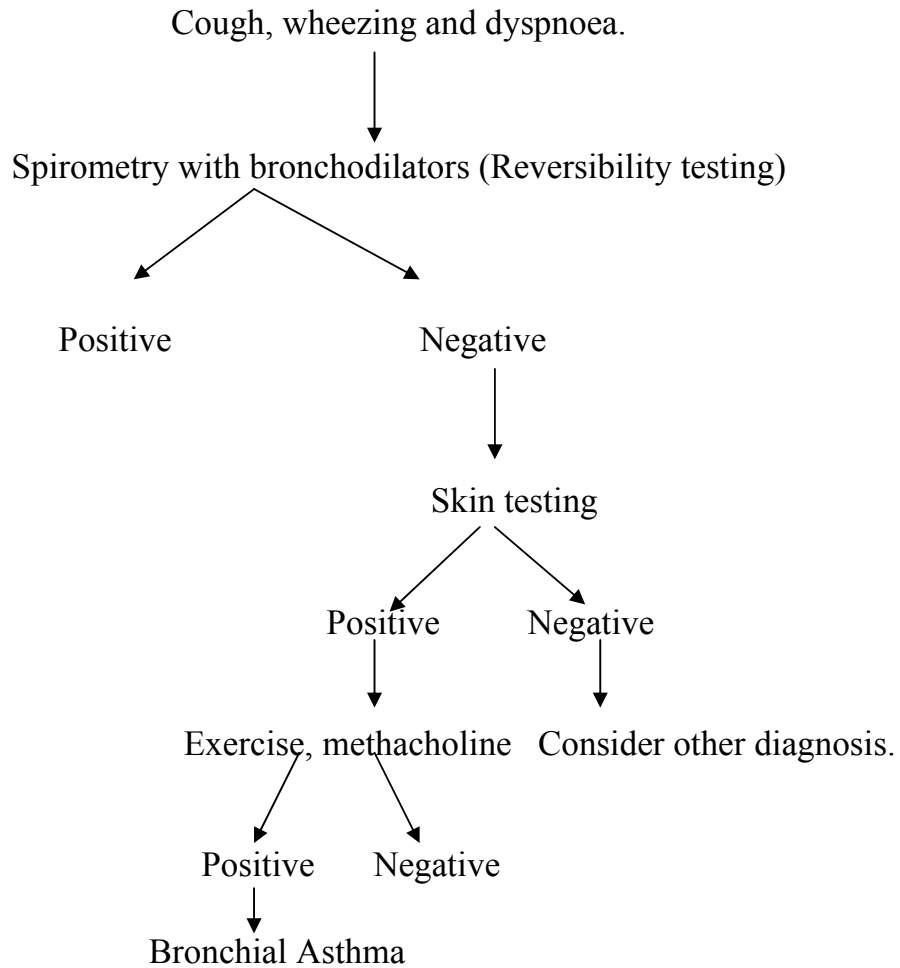
**Other investigations that may be helpful:**

- Rhinoscopy
- Sinus x-ray
- Broncho provocation test
- Provocative challenge
- Occupational allergens
- Evaluation of pH for gastro-oesophageal reflux.

**DIFFERENTIAL DIAGNOSIS OF BRONCHIAL ASTHMA**

1. COPD
2. Cardiac diseases
3. Laryngeal tumors
4. Tracheal tumors
5. Bronchogenic carcinoma
6. Bronchiectasis
7. Foreign body
8. Interstitial lung disease
9. Pulmonary embolism
10. Aspirations
11. Vocal cord dysfunction
12. Pulmonary infiltrations with Eosinophil
13. Cough due to drugs (  $\beta$  Blockers and ACE inhibitors)
14. Hyperventilation.

## DIAGNOSTIC WORK UP FOR BRONCHIAL ASTHMA



### Difference between Cardiac Asthma and Bronchial Asthma

NO	FACTORS	CARDIAC ASTHMA	BRONCHIAL ASTHMA
1	Past history	Hypertension aortic or coronary disease	Previous attacks of asthma or other allergic conditions in patients of other members of the family

2	Age	Onset usually after 50 yrs	Any age
3	Precipitating factors	May be precipitated by exertion or acute myocardial infarction or hypertension	Trigger factors may be infected non specific irritants, external, allergies, exercise of emotional factors,
4	<b>Symptoms:</b> a. Cough	Cough and dyspnoea, cough associated with watery expectoration which increases in intensity towards end of attacks.	Starts with dyspnoea, expectoration of small sticky sputum, paroxysm of wheezes when cough becomes profuse
	b. Wheezing	Rare	Usual
	c. Sweating	Prominent	Rare, unless status asthmaticus
5	<b>Signs:</b>		
	a. Inspection i. Accessory muscles of respiration	Not active	Active
	ii. Shape of the chest	Normal	Emphysematous
	b. Palpation	Heart often enlarged having palpable apex beat	Heart not enlarged, if long standing disease, right ventricular enlargement

	c. Auscultation	S <sub>2</sub> may be loud. Left ventricular gallop. Expiration not unduly prolonged, rales more than bronchitis, signs in early stage at base of the lungs, gradually ascending up with progress of the attack	Normal A <sub>2</sub> sound, right ventricular gallop is later feature of severe bronchial asthma. Expiration markedly prolonged rhonchi more than rales. Signs diffuse all over the lungs.
	d. Pulse	Full and bounding	Feeble and rapid
	e. B.P	Usually elevated	Normal or low
	f. Signs of underlying disease	Hypertension or coronary disease	No evident of cardiovascular disease
	h. Urine	Generally clear, then may be mild albuminuria.	Clear
6.	<b>Investigation:</b> Eosinophil	None	Common

### **Difference between Tropical Eosinophilia and Bronchial Asthma**

<b>NO</b>	<b>FACTORS</b>	<b>TROPICAL EOSINOPHILIA</b>	<b>BRONCHIAL ASTHMA</b>
1	Age	Any age	Usually starts before 3 yrs of age
2	Duration of symptom	Short duration	Long duration
3	Cough and dyspnoea	Dyspnoea more than cough, breathlessness particularly after cough	Paroxysmal cough more than dyspnoea.

4	Fever	Common	Rare
5	Loss of weight	Fairly common	Seldom
6	Auscultatory signs	Disproportion between cough and breathlessness	Compatible with degree of cough and breathlessness
7	Blood	Leukocytosis, Eosinophilia	Normal WBC count eosinophilia 8 to 15%

### **Difference between Pulmonary Tuberculosis and Bronchial Asthma**

<b>NO</b>	<b>FACTORS</b>	<b>PULMONARY TUBERCULOSIS</b>	<b>BRONCHIAL ASTHMA</b>
1	Age	Generally aged persons	Usually starts before 3 yrs of age
2	History	History of chronic cough	History of previous attacks.
3	Duration of symptoms	May last longer	May last upto old age
4	Time of onset	-	Early onset
5	Mode of onset	May be precipitated lay infection	May be precipitated by allergy
6	Loss of weight	Common	Seldom
7	<b>Symptoms:</b>		
	i. Fever	Various extent	Rare

	ii. Cough	Frequent, sharp, short, may be dry in the early stages, later it is persistent with copious, purulent, expectoration dyspnoea is later feature.	Paroxysmal cough more than dyspnoea
	iii. Wheezing	localised wheezing due to bronchial narrowing by tuberculous lymph nodes	wheezing present all over the field
	iv. Sweating	Especially during night	rare, unless in asthmatics
	v. Haemoptysis	Early stage blood stained sputum	Nothing relevant
8	Inspection	Affected side of chest flattened with displaced, apex impulse to the side of lesion, clubbing of fingers, present	No flattening of the chest, apical impulse in position.
9.	Palpation	Movement of chest in affected side, vocal fremitus diminished, increased in consolidation, lymphadenopathy is noted.	In longstanding cases, right ventricular enlargement.
10	Percussion	Dull note in the apex, others impaired	Normal
11	Auscultation	Breath sounds bronchial early wheezing, late crackling rales, diminished vocal resonance in early and increased in later conditions	Prolonged expiration wheezing rhonchi heard all over the field



	a. Pulse	Increased or low	Normal or low
	b. B.P.	Low	Normal or low
	c. Signs of underlying disease	-	No evidence of cardio vascular disease
	d. Sputum	hard, thick, tenacious sputum, positive in culture.	Sticky pellets.
	e. Blood	Lymphocytosis, raised ESR	Eosinophils, ESR normal.

## **DIAGNOSIS AND INVESTIGATION**

An account of episode wheeze, breathlessness interpreted with period of normality is sufficient evidence to suspect asthma and further evidence comes from a history of marked variability, attacks in small hours of the night, provocation by strong exercise and allergens and paroxysmal cough, productive small amount of sticky sputum.

## **CONFIRMATION OF THE DIAGNOSIS**

### **PHYSICAL SIGNS OF THE CHEST**

During an attack the chest is held near the position of full inspiration and the percussion note may be hyper resonant. Breath sounds when not obscured by numerous high pitched polyphonic in expiratory and inspiratory rhonchi is vascular in character with prolonged expiration. In very severe asthma airflow may be insufficient to produce rhonchi; a silent chest in such a patient is an ominous sign. These are usually no abnormal physical signs between attacks except in patients with chronic asthma who are seldom without expiratory rhonchi; severe asthma persisting from childhood may cause a 'pigeon chest' deformity.

## **RADIOLOGY EXAMINATION**

In an acute attack of asthma, the lungs appear hyper inflated. Between episodes the chest x-ray is usually normal. In long standing cases, the appearance may be indistinguishable from hyper-inflation caused by emphysema and a lateral view may demonstrate a 'pigeon chest' deformity. Occasionally when a large bronchus is obstructed by tenacious mucus, there is opacity caused by lobar or segmental collapse.

A chest x-ray should be performed if possible in all patients with severe acute asthma to exclude pneumothorax a rare but potentially fatal complication of the pulmonary hyper-inflation produced by severe airflow obstruction in asthma. The chest x-ray show mediastinal and subcutaneous emphysema in very severe disease.

## **LUNG FUNCTION TESTS**

The physiological function of the lung can be accurately by lung functional testing. The commonest reason for performing a lung function test is to reach a diagnostic.

## **SPIROMETRY**

There are many types of spirometry available and all record similar information. The traces are obtained by a deep inspiration followed by the fastest possible and maximal expiration.

The normal forced vital capacity (FVC) is fully exhaled in less than 3 seconds more than three quarter is exhaled in the first second. In addition to measuring the vital capacity in litres, the volume expired in the first second or the first expiratory volume at 1 second ( $FEV_1$ ) can be measured and the ratio of  $FEV_1 : FVC$  can be calculated. The results are compared with predicted values based on age height and ethnic group.  $FEV_1/FVC$  ratio is normally greater than 70%.

If diffuse airflow obstruction is present, the rate at which the air can be exhaled is diminished throughout expiration. The length of expiration is prolonged and FEV<sub>1</sub> is much reduced. This is an obstructive pattern and is seen in as time, chronic bronchitis and emphysema. In patients with asthma this obstruction may be reversible by treatment with bronchodilators or even corticosteroids.

In a restrictive pattern, there is a reduced lung volume, perhaps as a result of pulmonary fibrosis or that wall deformity but there is no obstruction to airflow and FEV<sub>1</sub> is normal.

### **PEAK EXPIRATORY FLOW METER**

It is a popular instrument for assessing airflow obstruction there is a cheap, simpler version called the mini peak flow meter which is suitable for use at home by individual patients. These machines measure the maximal rate of flow which is achieved during a forced expiration and most healthy people will achieve values of greater than 400 litres/min. Patients with lung fibrosis and restriction changes on the spirogram may also have normal expiratory flow rates. Patients with airflow obstruction will have reduced flow rates, with values below 200 litres/min being very significant and those below 100 litres/min extremely severe.

### **FLOW VOLUME CURVES**

The plotting of flow versus volume during both maximal expiratory and inspiratory manoeuvres is of major help in differentiating central airflow obstruction from diffuse airflow obstruction as seen in COPD and asthma.

## **LUNG VOLUMES**

Measurement of total lung capacity and residual volume is best performed using a whole body plethysmograph, but can be measured by a helium dilution method. In general, restrictive defects lead to reduced values, and obstructive defects to increased values.

## **MEASUREMENT OF DIFFUSING CAPACITY**

The diffusing capacity (DLCO) is a measure of the lung's ability to transfer gas from alveoli to blood. The test utilized uptake of carbon monoxide from a single breath of 0.3% mixture in air; this gas is chosen because it combines rapidly with hemoglobin and provides a true estimate of diffusion across the alveolar capillary membrane. The diffusing capacity is reduced in patients with disease principally affecting alveoli such as fibrosing alveolitis or emphysema. The transfer coefficient (KCO) is a measure of diffusing capacity expressed per volume of ventilated lung during the single breath test and is useful to confirm that a low DLCO is due to alveolar disease rather than misdistribution of ventilation. High values of DLCO may be seen in alveolar hemorrhage.

## **ARTERIAL BLOOD GASES AND OXIMETRY**

The measurement of hydrogen ion concentration,  $\text{PaO}_2$  and  $\text{PaCO}_2$ , and derived bicarbonate of arterial blood are essential in assessing the degree and type of respiratory failure and for measuring overall acid-base status. Use of a pulse oximeter allows a non-invasive continuous method of assessing oxygen saturation in patients who require continuous monitoring in order to assess hypoxaemia and its response to therapy, including supplemental oxygen.

## **EXERCISE TESTS**

Formal exercise testing with measurement of metabolic gas exchange and respiratory and cardiac response using cycle ergometry or treadmill exercise is useful in providing a detailed analysis of both pulmonary and cardiac function in the breathless patients. Exercise challenge with measurement of spirometry before and after can also be helpful in demonstrating exercise – induced asthma. Finally, the 6 minute walk test or ‘shuttle’ test can provide a simple but objective assessment of disability and response to treatment.

## **SKIN HYPERSENSITIVITY TESTS**

A prick is made in the skin with a fine needle through a drop of an aqueous extract of the substance to be tested. A positive reaction is indicated by the development of a wheal and flare, which begins to appear within few minutes. Tests are usually performed with a group of common allergens known to cause bronchial asthma. It is seldom possible with these tests to identify one particular allergens as the causes of asthma are an individual patient and their chief value is to distinguish atopic from non-atopic subjects.

## **SPUTUM EXAMINATION**

Sputum eosinophilia is a useful indication of an asthmatic type of airway reaction. Stained sections of sputum fixed in alcohol or formalin is probably a severe indication of asthma than a sputum eosinophil count. This is useful for the demonstration of *Aspergillus fumigatus*. Eosinophils are a prominent feature of the inflammatory exudates within the airway lumen lies a thick tenacious mucus which under the microscope is seen to contain strips of desquamated epithelial cells (Curschman’s spirals) eosinophils, isolated metaplastic epithelial cells

(Creola bodies) & crystalline materials consisting largely of major basic protein derived from eosinophilic granules. (Charcot - leydon crystals)

### **COMPLICATIONS**

Mortality is uncommon in asthma but a severe attack may result in respiratory failure and death.

This is more in 'status asthmaticus'. Other complications include frequent respiratory infection, pulmonary collapse due to obstruction by viscid secretions, pneumothorax, and emphysema and cough fracture (fracture of ribs due to violent coughing), children with asthma may show retardation of growth, especially if treated with corticosteroid on a long term basis. Long standing bronchial asthma, punctuated with frequent expiratory infections may lead to emphysema and chronic carpulmonale.

### **PROGNOSIS**

The prognosis of the individual attack is good, except in severe acute asthma, when there is occasionally a fatal outcome, especially if treatment is inadequate or delayed. Spontaneous remission is fairly common in episodic asthma, particularly in children, but rare in chronic asthma, which can lead to irreversible airflow obstruction. Seasonal fluctuation can occur in both types of asthma. Asthmatic subjects with episodic asthma are usually worse in the summer, when they are more heavily exposed to antigens, while chronic asthmatics are usually worse in winter months, because of the increased frequency of viral infections.

### **Avoidance of allergens**

There are few instances, in which a single agent can be identified as the cause for attacks of asthma. These allergens include grass pollens, mites, animal dander drugs, industrial chemicals such as isocyanates and certain articles of diet. The vast majority of patients are hypersensitive to a wide range of allergens and attempts to avoid all of them are impracticable.

## **MATERIALS AND METHODS**

### **Clinical Study**

The clinical study of **Swasakasam** was carried out during the year 2006-2007 at the Post Graduate Department of Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai. In this study, twenty patients of both sexes were selected in the Out-Patient Department, admitted in the In-Patient ward and were treated with the trial medicine and guided and clearly observed under the supervision of Professor, Reader, Lecturer and Assistant Lecturer in the Post Graduate Department of Pothu Maruthuvam. After discharge, all the twenty patients were followed as the Out-Patients in the Out-Patients Departments. The medicine was also subjected to trial with twenty Out-Patients in the Out-Patients department after detailed investigation, under the guidance and observation of Professor, Reader, Lecturer and Assistant Lecturer.

### **SELECTION OF THE PATIENTS:-**

The patients were selected on the basis of the clinical findings of cough with expectoration, dyspnea, tightness of chest, wheezing, hardly expectoration of scanty, mucoid sputum, flatulence.

Detailed history of the patient contains past, personal and family histories, socio-economic status, diet, habits, occupational history, exposure to chemical hazards, smoke, dust and cold.

### **Diagnosis:**

Siddha methods of diagnosis were employed with the following methods; Mukkutra nilaigal, En vagai Thervugal, Nilam, Kaalam, Udal kattugal, Poriyal arithal, Pulanal arithal and Vinathal.

## **INVESTIGATIONS:**

The following laboratory investigations were done in the college hospital for all the patients.

1. Blood test (TC, DC, ESR, HB %)
2. Urine Analysis (Albumin, Sugar, Deposits)
3. Motion test (Ova, Cyst)
4. Sputum for AFB
5. X-ray chest (PA View)
6. Mantoux test
7. Peak Expiratory Flow Rate.

To establish the efficacy of the trial medicine, biochemical analysis and pharmacological studies were conducted in the Department of Biochemical and Pharmacology separately in the Government Siddha Medical College, Palayamkottai and anti-bacterial activity was done at Malar Micro Diagnostic Centre, Palayamkottai.

## **TREATMENT:**

The trial medicine used in the present clinical study is 'Mungiluppu Chooranam' (4gm, twice daily with honey, after meals). All the patients were advised strictly to follow the Pathiyam (Dietary Regimen), Pranayamam and Yogic Exercise was also prescribed for the speedy recovery of "Swasa kasam".



## **RESULTS AND OBSERVATION**

Results were observed with respect to the following criteria

1. Sex Distribution
2. Age Distribution
3. Kaalam Distribution
4. Dehi Distribution
5. Gunam Distribution
6. Religion Distribution
7. Paruva Kaalam Distribution
8. Sirupoludhu Distribution
9. Thinai Distribution
10. Occupation Distribution
11. Socio - economic status
12. Aetiological factors
13. Mode of Onset
14. Clinical features
15. Duration of Illness
16. Other System involvement
17. Family history
18. Diet factor
19. Habits
20. Gnanendhiriyam (Imporigal)
21. Kanmendhiriyam
22. Kosam
23. Mukkutram a)Vadha b)Pitha c) Kaba
24. Ezhu Udal Kattugal
25. Envagai Thervugal
26. Neerkuri
27. Neikuri
28. Laboratory Analysis
29. Gradation of Results

For this study 20 In-Patients and 20 Out-Patients were selected.

## 1. SEX DISTRIBUTION:

**Table 1** illustrates the distribution of Sex

**Table 1**

S. No	Sex	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Male	12	60%	17	85%
2.	Female	8	40%	3	15%

Males are affected in 60% of the In - Patients and 85% of the Out-Patients. Females are affected in 40% of the In- Patients and 15% of the Out- Patients.

The table showed that males were affected more than females.

## 2. AGE DISTRIBUTION:

**Table 2** illustrates the distribution of Age

**Table 2**

S. No	Age in years	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	0-20	0	-	2	10 %
2.	21-30	1	5%	-	-
3.	31-40	1	5 %	2	10%
4.	41-50	4	20 %	3	15 %
5.	51-60	4	20 %	6	30 %
6.	61 and above	10	50 %	7	35 %

Among the In- Patients 50% of the patients were observed in the age group of 61 and above, 20 % of the patients were observed in the age

group of 51-60 years and 41 - 50, 5 % of the patients were observed in the age group of 31-40 years and 21 – 30 years.

Among the Out - Patients, 35% of the patients were observed in the age group of 61 and above, 30% of the patients were observed in the age group of 51 – 60 years, 15% of the patients were observed in the age group of 41 - 50 years, 10 % of the patients were observed in the age group of 31-40 years and 0 – 20 years.

The table showed predominance of distribution in the age group 61 and above years among the In-Patients and Out - Patients.

### 3. KAALAM DISTRIBUTION:

**Table 3** illustrates the distribution of Kaalam

**Table - 3**

S. No	Kaalam	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vadha Kaalam (1-33 Years)	1	5 %	3	15%
2.	Pitha Kaalam (34-66 years)	15	75 %	13	65%
3.	Kaba Kaalam (67-100 years)	4	20 %	4	20%

Among the In - Patients 75% were affected in Pitha Kaalam, 20% were affected in Kaba Kaalam and 5 % in the Vadha Kaalam.

Among the Out - Patients 65% were affected in Pitha Kaalam, 20% were affected in Kaba Kaalam and 15% were affected in Vadha Kaalam.

The table showed the increased incidence of the disease in the Pitha Kaalam i.e 34 - 66 years.

#### 4. DEHI DISTRIBUTION:

**Table 4** illustrates the distribution of Dehi,

**Table 4**

S. No	Dehi	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vadha Dehi	-	-	-	-
2.	Pitha Dehi	-	-	-	-
3.	Kaba Dehi	-	-	-	-
4.	Dhontha Dehi	20	100%	20	100%

From the table, it was observed that all the patients, i.e. 100% come under the Dhontha Dehi, among both the In-Patients and Out-Patients.

This showed the incidence of this disease only in the Dhontha Dehi.

## 5. GUNAM DISTRIBUTION:

Table 5 illustrates the distribution of Gunam

**Table 5**

S. No	Gunam	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Sathuva Gunam	-	-	-	-
2.	Rajo Gunam	20	100%	20	100%
3.	Thamo Gunam	-	-	-	-

100% of both In-Patients and Out-Patients fall under the type of Rajo Gunam.

This showed the incidence of this disease only in the Rajo Gunam persons.

## 6. RELIGION DISTRIBUTION:

Table 6 illustrates the distribution of Religion among the patients

**Table 6**

S. No	Religion	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Hindus	19	95%	19	95%
2.	Muslims	-	-	-	-
3.	Christians	1	5%	1	5%

Among the In-Patients and Out – Patients 95% were Hindus, 5% were Christians.

## 7. PARUVAKAALAM DISTRIBUTION:

Table 7 illustrates the distribution of the disease among the Paruva Kaalam

Table 7

S. No	Paruva Kaalam	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Kaar Kaalam	4	20 %	10	50%
2.	Koothir Kaalam	13	65 %	-	-
3.	Munpani Kaalam	-	-	-	-
4.	Pinpani Kaalam	-	-	-	-
5.	Elavenil Kaalam	-	-	-	-
6.	Muthuvenil Kaalam	4	20 %	10	50 %

Among the In-Patients, 65 % of the disease comes under the Koothir Kaalam and 20% of the incidence comes under the Kaar and Muthuvenil Kaalam.

Among the Out-Patients, 50 % of the incidence comes under the Kaar and Muthuvenil Kaalam.

The table showed the prevalence of disease under Koothir Kaalam among the In-Patients and Kaar and Muthuvenil Kaalam among the Out – Patients.

## 8. SIRUPOLUDHU DISTRIBUTION:

Table 8 illustrates the distribution of the disease among the Sirupoludhu.

Table 8

S. No	Sirupoludhu	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vaigarai	18	90%	17	85%
2.	Pagal	-	-	-	-
3.	Nan Pagal	-	-	-	-
4.	Pirpagal	-	-	-	-
5.	Maalai	2	10%	3	15%
6	Yamam	-	-	-	-

Among the In-Patients, 90% of the incidence occurs during Vaigarai Poludhu and 10% occurs during Maalai Poludhu.

Among the Out-Patients, 85% of the incidence occurs during Vaigarai Poludhu and 15% occurs during Maalai Poludhu.

The table showed the prevalence of disease during Vaigarai Poludhu in all patients.

## 9. THINAI DISTRIBUTION:

Table 9 illustrates the distribution of the disease among Thina

Table 9

S. No	Thinai	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Kurinji	-	-	-	-
2.	Mullai	-	-	-	-
3.	Marutham	19	95%	20	100%
4.	Neithal	1	5%	-	-
5.	Palai	-	-	-	-

Among the In-Patients, 95% belonged to the Marutham and 5% belonged to the Neithal.

Among the Out-Patients, all the 100% belonged to the Marutham.

The table indicated that Marutham was the place of incidence of the disease.

## 10. OCCUPATION:

**Table 10** illustrates the distribution of Occupation among the patients

**Table 10**

S. No	Occupation	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Agri labour	-	-	3	15 %
2.	Mill Worker	2	10 %	4	20 %
3.	Cook	1	5 %	-	-
4.	Mason	1	5%	-	-
5.	Tea Shop labour	-	-	1	5 %
6.	Auto Driver	7	35 %	7	35 %
7.	Teacher	-	-	1	5%
8.	Student	-	-	2	10%
9.	House Wives	7	35%	2	10%
10.	Garment employee	2	10 %	-	-

Among the In-Patients 35% were Drivers and Housewives, 10% were Rice Mill workers and Garment Employees and 5 % were Cooks and Mason. Among the Out-Patients 35% were Drivers, 20% were Rice



Mill workers, 10% were Students and House wives, 5% were teachers and teashop workers.

The table indicated increased incidence of the disease in drivers and rice mill workers.

### 11. SOCIO - ECONOMIC STATUS:

**Table 11** illustrates the Socio - Economic Status of the patients

**Table 11**

S. No	Socio - Economic Status	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Rich	-	-	-	-
2.	Middle Class	-	-	5	25 %
3.	Poor	20	100%	15	75 %

Among In – Patients 100 % of the cases are economically poor.

Among the Out – Patients 65 % are economically poor and 25 % belong to the middle class.

## 12. AETIOLOGICAL FACTOR (ALLERGEN):

Table 12 illustrates the Aetiological Factor for the disease

**Table 12**

S. No	Aetiology	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Dust	5	25%	7	35%
2.	Smoke	2	10%	2	10%
3.	Husks of grains	-	-	5	25%
4.	Dust and cold exposure	6	30%	-	-
5.	Husks of grains and cold exposure	5	25%	-	-
6.	Others	2	10%	6	30%

Among the In-Patients, 30% of the patients had dust and cold exposure collectively as their aetiological factor, 25% each had dust and husks of grains and cold exposure collectively as their aetiological factors, 10% of the patients had smoke as their aetiological factor. Another 10% of the patients had other factors such as cotton fibre and exposure to sulphur and lead as their aetiological factors.

Among the Out-Patients, 35% of the patients have dust as their aetiological factor, 25% had husks of grains as their aetiological factor, and 10% had smoke as their aetiological factor. Another 30% had other factors such as cotton fibre, soap as their aetiological factors.

The above table should that, dust and cold exposure, dust were the main aetiological factors among these patients.

### 13. MODE OF ONSET:

Table 13 illustrates the Mode of Onset of the disease

Table 13

S. No	Mode of onset	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Sudden	-	-	-	-
2.	Gradual	20	100%	20	100%

The table showed that the mode of onset was gradual in all the 100% of both In-Patients and Out- Patients.

### 14. CLINICAL FEATURES:

Table 14 illustrates the distribution of Clinical Features.

Table 14

S. No	Clinical Features	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Running Nose	4	20%	8	40%
2.	Sneeze	6	30%	8	40%
3.	Tightness of Chest	20	100%	20	100%
4.	Wheeze	20	100%	20	100%
5.	Cough with Expectorations	20	100%	20	100%
6.	Fever	8	40%	6	30%
7.	others				
	(i) Tachycardia	20	100%	20	100%
	(ii) Urticaria	2	10%	2	10%
	(iii) Clubbing	8	40%	6	30%
	(iv) Cyanosis	-	-	-	-

Among the In-Patients and Out-Patients, there was 100% incidence of clinical features of tightness of chest, wheeze, cough with scanty expectoration and tachycardia.

Among the In-Patients, there was 40% incidence of fever and Clubbing. 30 % incidence of Sneeze, 20 % incidence of running nose and 10 % incidence of Urticaria.

Among the Out-Patients, there was 40% incidence of Sneeze and Running nose, 30 % incidence of Fever and Clubbing and 10 % incidence of Urticaria.

#### 15. DURATION OF ILLNESS:

**Table 15** illustrates the distribution of Duration of Illness

**Table 15**

S. No	Duration of Illness	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Below 3 months	2	10%	2	10%
2.	3 - 6 months	2	10%	-	-
3.	6 months – 1 year	5	25%	6	30%
4.	1 - 2 years	5	25%	3	15 %
5.	2 - 3 years	2	10%	1	5%
6.	Above 3 years	4	20 %	8	40 %

Among the In-Patients, there was 25 % incidence for the duration of 6 months – 1 year and 1 – 2 years, 20% incidence of duration above 3 years and 10 % incidence of duration below 3 months, 3 - 6 months and 2 – 3 years.

Among the Out-Patients, 40% incidence showed the duration of above 3 years and 30% incidence showed the duration of 6 months -1 year, 15% incidence showed the duration of 1 - 2 years and 10 % incidence showed the duration of below 3 months and 5 % incidence showed the duration of 2 -3 years.

The table indicated highest incidence of duration of illness among both In-Patients and Out-Patients was above 3 years.

#### 16. OTHER SYSTEM INVOLVEMENT:

**Table 16** illustrates the distribution of Complications involving Other Systems.

**Table 16**

S. No	System	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Cardio Vascular System	1	5 %	-	-
2.	Gastro Intestinal System	8	40 %	7	35 %
3.	Musculo Skeletal System	1	5 %	-	-
4.	Central Nervous System	-	-	-	-

Among the In – Patients 40 % of the patients had Gastro-intestinal disturbance and 5 % of the patients had Cardiovascular system disturbance and 5 % of patients with Musculo Skeletal disturbance.

Among the Out – Patients 35 % of the patients had Gastro-intestinal disturbance.

The table showed the presence of Gastro-intestinal disturbance both in In – patients and Out – patients.

## 17. FAMILY HISTORY:

Table 17 illustrates the distribution of Family History

**Table 17**

S. No	Family History	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Positive	5	25%	5	25%
2.	Negative	15	75%	15	75%

Among the In-Patients and Out – Patients 75% of the patients had negative family history and 25% of the patients had positive family history.

The table showed that most of the patients had a negative family history.

## 18. DIET:

Table 18 illustrates the distribution of diet among the patients

**Table 18**

S. No	Diet	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vegetarian	1	5%	-	-
2.	Mixed diet	19	95%	20	100%

Among the In-Patients, 95% of the patients had mixed diet and 5% had vegetarian diet.

Among the Out-Patients 100% of the patients had mixed diet.

The table showed the highest incidence of the disease for the patients with mixed diet.

## 19. HABITS:

Table 19 illustrates the distribution of habits

**Table 19**

S. No	Habits	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Smoker	12	60%	14	70%
2.	Tobacco Chewer	8	40%	13	65%
3.	Betel nut chewer	8	40%	4	20%
4.	Alcoholic	12	60%	12	60%

Among the In-Patients 60% of the patients were smokers and alcoholics, 40% of the patients were tobacco chewers and betel nut chewers.

Among the Out-Patients 70% of the patients were smokers, 65 % of patients were tobacco chewers, 60 % of the patients were alcoholics and 20 % of the patients were betel nut chewers.

The table showed the highest incidence of the disease in the smokers.

## 20. IMPORIGAL (GNANENDHIRIYAM):

Table 20 illustrates the distribution of disease with Imporigal.

**Table 20**

S. No	Imporigal	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Mei	2	10%	-	-
2.	Vai	-	-	-	-
3.	Kann	12	60%	10	50%
4.	Mookku	10	50%	10	50%
5.	Sevi	1	5 %	-	-

Among the In – Patients, Kann was affected in 60% of the patients, Mookku was affected in 50% of the patients, Mei was affected in 10% of the patients and Sevi was affected in 5 % of the patients.

Among the Out - Patients, Kann and Mookku was affected in 50% of the Patients.

The table showed that Kann and Mookku was affected in most of the patients.



## 21. KANMENDHIRIYAM:

Table 21 illustrates the distribution of disease with Kanmendhiriyam.

**Table 21**

S. No	Kanmendhiriyam	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Kai	-	-	-	-
2.	Kaal	12	60%	10	50%
3.	Vai	-	-	-	-
4.	Eruvai	10	50%	10	50%
5.	Karuvai	-	-	-	-

Among the In-Patients, Kaal was affected in 60% of the patients and Eruvai was affected in 50% of the patients.

Among the Out- Patients, Kaal and Eruvai was affected in 50% of the patients.

The table showed that Eruvai and Kaal were affected in most of the cases.

## 22. KOSAM:

Table 22 illustrates the distribution of Kosam.

**Table 22**

S. No	Kosam	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Annamayakosam	8	40 %	7	35%
2.	Pranamayakosam	20	100%	20	100%
3.	Manomayakosam	-	-	-	-
4.	Gnanamayakosam	-	-	-	-
5.	Anandhamayakosam	-	-	-	-

In all the In-Patients and Out- Patients Pranamayakosam was affected. Annamayakosam was affected in 40% of the In-Patients as well as 35% of the Out-Patients.

## 23. MUKKUTRAM a.VADHA b.PITHA c.KABA

### 23.a. VADHA

Table 23.a illustrates the distribution of Vadha in the disease.

**Table 23 .a**

S. No	Types of Vadha	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Pranan	20	100%	20	100%
2.	Abanan	5	25%	10	50%
3.	Viyanan	12	60%	10	50%
4.	Udhanan	10	50%	10	50%
5.	Samanan	8	40%	7	35%
6.	Nagan	-	-	-	-
7.	Koorman	10	50%	10	50%
8.	Kirugaran	20	100%	20	100%
9.	Devathathan	6	30%	5	25%
10.	Dhananjeyan	-	-	-	-

Pranan and Kirugaran were affected in all the 100% of In - Patients and Out - Patients. Udanan and Koorman were affected in 50 % of the In-Patients and Out- Patients; Abanan was affected in 25 % of the In-Patients and 50 % of the Out- Patients. Viyanan was affected in 60% of the In-Patients and 50 % of the Out-Patients. Devathathan was affected in 30% of the In-Patients and 25 % of the Out-Patients. Samanan was affected in 40 % of the In – Patients and 35 % of the Out – Patients.

The table showed that the Pranan and Kirugaran were affected completely in this disease.

### 23. b. PITHA

**Table 23.b** illustrates the distribution of Pitha in the disease.

**Table 23.b**

S. No	Types of Pitha	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Analpitha	8	40%	7	35%
2.	Ranjagapitha	5	25 %	5	25 %
3.	Sadhagapitha	5	25%	4	20%
4.	Aalosagapitha	12	60%	10	50%
5.	Prasagapitha	2	10%	-	-

Analpitha was affected in all the 40 % of Out- Patients and 35 % of In – Patients. Ranjagapitha was affected in 25 % of In – patients and Out – Patients. Sadhagapitha was affected in 25 % of the In – Patients and 20 % of the Out- patients. Aalosagapitha was affected in 60 % of In – patients and 50 % of the Out – patients. Prasagapitha was affected in 10 % of the In – patients.

The table showed that the Aalosagapitha was affected in all the patients in this disease.

### 23. c. KABA

Table 23.C illustrates the distribution of Kaba in the disease.

**Table 23.c**

S. No	Types of Kaba	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Avalambagam	20	100%	20	100%
2.	Kilethagam	8	40 %	7	35 %
3.	Pothagam	-	-	-	-
4.	Tharpagam	2	10 %	2	10 %
5.	Sandhigam	12	60 %	10	50 %

Avalambagam was affected in all the 100% of In-Patients and Out-Patients. Kilethagam was affected in 40 % of the In-Patients and 35% of the Out-Patients. Tharpagam was affected in 10 % of the In – patients and out – patients. Sandhigam was affected in 60 % of the In – Patients and 50 % of the Out – Patients.

The table showed that the Avalambagam was affected in all the patients in this disease.

## 24. EZHU UDAL KATTUGAL:

Table 24 illustrates the distribution of derangement of Udal Kattugal in the disease.

**Table 24**

S. No	Ezhu Udal Kattugal	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Saaram	20	100 %	20	100%
2.	Senneer	5	25 %	5	25 %
3.	Oon	8	40 %	5	25 %
4.	Enbu	12	60 %	10	50%
5.	Kozhuppu	12	60%	10	50%
6.	Moolai	-	-	-	-
7.	Sukkilam / Suronitham	-	-	-	-

Saaram was affected in all the 100% of the In-Patients and Out – Patients. Among the In -Patients, Senneer was affected in 25% of the patients, Oon were affected in 40% of the patients. Enbu and Kozhuppu was affected in 60 % of patients.

Among the Out – Patients, Oon was affected in 25 % of patients, Enbu and Kozhuppu was affected in 50 % of the patients.

The table showed that Saaram was affected in most of the patients in this disease.

## 25. EN VAGAI THERVUGAL:

Table 25 illustrates the distribution of En Vagai Thervugal in the disease.

Table 25

S. No	En Vagai Thervugal	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Naa	9	45%	8	40%
2.	Niram	-	-	-	-
3.	Mozhi	-	-	-	-
4.	Vizhi	8	40%	6	30%
5.	Malam	10	50%	10	50%
6.	Moothiram	5	25 %	2	10 %
7.	Sparisam	2	10 %	-	-
8.	Naadi				
	a.Vadha Kaba	17	85%	19	95%
	b.Pitha Kaba	2	10%	-	-
	c.Kaba Vadha	1	5%	1	5%

Naa was affected in 45 % of the In-Patients and 40 % of the Out-Patients. Vizhi was affected 40% of the In-Patients and 30% of the Out-patients. Malam was affected 50 % of both In-Patients and Out-Patients. Moothiram was affected in 25 % of In-patients and 10 % of the Out – Patients. Sparisam was affected in 10 % of the In – Patients. In Naadi, 85% of In-Patients and 95% of Out-Patients had Vadha Kaba Naadi, 10% of the In-Patients had Pitha Kaba Naadi. 5% of In-Patients as well as Out-Patients had Kaba Vadha Naadi.

The table showed that Naa and Vizhi were affected in most of the patients. In Naadi, Vadha Kaba Naadi showed higher frequency than the others.

## 26. NEER KURI:

**Table 26** illustrates the distribution of Neer Kuri in the disease.

**Table 26**

S. No	Neerkuri	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Niram	10	50%	12	60%
2.	Edai	-	-	-	-
3.	Manam	-	-	-	-
4.	Nurai	-	-	-	-
5.	Enjal	-	-	-	-

Niram was affected in 50% of the In-Patients and 60% of the Out-Patients. Edai, Manam, Nurai, Enjal were not affected.

## 27. NEIKURI:

**Table 27** illustrates the distribution of Neikuri in the disease.

**Table 27**

S. No	Neikuri	In-Patients		Out-Patients	
		No.of.Cases	Percentage	No.of.Cases	Percentage
1.	Vadha Neer	6	30%	4	20%
2.	Pitha Neer	3	15%	1	5%
3.	Kaba Neer	3	15%	2	10%
4.	Vadha Pitha Neer	4	20%	1	5%
5.	Vadha Kaba Neer	3	15%	1	5%
6.	Pitha Vadha Neer	1	5%	1	5%

In Neikuri, 30% of In-Patients and 20% of Out-Patients showed Vadha Neer, 15% of In-Patients and 5% of Out-Patients showed Pitha Neer. 15% of In-Patients and 10% of Out - Patients showed Kaba Neer. 20% of In-Patients and 5% of Out-Patients had Vadha Pitha Neer. 15% of In-Patients and 55% of Out - Patients had Vadha Kaba Neer. 5% of each In-Patients and Out-Patients showed Pitha Vadha Neer.

The table showed that Vadha Neer was found in most of the cases.

## **28. LABORATORY INVESTIGATIONS:**

Table 28 – A, 28 – B, 28 – C and 28 – D illustarte the laboratory investigations.

Sputum for AFB and Mantoux test were found to be negative in all the 100% of In-Patients and Out-Patients.

## **29. GRADATION OF RESULTS:**

**Table 29 illustrates the Gradation of Results**

**Table 29**

<b>S. No</b>	<b>Result</b>	<b>In-Patients</b>		<b>Out-Patients</b>	
		<b>No.of.Cases</b>	<b>Percentage</b>	<b>No.of.Cases</b>	<b>Percentage</b>
1.	Good	10	50%	9	45%
2.	Fair	8	40%	9	45%
3.	Poor	2	10%	2	10%

Goods results were found in 50% of the In-Patients and in 45% of the Out-Patients. Fair results were found in 40% of the In-Patients and in 45% of the Out - Patients. Poor results were found in 10% of the In-Patients and 15% of the Out - Patients.



## **DISCUSSION**

Millions of people are affected all over the world by the disease Swasakasam, which is similar to Bronchial Asthma, occurring due to pollution of environment and changes in life style.

Efficacy of Siddha system in curing the respiratory disease prompted the author to carry out clinical and scientific study in this subject.

For the clinical study 20 selected patients were admitted as In-Patients in Post Graduate Department of Maruthuvam and were treated with the trial medicine. After discharge all the twenty patients were followed as the Out-Patients.

The medicine was also trialed with 20 Out-Patients in the Out-Patients Department.

The results were clearly observed and recorded under the supervision of Professor, reader, Lecturer and Assistant Lecturer. The observed results were discussed here.

### **1. SEX DISTRIBUTION:**

Males are affected in 60% of the In - Patients and 85% of the Out-Patients. Females are affected in 40% of the In- Patients and 15% of the Out- Patients.

This indicated that males were affected more than females.

### **2. AGE DISTRIBUTION:**

Among the In- Patients 50% of the patients were observed in the age group of 61 and above, 20 % of the patients were observed in the age group of 51-60 years and 41 - 50, 5 % of the patients were observed in the age group of 31-40 years and 21 – 30 years.

Among the Out - Patients, 35% of the patients were observed in the age group of 61 and above, 30% of the patients were observed in the age group of 51 – 60 years, 15% of the patients were observed in the age group of 41 - 50 years, 10 % of the patients were observed in the age group of 31-40 years and 0 – 20 years.

It showed predominance of distribution in the age group 61 and above years among the In-Patients and Out - Patients.

It is indicated that the increased incidence of disease during old age was due to decreasing lung capacity, lung function and immunity.

### **3. KAALAM DISTRIBUTION:**

Among the In - Patients 75% were affected in Pitha Kaalam, 20% were affected in Kaba Kaalam and 5 % in the Vadha Kaalam.

Among the Out - Patients 65% were affected in Pitha Kaalam, 20% were affected in Kaba Kaalam, and 15% were affected in Vadha Kaalam.

It showed the increased incidence of the disease in the Pitha Kaalam i.e 34 - 66 years.

### **4. DEHI DISTRIBUTION:**

From the table, it was observed that all the patients, i.e. 100% come under the Dhontha Dehi, among both the In-Patients and Out-Patients.

This showed the incidence of this disease only in the Dhontha Dehi.

### **5. GUNAM DISTRIBUTION:**

100% of both In-Patients and Out-Patients fall under the type of Rajo Gunam.

This showed the incidence of this disease only in the Rajo Gunam persons.

## **6. RELIGION DISTRIBUTION:**

Among the In-Patients and Out – Patients 95% were Hindus, 5% were Christians.

## **7. PARUVAKAALAM DISTRIBUTION:**

Among the In-Patients, 65 % of the disease comes under the Koothir Kaalam and 20% of the incidence comes under the Kaar and Muthuvenil Kaalam.

Among the Out-Patients, 50 % of the incidence comes under the Kaar and Muthuvenil Kaalam.

It showed the prevalence of disease under Koothir Kaalam among the In-Patients and Kaar and Muthuvenil Kaalam among the Out – Patients.

## **8. SIRUPOLUDHU DISTRIBUTION:**

Among the In-Patients, 90% of the incidence occurs during Vaigarai Poludhu and 10% occurs during Maalai Poludhu.

Among the Out-Patients, 85% of the incidence occurs during Vaigarai Poludhu and 15% occurs during Maalai Poludhu.

It showed the prevalence of disease during Vaigarai Poludhu( Early morning ) in all patients.

## **9. THINAI DISTRIBUTION:**

Among the In-Patients, 95% belonged to the Marutham and 5% belonged to the Neithal.

Among the Out-Patients, all the 100% belonged to the Marutham.

It showed that Marutham was the place of incidence of the disease.

## **10. OCCUPATION:**

Among the In-Patients 35% were Drivers and Housewives, 10% were Rice Mill workers and Garment Employees and 5 % were Cooks and Mason. Among the Out-Patients 35% each were Drivers, 20% were Rice Mill workders, 10% were Students, 5% were teachers and teashop workers.

This observation indicated increased incidence of the disease in drivers and rice mill workers.

## **11. SOCIO - ECONOMIC STATUS:**

Among In – Patients 100 % of the cases are economically poor.

Among the Out – Patients 65 % are economically poor and 25 % belong to the middle class.

## **12. AETIOLOGICAL FACTOR (ALLERGEN):**

Among the In-Patients, 30% of the patients had dust and cold exposure collectively as their aetiological factor, 25% each had dust and husks of grains and cold exposure collectively as their aetiological factors, 10% of the patients had smoke as their aetiological factor. Another 10% of the patients had other factors such as cotton fibre and exposure to sulphur and lead as their aetiological factors.

Among the Out-Patients, 35% of the patients have dust as their aetiological factor, 25% had husks of grains as their aetiological factor, and 10% had smoke as their aetiological factor. Another 30% had other factors such as cotton fibre, soap as their aetiological factors.

The above data illustrated that, dust and cold exposure, dust were the main aetiological factors among these patients.

According to literature, the aetiological factors are excessive

inhalation of smoke, cold climate, noisy wind, husk of grains. Thus above data coincided with the literature.

### **13. MODE OF ONSET:**

The observation illustrated that the mode of onset was gradual in all the 100% of both In-Patients and Out- Patients.

### **14. CLINICAL FEATURES:**

Among the In-Patients and Out-Patients, there was 100% incidence of clinical features of tightness of chest, wheeze, cough with scanty expectoration and tachycardia.

Among the In-Patients, there was 40% incidence of fever and Clubbing. 30 % incidence of Sneeze, 20 % incidence of running nose and 10 % incidence of Urticaria.

Among the Out-Patients, there was 40% incidence of Sneeze and Running nose, 30 % incidence of Fever and Clubbing and 10 % incidence of Urticaria.

### **15. DURATION OF ILLNESS:**

Among the In-Patients, there was 25 % incidence for the duration of 6 months – 1 year and 1 – 2 years, 20% incidence of duration above 3 years and 10 % incidence of duration below 3 months, 3 - 6 months and 2 – 3 years.

Among the Out-Patients, 40% incidence showed the duration of above 3 years and 30% incidence showed the duration of 6 months -1 year, 15% incidence showed the duration of 1 - 2 years and 10 % incidence showed the duration of below 3 months and 5 % incidence showed the duration of 2 -3 years.

The data indicated highest incidence of duration of illness among both In-Patients and Out-Patients was above 3 years.

#### **16. OTHER SYSTEM INVOLVEMENT:**

Among the In – Patients 40 % of the patients with Gastro-intestinal disturbance and 5 % of the patients with Cardiovascular system disturbance and 5 % of patients with Musculo Skeletal disturbance.

Among the Out – Patients 35 % of the patients with Gastro-intestinal disturbance.

The data showed the presence of Gastro-intestinal disturbance both in In – patients and Out – patients.

#### **17. FAMILY HISTORY:**

Among the In-Patients and Out – Patients 75% of the patients had negative family history and 25% of the patients had positive family history.

This showed that most of the patients had a negative family history.

#### **18. DIET:**

Among the In-Patients, 95% of the patients had mixed diet and 5% had vegetarian diet.

Among the Out-Patients 100% of the patients had mixed diet.

The data showed the highest incidence of the disease for the patients with mixed diet.

According to Yugivaidhya Chinthamani dietary factors are improperly cooked food and non vegetarian diet. Here the observation coincides with yugi's concept.

## **19. HABITS:**

Among the In-Patients 60% of the patients were smokers and alcoholics, 40% of the patients were tobacco chewers and betel nut chewers.

Among the Out-Patients 70% of the patients were smokers, 65 % of patients were tobacco chewers, 60 % of the patients were alcoholics and 20 % of the patients were betel nut chewers.

This showed the highest incidence of the disease in the smokers.

## **20. IMPORIGAL (GNANENDHIRIYAM):**

Among the In – Patients, Kann was affected in 60% of the patients, Mookku was affected in 50% of the patients, Mei was affected in 10% of the patients and Sevi was affected in 5 % of the patients.

Among the Out - Patients, Kann and Mookku was affected in 50% of the Patients.

This showed that Kann and Mookku were affected in most of the patients.

## **21. KANMENDHIRIYAM:**

Among the In-Patients, Kaal was affected in 60% of the patients and Eruvai was affected in 50% of the patients.

Among the Out- Patients, Kaal and Eruvai was affected in 50% of the patients.

This showed that Eruvai and Kaal were affected in most of the cases

## **22. KOSAM:**

In all the In-Patients and Out- Patients Pranamayakosam was affected. Annamayakosam was affected in 40% of the In-Patients as well as 35% of the Out-Patients.

Pranamayakosam is made up of Pranana and Kanmendhiriyam. Since, Pranana and Eruvai of the Kanmendhiriyam were affected; Pranamayakosam was affected in this disease.

Manomayakosam is made up of Manana and Gnanendhiriyam.

Gnanamayakosam is made up of Buddhi and Gnanendhiriyam.

Anandhamayakosam is made up of Pranana and Suluthi. Since, Pranana was affected; Anandhamayakosam was affected in this disease.

## **23. MUKKUTRAM a. VADHA b.PITHA c.KABA**

### **23. a.VADHA :**

Pranana and Kirugaran were affected in all the 100% of In - Patients and Out - Patients. Udanana and Koorman were affected in 50 % of the In-Patients and Out- Patients; Abanana was affected in 25 % of the In-Patients and 50 % of the Out- Patients. Viyanana was affected in 60% of the In-Patients and 50 % of the Out-Patients. Devathathan was affected in 30% of the In-Patients and 25 % of the Out-Patients. Samanana was affected in 40 % of the In – Patients and 35 % of the Out – Patients.

Pranana is responsible for respiration. In Swasakasam, this vayu was affected leading to difficulty in breathing, cough and sneeze is also caused by Pranana.

Viyanana's main function is distribution of Saaram in the body. Since, Saaram of Ezhu Udal Kattugal was affected; this vayu was affected in this disease.



Udhanan is responsible for speech, strength of the mind and the body. Since, there was low pitched voice, decrease in strength of body and mind, this vayu was affected in this disease.

Samanan is responsible for controlling other vayus and digestion. Since, Samanan cannot control other vayus; it was affected in this disease.

Kirugaran is responsible for appetite, sneeze, cough and running nose. It was affected in this disease.

Koorman is responsible for vision. This was due to aging.

Devathathan is responsible for tiredness after sleep and emotion. This was due to severity of the asthmatic episode.

### **23. b. PITHA:**

Analpitha was affected in all the 40 % of Out- Patients and 35 % of In – Patients. Ranjagapitha was affected in 25 % of In – patients and Out – Patients. Sadhagapitha was affected in 25 % of the In – Patients and 20 % of the Out- patients. Aalosagapitha was affected in 60 % of In – patients and 50 % of the Out – patients. Prasagapitha was affected in 10 % of the In – patients.

Sadhaga Pitha influences the normal day to day activities with the help of mind and brain. Since dyspnoea and restlessness is present, this Pitha was affected in this disease.

Anal Pitha is responsible for appetite. Since, there was loss of appetite; this Pitha was affected in this disease.

Aalosaga Pitha is responsible for clear vision. Since, there was diminished vision, this was affected. It was due to their aging.

### **23. c. KABA:**

Avalambagam was affected in all the 100% of In-Patients and Out-Patients. Kilethagam was affected in 40 % of the In-Patients and 35% of the Out-Patients. Tharpagam was affected in 10 % of the In – patients and out – patients. Sandhigam was affected in 60 % of the In – Patients and 50 % of the Out – Patients.

Avalambagam is residing in lungs and helps other four types of Kaba to function. It was deranged due to the presence of tightness of chest, cough, wheezing and dyspnoea.

Sandhigam resides in the joints and helps for movement. Since, there was joint pain, it was affected. It was due to their aging.

### **24. EZHU UDAL KATTUGAL:**

Saaram was affected in all the 100% of the In-Patients and Out – Patients. Among the In -Patients, Senneer was affected in 25% of the patients, Oon were affected in 40% of the patients. Enbu and Kozhuppu were affected in 60 % of patients.

Among the Out – Patients, Oon was affected in 25 % of patients, Enbu and Kozhuppu was affected in 50 % of the patients.

Saaram strengthens the body and mind. Since, there is loss of appetite causing body tiredness.

Enbu and Kozhuppu are responsible for the movements of the body and gives lubrication to the joint cavities. Since, there was joint pain; these two were affected due to aging.

## **25. EN VAGAI THERVUGAL:**

Naa was affected in 45 % of the In-Patients and 40 % of the Out-Patients. Vizhi was affected 40% of the In-Patients and 30% of the Out-patients. Malam was affected 50 % of both In-Patients and Out-Patients. Moothiram was affected in 25 % of In-patients and 10 % of the Out – Patients. Sparisam was affected in 10 % of the In – Patients. In Naadi, 85% of In-Patients and 95% of Out-Patients had Vadha Kaba Naadi, 10% of the In-Patients had Pitha Kaba Naadi. 5% of In-Patients as well as Out-Patients had Kaba Vadha Naadi.

This showed that Naa and Vizhi were affected in most of the patients. In Naadi, Vadha Kaba Naadi showed higher frequency than the others.

## **26. NEERKURI:**

Niram was affected in 50% of the In-Patients and 60% of the Out-Patients due to the vitiating Kaba. Edai, Manam, Nurai and Enjal were not affected.

## **27. NEI KURI:**

In Neikuri, 30% of the In-Patients and 20% of the Out-Patients had Vadha Neer, 15% of the In-Patients and 5% of the Out-Patients had Pitha Neer. 15% of the In-Patients and 10% of the Out-Patients showed Kaba Neer. 20% of In-Patients and 5% of Out-Patients showed Vadha Pitha Neer. 15% of the In-Patients and 5% of the Out-Patients had Vadha Kaba Neer. 5% of each In-Patients and Out-Patients had Pitha Vadha Neer.

This showed the Vadha Neer was found in most of the cases.

## **28. LABORATORY INVESTIGATIONS:**

Routine investigations of blood and urine were done during the admission and at the end of the treatment for every case.

Blood sugar, urea and serum cholesterol were found to be in normal range before and after treatment.

X-Ray Chest PA view showed normal for 85% of the cases and 15% of the cases showed bronchitis in In-Patients.

In the Out-Patients, X -Ray Chest PA view showed 100% normal.

The Peak Flow Meter Reading showed diurnal variation. The Peak Flow Meter Reading ranged from 60L/min. to 150 L/min. before treatment among the In-Patients. After treatment it ranged from 150L/min. to 350L/min.

Among the Out-Patients, the Peak Flow Meter Reading ranged from 60L/min. to 250L/min. before treatment, and after treatment, it ranged from 160L/min. to 340L/min.

Pulmonary Function Tests are done for one patient each from In-Patients and Out-Patients. The results showed clinically co-related Obstructive Lung Disease.

Urine examination showed nil albumin and sugar, but 10% In-Patients showed pus cells and epithelial cells in their urine.

Urine examination in Out-Patients showed 15% of the patients had epithelial cells, and after treatment it was NAD, with appropriate medicine.

Blood investigation of In-patients showed total count of WBC within the normal range. Eosinophils count was increased and ranged from 2% to 22% cells before treatment, and after treatment it ranged 1% to 10%. ESR was raised and Hb content was normal before treatment. ESR was reduced after treatment.

Blood investigation of Out-Patients showed TC with in the normal

range. Eosinophils count was raised and should the range of 2% to 12% cells before treatment, and after treatment it ranged 1% to 6%. Hb content was in normal range.

Motion test revealed that 100% of both In-Patients and Out-Patients had nil result before treatment as well as after treatment.

Sputum examination was found to be negative for all the 100% of both the In-Patients and Out-Patients.

Mantoux test was found to be negative for all the 100% of both the In-Patients and Out-Patients.

### **MODERN MEDICINE COMPARISON:**

According to Modern Medicine, the aetiological factors for the disease are exposure to dust, smoke, pollens, grains, chemicals, and cold exposure, stress and food habits.

In our literature, Yugi said more or less the same reasons for the disease.

The signs and symptoms of the disease **Bronchial Asthma** are closely matched with **Swasakasam** as explained by Yugi Muni.

### **TREATMENT:**

On the previous day of treatment, laxative Nilavagai Choornam 5 gm with hot water at the bed time before starting the internal medicine.

On the first day, the trial medicine “**Mungiluppu Chooranam**” - 4 gm twice daily with honey after meals was prescribed and was given till the end of their treatment.

**DIET REGIMEN:**

Patients were advised to avoid watery vegetables and fruits, bitter guard, jack fruit, dry fish, fish, onion, butter, curd, coconut, cold water and cold food.

Patients were recommended to take vegetables such as brinjal, drumstick, ginger, garlic, raddish, greens, such as Eclipta Alba etc., and goat ghee.

**PRAHNAYAMAM:**

Patients were advised to do Prahnyamam breathing exercise 20 counts twice daily for better results.

**YOGA THERPY:**

Yogasanam such as Bujangasanam, Chakrasanam, Machasanam, Mayurasanam, Patha Hasthasanam, Arai Machayenrhirasanam, Trikonasanam and Savasanam were advised to be practiced for quick relief.

**GRADATION OF RESULTS:**

Good results showed that the asthmatic episodes in a week are reduced more than 50% and markedly increased PFMR, after treatment. 50% of the In-Patients and 45 % of the Out-Patients were observed good results.

Fair results showed that the asthmatic episodes in a week are reduced more than 80% and greatly increased PFMR, after treatment. 40% of the In-Patients and 40% of the Out-Patients were observed fair results.

Poor results showed that the asthmatic episodes in a week are reduced below 50% or no improvement and only a minimal increase in

PFMR after treatment. 10% of the In-Patients and 10 % of the Out-Patients are observed in poor results.

Biochemical analysis showed the presence of sulphate, ferrous iron, tannic acid and unsaturated compound in the trial medicine "**Mungiluppu Chooranam**".

Pharmacological analysis revealed that the trial medicine "**Mungiluppu Chooranam**" had significant anti-histamine and anti-spasmodic activities.

Anti-microbial study showed the trial medicine is sensitized to **Staphylococcus aureus**.

Clinically no side effects and adverse effects were noted for the maximum 60 days of therapy.

## SUMMARY

**Swasa kasam** is the common respiratory disease seen in day to day clinical practice.

Sincerity, charity and skill are the basis of practice of medicine. Further loving tender care is essential for winning co-operation and confidence of the patients for the ultimate recovery.

Economy is more important in the rising cost of living. Mungiluppu Chooranam is easily preparable, low economic and purely herbal, the author had selected as the trial medicine.

The aetiology, pathology, classification, clinical features, diagnosis, complications, prognosis, treatment and preventive measures were selected from Siddha and Modern Systems of Medicine.

In this study, 20 patients of both sexes of varying age groups were selected as In-Patients and 20 patients as Out-Patients.

From the observation and results, we were clear that the disease was common in the following aspects.

- ❖ Males were mostly affected than Females. Age incidence has commonly found in all decades. Increased incidence during their occupational period.
- ❖ All the patients had Dhontha Dehi, Rajogunam and Poor Socio-Economic status.
- ❖ Majority of the cases were affected in Koothir Kaalam (Iyppasi & Karthigai).
- ❖ Most of the cases got the acute symptoms during Vaigarai Poludhu i.e., 4 hours before sunrise. Most of the patients belonged to the Thinai Marutham.
- ❖ In the occupation, Drivers and rice mills workers were mostly affected.
- ❖ Aetiological factors were mostly dust, smoke & cold exposure.



- ❖ All the patients had gradual onset of the disease.
- ❖ All the patients were affected with clinical features of running nose, sneeze, tightness of chest, and cough with scanty mucoid sputum.
- ❖ Duration of illness ranged from 3 months – above 3 years.
- ❖ Gastro Intestinal system was mostly affected in this disease.
- ❖ 75% of the In-Patients and Out-Patients had negative family history.
- ❖ Smokers were mostly affected in this disease.
- ❖ In Imporigal, Mookku and Kann was affected in all the cases of In-Patients and Out-Patients
- ❖ In Kanmendhiriyam, Eruvai and Kaal were mostly affected.
- ❖ In Kosam Pranamayakosam was affected in all the patients
- ❖ In Mukkutram,
  - In Vadha, Pranan, Kirugaran were affected in all the 100% of the both In-Patients and Out-Patients.  
Abanan, Samanan, Viyanan, Udhanan, Devathathan were affected in many cases of both In-Patients and Out-Patients.
  - In Pitha Aalosagapitha and Analpitha Pitha was affected in most of the patient.
  - In Kaba, Avalambagam was affected in all the cases.  
Sandhigam and Kilethagam were affected in few cases.
- ❖ In Ezhu Udal Kattugal, Saaram was affected in most of the cases.  
Oon, Enbu and Kozhuppu were affected in few cases.
- ❖ In En Vagai Thervugal,
  - Naa, Vizhi and Malam were affected in most of the patients.
  - Neerkuri showed transparent urine.
  - Neikuri showed derangement of Mukkutram. Vadha Neer was found in most of the cases.

- ❖ Laboratory Investigations showed normal blood sugar, urea, serum cholesterol, normal Hb content, TC count, raised Eosinophil count and ESR. Raised Eosinophil count and ESR were reduced after treatment.
- ❖ Urine analysis showed Epithelial cells in few cases
- ❖ Sputum analysis showed negative AFB and Mantoux test was negative in all the patients.
- ❖ X-Ray Chest PA view was normal in most of the cases.
- ❖ Peak Flow Meter Reading among the In-Patients ranged from 60 L/min. to 150 L/min. before treatment and 150 L/min. to 350 L/min after treatment. Among the Out-Patients it ranged from 60 L/min. to 250 L/min. before treatment and after treatment it ranged from 160 L/min. to 340 L/min. It showed better prognosis of the disease.
- ❖ Pulmonary Function Tests showed the results for 2 patients that they had obstructive lung disease.
- ❖ The efficacy of the trial medicine, Mungiluppu Chooranam was studied and observed during the dissertation period.
- ❖ All the patients were advised to follow strict diet restrictions and advised to practice prahnayama and yoga therapy for fast relief.
- ❖ Clinically 50% of the patients showed good results.
- ❖ No side effects and adverse effects were noticed during the period of study.
- ❖ Biochemical analysis showed the presence of the starch, ferrous iron, amino acid and unsaturated compound.
- ❖ Pharmacological analysis showed that the trial medicine had significant anti-spasmodic and anti-histamine activities.
- ❖ Anti-microbial studies showed that the drug Mungiluppu Chooranam was sensitised to the Staphylococcus aureus.

## CONCLUSION

- ❖ 50% of the patients showed good results, 40% showed fair results and 10% showed poor results in this trial study.
- ❖ The trial medicine **Mungiluppu Chooranam** has the tastes of Kaippu, Kaarppu and Inippu altogether.

**Suvai** : Kaippu, kaarppu, Inippu

**Thanmai** : Veppam

**Pirivu** : Kaarppu

As the tastes Kaippu, Kaarppu both antagonise the excessive Kaba, and Inippu has the soothing effect on throat which have been irritated by the chronic cough, **Mungiluppu Chooranam** act as an Anti – Kaba medicine.

And the trial medicine has its Thanmai as Veppam. All the Veppam natured drugs will eliminate the excessive Kaba in Kaba pateints. This medicine also acts as an Anti-Kaba medicine.

After ingestion while the trial medicine reaches the gastric juice, it will change into the Vibagam- Kaarppu. At this stage also, the medicine will act as an Anti-Kaba medicine due to the Vibagam- Kaarppu.

- ❖ Mungiluppu Chooranam acts as an Anti – Kaba medicine on the basis of Suvai, Thanmai, Pirivu and Vibagam and on the basis of Edhir Urai to Kaba diseases.

Thus Mungiluppu Chooranam yield good prognosis in Swasakasam patients.

- ❖ The trial medicine was simple to prepare low economic and free from side effects.
- ❖ Further follow up of these patients showed sense of well being and complete disappearance of symptoms.
- ❖ So, **Swasakasam** is controllable with **Mungiluppu Chooranam**.

**PREPARATION OF THE TRIAL MEDICINE**  
**MUNGILUPPU CHOORNAM**

**INGREDIENTS:**

1. MUNGILUPPU - 4 parts
2. TIPPILI - 2 parts
3. YELAM - 1 part
4. ELAVANGAM - ½ parts
5. SAKKARAI - 8 parts

**METHOD OF PREPARATIONS:**

All the ingredients except mungiluppu are dried, cleaned, slightly fried and finally powdered and mixed with equal quantity of sugar.

**DOSAGE**

4 gm, twice daily, after food

**ADJUVANT:**

Honey

**INDICATION:**

Cough, asthma and kaba diseases.

**DETAILS OF THE INGREDIENTS**

**மூங்கில் உப்பு**

Common name	:	மூங்கிலுப்பு
Synonyms	:	ஆம்பல், கனை, கீசகம், பனை, முடங்கள், வேல், வேணு
Botanical name	:	Bambusa arundinaceae
Family	:	Graminae
Parts used	:	salt of mungil

**Characters:**

Suvai : Thuvarppu

Thanmai : Veppam

Pirivu : Kaarppu

**Chemical constituents:**

Cholin, betain, nuclease, urease, proteolytic enzyme, diastatic and emulsifying enzyme and oxalic acid.

**Therapeutic action:**

Stimulant, astringent, tonic, antispasmodic and aphrodisiac

**Uses:**

Used in cough and asthma

**ஏலம்**

Common name : Yelam

Synonyms : ஆஞ்சி, கோரங்கம், குடி

Botanical name : Elettaria cardamomum

Family name : Zingiberaceae

Parts used : seeds

**Character:**

Suvai : Kaarpu

Thanmai : Veppam

Pirivu : Kaarpu

**Chemical constituents:**

Essential oils – the principal constituents of oil are cineol, terpineol, terpinene, limonene, sabinene and terpineol in the form of formic and acetic acids.

**Therapeutic action:**

Stimulant, carminative, stomachic, aromatic and diuretic

### Uses:

”தொண்டை வாய் கவுள் தாலுகு தங்களில்  
தோன்றும் நோயதி சாரம் பன் மேகத்தால்  
உண்டை போல் எழுங் கட்டி கிரிச்சரம்  
உழலை வாந்தி சிலந்தி விஷஞ்சுரம்  
பண்டை வெக்கை விதாகநோய் காசமும்  
பாழுஞ் சோமப் பிணி விந்து நட்டமும்  
அண்டை யீளை வன் பித்தம் இவைக்கெல்லாம்  
ஆலமாங்கமழ் ஏல மருந்ததே

- தேரையர் குணவாகடம்

It is given in sore throat, oral diseases, cough, diarrhoea, burning micturition, cough and asthma.

### இலவங்கம்

Common name : Lavangam  
Synonyms : அஞ்சுகம், உற்கடம், திரளி, கருவாய் கிராம்பு,  
சோகம், வராங்கம்  
Botanical name : Syzygium aromaticum  
Family : Myrtaceae  
Parts used : dried flower buds.

**Chemical constituents:** Essential oil containing eugenol.

**Therapeutic action:** Antispasmodic, carminatives, stomachic, aromatic

### Uses:

தாதுநட்டம் பேதி சருவ விஷம் ஆகிய நோய்  
பூதகிரகஞ் சிலந்திப் பூச்சிவிடஞ் - சாதிவிடம்  
ஆட்டு மிரைப் போடிருமல் ஆகிய நோய்க்கூட்டமற  
ஓட்டுமில வங்கத் தூரி

- அகத்தியர் குணவாகடம்

It is used in poisons, snake bite, cough, asthma and bronchitis.

## திப்பிலி

Common name	:	Thippili
Synonyms	:	, iÁý, Ái,¼ç, s,i"ÆÁÛì,ç, -¼ç ÁÕóÐ
Botanical name	:	Piper longum
Family	:	Piperaceae
Parts used	:	Fruit (Dried)

### Characters

Suvai	:	Kaarppu
Thanmai	:	Veppam
Pirivu	:	Inippu

### Chemical constituents :

Alkaloid - Piperine, Resin, Volatile oil, Starch, Gum, Fatty oil and Inorganic matter.

### Therapeutic action :

Stimulant, Good Expectorant Carminative, Alterative and tonic.

### Uses:

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- À¼i÷ò¼ Ì½Áççì,ò

It is used in chest affections, cough, cold, and asthma, diseases of respiratory system, dry cough, chronic cough and chronic bronchitis.

### **சர்க்கரை**

Common Name	:	Karumbu sarkarai
Synonyms	:	ÒÉ÷â°õ, þîÏ, §Åö
Botanical Name	:	Saccharum officinarum
Family	:	Graminae
Parts Used	:	Crystallised sugar obtained from the juice.

### **Characters**

Suvai	:	Inippu
Thanmai	:	Seetham
Pirivu	:	Inippu

### **Chemical Constituents :**

Water, Mucilage, Resin, Fat and Albumen

### **Therapeutic Action :**

Preservative, Demulcent, Anti- septic, Laxative, Diuretic

### **Uses:**

It is used to preserve food and medicines. It is used in sore throat and commencing cold.



# BIO - CHEMICAL ANALYSIS OF MUNGILUPPU

## CHOORANAM

### PREPARATION OF THE EXTRACT

5gms of chooranam was weighed accurately and placed in a 250ml clean beaker. Then 50ml distilled water was added and dissolved well. Then it was boiled well for about 10 minutes. It is cooled and filtered in a 100ml volumetric flask and then it is made up to 100ml with distilled water. This fluid was taken for analysis.

### QUALITATIVE ANALYSIS

S.NO	EXPERIMENT	OBSERVATION	INFERENCE
1.	<u>TEST FOR CALCIUM</u> 2ml of the above prepared extract is taken in a clean test tube. 2 ml of 4% Ammonium oxalate solution is added to it.	No White precipitate is formed.	Absence of calcium.
2.	<u>TEST FOR SULPHATE:</u> 2ml of the extract is added to 5% barium chloride solution.	No white precipitate is formed.	Absence of sulphate.
3.	<u>TEST FOR CHLORIDE</u> The extract is treated with silver nitrate solution.	No white precipitate is formed.	Absence of chloride.
4.	<u>TEST FOR CARBONATE</u> The substance is treated with concentrated HCL.	No brisk effervescence is formed.	Absence Of Carbonate.
5.	<u>TEST FOR STARCH</u> The extract is added with weak iodine solution.	Blue colour is formed.	Indidcats the presence of starch.
6.	<u>TEST FOR IRON-FERRIC</u> The extract is treated with concentrated Glacial acetic acid and potassium ferro cyanide.	No blue colour is formed.	Absence of ferric iron.

7.	<u>TEST OF IRON</u> <u>FERROUS:</u> The extract is treated with concentrated Nitric acid and ammonium thio cyanate.	Blood red colour is formed.	Indicates the presence of ferrous iron.
8.	<u>TEST FOR PHOSPHATE</u> The extract is treated with ammonium Molybdate and concentrated nitric acid.	No yellow precipitate is formed.	Absence of phosphate.
9.	<u>TEST FOR ALBUMIN</u> The extract is treated with Esbach's reagent.	No yellow precipitate is formed.	Absence of Albumin.
10.	<u>TEST FOR TANNIC ACID</u> The extract is treated with ferric chloride.	No Blue black precipitate is formed.	Absence of Tannic acid
11.	<u>TEST FOR UNSATURATION</u> Potassium permanganate solution is added to the extract.	It gets decolourised.	Indicates the presence of unsaturated compound.
12.	<u>TEST FOR THE REDUCING SUGAR</u> 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.	No colour change occurs	Absence of reducing sugar
13.	<u>TEST FOR AMINO ACID:</u> One or two drops of the extract is placed on a filter paper and dried it well. After drying, 1% Ninhydrin is sprayed over the same and dried it well.	Violet colour is formed.	Indicates the presence of Amino acid.

## **PHARMACOLOGICAL ANALYSIS**

### **1. ANTI - HISTAMINIC EFFECT ON “MUNGILUPPU CHOORANAM”**

#### **AIM:**

To study the anti - histaminic effect of “Mungiluppu Chooranam”.

#### **PREPARATION OF THE TRIAL MEDICINE:**

1gm of the Mungiluppu Chooranam was taken and mixed with 5ml of water and 5ml of honey and filtered.

#### **PROCEDURE:**

A guinea pig weighing about 350gm was starved for 48 hours and only water was allowed.

It was killed by stunning with a sharp blow on the head and cutting its throat to bleed to death. The abdomen was quickly opened and the viscera inspected and loops of intestine identified using the patch as a landmark. Then, the ileum was removed and placed in a shallow dish containing warm tyrode solution (37°C) and continuously aerated. The contents of the lumen of the ileum were washed and utmost care was taken to avoid any damage. It was cut into segments of 4 cm in a fully relaxed state and sutures were made with needle and tied on either side and the segment was suspended in an isolated organ bath. It was aerated by an oxygen tube immersed in tyrode solution. Drugs were given to study the inhibitory effect of histamine, induced contractions.

#### **INFERENCE:**

The test drug “**Mungiluppu Chooranam**” had significant effect.

## **2. ANTI - SPASMODIC EFFECT ON “MUNGILUPPU CHORANAM”**

### **AIM:**

To study the anti - spasmodic effect on “Mungiluppu Chooranam”.

### **PREPARATION OF THE TRIAL MEDICINE:**

1gm of the Mungiluppu Chooranam was taken and mixed with 5ml of water and 5ml of honey and filtered.

### **PROCEDURE:**

A rabbit weighing about 350 gm was starved for 48 hours and only water was allowed.

It was killed by stunning with a sharp blow on the head and cutting its throat to bleed to death. The abdomen was quickly opened and the viscera inspected and loops of intestine identified using the patch as a landmark. Then, the ileum was removed and placed in a shallow dish containing warm tyrode solution (37°C) and continuously aerated. The contents of the lumen of the ileum were washed and utmost care was taken to avoid any damage. It was cut into segments of 4 cm in a fully relaxed state and sutures were made with needle and tied on either side and the segment was suspended in an isolated organ bath. It was aerated by an oxygen tube immersed in tyrode solution. Drugs were given to study the inhibitory effect of Acetyl Choline.

### **INFERENCE:**

The test drug “**Mungiluppu Chooranam**” had significant effect.

## **MICROBIOLOGICAL STUDIES**

### **ANTI MICROBIAL STUDY OF MUNGILUPPU CHOORANAM**

#### **AIM:**

To study the anti microbial action of MUNGILUPPU CHOORANAM

#### **PROCEDURE:**

To prepare the chooranam 40 mg concentration of the drug, 4 grams of the drug was dissolved in 1 ml of sterile distilled water and from this master solution 40 micro litre was loaded on the disc.

#### **PREPARATIONS OF STANDARD STRAINS:**

Standard laboratory referral strains such were initially grown in nutrient agar and maintained at 37°C.

Before antibacterial testing each strain was inoculated in 5ml of Brain Heart Infusion broth (B.H.I) and incubated at 37°C for 30 minutes.

#### **ANTIMICROBIAL ACTIVITY TESTING BY KIRBY - BAUER DISC DIFFUSION METHOD:**

For antimicrobial activity 90mm petriplates with Muller Hinton. Agar (M.H.A) was used, for each organism, one M.H.A plate was used. The organisms grown in B.H.I Broth with 0.5 Mcfarland turbidity standard was poured on the M.H.A plate and allowed to spread uniformly. The excess broth was drained aseptically.

The disc which contains 40mg concentration of the drug was placed in M.H.A and incubated at 37°C for 24 hours.

#### **INTERPRETATION:**

Readings were taken after 24 hours of incubation. The inhibitory zone diameter was measured in millimeter scale.

#### **RESULT:**

MUNGILUPPU CHOORANAM was compared with standard antibiotics. The medicine was well sensitive against Staphylococcus aureus.

**PROFORMA OF CASE SHEET**  
**GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL**  
**DEPARTMENT OF POST GRADUATE POTHU MARUTHUVAM**  
**PALAYAMKOTTAI - TIRUNELVELI - 627002**  
**CASE SHEET PROFORMA FOR "SWASAKASAM" - (IP)**

Ward :	Occupation :
I.P No :	Income :
Bed No :	Nationality :
Name :	Religion :
Age/Sex :	Date of admission :
Address :	Date of discharge :
	Result :
	Diagnosis : <b>SWASAKASAM</b>
	Total No of days Treated:
	Medical officer :

COMPLAINTS AND DURATION:

HISTORY OF PRESENT ILLNESS:

HISTORY OF PAST ILLNESS:

PERSONAL HISTORY:

FAMILY HISTORY:

HABITS:

OCCUPATIONAL HISTORY:

**GENERAL EXAMINATION:**

Consciousness :  
 Decubitus :  
 Nourishment :  
 Anaemia :  
 Jaundice :  
 Clubbing :  
 Cyanosis :  
 Lymphadenopathy :  
 JVP :  
 Pedal Oedema :  
 Congenital anomaly :

**VITAL SIGNS** :  
 Temperature :  
 Pulse Rate :  
 Respiratory Rate :  
 Blood Pressure :

**❖ SIDDHA ASPECTS**

<b>NILAM</b> :	<b>PARUVAKALAM</b> :	<b>SIRUPOLUDHU:</b>
Kurinji :	Kaar :	Vaigarai :
Mullai :	Koothir :	Pagal :
Marutham :	Munpani :	Nanpagal :
Neithal :	Pinpani :	Pirpagal :
Palai :	Ilavenil :	Maalai :
	Muthuvenil :	Yamam :

**IMPORIGAL & IMPULANGAL**

Mei :  
 Vai :  
 Kann :  
 Mookku :  
 Sevi :

**KANMENTHIRIYAM & KANMAVIDAYAM**

Kai :  
 Kaal :  
 Vai :

Eruvai :

Karuva :

**KOSAM:**

**Annamayakosam** :

*(Digestive System)*

**Pranamayakosam** :

*(Respiratory System)*

**Manomayakosam** :

*(Cardio Vascular System)*

**Gnanamayakosam** :

*(Central Nervous System)*

**Aananthamayakosam** :

*(Reproductive System)*

**UYIR THATHUKKAL:**

**VATHAM**

Pranan :

Abanan :

Viyanan :

Uthanan :

Samanan :

Nagan :

Koorman :

Kirugaran :

Devathathan :

Dhananjayan :

**PITHAM**

Anarpitham :

Ranjagam :

Sathagam :

Alosagam :

Prasagam :



## **KABAM**

Avalambagam	:
Kilethagam	:
Tharpagam	:
Pothagam	:
Sandhigam	:

## **UDAL KATTUGAL**

Saaram	:
Senneer	:
Oon	:
Enbu	:
Kozhuppu	:
Moolai	:
Sukkilam / Suronitham	:

## **ENVAGAI THERVUGAL**

Naadi	:
Sparism	:
Naa	:
Niram	:
Mozhi	:
Vizhi	:
Malam	:
Moothiram	:

### ***(a) Neerkuri:***

i. Niram	:
ii. Manam	:
iii. Edai	:
iv. Nurai	:
v. Enjal	:

### ***(b) Neikuri:***

## ❖ MODERN ASPECTS

### I. Inspection

1. Trachea :
2. Chest wall symmetry :
3. Chest wall abnormality :
4. Harrison's sulcus :
5. Apical impulse :
6. Pulsatile swelling :
7. Intercostal Muscle Wasting :
8. Drooping of shoulder :
9. Intercostal bulging :
10. Respiratory Movement :
11. Measurements – AP :  
                                Transverse :
12. Suprasternal pulsation :
13. Carotid pulse :
14. Jugular venous pulsation:

### II. Palpation

1. Tracheal position (Tracheal sign) :
2. Apical impulse :
3. Respiratory Movements :
4. Local tenderness :
5. Tactile Fremitus :
6. Vocal Fremitus :

### III. Percussion

1. Abnormal Dulness :
  - i. Hyper Resonance :
  - ii. Dullness :
  - iii. Stony Dullness :
  - iv. Tidal percussion :
  - v. Ellie's 'S' shaped curve :

- vi. Straight line dullness :
- vii. Shifting dullness :
- viii. Succussion splash :
- 2. Traube's space :
- 3. Upper border of liver dullness :
- 4. Cardiac border :
- 5. Kronig's isthmus sign :

**Auscultation**

- 1. Breath sounds :
  - NVBS :
  - Bronchial breathing :
    - I. Tubular breathing :
    - II. Cavernous breathing :
    - III. Amphoric :
- 2. Adventitious sounds :
  - Wheeze continuous :
  - Crackles interrupted :
  - Pleural rub :
- 3. Vocal Resonance :
  - Aegophony :
  - Bronchophony :
  - Whispering pectoriloguy :

**IV. Other systemic Examination**

- Cardio Vascular System :
- Gastro Intestinal System :
- Central Nervous System :
- Musculo Skeletal System :

**VI. Lab Investigations**

<b>Blood</b>	<b>BT</b>	<b>AT</b>
TC	:	
DC	:	
ESR	:	
Hb	:	
Blood Sugar(R)	:	
Blood Urea	:	
Serum Cholesterol	:	
<b>Urine</b>		
Albumin	:	
Sugar	:	
Deposits	:	
<b>Motion</b>		
Ova	:	
Cyst	:	

**VII. Other Investigations**

- a) Mantoux test :
- b) Sputum AFB :
- c) X-Ray Chest PA View :
- d) Pulmonary Function Test :
- e) Arterial Blood Gases  
and oximetry :
- f) Absolute Eosinophil count :

**Treatment:**

- Diet :
- Advice :

**GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL,  
PALAYAMKOTTAI  
DEPARTMENT OF POST GRADUATE - POTHU MARUTHUVAM  
DISCHARGE CASE SHEET - 'SWASA KASAM'**

I.P.No : Occupation :  
 Bed No : Income :  
 Ward No : Nationality :  
 Name : Religion :  
 Age/Sex : Date of Admission :  
 Address : Date of Discharge :  
 No.of.Days treated:  
 Diagnosis : **'SWASA KASAM'**  
 Result :  
 Medical officer :

**Clinical Features:**

S.No	Signs and Symptoms	During Admission	During Discharge
1.	Running Nose		
2.	Sneezing		
3.	Difficulty in breathing		
4.	Cough with expectoration		
5.	Tightness of chest		
6.	Clubbing		
7.	Cyanosis		
8.	Sweating		
9.	Tachycardia		
10.	Fever		
11.	Eosinophil		
12.	Peak expiratory flow meter rate		

O.P No during Follow up :  
 No. of Days I.P Treated :  
 Total No.of Days Treated :

**PROFORMA OF CASE SHEET**  
**GOVERNMENT SIDDHA MEDICAL COLLEGE AND HOSPITAL**  
**DEPARTMENT OF POST GRADUATE POTHU MARUTHUVAM**  
**PALAYAMKOTTAI - TIRUNELVELI - 627002**

CASE SHEET PROFORMA FOR "SWASAKASAM" - O.P

O.P No : Occupation :  
Name : Income :  
Age/sex : Treatment Starting Date :  
Address : End of the Treatment Date :  
Total No. of Days Treated :  
Result :  
Diagnosis : **Swasakasam**  
Medical Officer :

**COMPLAINTS AND DURATION**

1. Running nose :
  2. Sneezing :
  3. Difficulty in breathing :
  4. Cough with expectoration :
  5. Tightness of chest :
  6. Fever :
  7. Flatulence :
  8. Excessive salivation :
  9. Throat irritation :
  10. Sweating :
  11. Tachycardia :
  12. Sputum :
- DURATION OF ILLNESS :  
PAST HISTORY :  
OCCUPATIONAL HISTORY :

## **GENERAL EXAMINATION**

Consciousness :  
Temperature :  
Decubitus : Pulse rate :  
Nourishment : Heart rate :  
Anaemia : Respiratory rate :  
Jaundice : Blood Pressure :  
Clubbing :  
Cyanosis :  
Lymphadenopathy :  
Pedaloedema :  
JVP :

## **ENVAGAI THERVUGAL**

Naadi :  
Sparisam :  
Naa :  
Niram :  
Mozhi :  
Vizhi :  
Malam :  
Moothiram :

### **a) Neerkuri**

- i. Niram :
- ii. Manam:
- iii. Edai :
- iv. Nurai :
- v. Enjal :

### **b) Neikuri :**

**Lab Investigations:**

<b>Blood</b>		<b>BT</b>	<b>AT</b>
TC	:		
DC	:		
ESR	:		
Hb	:		
Blood Sugar(R)	:		
Blood Urea	:		
Serum Cholesterol	:		
<b>Urine</b>			
Albumin	:		
Sugar	:		
Deposits	:		
<b>Motion</b>			
Ova	:		
Cyst	:		

**OTHER INVESTIGATIONS:**

- a. Mantoux Test :
- b. Sputum AFB :
- c. X - Ray Chest PA view :
- d) Pulmonary Function Test :
- e) Arterial Blood Gases  
and oximetry :
- f) Absolute Eosinophil count :

**EXAMINATION OF RESPIRATORY SYSTEM**

Trachea	:
Respiratory Movements	:
Auscultation	:
Breath Sounds	:
NVBS	:
Bronchial breathing	:



- I. Tubular breathing :
- II. Cavernous breathing :
- III. Amphoric :
- Added sounds :
- Peak Expiratory Flow Meter Rate :

**OTHER SYSTEMIC EXAMINATION**

- Cardio Vascular System :
- Gastro Intestinal System :
- Central Nervous System :
- Musculo Skeletal System :

**Treatment :**

**Diet :**

**Advice :**

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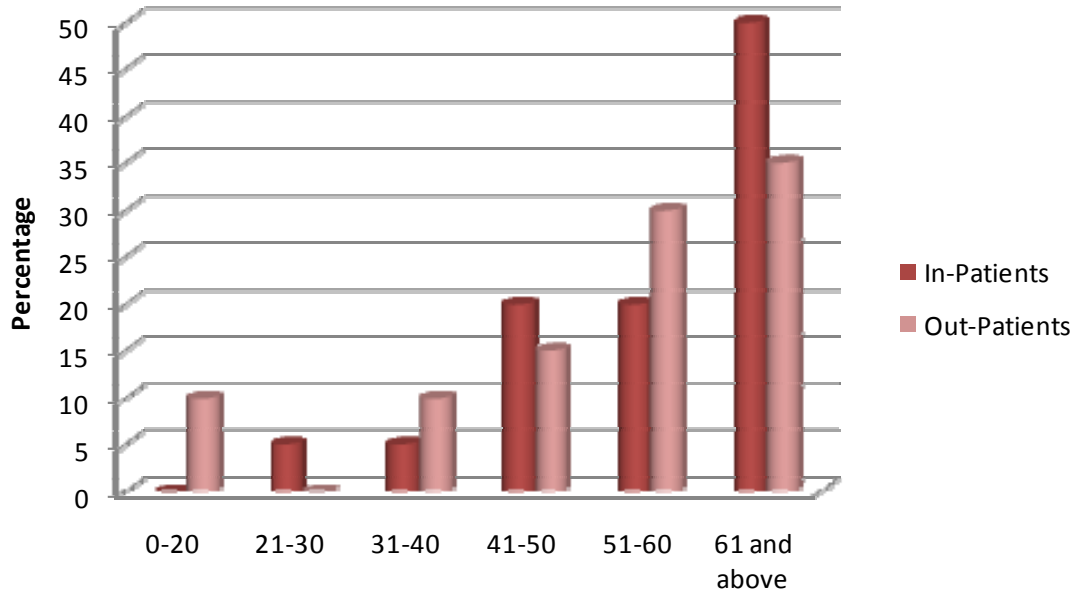
TABLE 28C

S. No	IP. No	BT				AT				BT ESR		AT ESR		Urine BT			Urine AT			Motion BT		Motion AT	
		TC/ cu-mm	DC			TC/ cu-mm	DC			1/2hr	1hr	1/2hr	1hr	Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst
			P%	L%	E%		P%	L%	E%														
1	1598	8400	70	38	8	8200	60	34	6	10	20	8	17	Nil	Nil	1-2 dep	Nil	Nil	1-2 dep	Nil	Nil	Nil	Nil
2	1817	8900	68	30	9	8800	64	30	6	9	18	7	15	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
3	1930	9700	68	38	8	9600	62	34	4	15	30	9	17	Nil	Nil	1-2 dep	Nil	Nil	1-2 dep	Nil	Nil	Nil	Nil
4	1972	9100	70	40	8	8900	60	35	5	6	12	4	9	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
5	2408	9800	74	28	5	9700	73	24	3	15	28	7	13	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
6	2489	8700	68	32	6	8900	70	26	4	7	13	5	12	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
7	2545	s	62	58	10	9200	58	34	8	10	20	8	15	Nil	Nil	OCC EC	Nil	Nil	OCC EC	Nil	Nil	Nil	Nil
8	2546	9000	50	52	6	9000	48	48	4	30	50	10	25	Nil	Nil	Few EC	Nil	Nil	Few EC	Nil	Nil	Nil	Nil
9	2609	8400	68	37	6	8200	55	38	4	30	60	25	50	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
10	2652	9100	72	38	7	9300	68	34	5	20	38	15	30	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
11	2670	8700	72	35	7	8500	70	25	5	8	14	6	12	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
12	2687	8500	69	40	5	8300	67	30	3	5	10	2	4	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
13	2695	8900	68	42	8	8700	54	40	6	24	38	20	35	Nil	Nil	OCC PC	Nil	Nil	OCC PC	Nil	Nil	Nil	Nil
14	2719	9100	72	46	7	9200	68	42		15	28	10	20	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
15	2879	8800	68	42	6	7900	60	37	3	20	38	18	32	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
16	2910	9100	72	26	5	9200	71	27	2	7	14	5	10	Nil	Nil	Few PC	Nil	Nil	Few PC	Nil	Nil	Nil	Nil
17	2915	8400	58	32	7	8600	48	48	4	60	100	40	80	Nil	Nil	Few EC	Nil	Nil	Few EC	Nil	Nil	Nil	Nil
18	2949	9100	72	40	5	8400	69	30	1	9	18	7	15	Nil	Nil	Few EC	Nil	Nil	Few EC	Nil	Nil	Nil	Nil
19	2958	8700	70	32	4	9200	68	30	2	10	20	7	15	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil
20	2975	8300	64	28	6	7200	65	30	3	30	60	25	48	Nil	Nil	NAD	Nil	Nil	NAD	Nil	Nil	Nil	Nil

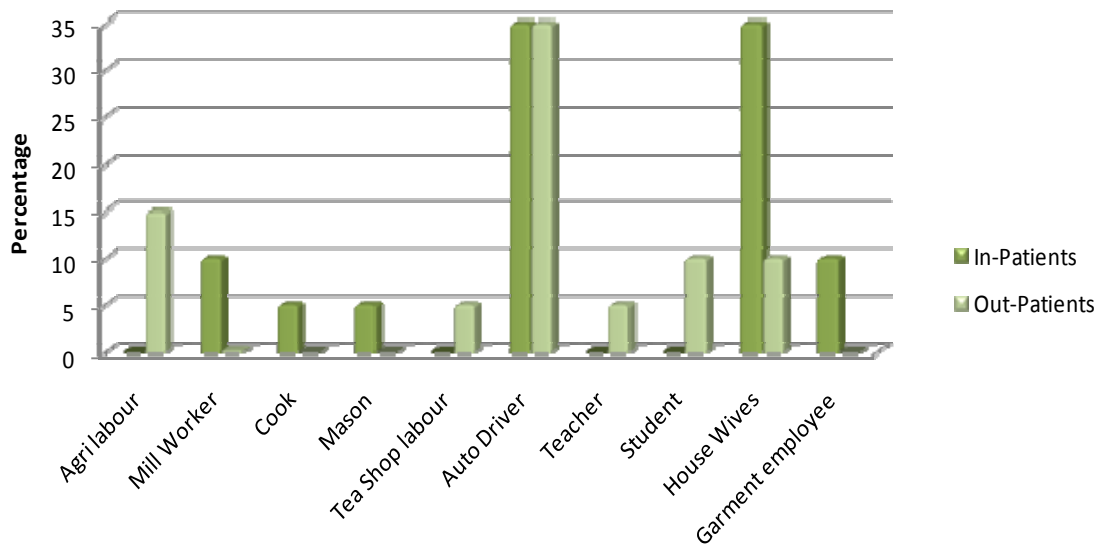
PC - Pus cells

EC - Epithelial Cells

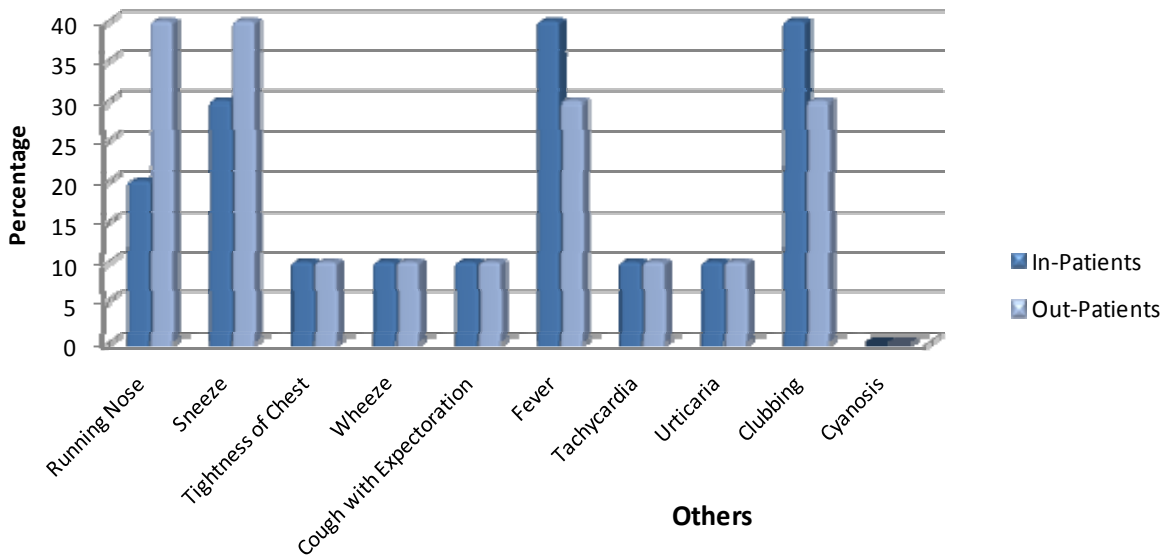
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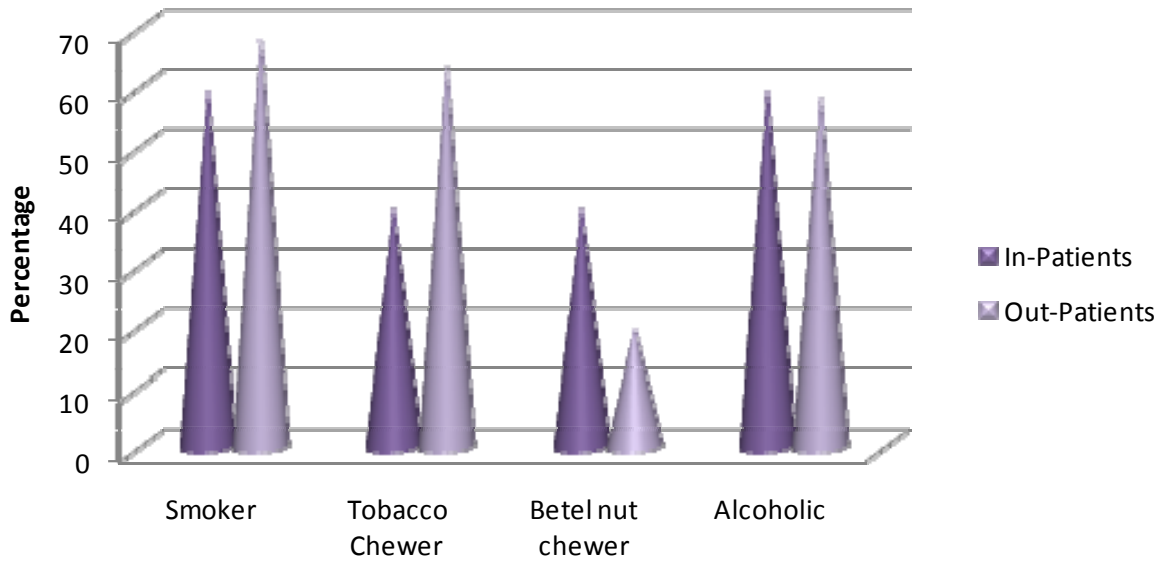
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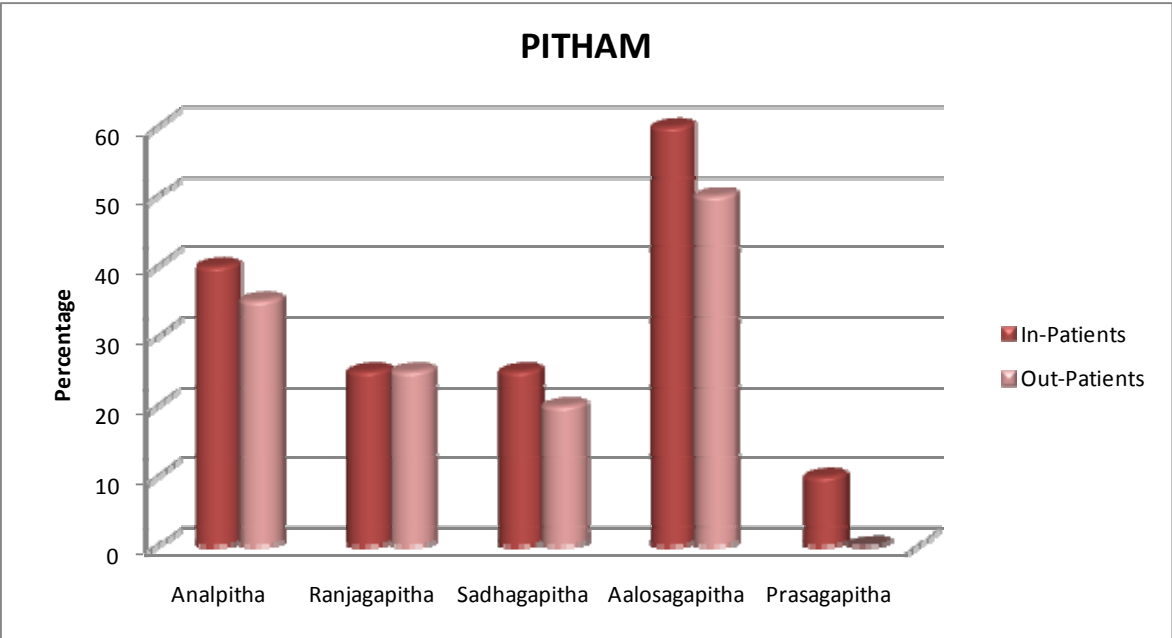
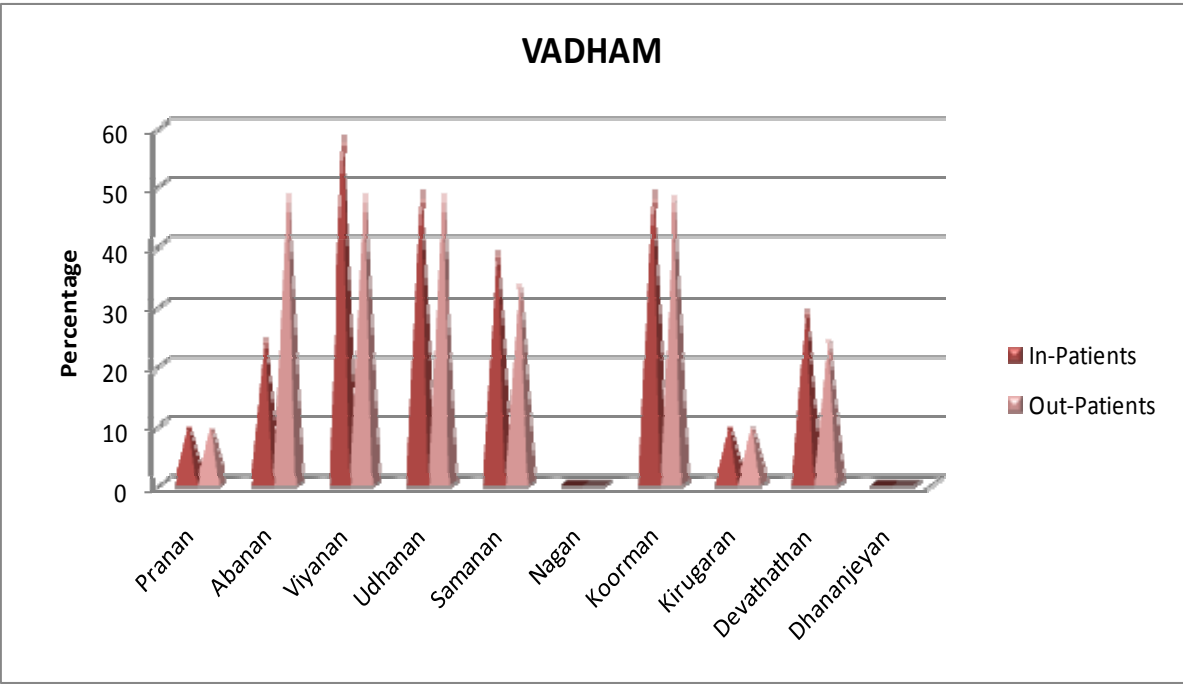


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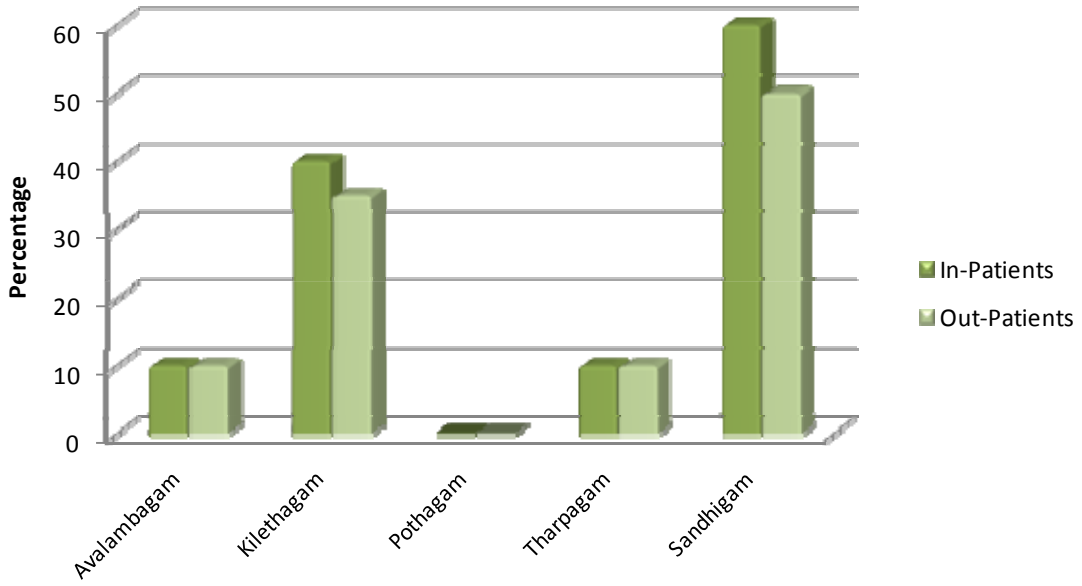


### HABITS

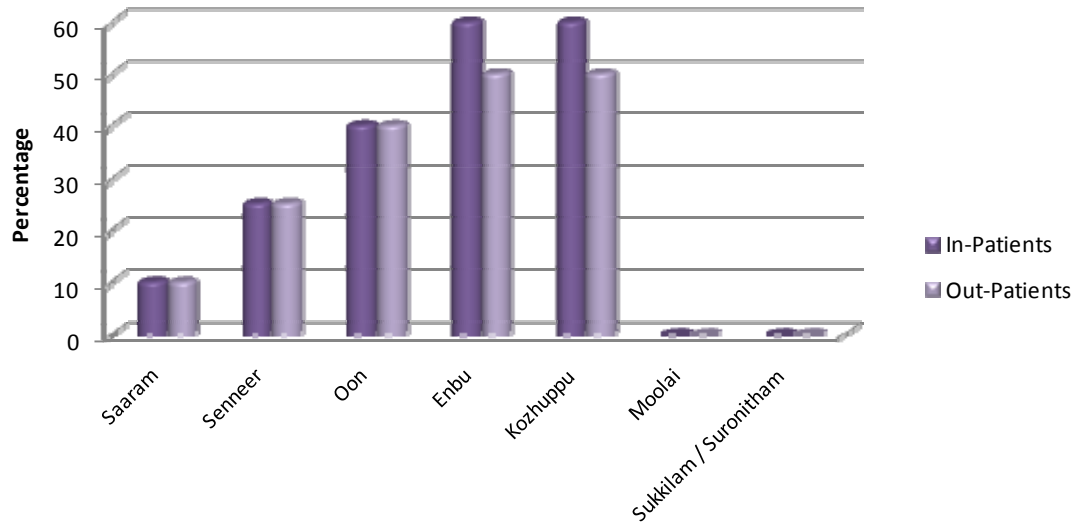




### KABAM

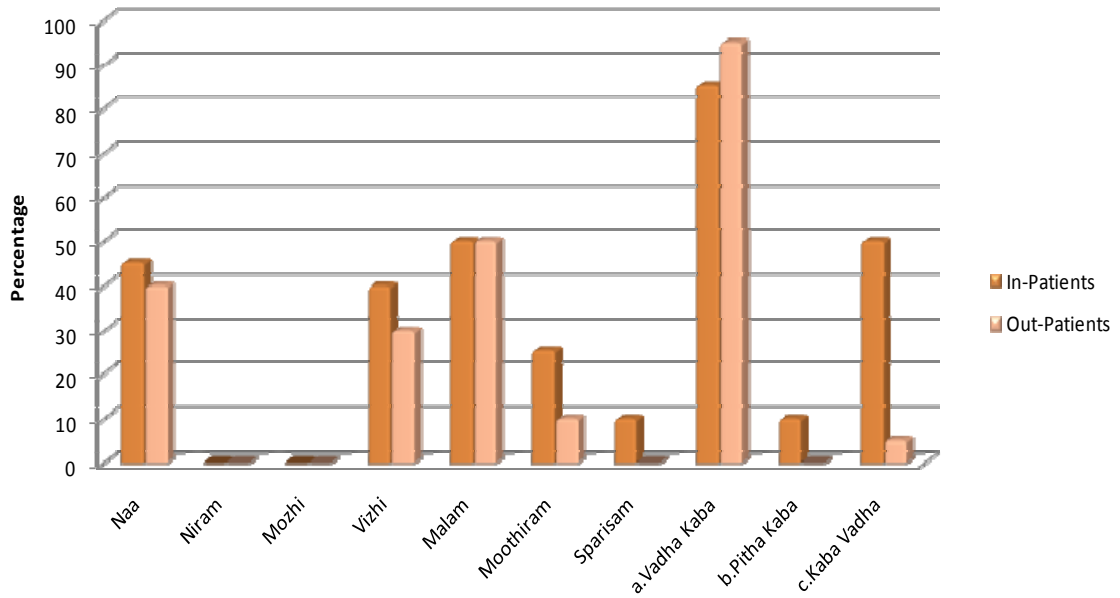


### EZHU UDAL KATTUGAL





### EN VAGAI THERVUGAL



### Gradation of results

