A STUDY ON
VADHA UBHAKATHAM

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INTRODUCTION

Medicine as every one knows is not merely as science but an art as well. There are different systems of medicine in the world according to their way of life with their geographical conditions.

The siddha system of medicine is one of the two pillars of Indian system of medicine, the other one is ayurvedha, siddha system of medicine is dedicated by siddhars to the human society to live long & free from disease.

Siddhars were men of highly cultured, intellectual and spiritual faculties combined with supernatural powers.

The siddha system considers not merely body alone but also the soul.

The siddha system considers body as a whole, made up of five basic elements namely.

1. Prithivi
2. Appu
3. Theyu
4. Vayu
5. Agayam

These five elements are considered as the fundamental principles of all creations of god. This can be stated in the sathaga naadi as ""

 Traditional system of medicine all over the world believed above."
According to this the natural forces acting in and through the various organs of the human body are intimately related to the similar or corresponding forces acting in and through the organism of the universe.

According to siddha literatures the total number of diseases which affects the mankind is 4448. One among them is vadha ubhakatham.

The evidence of the disease Vadha ubhakatham is selected from yugi vaidhya chinthamani – 800.

The author has chosen the disease ‘Vadha Ubhakatham’ for this dissertation work. It is now common in occurrence due to prevalence of madhumega noi as their complications. The disease is correlated with the ‘peripheral neuritis’ in allopathic view.

Principles of Treatment

When patient, doctor, pharmacist and nurse all act in coordination the disease will be cured.

In this unique system treatment is based upon the principles of arusuvai, mukkutram and panchaboothas. Also the seasonal variation (Paruvakalam), personal habits (Nall ozhukkam) etc., are also considered.
Siddha medicines are classified mainly into two major divisions.

- Internal medicine – 32
- External medicine – 32

Pathiyam is a medical advice which includes dietary restrictions and lifestyle modifications according to the diseased conditions.

The unshakable belief among the people regarding the vadha disease that it can be effectively managed only by siddha medicine than other systems of medicine also initiates the author to choose it for dissertation.

The author’s choice of medicine for the clinical study were

- **Pathiriver chooranam 5 gm , B.d with hot water**
  
  **Gunapadam Mooligai Vahuppu**

- **Medicated vennai for external use.**
  
  **Sarabendhirar Pitha roha sihitchai**

So the author hopes that this dissertation work will arise new horizons in the field of siddha medicines in managing vadha disease.
AIM AND OBJECTIVES

The chief aim of this study about vadha ubhakatham is to establish the disease scientifically, because

In siddha medicine vadham is considered as the most effective cause of disease. It is common in India and other countries.

As the patients are disturbed both functionally and emotionally, diagnosing the disease with proper siddha and modern parameters is necessary to evaluate the disease because if it is not diagnosed and treated it may leads to so many complications.

The disease vadha ubhakatham produces Gnawing sensation over legs and hands, numbness, paresthesia, sensation of walking on cotton wool, pain in the extremities, polyphagia and giddiness.

The purpose of the author is to elucidate a good medicine from ancient siddha literatures and to create hope and faith in their treatments.

The author extent a correlation of aetiology, signs and symptoms of vadha ubhakatham in siddha aspect with peripheral neuritis in modern science.

There are various causes for vadha ubhakatham. The patients with vadha ubhakatham were selected for dissertation work that had (madhumegam) diabetes mellitus anaemia ($B_{12}$ defeciency) nutrional disorder and alcoholic abuse.

To collect various siddha literatures aetilogy pathology, clinical features, diagnosis, prognosis, complications and the treatment by making use of siddha concept.

To express the unique diagnostic method mentioned by siddhars to know how the disease vadha ubhakatham deranges the
normal muththadhukkal, poripulangal, udal kattugal, and envagai thervugal and naadi nadai.

To have an idea about the incidence of disease with age, sex, habit, socio economic status, family history and climate conditions.

To have an idea about paruva kalam, environmental changes, Thinai, Thegi for this disease.

To have a detailed clinical investigations.

To have a clinical trial on vadha ubhakatham with Pathiri ver chooranam internally and Medicated vennai externally.

To apply the principle of management as advised by siddhars, recording the cause, prevention and treatment of disease.

To evaluate the biochemical and pharmacological effects of the trial medicines.

To use modern parameters to confirm the diagnosis and to access the prognosis of the disease.

To give yoga therapy (Mayurasanam) along with medicines for relaxation of brain centers to achieve good results.

To apply the principle of pathiyam specific to this disease.
ABSTRACT

Keeping in mind the need for bringing out an effective siddha therapy for “Vadha Udbhakatham” since the commonest disease in the society, number of sufferers increasing day by day so I have selected this disease for my dissertation work.

I studied the clinical course of the disease on basis of siddha literature with the help of diagnostic methods mentioned by siddhars and how the disease alter the normal conditions of uyir thathukkal, poripulangal, seven udal kattugal and envagai thervugal.

Signs and symptoms of “vadha ubhakatham” with peripheral neuropathy in modern science.

20 out patients and 20 in patients of either sex were selected and administered with the trail medicine.

I studied the incidence of the disease with age, sex, socio-economic, status, family history and seasonal variation.

I utilized the modern parameters for confirmation and prognosis of the disease.

I had clinical trail on vadha ubhakatham with

- Pathiri ver chooranam (Internally)
- Medicated vennai (Externally)

The trial medicine was subjected to bio-chemical and pharmacological analysis.

At the end of the trial study the majority of the cases showed good results.
REVIEW OF LITERATURES

SIDDHA ASPECTS

According to siddha system of medicine a human being is composed of 96 basic principles. Among them the first thirty is considered very vital and the rest are considered to be the extension of the first thirty principles. This not only consists of the physical principles components of the human body but also the mental, intellectual components like passions, qualities, knowledge, functions of the sense organs and motor organs and their coordination.

Pancha pootha Theory

The five elements vinn, kall, anal, punal, and mann are the basis for the world and the human being. These five elements are in subtle states (Suthchuma nilai). They manifest, into gross state (sthula nilai) and become visible. The manifestation of the five elements from the subtle state to gross state are called as panchapootha panchi karanam.

Three humours theory

The three “humours” as described in siddha medicine is a golden line continuous in physiology, pathology and treatment. The three humours are vadham, pitham and kabham, whose balance is essential for the maintenance of good health. These are also made up of five elements.

Vadham is formed by the combination of vayu and akash (kall and vinn).

Pitham is formed by the theyu (anal)
Kabham is formed by the combination of prithvi and appu (mann & punal)

These three humours exist in the ratio of 1: ½ : ¼ when these three humours are in equilibrium they are called as uyir thathu while they get deranged they are called kutras or doshas.

When an individual has predominant vadha he is said to have a vadha temperament. Like wise other two, this temperament of body is determined during fertilization.

Aetiopathology of disease

Siddha humoural pathology explains that all diseases are caused due to the derangement of the three humours. When they get deranged they bring about diseases peculiar to their influence. The humours by themselves are not the producers of diseases while functioning normally. But they give rise to diseases if they are vitiated by other factors. This is quoted in Thirukkural as

"பின்புறை கருநிதி வேளும் உடையிடம் நாரையார்கள்
கொண்டு வந்திருந்தான் பெரும்"
- திருக்குரால்

Any change in the proportion of the three humours is sure to bring about disease but the maintenance of their normal proportion gives vitality to the organism and assures the preservation of health and longevity of life.
EXPLANATION ABOUT TRIDHOSHAS

FORMATION OF TRIDHOSHAS

The formation of vadham, pitham and kabham has been explained in kannusamiyam as follows.

¬ Abanan in conjunction with edagalai produces vadham.
¬ Piranan in conjunction with pingalai produced pitham.
¬ Samanan in conjunction with suzhumunai produces kabham
¬ Thus vadham, pitham and kabham are formed.
VADHAM

**Synonyms:**

The term vadham denotes vayu, dryness, pain, flatulence & lightness.

**Definition:**

The three basic factors vadham, pitham and kabham working in physiological conditions are called muththadhukkal (or) uyir thattukkal. Among the three, the biological air humour is called vadham. In terms of etymology it means that which moves thing it is the motivating force behind the other two humours which are considered to be lame incapable of movement without it.

Vadha may be increased (or) decreased when the equilibrium state is disturbed; if vadha is altered the other two are also altered leading to vadha disease.

According to this the human body is composed of 72,000 naadi, narambugal among this 72,000 the ten are prominently naadies (dhasanaadies) of this ten naadies edagalai, pingalai, shuzhumunai are known as moolathara naadies.

Among the ten vayus five are important, they are piranan, abanan, viyanan udhanan, samanan.

These three humours are thadhu (i.e) vadha, pitha & kabha are the functional principles in the composition and substance of the body.
Location of vadham

Vadham lives in

"தொகைகளில் மாணவர் விளக்கத்து பாடல்

சீனக்கிளச்சில் இருந்துச் சீனான் கிளான்

தர்க்ககிளச் முடித்து பாடல்

சேலமாவின் பெண்களின் வழக்கத்தின் பாடல்

வாழ்க்கை செய்யும்படி முகாம் காய்

சீனக்கிளச் குறிப்பாக எழுதிய நூற்று

சீனக்கிளச் பாடலில் புராணக் கால

சீனக்கிளச் பாடலில் புராணக் கால

சீனக்கிளச் பாடலில் புராணக் கால

சீனக்கிளச் பாடலில் புராணக் கால"

- தமிழில் கூறு 55,56

☞ Abanan
☞ Stools
☞ Edakalai
☞ Undhiyin keel moolam
☞ Kaamakodi
☞ Hip bone and Joints
☞ Skin
☞ Nerves
☞ Hair follicles
☞ Muscles

"அகுல் செழுமையை காண்பதற்கான

சீனக்கிளச் செழுமையை - நீண்டநூறு

வாழ்க்கையாருக்கு செழுமை கைத்தையை அறப்பது

சீனக்கிளச் அறப்பது"

- தமிழில் கூறு

"அகுல் செழுமையை காண்பதற்கான

சீனக்கிளச் செழுமையை அறப்பது

வாழ்க்கையாருக்கு செழுமை

சீனக்கிளச் அறப்பது"

-தமிழில் கூறு
According to the maruthuva thanippadal vadha lives in the digestive system, bones, ear, thigh, skin and hip, below navel region.

**Nature of vadham:**

- Giving briskness
- Respiration
- Functioning the mind thought & body
- Regulation of fourteen physiological reflexes.
- Uniform functioning of the seven udal thathukkal.
- Strengthening the five sensory organs.

**Qualities of vadham**

- Hardness
- Dryness
- Lightness
- Coolness
- Mobility
- Subtleness

**Opposite qualities of vadham**

- Softness
- Greasy
- Heaviness
» Hotness
» Stable
» Solid State

**Functions of vadham**

» Body ache
» Pricking pain
» Tearing pain
» Nerve weakness
» Shivering
» Mental distress
» Dryness
» Movements
» Weakness
» Joint pain
» Traumatic pain
» Dislocation of joints
» Weakness of organs
» Pilo – erection
» Paralysis of limbs
» Polydipsia
» Severe pain in leg and thigh muscles bony pricking pain
» Constipation
» Unable to do flexion and extension of the limbs
» All tastes to be like astringent
» Excess salivation
» Darkness of skin, eyes and urine.

**Description of vadham**

The siddha classical texts divide the general principles of vadham into ten subsidiary forms that differ from one another by
their location in the body (anatomical) and by their particular functions (Physiological).

They are

1. **Piraninan (Heart Centre) - Uyirkkal**

   It corresponds to the cardiac plexus and refers to the chest. It maintains the action of the heart the functioning of the mental faculties of perception and concentrations and also cares for the arteries, veins & nerves. It regulates the respiration & digestion. It otherwise called as “Uyirkkal”

2. **Abaanan (Moolaadharam centre) – Kezhnokkukal**

   It corresponds to the pelvic plexus and controls the excretion. It is focused in the lower part of the gut and also occupies the sites in the bladder and gential organ. It has a tendency to travel downwards. It moves in the whole gentio urinarytract and regulates the defecation, micturation, mensuration parturition and ejaculation.

3. **Viyanan (Fore head centre) - Paravukal**

   It corresponds to the nasociliary plexus at the root of the nose and base of the skull and controls the will. It helps in the circulation of energy through out the entire nervous system and the movements of various parts of the body. It also transports nutrients and blood through out the entire body. It is also know as “paruvakal”.

4. **Udhanan (Throat centre) - Melnokkukal**

   This corresponds to the pharyngeal plexus in the throat region and controls speech, and breathing. It is also responsible for
the physiological reflex actions like vomiting, hiccup, cough etc., It is otherwise named as melnokukaal.

5. Samaanan (Nerves Centre) - Nadukkal

It corresponds to the solar plexus in the navel region and controls digestion. It selects useful substances from the swallowed food and supplies them to the whole body. It balances the other vayues it is also called nadukkal.

6. Naagan

It is responsible for the intelligence of an individual winking, singing and pilo erection.

7. Koorman

It is responsible for yawning, closing of mouth, winking, shedding of tears, vision and opening of the eyes.

8. Kirugaran

It is responsible for salivation & nasal secretion. It helps in digestion and medication. It produces cough & sneeze.

9. Dhevathathan

It is responsible for laziness, lassitude, to quarreling arguing, begging and also for much anger. It helps movements of the eye ball in various directions and is present in genitallia & anal region.

10. Thananjeyan

It is present in nose and responsible for swelling of the body and tinnitus. It leaves from the body by blowing up cranium only on the third day after death.
PITHAM

The term pitham denotes gastric juice, bile, energy, heat, anger etc.,

Location of pitham

Head, heart, bladder, abdomen, umblicus, stomach, saliva, sweat, blood, eyes and skin are the sites of the pitham.

Effects of vitiated pitham

Excessive heat in the body, improper digestion, excessive sweat, giddiness, syncope and immoral behaviour are some of the ill effects of vitiated pitham.

Types of pitham

According to the functions and location, pitham is classified into five types. They are,

Anar Pitham - It is located in the stomach and intestines and is responsible for proper digestion.
Ranjaga Pitham - It is located in the intestine and is responsible for the colour of blood.
Sadhaga Pitham - It is located in the heart and controls the functions of the body.
Alosaga Pitham - It is located in the eyes and is responsible for proper vision
Prasaga Pitham - It is located in the skin and is responsible for the complexion of skin
KABHAM

Location of kabham

It is located in the tongue, chest, blood, bone marrow, bones nerves, brain, large intestine, eyes and joints.

Functions of kabham

The important functions of kabham are maintaining the viscosity and proper functioning of the joints.

Effects of vitiated kabham

Pain in the long bones, dysfunction of joints improper digestion, excessive sleep and inhibition of under stating capacity.

Types of kabham

Avalambagam :

It is located in the heart and it controls the other four kabham.

Kilethagam :

It is located in the stomach and is responsible for proper digestion.

Pothagam :

It is located in the tongue and helps to feel the sensation of taste.

Tharpagam :

It is present in the head and keeps the eyes cool.

Santhegam :

It is present in the joints and is responsible for proper functioning of the joints.
Fate of three humours

"அறிக்கித்து வாதம் நுழைவுகளும் பிரிந்து விளக்கு வாதம் பரிப வாதத்து வாதத்தில் வாதம் வாதம் வாதந்த விளக்கில்”

From this it is clear that the three humours can be discharged through the following routes.

Vadham - faeces
Pitham - Urine
Kabham - Semen / Suronidham

The features of exaggeration of vadham

- Body weakness and darkness
- Shivering
- Constipation
- Dimmunciation of immunity
- Giddiness
- Insomnia
- Laziness

The Features of diminution of vadham

- Body ache
- Hoarseness of voice
- Loss of memory
- Semi Consciousness
- Difficulty to do any work
- Paleness and coolness of body
- Anorexia
BEHAVIORAL CHARACTERISTICS OF VADHA DHEGI

Perform activity - very rapidly
Motivated enthusiastic
and excitable - very easily
Moods - Changes quickly
Learns - very quickly & easily
Quality of mind - quick, creative & imaginative
but restless
Memory - good, short term
Digestion - in consistent varies between
  weak & strong
Appetite - variable can ship meals occasionally
Quantity of food - variable
Taste preference - sweet, sour and salty
Thirst - varies
Food preference - warm, moist food
Drink preference - hot
Frequency of bowel movement - irregularly
Consistency of feces - hard, dry stools
Perspiration - moderate
Sexual desire - less
Amount of sleep - usually 5-6 hours
Quality of sleep - light, easily, interrupted
Types of dreams - fear, flying, running, jumping,
  climbing trees and mountains.
Response to challenge - uncertain, worried and indecisive
Speech - fast, omitting words and digressing
Gait - fast with a light step
**Physical characteristics of Vadha Dhehi**

- Shape of face: thin body and elongated plain looking
- Complexion: dark, brownish (or) black
- Involuntary bodily movement: twitching, jerking and fine tremours
- Body weight: Light and below normal
- Built: lean, thin, tall (or) short
- Texture or quality of skin: dry, worse, rough, cracked (or) scaling and birth marks.
- Skin moisture: dry
- Body temperature: low, cold extremities
- Stamina: short
- Shape & quality of eyes and lashes: small bulging and deep set with thin scanty eye lashes
- Characteristic of eyes: dry, frequent blinking
- Teeth: very small (or) protruding crocked, easily cracked.
- Nails: short, rough, brittle dark & lusterness
- Lips: dark, dry and cracked.
- Size and shape of fingers: very, short (or) very long stubby and thick.
- Colour & texture: thin course dry and wiry, darker in colour or balding
- Body hairs: scanty
- Joints: loose (or) rigid, pronounced crack.
- Veins: prominent (or) branching close to surface
- Chest: long, sunken thin ribs easily visible
AETIOLOGY

The common aetiological factors for all types of vadha disease are applicable for vadha ubhakatham also.

Though yugi has not mentioned any specific cause for vadha ubhakatham he has summarized the causative factors for all types of vadha diseases as.

"commended maasserta viddadham karan
 proclaim sexu variyakum kuthum
 inebes revi paraanadham thiladum
 panduvariantha pugazhatharad tamathikum
 bavanan dhugum vachechi viradum
 sattu pura thodhum magam pirakum
 kandaikum visadham kuthukal vekkaram
 karthikum tattakkilum maattakkilum"
- pukki varakkum புராண வரக் 243

According to this poem, dacoity, abusing bolymen, exploiting properties of charities, forget the parents and teachers and desecrating holy scripts will cause disturbance of vadham.

"kandaiyam cudinam tirumam kathum
 kathuyam cilakumaluthu sambadu manubodam
 akandaiyam padiyam cilakumkum ambigum
 ambavana cikalum kudhum ambigum
 parangal kuduthukum melegillum
 parangal kuduthukum melegillum
 parangal kuduthukum melegillum
 parangal kuduthukum melegillum
 parangal kuduthukum melegillum
 parangal kuduthukum melegillum"
- pukki varakkum புராண வரக் 244
Excessive intake of bitter, astringent and pungent foods, rotten foods, starvation, drinking rain water, sleeping during day time waking at night etc., may cause disturbance of vadha.

"அரைத்த மாடம் சாதுக்கை குபித்
பெருமாள் தண்டுதலியலும் பெருமாள் தேர்மை
தரும் பேலைக்கு முன் தம்பிலின்
கல்கல்கா மரற்றை பெரித்து பலர்
மறைமுன் அறுவாள் சியம்பு சாண்டர்
மறையுள்ளது மராத்திய இயநூற்றாண்டை
முதலில் தூரத்தை மூன்றாண்டு குரலாட்சியில்"
- புத்தி சாம்சிய சிக்ககோணி 285

Indulging in excessive sex, walking for a long distance exposure to extremes of weather and in appropriate food habits will lead to vadha disorders.

"அரசன் வொருக்கல்களைப் பெருமாள் உருவா
அரசன் தூரத்தியும் வெறும் வெறும்
சாண்டருடன் பொறுப்பையும் உருவை உருவை
அரசன் சாண்டரின் உருவை இருந்து
அரசன் சாண்டரின் உருவை வெறும்
- புத்தி சாம்சிய சிக்ககோணி 253

Refusing food for destitutes and sanayasis, forgetting the advice of teacher’s murder, decoity, lying and excessive sexual activity may also have to vitiation of vadha.
Accordingly barrenning the land of young green, trees, laming living beings, chopping the branches of a living tree, removing the barks and leaves of trees may lead to vadha diseases.

According to this verse fear, anger, sorrow, excessive work & climate changes lead to vitiation of vadham.

Sabapathi kaiyedhu attributes the causes of vadha diseases to in appropriate food habits exposure to cold and indulgence in excessive sex.
Brain disease
Renal disease
Sexually transmitted disease
Disease of the vertibral column & spinal cord
Compression of sensory nerves
Menorrhogia

Taking improperly prepared medicine of mercury and lead will cause vadha disease.
Other Causes:

1. Consumption of bitter, astringent
2. Eating previously cooked food
3. Drinking polluted water
4. Changing sleep rhythm
5. Excessive starvation
6. Excessive rest
7. Walking long distance
8. Living in chill environment
9. Excessive consumption of tubers, fruits, curd etc.,

Alteration of udal vanmai

Udalvanmai is described as iyarkai vanmai, cheyarkai vanmai and kaala vanmai

- Iyarkai vanmai is considered with three gunangal (Sathuva, rajo, thamo gunam)
- Kaala vanmai is considered with age, season, most of the vadha disese occurs in old age because the kaala vanmai is diminished in old age.
- Changes in cheyarkai vanmai also play a major role. Wrong postures, style and improper foods, life style modification causes the peripheral neuritis.

"மாசி பதிற்ற நெவாையும் ....
- ஒப்பேச்சிக்கு குரத்து தமு ஜூது

"பாதிக்கக்கினு குறைப்புதை பகிர்த்து வாகமு....
- கார்பேச்சிக்கும்
பதிற்ற, நெபா அருக்குரு உணர்வுகோ மரகமு மிக்கும்
SIGNS AND SYMPTOMS OF VADHA DISEASES

The signs and symptoms of vadha diseases have been given in many siddha classical text books as follows.

Pricking pain, dull aching pain termours, palpitation, spasm, dryness, dehydration, dislocation of joints, weakness of the body, paralysis, constipation, oliguria, excessive thirst horpliation, difficulty in flexion and extension of limbs, astringent taste in the mouth and excretions like stools, urine, tears and sweat are black in colour.

In Agasthiyar 2000

"ஒருங்குறிங்கள் வேளவியசார்களின்
அனைத்துக்கும் குறிப்பிட்டதும்
பாதுகாப்பு பொருளிலே குறிப்பிட்டது
நூற்றாண்டு முறையில் புரோஸ்வாதி செய்யப்படுதல் தொடங்கி
பாதுகாப்புச்சார்கள் வாழ்க்கையும் பாதுகாப்பும் பெறுதலாம்"

- Giddiness
- Stabbing pain in the face
- Redness of eyes
- Peptic ulcer
- Abdominal distension
- Joints pain in upper & lower limbs.
- Numbness in the limb
- Oliguria
- Drowsiness
- Chillness of body
Agasthiyar Naadi

"அகாயத்தக்க மரம் விளைந்தார்
செஞ்சங்களின் மாறு விளைந்து வீழ்வது
பல சக்தியை அகற்ற புக்கைக்
போகும் பலத்தை நிறைய அகாயத்தக்க மரத்தை வீழ்வது "
- அகாயத்தக்க மரம்

- Weakness of the limbs
- Sluggishness
- Stiffness
- Numbness

In Theraiyar Vaagadam

"மகரத்திற்கு உண்மையான தக்கத்தை மட்டையையே வீழ்வது
நேரடி கலர் கம்பியை வீழ்வையே வெளியிருக்கும்
துர்களில் மரம் கூடமை விளைத்துக் கூட்டியது
இன்றைய வனவிலங்கு ஓருவன் தேசியும் கிட்டத்தடுகி "
- செலும்பழம் மரம்

- Loss of appetite
- Back ache
- Fever
- Cough
- Sleeplessness
- Shivering
- Pain in the joints

"சத்தம் மரம் வனவிலங்கில் ஓருவன் தேசியக் கிட்டத்தடு
பெரும் வனவிலங்கு ஓருவன் தேசியக் கிட்டத்தடு
சத்தம் மரம் வனவிலங்கு ஓருவன் தேசியக் கிட்டத்தடு
பெரும் வனவிலங்கு ஓருவன் தேசியக் கிட்டத்தடு"
- செலும்பழம் மரம்

- Pain in the joints
Head ache
Excessive yawning
Constipation
Burning sensation of the body
Paralysis
Excessive salivation
Chillness
Tremour

In Padhinen siddharkal naadi sasthiram

"The vitiation of vadha results in dyspepsia, pain in the joints & dysuria constipation, abdominal colic, fever, rigor, body ache and excessive sweating."
CLASSIFICATION OF VADHA DISEASES

In classification of vadha diseases we can find contradictory view regarding the number, in various books.

In yoogivaidhya chinthamani – 800

"நான்கு வகையான பட்டைத்துறை என்பது"

Eighty types of vadha diseases are described.

In “Agasthiyar Rathina Surukam” – 500 Eighty four types of vadha diseases are reported

"அனந்தன் பார்த்துறையும் வட்டம்
சாத்தாரா நூறாக என்று"

- அல்லது வழிந்து குறிப்பிட்டு

But while concluding the number has been given as eighty four by the yoogi vaidhya chinthamani – 800.

"ஆனந்தன் பார்த்துறையும் வட்டம்
ஆனந்தன் வழிந்து குறிப்பிட்டு"

- புது வகையான சிரைப்படை

In Astaanga sangiragam and noi naadal noi mudhal naadal thrattu part I. Vadha diseses have been classified as 85 types on the symptamatology and involvement and different parts of the body.

In Theraiyar Vaagadam 81 types of vadha diseases been described
“Dhanvandhiri vaidhiyam” and “Jeeva Rakshamiraham” 88 types of vadha diseases have been noted.

“Agasthiyar 2000” forty types of vadha diseases are in the upper half of the body and in forty lower half of the body and the total number in 80.

In Bohar Vaidhiyam 700, 80 vadha diseases are told.

In Agasthiyar Gurumuni – 235, 85 vadha diseases have been reported.
CLINICAL FEATURES OF VADHA UBHAKATHAM

Uba means Subnormal

Katham means Pain full condition.

Yugi in yugi vaidhya chinthamnai, has described the clinical features as follows.

Vadha ubhakatham is one of the vadha disease.

An increased feeling of numbness is vigorously present in the legs & in the hands joints and all over the body.

A sensation of smearing the dung of animals as armour.
Hastening of numbness with gnawing sensation.

A sense of heat in the leg during movements due to burning sensation.

As the worsening of numbness progress they have difficulty in walking with delusion & drowsiness.

All the above symptoms are called vadha ubhakatham.

Summary

An increased feeling of numbness and a sensation smearing the dung of animals is vigorously present in the legs and in the hands joints and all over the body. The numbness a condition of insensibility increases in its density with restlessness of the body. As a result a sense of heat in the feet become prominent. As the worsening progresses they are difficult in walking, with mental delusion and drowsiness.
MUKKUTRA VERUPAADUGAL

Pathogenesis

1. By any one (or) other etiological factors vadham is vitiated first.

2. Then it affects the other dhoshas pitham and kabham which are in three equilibrium.

3. And then the ten vayus, seven udarkattugal and other structures are also affected according to the severity of the illness.

4. By the affection of piranan wheezing cough, dyspnoea, nasal congestion and indigestion may occur.

5. By the vitiation of abaanan constipation, oliguria and menstrual disorders may occur.

6. By the affection of udhanan heart, chest and eyes are affected hiccup vomiting & heart burns are formed.

7. By the vitiation of viyanan muscle wasting loss of sensation, giddiness coma, body ache, numbness, itcinc & tingling sensation are formed.

8. By the affection of samanan disturbances of other vayus abdominal distension, anorexia, malnutrition and indigestion may occur.

9. When saaram is affected anorexia, laziness lassitude weakness and dryness of the skin are formed.

10. When senneer is affected nerve weakness, dryness, mental disorder, haematuria, jaundice anaemia, anorexia, spleenomegaly & skin diseases may occurs.
11. When oon is affected muscle wasting dropsy, body ache, oedema, weakness of the five sensory organs are formed.

12. When kozhuppu is affected body debility body ache, joints pain, spleenomegaly, tiredness may occur.

13. When enbu is affected arthritis, joint pain, osteophytic formation and other bone diseases are formed.

14. When moolai is affected blurring of vision, ulcers, heaviness burning micturation, the body & bone diseases may occurs.

15. When sukkilam is affected lustfulness urinary calculus, bleeding during coitus orchitis and diseases of genitalia are formed.

16. When pitham is affected anorexia, anaemia, indigestion, blurring vision, dryness and darkness of skin. Vomiting, giddiness, burning sensation of the body and difficulty to do works are formed.

17. When kabham is affected respiratory disorders, indigestion, tastelessness, burning sensation of eyes and joint disease may occurs.

In vadha disease abanan, viyanan, samanan, naagan, koorman, dhevathan are affected, generally saaram, senneer, oon kozhuppu enbu and moolai are also affected one by one.

Among the five types of pitha the rhythm of sadhaha pitham gets affected mainly causing difficulty in performing the activities of daily life. (i.e.,) difficulty in walking etc., Among the five types of kabham, tharpagam is affected mainly in Madhumegam patients.
**Relationship between tridhoshas suvai and panchabootham:**

There are six types of suvai. Each suvai is formed by the combination of two boothas.

- Inippu (Sweet) – Earth + water
- Pulippu (Sour) – Earth + fire
- Uppu (Saline) – Fire + water
- Kaippu (Bitter) – Air + space
- Karppu (Pungent) – Air + fire
- Thuvarpu (Astringent) – Earth + Air

They are also classified into 2 types of veeriyam (Potency)

- Veppa veeriyam
- Seetha veeriyam

Veppa veeriyam suvigał – Pullipu, karpu, uppu
Seetha veeriyam suvigał – Inippu, thuvarpu, kaippu

By knowing the veeriyam medicines are administered so that the deranged kutrams are normalized
PINIYARI MURAIMITAI (DIAGNOSIS)

Pini means the disease which affect the body and any interruption of the normal functions of any body part.

Ari means identify

Muraimai means rules

Piniyarai murimate is the method of diagnosing the disease affecting the people is based upon the following aspects

I. Poriyalarithal
II. Pulanarithal
III. Vinathal
IV. Envagai thervugal
V. Naadi Paritchai

I. Poriaal aridhal

The physician should examine the patient’s porigal by his porigal.

Mei - feels all types of sensations
Vaai - for knowing taste
Kan - meant for vision
Mooku - for knowing the smell
Sevi - for hearing

II. Pulannal aridhal

The physician should examine the patient’s pulangal by his pulangal

Hearing - ear
Vision - eye
Taste - tongue
Sensation - skin
Smell - nose

III. Vinadhal (Interrogation)

The physician should interrogate about the patient’s name, age, sex, occupation, native, socio economic status, dietary habits prone to any allergers, complaints history of previous illness, history of habits and frequency of attacks. If the patient is in the stage of inability to speak or a child to physician should interrogate the details with his immediate relatives who are taking care of him.

☞ Naadi (Pulse)
☞ Sparisam (Palpation)
☞ Naa (Tongue)
☞ Niram (Colour of the body)
☞ Mozhi (Speech)
☞ Vizhi (Eye examination)
☞ Malam (Motion examination)
☞ Moothiram (Urine Examination)

Envagai thervugal in the siddha literature gunvagada naadi is as follows.

"குரேசுத்தில் விஷாலியில் குறுக்க பாடாங்கத்தில்
கார் கௌரி வேதகம் வேதங்கள் குணமனிகம்
சீலோசும் விளையுறுக்கைகள் குத்துலந்தில்
குரவரை பாடித் சுருங்கம் சுருக்கம்"
Eight different kinds of tests to be applied or attend by a physician before arriving a correct diagnosis. These are also called attavitha parichai (or) attathana parikshai.

Envagai thervugal is considered as physician’s instruments.

"பது பரிக்கு வந்து செய்வது விளை
மேலு பற்றையும் மெதும்பொற்கும்"

"பெய்த்து பிறந்திருக்கும் நிலையிலும் மேலு மக்கள்"

- கோய்யானா

In agasthiyar vallathi 600, Envagai thervugal has been mentioned as atta vidha paritchai.

"தன்னாலினால் அழிக்கிற பெரும் காலம்
தன்னாலினால் பரிக்கிற விளையாட்டு
பெருமான் மாகாணம் பிற்குரையா
பெருமான் மாகாணத்தையலாம் மாகாணம் பருது
செய்தக்கு விளங்கான வரும்
வருமான தரமானது பிறந்துக்கும் வருமான
செய்தக்கு வந்துபடுத்தப்பட்டு பருத்து வருமான
செய்தக்கு வருமான காலம் பருத்து வருமான
செய்தக்கு வருமான காலமாகத்தை விளையாட்டு மாட்டு"

- அரங்கு மாணவர் 600
According to thirumoolar, feeling the naadi in various sites are mentioned as

Naadi (Pulse)

Naadi is the vital force. Any changes in the three dhoshas are best diagnosed feeling the naadi. Naadi is an important observation for diagnosis and prognosis. Naadi is responsible for the existence of life and can be felt one inch below the wrist on the radial side by means of palpation with the tips of index, middle ring finger corresponding to vadham, pitham and kabham site to feel naadi and its procedure is

"கார்ஷோக தண்டனை வாய்ந்ததிக்
செஞ்சிக் நல் பார்வைக்
போர்ந்தனாக்கிக்
பிருஷோ் குரோண் கூர்ப்பல்
குழுற்று விளைந்த பார்வை
பார்வை நிருத்திக் பிள்ளை
சித்தமன் வாண்டு விளைந்த
சித்தமன் பார்வை காணோ
- சித்தமன் தழந்

Formation of Naadi

(Naadi) + (Vayu) = Uyirthathu
Idakalai + Abanan = Vadham
Pinkalai + Pranan = Pitham
Suzhumunai + samanan = Kabham

Normally the three humours of vadham, pitham and kabham exist the ratio 1: ½ : ¼ . The derangement in these ratio leads to various disease entities and is best diagnosed by feeling the naadi.

1. Naadi (Pulse)

Naadi is the vital force. Any changes in the three dhoshas are best diagnosed feeling the naadi. Naadi is an important observation for diagnosis and prognosis. Naadi is responsible for the existence of life and can be felt one inch below the wrist on the radial side by means of palpation with the tips of index, middle ring finger corresponding to vadham, pitham and kabham site to feel naadi and its procedure is
Character of pulse

From this poem it is evident that the character of vadha naadi resembles the walk of goose, hen, and peacock. Pitha resembles the walk of tortoise and leech and kabha resembles the walk of frog snake etc.,

In cases of vadha diseases the following stages of naadi are seen.

Vatha Pitha Nadi

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In cases of vadha diseases the following stages of naadi are seen.

Vatha Pitha Nadi
Vadha Kabha Naadi

"பாரம்பரிகான மார்காநிதியாக இருக்கும் துன்பாம்
மருந்தாக கிருட்டம் பொறைச்சிச்சாதனம்
எண்முக்த சித்தியம் மற்றும் கருத்தியால்
சொன்னது விளம்பு செய்ய தேடி பிரச்சினைகள்
மருந்தாக ஆற்றம் போன்றாக செய்யப்படும்
மேலாகும் புகழ்பெற்றிய முன்னிலாம்
துறைநாளின்றை பெருக்க காரணமாக
"கம்சத்தின் சாத்தியாக்கில் மனிதம் விழாவாடலாக
பணிக்கும் கடவுட்டிகள் பிற்பானியும் கதாகங்கள் அமைப்பிட்டு
ங்கும் பிற்பானியும் கிளையார்வன் தேடியே
பணிக்கும் வள்ளு வெளியே விழாவாடல் விழாவாடல்""
- பண்டிகை கோவை

Kabha Vadha Naadi

"கம்சத்தின் சாத்தியாக்கில் மனிதம் மற்றாக
காண்டவரிசிய மனிதமாரணம் செய்யக் காட்சிகள்
"கம்சத்தின் சாத்தியாக்கில் காண்டவரிசிய
c. மனித மகாநார் காண்டவரிசிய
c. மனித மகாநார் காண்டவரிசிய
விழாவாடல் பிற்பானியும் இருக்கும் பாரம்பரிகம்
விழாவாடல் கிளையார்வன்
சீராய்கவுளத்து துறைநாளின்றை
சீராய்கவுளத்து துறைநாளின்றை
"- பண்டிகை கோவை

"பாரம்பரிகான மார்காநிதியாக மார்காநிதியாக
பராமரிப்பாளர் கொல்லும் பராமரிப்பாளர் மற்றும்
பாரம்பரிகான பெருமான்கள் மற்றும்
பாரம்பரிகான பெருமான்கள்
"- பண்டிகை கோவை
In all vadha ubhakatham patients vadha, kabha, thontha naadi was noted.

Selection of hand for both

This poem says that pulse should be seen on the right hand for males and left hand for females.

This is because of the position of nabikoormam. It is upwards in females and downwards in males.

Sparisam (Skin)

Examination of skin can be made out of inspection and touch it reveals whether the skin is warm, chill, dry, weeping, rough, smooth, hard, tender, presence of ulcers, swelling, wrinkles hair, pigmentation etc.,

Naa-tongue

The following should be noted colour, clearness, pallor, any coating excessive salivation, dryness, ulceration, fissures, thickening any growths, dents, conditions of teeth, and gums, speech any deviation of tongue etc.,

Niram (Complexion)

Colour of skin, flushing pallor discolouration etc., should be noted.

Mozhi (Speech)

Disorder of speech tone, hoarseness, laughter, slurring, incoherent, speech making unusual noises etc., should be noted.
**Vizhi (Eyes)**

Redness, ulceration, pallor, sunken state, discolouration, excessive lacrimation falling of eye lashes, condition of vision, burning of eyes other motor and sensory disturbance etc are noted.

**Malam (Stools)**

Colour, consistency, smell etc., are noted.

**Moothiram (Urine)**

Urine is examined for
Niram – Colour of urine
Manam – Smell
Edai – Specific gravity of urine
Nurai – frothy nature
Enjal – Quantity

**Neikuri**

Siddhars have explained a wonderful method to diagnose a disease by examining the urine with gingelly etc.,

"அனையற்றுக்காம் அனிவர்க்கம்
அனையற்றுக்காம் அசைவுக்காம் காற்றுக்காம்
ஏக்கரையில் கூரிய தோல்கை
அந்தக் காண்களை அனைவு போம்
நீர் அழுகைக்காம் கனவா பயிரிக்கா
பிள்ளை பெருவுக்காம் பிள்ளைகளா காணா"
- சிதழேசன்
**Method**

Prior to the day of urine examination the patient is advised to take a balanced diet and he should have a good sleep. After waking in the morning urine voided first is collected in a glass contained and is analysed within 1½ hours. A drop of gingelly oil is dropped without any oscillation in direct sunlight and the nature of its spread is noted.

"அர்ப்பா வழங்காது காண்வாற்றி விளக்கி"

Though urine should be examined in the morning only during emergency it may be done at any time.

"அசையாம் இல்லையாம் மாட்டும்"

If the drop of oil spread like a snake it indicated vadham.

"ஆலிபாம் பரிகாம் அறிக்கை"

If the oil spreads like a ring it indicates pitham

"மென்பாத்து பரிகாம் அறிக்கை"

If the oil remains like a pearl it indicates kabham.

Besides Envagai thervugal paruvakalangal and thinai should also be taken into consideration to arrive at a perfect and correct diagnosis.
**Paruvakalam (Seasons)**

In siddha system of medicine siddhars have classified a year into six seasons each having two months.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Kalam</th>
<th>Kuttram</th>
<th>State of Kuttram</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Kar kalam (Avani, Purattasi)</td>
<td>Vadham</td>
<td>Vettrunilai</td>
</tr>
<tr>
<td></td>
<td>(Aug 17 – Oct 17)</td>
<td>Pitham</td>
<td>Valarchi</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thannilai</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Valarchi</td>
</tr>
<tr>
<td>2</td>
<td>Koothirkalam (Iyppasi &amp; Karthigai)</td>
<td>Vadham</td>
<td>Thannilai</td>
</tr>
<tr>
<td></td>
<td>(Oct 18 – Dec 15)</td>
<td>Pitham</td>
<td>Valarchi</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Vettrunilai</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Valarchi</td>
</tr>
<tr>
<td>3</td>
<td>Munpani Kalam (Margali &amp; Thai)</td>
<td>Pitham</td>
<td>Thannilai</td>
</tr>
<tr>
<td></td>
<td>(Dec 16 – Feb 12)</td>
<td></td>
<td>Adaidhal</td>
</tr>
<tr>
<td>4</td>
<td>Pinpani Kalam (Masi &amp; Panguni)</td>
<td>Kabham</td>
<td>Thannilai</td>
</tr>
<tr>
<td></td>
<td>(Feb 13 – April 13)</td>
<td></td>
<td>Valarchi</td>
</tr>
<tr>
<td>5</td>
<td>Elavnenir Kalam (Chithirai &amp; Vaikasi)</td>
<td>Kabham</td>
<td>Vettrunilai</td>
</tr>
<tr>
<td></td>
<td>(April 14 – June 14)</td>
<td></td>
<td>Valarchi</td>
</tr>
<tr>
<td>6</td>
<td>Mudhuvenir kalam (Aani &amp; Aadi)</td>
<td>Vadham</td>
<td>Thannilai</td>
</tr>
<tr>
<td></td>
<td>(June 15 to August 16)</td>
<td>Kabham</td>
<td>Valarchi</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thannilai</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Adaidhal</td>
</tr>
</tbody>
</table>
Environmental changes

(a) Seasonal changes of humours

Humours  ↑↑  ↓→

Vadham  Mudhuvenir Kalam  Kaarkalam  Koodhirkalam
Pitham  Kaarkalam  Koodhirkalam  Munpanikalam
Kabham  Pinpanikalam  Elavnil kalam  Mudhuvenil kalam

↑ - Thannilai valarchi

↑↑ - Piranilai valarchi

→ - Thannilai adaithal

Regional changes of humours

Thinai (land and place)

The geographical distribution of the land is classified into five regions.

Kurunji  - Mountain and surroundings - Kabha disease
Mullai  - Forest and adjacent area - Pitha disease
Marutham  - Fields and surroundings - No disease will occur
Neithal  - Sea and surroundings - Vadha disease
Palai  - Desert and its surroundings - Mukkuttra disease

Each region has its own characters which influence the inhabitants physical mental, economic, occupational and cultural activities. In each region some ailments are common in neithal nilam. Palai nilam is a common place for all type of disease and marutha nilam is good for all types of treatment and health.
ASSOCIATED DISEASES

Regarding to Diagnostic aspect of siddha system

According to Maan murugheeyam this disease vadha ubhakatham is mentioned as follows.

"அம்முறையில் பெரும் பாதிகள் விளைவுகள் கற்பிக்கும் குறைவுகள் குறைவுகள்
முருங்கு சுட்டு குறைவுகள் பெரும் சுட்டுகள்
சுட்டு குறைவுகள் பெரும் சுட்டுகள்
சுட்டு குறைவு பெரும் சுட்டு
சுட்டு குறைவு பெரும் சுட்டு"  
- முந்தோங்கா-மானத்தீவியம் பகுதியால் 33-83, 84-85

This disease due to disorders of the haemopoietic system, especially due to anaemia (In peripheral neuritis one of the cause is anaemia)

The symptoms are

1. The pallor of the skin and mucous membrane
2. Loss of body weight
3. Fatigue
4. Loss of taste sensation
5. Burning and pricking sensation over arms, palms legs and soles.
பாசத்திள்சம் (In anaemia)

"अंसारी 's वायुस्र्व अंसारी 's अंसारी अंसारी अंसारी 's अंसारी अंसारी अंसारी 's अंसारी अंसारी अंसारी 's अंसारी अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी अंसारी 's अंसारी

- अंसारी-इंसारी इंसारी 33-83, 84-85

பாசாத்திள்சம் (In leprosy)

"குருங்கை விளக்கத் தொடுகல்

பாசாத்திள்சம் விளக்கத் தொடுகல் குருங்கை விளக்கத் தொடுகல்

- பாசாத்திள்சம் 3977

முப்பு முப்பு முப்பு (Post - Menopausal)

"தொண்டத்தொண்ட தொண்டத்தொண்ட மூசகம்

மூசகம் மூசகம் மூசகம் மூசகம்

- பாசாத்திள்சம் 1056
Differential Diagnosis

Dung coated sensation in both feet numbness and heaviness along course of sciatic nerve. In ability to maintain the upright posture. Lower limbs pain. This is the signs and symptoms of Vadhakarshanam.

In Vadhaubhakatham

Complaints such as numbness and heaviness along course of sciatic nerve. In ability to maintain the upright posture is not present. Thus it differentiated from Vadhakarshanam.
Burning sensation in both lower limbs, generalised paleness of body sensory disturbance, difficulty in walking, heaviness of the body, this is the signs and symptoms of Karasthamba vadham

**In Vadhaubhakatham**

Heaviness of the body is not present thus it differentiated from the Karasthambhavatham

The disease is specially localized to gluteal and thigh region. Around waist, hip, legs and fingers there is a feeling of dung coated sensation, heaviness of head, generalised bulkiness and numbness. The walk is diminished. These are the sings and symptoms of voorusdhambham. Which is due to decreased vadham

**In Vadha Ubhakatham**

It is not specified for gluteal and thigh region. Heaviness of head is not present thus it differentiated from Oorusthambavadham.
Treatment of vadha ubhakatham

In siddha system of medicine the main aim of the treatment is removal of udalpinigal (due to alterations of uyir thadhukkal and udal thadhukkal) and ulappinigal (due to alternation of mind). Treatment is not only for removal of disease but for the prevention and improving the body condition also. This said to as follow.

- Kaapu
- Neekam
- Niriappu

Ayyan Thiruvalluvar says about physicians duty as “study the disease, spy the cause, seek subsiding ways and do what is proper and effective” the man wellversed in medical lore would measure the patients, disease and time before the healing work begins.

" செய்து வைத்து செய்து வெளியுவினு ஆதிக்காதே மறு வைத்து மறுப்பைத்தே"  - திருத்தல்

" காற்றோர் காற்றோர் விளைவாயினும் காற்றோர் காற்றோர் ஓழும்"  - திருத்தல்

So its essential to know the disease the cause, the nature of the patient, severity of illness, the seasons and time of occurrence must be observed clearly.

The treatment is divided into three types in siddha system of medicine namely.
Dheva Maruthuvam

It includes medicines, made out of organic and inorganic ores such as chunnam, parpam, chenduram, kalangu, kattu, mathirai, melugu.

Maanida Maruthuvam

It is the medicine method made out of plants such as kudineer, churanam, surasam, pittu, vadagam, ilaham.

Asura Maruthuvam

It is named after the method of treatment. It include aruvai (surgery) keeral (scratching of blood vessels and abcess) attai vedal (Leech therapy) kuridhi vangal (blood letting therapy) kombu kattal (setting of bone with support).

Purgatives

It corrects the vitiated vadham

"புர்ப்புணகம் வாதம் கருதும்"

Vellai ennai – 15ml at early morning is given one day before starting the main treatment usually otherwise we used Nila vagai chooranam 10gm with hot water at bed time.

"நீல வாகை சோராணத்து 10 மிளில் வெளி முன் பெள்ளித்தொடர்கள் மேலும் வெளிப்படுத்தும் வெள்"
Medicines

The anti vadha medicines both internal medicines and external applications are given to relieve the symptoms and strengthen the affected parts.

Pathiyam

Pathiyam is also an important part of treatment. It is divided into three types namely

1. Echcha pathiyam
2. Kadum Pathiyam
3. Migakkadum Pathiyam

Uppila pathiyam is also mentioned in many ancient siddha literatures especially for vadha disease.

These diet regimen will produce harmful effects so they should be avoided.
Diet and advise

- It must be easily digestible
- Avoid fat contents and spicy foods
- Diet must contain adequate fibre contents to facilitate easy evacuation of bowels
- Add food stuffs which neutralise vadham
- Diabetic patients avoid sweets and carbohydrate food.
MODERN ASPECT

Theoretical View of the Dissertation Topic in Modern Aspect

ANATOMY

The peripheral nervous system is formed by neurons and their processes present in all regions of the body. Neurons or nerve cells are structural and functional unit of the nervous system. It provides the links from and to the real world. Its ghostly white nerves thread through virtually every part of the body enabling the CNS to receive information and carry out its decisions. It includes all neural structures outside the brain and spinal cord that is the sensory receptors, peripheral nerves and their associated ganglionic and efferent motor endings.

Structure of neuron:-

The nerve cell is like any other cells in the body having almost all the organelles in the cytoplasm. However it is different from other cells because of the presence of the processes and absence of centrosomes (centrosome function is formation of cilia and flagellae and it forms the spindle of fibrillar protein during mitosis). The neuron is made up of,

1. Nerve Cells
2. Dendrites
3. Axon

The nerve cell body is also called as soma or perikaryon. The dendrite and axon together form the processes. Each neuron has only one axon. The axon arises from axon Hillock of soma.

The dendrites may be absent or if present it may be one or many in number. In general, the dendrites are short processes and the axons are long processes. The dendrites and axons are usually called nerve fibers.
1. **Nerve cell body**

   The nerve cell body is irregular in shape and, like any other cell it is constituted by a mass of cytoplasm called neuroplasm covered a cell membrane. The cytoplasm contains a large nucleus, Nissl bodies, neurofibrils, mitochondria and golgi apparatus.

   (i) **Nucleus**

   Each neuron has only one nucleus in the nerve cell body. It is located in the central part. It has one or two nucleoli which are prominent. The nucleus does not contain centrosome. So the nerve cell can not multiply like the other cells.

   (ii) **Nissl bodies**

   Nissl bodies or granules are basophilic in nature. These granules are present through out soma except axon Hillock. These bodies are responsible for spotted appearance of soma after suitable staining. The nissl granules flow into the dendrites from soma, but not into axon. By this axons can be distinguished from the dendrites.

   The nissl bodies are organelles responsible for synthesis of proteins. The formed proteins in soma are transported to the axon by axonal flow.

   The number of nissl bodies vary with the condition of the nerve. During fatigue or injury of neuron, these bodies fragment and disappear by a process called chromatolysis.

   (iii) **Neurofibrils**

   These are thread like structures present in the form of network in soma and the processes. These consist of microfilaments and microtubules.
iv. Mitochondria

It forms the powerhouse of the nerve cell where ATP is produced which is energy rich compound.

Golgi apparatus is concerned with package of proteins into granules.

2. Dendrites

The dendrites are the branched processes of the neuron and are branched repeatedly. The dendrite may be present or absent. The dendrites have nissl granules and neurofibrils.

Dendrites are conductive in nature and transmit impulses towards the nerve cell body.

3. Axon

The axon is the longer process of the nerve cell. This arises from axon Hillock of the nerve cell body and is devoid of nissl granules. The axon may extend for a long distance away from the nerve cell body. The length of the longest axon is about one metre.

(i) Structure of axon

With in a nerve, each axon is surrounded by a delicate layer of loose connective tissue called endoneurium, which also encloses the fibre’s associated myelin and or neurilemma sheath. Group of fibers are bound into bundles or fascicles by a coarser connective tissue, wrapping the perineurium. Finally, all the vesicles are enclosed by a tough fibrous sheath, the epineurium, to form the nerve.

Neuron processes constitute only a small fraction of a nerve’s myelin and the protective connective tissue wrappings. Blood vessels and lymphatic vessels and also found within a nerve.
Internal structure of axon:-

Axon cylinder:-

The axon has long central core of cytoplasm called axoplasm. The axoplasm is covered by membrane called axolemma. Axoplasm contains mitochondria, neurofibrils and axoplasmic vesicles. Most of the axons are insulated by myelin sheath called as myelinated nerve fiber. Those, without myelin are called non-myelinated nerve fibers.

Myelin sheath

It does not form a continuous sheath and is absent at regular intervals. The area where myelin sheath absent is called node of Ranvier. It is responsible for white colour of nerve fibers.

Neurilemma:

Surrounding the myelin sheath, there is thin membrane called as neurilemmal sheath. This is also called as neurilemma or sheath of Schwann. This contains Schwann cells which have flattened and elongated nuclei. One nucleus is present in each internode of axon. The nucleus is situated between myelin sheath and neurilemma.

Schwann cells (Lemmocytes):-

Schwann cells are satellite cells of the peripheral nervous system all peripheral axons are ensheathed by them and are separted from the endoneurrum by the schwann cell plasma membranes. Schwann cells participate in the supply of metabolises and trophic factors 10 axons in the maintenance of the ionic state of the periaxonal space and possibly to the distribution of neurotransmitters, also to the sitting of sodium channels along the axolemma.
PHYSIOLOGY

Nervous system controls all the activities of the body. It is quicker than the other control system in the body namely the endocrine system. Primarily the nervous system is divided into central and peripheral nervous system. The central nervous system includes brain and spinal cord.

Peripheral nervous system divides into somatic and autonomic nervous system. The somatic nervous system controls the movements of the body by acting on the skeletal muscles. The autonomic or involuntary nervous system is concerned with regulation of visceral or vegetative functions. It consists of sympathetic and parasympathetic division. Groups of neuronal cell bodies in peripheral nervous system are called as ganglia.

Classification of Neurons:-

The neurons classified by three different methods which are
1. Depending upon the number of poles divided into unipolar, bipolar and multipolar neurons.
2. Depending upon the function divided into motor and sensory neurons.
3. Depending upon the length of axon, divided into.
   ✏ Golgi type I neurons and
   ✏ Golgi type II neurons.

According to their function.
1. Motor (efferent or effector) neurons.
2. Sensory (afferent (or) receptor) neurons.
3. Connecting neurons.
Axons of the motar neurons transmit impulses from the CNS to stimulate muscles (or) glandular tissue.

The axons of sensory neurons transmit impulses to areas of the brain or spinal cord from the periphery.

Connecting neurons which occur only in the grey matter of the brain and spinal cord convey incoming stimuli to neurons of various integrating centers of the CNS.

Neurons are designed to initiate receive and react to stimuli transmit impulse process and store information neuronal activity results in a wide variety of responses ranging from a simple reflex to complex behaviors requiring central co-ordination.

**Nerve Degeneration and Regeneration of nerve**

When a peripheral nerve is cut the part of the nerve separated from the cell body shows as series of chemical and physical degenerative changes. At the same time the fibers of the proximal stump of the nerve those still attached to their cell bodies, grow distally toward the separated part of the nerve these changes constitute the process of regeneration.

**Conduction in Peripheral nerves**

When a nerve is stimulated, the sum of its action potentials the compound action placed on the surface. Since action potentials travel at different diameters. The compound action potential measured at a distance from the point of stimulation is a complex mixture with a least four sequential waves of different amplitude and velocity.

A number of different factors govern conduction velocities of nerve fibers is non myelinated fibers the action potential. Sweeps continuously over the axolemma as depolarization of one area of
membrane triggers depolarization of adjacent areas. The rate of spread is proportional to the nodes ranvier, from which ionic currents spreads to other nodes is sequence.

During nodal excitation the permeability of the expose nodal axolemma to sodium ions. In creases rapidly and these ions flow in along their concentration gradient generating longitudinal ionic currents inside the internodal axon to the next node. In this condition, the conduction velocity is increase from about 1 m/s is unmyelination axons to 60 – 70 m.s is the larges myelinated fibers.

**Functions of the Neurons**

The functions of the nerve cells are to receive, initiate and conduct messages' known as nerve impulses. An impulse is a combination of a -mechanical, chemical, or electronical change at some point in the immediate environment of the neuron. These changes consist of rapid fluxes of ions across the plasma membrane, against a back ground of steady, trans-membrane electrical potential difference.

**The resting potential**

The neuron is identical to the trans-membrane potential in non-excitabile cells. In most neurons it is about 80mv, inside negative. The resting potential can change either by graded potentials or action potentials. Graded potentials occur mainly across the membranes of dendrites and somata; they are typically transient increases or decreases in resting potential, (i.e) the cell is relatively hyperpolarized or depolarized. Action potentials are transient complete reversals of polarity across the membranes of axons.

**Graded potential variations**

May be excitatory or inhibitory to the neuron. When excitatory, they accompany an increased permeability to sodium or
calcium ions, which flow down their concentration gradient into the cell, progressively depolarizing the membranes towards zero potential. Inhibitory stimuli, believed to act mainly by an increasing inflow of negatively charged chloride ions, tend to increase: the membrane potential (hyper polarization) opposing or reducing the total excitatory state.

**The action potential**

It is seen in peripheral nerves in contrast a brief complete, reversal of polarity, due to the influx of sodium ions, followed by a rapid return to the resting potential as potassium ions flow out the whole process being completed in about 5 milli seconds. The action potential spreads rapidly; but unlike graded potentials, its size and timing do not alter. Graded and action potential is functionally inter-related at particular regions of neuronal surfaces.

**The Action potential transmission**

- Action potential is propagated to the terminal region of the nerve / synaptic region where it triggers the release of transmitter.
- The initiates a synaptic potential in the motor neuron.
- The depolarizing currents occur between the nodes of Ranvier and Saltatory conduction.
- At the nodes voltage - gated channels open reducing an action potential.
- In myelinated fibers the inter nodal distance increases which increasing fiber diameter, thus conduction increases in proportion to the fiber diameter.
- Conduction velocity in unmyelinated fibres is proportional to the square root of the fiber diameter
<table>
<thead>
<tr>
<th>Sensation</th>
<th>Receptor</th>
<th>First order Neuron in</th>
<th>Second order neuron in</th>
<th>Third order Neuron</th>
<th>Center</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fine touch, Tactile, Localization,</td>
<td>Meissner's corpuscles</td>
<td>Posterior nerve root ganglion – Fibers from</td>
<td>Nucleus gracilis and nucleus cuneatus internal arcuate</td>
<td>Ventral postero in lateral nucleus of</td>
<td>Sensory cortex</td>
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<td>Tactile discrimination, Tactile</td>
<td>merkel’s disc</td>
<td>fasciculus gracilis &amp; Fasiculus cuneatus</td>
<td>fibers</td>
<td>thalamus</td>
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<td>Vibratory sensation, Stereognosis</td>
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<td>Pressure crude touch</td>
<td>Pacian corpuscles</td>
<td>Post Nerve root ganglion</td>
<td>Chief sensory cells – fibes from anterior spinal thalamic</td>
<td>Ventral postero lateral nucleus of</td>
<td>Sensory cortex</td>
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<td>tract</td>
<td>thalamus</td>
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<td>Temperature</td>
<td>Warmth raffinis end bulb cold –</td>
<td>Post. Nerve root ganglion</td>
<td>Substantial gelatinaosa cells fibers form lateral spino</td>
<td>-</td>
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<td></td>
<td>krause’s end bulb</td>
<td></td>
<td>thalamic tract</td>
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<td>Conscious kinesthetic sensation</td>
<td>Proprioceptors muscle spindle</td>
<td>Post nerve root ganglion</td>
<td>Clarke’s column of cells and marginal cells fibers form</td>
<td>-</td>
<td>Cerebellum</td>
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<tr>
<td></td>
<td>golgi tendon apparatus</td>
<td></td>
<td>dorsal and ventral spino cerebellar tracts</td>
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<td>Pain</td>
<td>Free nerve endings</td>
<td>Posterior nerve root ganglion. Fast pain and slow pain</td>
<td>Fast pain – marginal cells, in spinal cord and slow pain</td>
<td>Ventrial postero lctal nucleus of</td>
<td>Sensory cortex</td>
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<tr>
<td></td>
<td></td>
<td>fibers</td>
<td>substantia gentinosa fibres form latral spino thalamic</td>
<td>thalamus reticular formation and midbrain</td>
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<td>tract</td>
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Presynaptic events of neuromuscular transmission

When the action potential reaches the presynaptic nerve terminal, voltage-gated calcium channels open, allowing influx of calcium ions (Ca^{2+}). This triggers release of acetylcholine from presynaptic vesicles into the synaptic cleft.

Active zone

The active zone is that specialized area of the presynaptic nerve terminal membrane visualized by freeze fracture electron microscopy that contains a series of particles aligned in two parallel rows. The particles are thought to represent the L-type voltage-gated Ca^{2+} channels (VGCCs) activated by motor nerve depolarization, which trigger the release of acetylcholine within the presynaptic vesicles into the synaptic cleft.

Postsynaptic events of neuromuscular transmission

The binding of two acetylcholine molecules, to each acetylcholine receptor opens a channel within the receptor, allowing Na^{+} influx and generating small, sub threshold endplate potentials at the post synaptic membrane known as miniature endplate potentials (MEPPs). The amplitude of the summated endplate potential (EPP) for each muscle fiber is proportional to the total number of MEPPs generated by the activation of many different acetylcholine receptors at the same time. When a sufficient number of receptors activated simultaneously, the endplate potential becomes large enough to trigger an action potential. The action potential then propagates along the muscle sarcoplasmic membrane to the T-tubule system, leading to the release of Ca^{2+} from the Sarcp Plasmic reticulum, ultimately resulting in muscle contraction.
Most common diseases affecting the peripheral nerves

- Diabetes
- Alcohol
- Nutritional
- Guillain-Barre
- Trauma
- Rheumatic (collagen vascular)
- Hereditary
- Amyloid
- Environmental
- Paraneoplastic toxins and Infections drugs
- Systemic disease
- Tumors

Peripheral Neuropathies with Cranial Nerve’s Involvement

<table>
<thead>
<tr>
<th>NEUROPATHIES</th>
<th>MOST COMMONLY INVOLVED CRANIAL NERVES</th>
<th>LESS COMMONLY INVOLVED CRANIAL NERVES</th>
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<tr>
<td>Diphtheria</td>
<td>IX</td>
<td>II, III</td>
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<tr>
<td>Sarcoid</td>
<td>VIII</td>
<td>I, III, IV, VI</td>
</tr>
<tr>
<td>Diabetes</td>
<td>III*</td>
<td>IV, VI, VII</td>
</tr>
<tr>
<td>Guillain-Barre</td>
<td>VI, VII</td>
<td></td>
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<tr>
<td>Miller-Fisher variant of GBS</td>
<td>1 1 1, IV</td>
<td></td>
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<tr>
<td>Sjogren syndrome</td>
<td>V</td>
<td></td>
</tr>
<tr>
<td>Polyarteritis nodosa</td>
<td>III, VII</td>
<td>VIII</td>
</tr>
<tr>
<td>Wegener</td>
<td>VIII</td>
<td></td>
</tr>
<tr>
<td>Lyme disease</td>
<td>VII, V</td>
<td>All but I</td>
</tr>
<tr>
<td>Porphyria</td>
<td>VII, X</td>
<td>III, IV, V, XI</td>
</tr>
<tr>
<td>Refsum’s disease</td>
<td>I, VIII</td>
<td></td>
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<tr>
<td>Primary amyloidosis</td>
<td>VII, V, III</td>
<td>VI, XII</td>
</tr>
<tr>
<td>Syphilis</td>
<td>III</td>
<td>IV, V, VII</td>
</tr>
<tr>
<td>Arsenic</td>
<td>V</td>
<td></td>
</tr>
</tbody>
</table>

* Pupil is usually not affected.
Most neuropathies begin distally, but a few may begin proximally:

**Sensory neuropathies:**
Porphyria and rare cases of Charcot-Marie-Tooth and Tangier disease

**Motor neuropathies:**

Most neuropathies present with symptoms in the feet. Once the symptoms in the lower extremities proceed to the middle of the calf, the neuropathies begin to appear in the hands. Although this pattern generally holds, some neuropathies may start in the upper extremity:

1. Compression/entrapment syndromes
2. Diabetes
3. Vasculitic neuropathy
4. Guillain-Barre syndrome
5. Multifocal motor neuropathy
6. Lead Toxicity
7. Porphyria
8. Sarcoidosis
9. Leprosy
10. Charcot-Marie-Tooth disease (rare)
11. Tangier disease
12. Inherited recurrent focal neuropathies
13. Some forms of familial amyloid polyneuropathy
DISORDERS OF PERIPHERAL NERVES

Peripheral nerves are made up of axons, which represent elongated processes originating from neurons in the anterior horn cells of the spinal cord and dorsal root ganglia. These are enveloped in a series of Schwann cells which form the fatty myelin sheath. Pathological process may affect cell bodies, the myelin sheath, or the connective tissue and blood vessels of peripheral nerves. Although these processes cannot be clinically distinguished, knowledge of the pathological nature of a neuropathy is helpful in assessing prognosis and deciding on treatment. The clinical classification of peripheral nerve lesions comprises involvement of one or more individual peripheral nerves or a generalized polyneuropathy.

Peripheral neuropathy and diseases of the lower motor neuron

Peripheral neuropathy manifests as paraesthesiae (pins and needles) pain, to severe pain. Actual sensory impairment may range from slight numbness with hypersensitivity to painful stimuli to gross loss of pain perception. In others the picture is essentially a motor one almost to the exclusion of sensory phenomena. The only constant physical finding is depression or absence of reflexes it is unlikely that he is suffering from a peripheral neuropathy.

Lower motor neuron (LMN)

The motor cells of the cranial nerve nuclei of the brainstem and the anterior horn cells of the spinal cord form the lower motor neurons. The axons from these cells reach the mixed peripheral nerve through the anterior nerve roots and supply the motor and
plates of a group of muscles. The anterior horn cell its axon and the group of muscle fibers, it supplies comprise a lower motor unit. All muscles are supplied by several such motor units, the lower motor neuron is an integral part of the spinal reflex arc. It is the final common path for all motor activity (both voluntary and reflex) of all muscles. Integrity of the LMN is essential for maintaining the normal nutrition and size of the muscle fiber and when the LMN is damaged the corresponding muscles undergo atrophy.

**Signs of LMN lesions**

1. Loss of all movements - voluntary and reflex.
2. Loss of tone - Flaccidity
3. Wasting - which appears within 2-3 weeks of the lesion
4. Loss of tendon reflexes and the corresponding superficial reflexes
5. The atrophy muscles undergo contractures.
6. Denervated muscle fibres contract spontaneously contraction of single muscle fibres is called fibrillation. This not visible, but can be detected by electromyography, groups of muscle fibres which are damaged but which are still capable of contracting spontaneously give rise to visible fasciculations.
7. Electrical activity of the muscle is altered and this can be detected by electromyography. The pattern of motor loss depends on the site of affection of the LMN. Lesions of the anterior horn cells or the anterior root give rise to segmental loss of function. Lesions in the peripheral nerve give rise to paralysis of the muscles supplied by that nerve often peripheral nerve lesions are associated with sensory disturbances.
PERIPHERAL NEUROPATHY

Alternative Names

Peripheral Neuritis.

Definition

Peripheral neuropathy is failure of the peripheral nerves that carry information to and from the brain and Spinal cord. This produces symptoms like pain, loss of sensation, and inability to control muscles, ("peripheral" - nerves beyond the brain and spinal cord; "Neuro"- nerves; "Pathy"- abnormal)

Peripheral neuropathy may manifest as paraesthesia (pins and needles) pain, muscle atrophy, sensory loss and cluminess usually due to a mixture of slight loss of dexterity and sensory impairment make the diagnosis more obvious even though the aetiology often remains obscure.

In all age groups a very detailed family history is vital. Sometimes several members of a family are found to have the same disease, masquerading under several different diagnostic labels. If there is any doubt it is worth examining other family members to establish whether or not an inherited disorder is present. A detailed history of all drugs used with in the previous two years and any possible chemical exposure, diabetus mellitus, dietary habits, previous surgical procedures and alcohol intake should be included.
PATHOLOGY

The pathological reaction of peripheral nervous system in response to injury may be in the form of one of the three types of degeneration causing “Peripheral Neuropathy”. The three types of degenerations are
1. Wallerian degeneration
2. Axonal degeneration
3. Segment demyelination

Degrees of injury

When a nerve fibre is injured various changes occur in the nerve fibre and nerve cell body. All these changes are together called the degenerative changes. The injury may occur due to the obstruction of blood flow, crushing of nerve fibre transaction of the fibre and by local injection of toxic substances in to the nerve.

1. First degree of injury

In this the axon is not destroyed but it losses the function for a short time. This is caused by the pressure. Over a nerve for a short period leadings to occlusion of blood flow and hypoxia. The function of nerve fibre returns with in few hours to few weeks.

2. Second degree injury

This occurs due to the severe, prolonged pressures applied over the nerve fibre. The endoneural tube remains intact.

3. Third degree of injury

In this the endoneural tubes are interrupted.

4. Fourth degree of injury

This initiates the degenerative changes which includes the chemical and physical changes.
Changes during degeneration

(a) Changes in nerve cell body

This nissl granule disintegrates into fragments by chromatolysis process. The Golgi apparatus is disintegrated. The cell body swells due to accumulation of fluid and become round. In this case the neuron atropies and disappears completely.

(b) Changes in the nerve fibres

The degenerative changes in the distal cut end of the nerve fibre are named after the discoverer called as wallerian degeneration.

The degeneration at proximal cut end along with degeneration of cell body is called “retrograde degeneration”

Reaction to injury

1. Wallerian degeneration

It occurs after transection of the axon which may be as a result of knife wounds. Compression, traction and ischemia. Following transection initially there is accumulation of granules in the proximal and distal ends of the transection sites. Subsequently the axon and myelin sheath distal to the transaction sites. Undergo disintegration up to the next node of ranvier followed by phagocytosis. The process of regeneration occurs by sprouting of axons and proliferation of schwanncells from the proximal end.

2. Axonal degeneration

In axonal degeneration of the axon beings at the peripheral terminal and proceeds backward towards the nerve cell body. Cytoplasmic disintegration induced by injury of the distal axon lie that portion of the axon farthest from the neuronal cell body is called “axonal degeneration” Degeneration beings at the single point
within the axon and progresses to involve more symptoms within few days after the injury all portions of the distal axon begin the process of disintegration.

Concurrently the axolemma becomes discontinuous and electrical propagation is interrupted. The myelin sheath disintegrates and axon fragments into small osm segments at all points distal into the site of injury. If the injury is in the immediate proximity of the neuronal cell body the latter degenerates, otherwise the cell body enlarges with a central eosinophilic region and displacement of the nissl substances to the periphery of the cell, termed axonal traction or chromatolytes.

Schwann cells actively participate in axonal degeneration and regeneration. Shortly after the onset of disintegration of the axonal cylinder. Schwann cells proliferate and encircle the axonal remains. Hacrophanges are recruited to the site of axonal degeneration and adhere to the outer surface of the fibre.

They participate in the phagocytosis of myelin and axonal debris. The debris is removed over a period of weeks as foamy macrophages migrate towards vascular spaces.

Evidence implicating fast axonal transport in the process of axonal degeneration more slowly than short axons, implying that material is depleted more rapidly in shorter axons. The interval from injury to total axonal fragmentation is generally less than 2 weeks. Thus an acute axonopathy is by abundant degenerating fibers while a chronic axonopathy features fiber loss.

The peripheral nervous system has a large capacity for regeneration if the injurious agent is eliminated. There are two patterns of axonal sprouting and regrowth. The first and most rapid is sprouting of intact axons contiguous to site by denervation. The sprouting occurs at the distal stump of the injured axon and
results in the formation of growth cones. Growth cones contains elevated levels of growth associated protein (GAP – 43) other growth factors including nerve growth factor (NGF) facilitate axonal regeneration NGF plays dual role in axonal regeneration, interacting with both axonal growth cones and schwann cells.

Shortly in axonal degeneration the cell body undergoes chromatolysis. There is Schwann cells proliferation in the region of axonal degeneration. The loss of axonal integrity occurs probably as a result of some primary metabolic chrstarbance with in axon itself. Changes similar to those seen is wallerian degeneration are present but regenerative reaction is limited or absent.

3. Segmental demyelination

Segmental demyelination is similar to demyelination within the brain. It is the demyelination between two consecutive nodes of raniver, leaving a denoted axon segment. The axon however remains intact Schwann cell proliferation. This results in remyelination of the affected axon. Repeated episodes of demylination and remyelintaion are associated with concentric proliferation of Schwann cells around axons producing onion bulbs found in hypertrophic neuropathy.

Clinical symptomatology

Clinical features may be due to affection of the motor, sensory, or autonomic fibres separately or in combination. 1. Sensory Phenomena

It may be positive, i.e. Irritative in nature or negative, i.e due to loss of function.
Positive phenomena

i. Spontaneous pain
   This occurs due to irritation of the small fibers which carry pain or due to loss of large fibers which usually inhibit the pain sensation carried by the small fibres,

ii. Paraesthesiae in the form of pins and needles,

iii. Contact dysesthesia: where contact with the affected part elicits a disturbing sensation,

iv. Hyperalgesia: increased appreciation of pain resulting from decrease in the sensory threshold for pain,

v. Burning feet: this is a frequent symptom but its mechanism is not clear. It may occur in neuropathies, accompanying alcoholism, diabetes and beriberi,

vi. Lancinating pains: These are sharp and paroxysmal pains occurring usually in the transverse plane in the lower limbs in patients suffering from tabes dorsalis and diabetes.

Negative Phenomena

Numbness is an important negative symptom which indicates that at least 50 percent of the nerve fibres are destroyed.

Patients may have a feeling as though they are walking on cotton wool. They may have unsteadiness of gait, particularly in the dark due to loss of joint and position sense. Non healing painless ulcers and neuropathic joints develop due to loss of protective reflexes when the protective influence of pain is absent, the joints undergo minor and major trauma due to over use and injuries.

Degenerative and destructive changes of joint structures develop, leading to disorganization of the joint (Charcot's joints) such joints may occur in severe peripheral neuropathy. The pattern of
sensory loss differs depending up on the site of affection. When a single peripheral nerve is affected the sensory and motor abnormalities occur in the distribution of that nerve (mononeuritis) (ex. Neural Leprosy). In classical Polyneuropathies there is symmetrical distal sensory loss in the extremities described as "gloved and stocking" anesthesia.

2. Motor Phenomena

These take the form of weakness, muscle wasting, fasciculations and cramps, compared to disease affecting the anterior horn cells, fasciculations are much less marked.

The tenden reflexes are lost early in Peripheral neuropathies which affect the large fibres. The reflexes may be spared in neuropathies which affect the small fibres alone. (eg) Neural leprosy.

3. Autonomic dysfunction

This manifests as loss of sweating postural hypo tension and trophic disturbances and impotence.

APPROACH TO A PATIENTS WITH A CLINICAL FEATURES

The clinical points which are most helpful in diagnosis are given below:

1. **Rate of Onset:**
   - Acute: less than 1 week.
   - Subacute: less than 1 month
   - Chronic: more than 1 month

2. **Type of nerve fibre involved**
   - Motor, Sensory, autonomic, or mixed.

3. **Distribution**
   - Proximal, distal or diffuse.

4. **Painful neuropathies**
   - Alcohol and nutritional deficiencies, DM, hereditary sensory neuropathy, arsenic.
Loss of tendon reflexes is a frequent in Peripheral neuropathy, and usually first affects the ankle jerks. Sensory symptoms and sensory loss in symmetrical neuropathies are usually distal in distribution, giving rise to the "Glove and stocking" pattern of involvement.

In the first, the impairment predominantly affects joint position sense, and vibration and touch-pressure sensibility, corresponding to a predominant loss of function in the larger myelinated nerve fibres.

In the second pattern of selective sensory loss, pain and temperature sensibility are predominantly affected, often associated with loss of autonomic function, corresponding to a predominant loss of autonomic function, corresponding to a predominant loss of small myelinated and unmyelinated axons. Trophic changes may complicate Peripheral neuropathies.

Paraesthesiae are a frequent feature in Peripheral neuropathy. These are usually of a tingling nature ('Pins and needles') but may involve thermal sensation, most often with a burning quality. The paraesthesiae may be aggravated by touching or rubbing the skin.

**Peripheral neuropathy** can note any disease of the peripheral nervous system (PNS). Involvement of a single peripheral nerve, it is a syndrome characterized by disturbances of the functions of peripheral nerves due to varied aetiology. In this section we are concerned with inherited, infective, metabolic, deficiency, toxic and obscure disorders in which the peripheral nerves are diffusely damaged. The patients with peripheral neuritis were selected for dissertation work who had diabetes mellitus, B12 deficiency, alcoholic abuse, Nutritional disorder.
Deficiency neuropathy

Cyanocobalamin (B12 deficiency) Definition

A progressive disease due to deficiency of vitamin B12 and associated with pernicious anaemia, in which white matter of the spinal cord degenerates, the effects being particularly in the posterior and lateral columns. There is an associated leads to peripheral neuropathy.

Symptoms

In most cases the symptoms appear indiously and without any recognized existing cause. The first symptom is usually numbness or tingling in the feet and a slight sensation of the same kind in the fingers. This often the sensation in the feet is one of swelling or coldness, or as if walking on cotton wool; and in few cases unsteadiness in walking is at first the only complaint.

Examination

At the time shows the weakness of the toes or in dorsiflexion of the feet, diminution or absence of the ankle-jerk, probably an extensor plantar reflex, and a variable degree of sensory loss. The superficial sensory loss is at first only over the feet, then it spreads up to a cover a ‘sock’ area, and later has ‘stocking’ distribution, pain and temperature impairment meanwhile being added to it.

Alcoholic peripheral neuropathy

Alcoholism is perhaps the commonest cause of peripheral neuritis. It is preceded by chronic gastritis due to constant drinking and as a result, there is failure of absorption of vitamin B1 by the damaged stomach leading to neuritis. The onset is insidious with tingling and numbness in the lower limbs followed by weakness and globar wasting of the muscles which is symmetrical in distribution. There is generally hypoaesthesia of the skin, with hyperaesthesia of the deeper structures, like tenderness
over the calf muscles. The tendon jerks are lost. The gait is characteristically of 'steppage' type due to bilateral foot-drop. It may occur and recovery may take years even with adequate vitamin replacement and abstention from alcohol.

DIABETIC NEUROPATHY

Definition

Diabetic neuropathy may be defined as a non-inflammatory, non-specific pathological or functional disorder of the peripheral nervous system.

Introduction

Diabetic neuropathy (DN) is one of the commonest metabolic complications of diabetes. (De-calvi, first showed the relationship between DM and peripheral nerve damage in 1864). It reviews current knowledge of the aetiology of diabetic neuropathy and the outcomes and limitations of previous trials and discusses future directions for the investigation of its prevention and treatment proposed mechanisms for the development of diabetic neuropathy have been widely studied. It has been shown that there is improvement of nerve function associated with some short term clinical trials of treatments that address a number of possible etiologic pathways. However, with the expectation of the diabetes control and complications Trial (DCCT long term trials with adequate statistical power to evaluate clinical outcome points have not been conducted. The changes in nerve function are similar in most of the clinical trials. For instance, in four clinical trials directed at separate mechanisms / improved glucose control, high myo-inositol diet, therapy with an aldose reductase inhibitor, and therapy with supplementary (lambda - linolenic acid) a similar improvement in
peroneal motor velocity of 1 - 2 m/s is observed. This implies that each of the proposed mechanisms contributes equally to the development of neuropathy or that there is some redundancy to their mechanisms.

Improvement with treatment has been measured with several markers including nerve conduction velocity, quantitative sensory testing, autonomic function testing, and morphometric changes, morphometry will predict improvement in long-term clinical outcomes such as impaired sensation, painful neuropathy insensitive feet, neuropathic ulceration and amputation. Neuropathy trials must consider present knowledge about complications in general and neuropathy specially.

Patients with mild to moderate neuropathy with presumably more metabolic than structural neuropathy would be referred subjects, and follow up of 3-5 years is likely to be needed. During the past 20 years, numerous clinical trials have evaluated the efficacy of different agents for the treatment of diabetic neuropathy.

Pathology of Human Diabetic neuropathy

They can occur in motor, sensory and autonomic nerves. There is a significant relationship between clinical measure of neuropathic severity and myelinated fibre loss. Perhaps the prime site of intermediate tissue damage resides in endoneural microvasculature. Endoneurial microangiopathy has been related, it various studies, to the severity of diabetes mellitus.

Pathological features
- Axonal degeneration of both myelinated and unmyelinated fibres.
  Early: Axon shrinkage
  Later: Axonal fragmentation ; regeneration
- Thickening of schwann cell basal lamina
- Patchy, segmental demyelination
- Thickening of basement membrane and microthrombi in intraneural Capillaries

**Aetiopathogenesis of diabetic neuropathy**

Factors involved are,
- Direct insult
- Nerve perfusion
- Regenerative capacity
- Auto immune

Studies in neuropathic patients using vasodilator approach have been extensive. Recent studies have noted alleviation of symptoms using prostaglandin analog II prost and angiotension converting enzymes inhibitor (ACEI) lisinopril. At present however nitric acid (Na) prostaglandin (PGI2) Angiotension II (All) and endothelin (CET) are the factors appearing to play significant role in aetiopathogenesis.

Metabolic factors in diabetes which contribute to the decrease of blood flow to endoneurium. They are likely to be dependent on hyperglycemia mainly, even though insulin may also play a part. The major metabolic changes caused by hyperglycemia are increased poly of pathway flux, elevated oxygen free radical formation, and advanced glycosylation. In addition, impaired insulin action / hyperglycemia may contribute to defective carnitine and w-6 essential fatty acid (EFA) metabolism.
The twin problems of chronic hyperglycemia and endoneurial hypoxia results in excessive oxidative stress. Peripheral nerves have chain breaking antioxidants suppressing free radical chain oxidation by molecular oxygen and comprises of superoxide dismutase (SOD), catalase, glutathione peroxidase and glutathione reductase. Of great interest is the fall of glutathione and its related enzymes to only 10% in diabetic rodent’s peripheral nerve. The potential sources of free radical generation in diabetes are ischemia, hyperglycemia, increased mitochondrial leak, catecholamine oxidation and leukocytes.

Hyperglycemia damages microvessels via rheologic mechanisms, advanced glycation end products (AGE) and reduction in nitric oxide, resulting in endoneurial hypoxia and multifocal glycation and lipid peroxidation.

The role of leukocytes in human diabetic neuropathy is suggested by the presence of round cell infiltration in acute autonomic neuropathy and subacute proximal neuropathy.
Oxidative stress also leads to mitochondrial damage and possibly mitochondrial DNA damage and mutations, which further worsen the mitochondrial dysfunction.

Improvement in experimental Diabetic neuropathy have been noted with probucol intravenous glutathione, carvidilol and ascorbic acid, lipid acid, a powerful lipophilic free radical scavengers has been shown to improve nerve blood flow in one study.

Hyperglycemia

<table>
<thead>
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<th>AGE</th>
<th>Metabolic abnormalities</th>
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<tbody>
<tr>
<td></td>
<td>Insulin glycemic</td>
</tr>
</tbody>
</table>

Control

Aminoguanidine ______ oxidative stress ______ polyol _______ EFA Pathway
              Metabolism

Antioxidant Aldose EPO
Transition Reductase
GLA/DGLA
Metal chelators Inhibitors

AGE - Advance Glycation End Products
EFA - Essential fatty acid
GLA - Gamma Linolenic acid
EPO - Evening Primrose oil
DGLA - Dihomo Gamma Linolenic acid

Distal symmetric neuropathy classically presents with stocking glove distribution of sensory loss with feet and hands
presenting earliest with more severe signs and symptoms. This indicates that neuron with the longest axons are the most vulnerable.

NGF and neurotrophin - 3 are both found to prevent at least some of the diabetes associated metabolic changes and is an important step in understanding the pathogenesis of diabetic neuropathy and may have practical repercussions for the development of therapy and prophylaxis.

**Pathogenesis of diabetic neuropathy**

The pathogenesis of diabetic neuropathy remains poorly understood and is split between the metabolic and vascular theories. Pathological changes include a loss of myelinated fibres and microvascular abnormalities, particularly basement membrane thickening and endothelial cell hyperplasia.

The metabolic theory suggests that diabetic neuropathy arises as a consequence of chronic hyperglycemia. The diabetes control and complications trial research group demonstrated that glycaemic control reduced the progression of neuropathy in type 1 diabetes and this is also seen in patients after pancreatic transplants. The most clearly defined inducers of nerve damage include activation of the polyol pathway, advanced glycation and oxidative stress, although the transducers linking these abnormalities to tissue malfunction and damage and unknown.
Diagnosis of Diabetic peripheral neuropathy

In our diabetes centre and high risk factor ulcer clinic, all patients with foot problems have a foot screen that includes a neuropathic and vascular assessment. The neuropathy disability score uses a Nuerotip, tuning fork, hot and cold rods and a tendon hammer. This simple assessment uses a scoring system in which the patient scores one point for each incorrect test and an extra point if the Achilles tendon reflexes are not determined with reinforcement. The maximum score for each foot is 5 points, and a score of greater than 6 out of 10 suggests neuropathy.

A 10 g Semmes Weinstein monofilament is used on the plantar surface of the great toe, first, third, and fifth metatarsal heads and heal, and the dorsum of the foot, patients who cannot feel the touch of a log monofilament are also at risk. This relatively inexpensive device is useful too for determining who is at risk of foot ulceration.

Vibration perception threshold (VPT) is also commonly measured using a neurothesiometer. A 'VPT' score of 25 volts on the pulp of great toe indicates neuropathy.

Peripheral neuropathy can be diagnosed using nerve conduction studies and quantitative sensory testing. The neuropathy symptom score can be used to assess the severity of neuropathic pain.
Assessment of diabetic patients using the neuropathy disability score

<table>
<thead>
<tr>
<th>Neuropathic assessment</th>
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<th>Lt</th>
<th>Disability score</th>
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</thead>
<tbody>
<tr>
<td>Neurotip discrimination</td>
<td>Hallux - dorsal surface proximal to the toe nail</td>
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<tr>
<td>Temperature discrimination</td>
<td>Hallux-dorsal surface proximal to the toe nail</td>
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<td></td>
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<tr>
<td>Reflexes</td>
<td>Achilles tendon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>128 KHZ tuning fork</td>
<td>Pulp of hallux</td>
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</tbody>
</table>

Neuropathy disability score total (out of 10) assessment of the severity of neuropathic using the neuropathy.

Neuropathy symptom score

Have you, in the past 6 months, Burning,
Had any pain or discomfort Numbness,
In your legs and feet tingling = 2
When you are not walking fatigue, cramping,
Aching = 1 other = 0

Is this pain and discomfort most felt in the feet = 2 Calves -1
Thighs -0

Are these symptoms at their worst during the Night = 2
Various times of day Night = 1 day = 0
Have these symptoms ever kept you awake at night
Yes = 1
No = 1

When you get this pain or discomfort is there
Yes, walk = 2
No, or stand up = 1
All others 0.

Neuropathy symptom score total (out of 9)

**Differential diagnosis**

NIDDM is a relatively common disorder and a chance association with a neuropathy from another etiology is possible. In a patient with a small fibre neuropathy, with autonomic features, amyloidosis may need exclusion, as may lepromatous leprosy in a patient from an endemic area. For pure sensory neuropathy with large fibre involvement, conditions such as vitamin B.12 deficiency, paraneoplastic neuropathy, or neuropathy related to dajerine syndrome are to be excluded. Nerve biopsies and serum paraprotein estimation may be help in differentiation.

In acute painful diabetic, third nerve palsy, intracranial aneurysm is the most important condition to enter into differentiates diagnosis. Sparing of papillary function in the former may come to rescue when this situation occurs. Truncal mononeuropathies or radiculopathies may need exclusion of compressive lesions. Diabetic amyotrophy has to be differentiated from cauda equina syndrome and lumbo sacral plexus malignant invasion. The finding of a markedly increased latency of the response in nerve conduction studies on femoral nerve electrical stimulation may help.
Investigation of the peripheral neuropathy

Nerve conduction studies

When a patient's symptoms include weakness or wasting, undue fatiguability, sensory impairment, or paraesthesiae, it is usually desirable, and often essential, to supplement clinical examination by the study electrical activity in nerve and muscle. For measuring motor and sensory nerve conduction lutionized the detection of mild neuropathy, the location of sites of compression in mononeuropathy, and allow demyelinating neuropathies to be distinguished from axonal degeneration.

Electro physiological studies

In generalized symmetrical neuropathies impairment of motor and or sensory conduction.

Electromyography (EMG)

Electromyography detects and distinguish between disorders of anterior horn cell root, plexus, peripheral nerve, neuro muscular junction, and muscle, to dermise their extent and severity and to relate neurophysiological abnormality to the clinical context is found specific pathological changes, such inflammation in muscle or demyelinationof nerve, can be inferred.

Nerve biopsy

Nerve conduction tests

Quantitative sensory testing (QST) uses the response to stimuli, such as pressure, vibration and temperature, to check for neuropathy.

Clinical diagnosis of Peripheral neuropathy

The different diagnosis of peripheral neuropathy is enormously wide and each patient requires careful clinical and electro physiological evaluation. A fundamental distinction should be made between generalized polyneuropathy, which
affects all peripheral nerve fibres, often in relation to their length, and focal neuropathy, which affects individual peripheral nerves singly or multiply. Recognized causes of (multi) focal peripheral neuropathy. In focal neuropathy the muscle wasting and weakness, reflex loss and sensory disturbance are restricted to the territories of the affected peripheral nerve(s) or root(s). Occasionally widespread vasculitic involvement of the peripheral nervous system may produce the clinical picture of symmetrical polyneuropathy rather than the multifocal neuropathy usually associated with vasculitis.

Nerve conduction velocity measurements and electromyography should be used to segregate polyneuropathies into those due to primary demyelination and those due to primary axonal generation. Clinical feature suggestive of demyelinating neuropathies include a relative lack of muscle wasting in relation to the degree of weakness. Weakness of proximal muscles and disproportionate loss of joint position and vibration sensation compared to the relative preservation of pain and temperature sensation,

Which are predominantly by unmyelinated fibres. Nerve conduction studies in demyelinating neuropathy show prolonged distal motor latencies, slowed motor conduction velocities, prolonged F-wave latencies, and particularly in acquired demyelinating neuropathy, evidence of conduction block. Demyelinating neuropathies generally hold a better prospect for recovery than axonal degeneration neuropathies.

Inherited neuropathy should be distinguished from acquired neuropathy. It usually has a poor prospect for recovery and other family members may require genetic counseling. Inherited neuropathy is suggested by a family history
of neuropathy or parent consanguinity. It tends to evolve slowly, and even marked degrees of weakness may not excite complaint by the patient. Positive primary symptoms, such as paraesthesias, spontaneous pain (or) sexual sensations suggest an acquired neuropathy, although features do occur in metachromatic leucodystrophy and disease. Often the inherited basis for a patient’s neurology can only be firmly established after clinical or electrical examination of relatives.

Clinical features may help in the often difficult distinction and lesions of roots, plexuses, and peripheral nerves, lesions and brachial or lumbo-sacral plexuses are suggested by patterns blackness, reflex loss, and sensory disturbance which cannot be generated to lesions of a single nerve root or peripheral nerve. Relation between plexus and multiple root lesions may be important on clinical grounds; however, the involvement of axon nerve fibers in plexus lesions tends to produce a warm, red foot. Furthermore, sensory nerve action potentials are stored in root lesions, because the dorsal root ganglion or general branch of the sensory axon is not affected. Proximal muscle weakness tends to be a feature of lesions involving rectus or roots, rather than a feature or lesions restricted to neural nerves. Lesions of single nerve roots may be distal from those of individual peripheral nerves by the topical distribution of cutaneous sensory disturbance.

**Treatment for peripheral neuropathy**

We are still looking for a way to heal nerves damaged by neuropathy. The treatment aims to make the symptoms of neuropathy better; therapy for peripheral neuropathy differs depending on the cause.
For example

Therapy for peripheral neuropathy caused by diabetes involves control of the diabetes. Tight control of glucose is important to prevent progression. As blood sugar is brought under control, symptoms may worsen temporarily. However, improved control often relieves pain and prevents or delays further problems. Good foot care is also essential part of treatment for neuropathy affecting the lower extremities.

Treatment for diabetic neuropathy is includes - drugs to relieve pain and treat neurological problems.
- Relief of pain - Hot packs or soaks or infra red light with care
- Topical creams apply
- Dietary modification
- Prevention of foot drop and wrist drop and contractures by splints or sand bags.
- Daily massage and passive movements as soon as calf muscles are less tender. Care should be taken not to over stain weak muscles.
- Check the feet daily
- Weakness - Occupational rehabilitation
- Nerve stimulation techniques
- Specific: - Adequate control of diabetes mellitus

Alcoholic Polyneuropathy:
Treatment of polyneuropathy in general combined with that of alcohol addition, the aim being the gradual withdrawl of alcohol & fully balanced dile.

B12 Defeciency Polyneuropathy:
General treatment combined with full, balanced diet, parentral intake of vit $B_{12}$
**Nutritional Deficiency:**

General Treatment combined with rich nutrition diet.

**Expectations** (prognosis)

The outcome greatly depends on the cause of the neuropathy. In cases where a medical condition **can** be identified and treated, the prognosis may be excellent.

**Prevention**

Neuropathy can be prevented, at least in some cases. For example: The Diabetes control and complications trial studied complications in people on tight control of blood sugar level prevents the development of neuropathy in diabetics and decreases the severity of symptoms. Take care of soles and hands.

In addition, regular foot care can prevent a small infection from progressing. Maintaining ideal weight, regular exercise. Follow good habits avoid alcohol and cigarette
MATERIALS AND METHODS

“Vadha ubhakatham” a type among 80 types of vadham has been dealt in the text book of yugi vaithya chindhamani vadha ubhakatham is siddha aspect and correlates with peripheral neuritis in modern medicine. There are various causes for vadha ubhakatham. The author had chose the patients with diabetis mellitus, anaemia (B12 defeciency), nutritional disorder, patients with alcoholic abuse.

Selection of the patients

For this clinical study 20 patients of both sexes and of varying age groups suffering from vada ubhakatham selected and admitted in the in patient ward, another twenty patients were treated in out patient’s ward of pothu maruthuvam, Post Graduate -department of Government siddha medical college, palayamkottai.

This clinical study was carried under the careful supervision and monitoring by professor, reader, and lecturer, Assistant lecturer of the department.

In this study the detailed clinical history, family history, related past history, personal habits, occupation were taken.

For this purpose the case sheets were prepared, based on both siddha concept and modern concept were maintained separately for all patients.

The Cases selected on the basis of

1. Gnawing sensation over legs, hands & all over the body

2. Numbness

3. Burning sensation present in the Upper & lower limbs and all over the body
4. Polyphagia
5. Restricted movements
6. Tingling sensation
7. Giddiness
8. Dung coated sensation over soles
9. Paresthesia
10. Pricking pain with pin and needle sensation.
11. Disturbed sensory perception of touch, temperature and pain
12. Difficulties to hold chappals
13. Difficulties to grasp things
14. Trophic ulcers

**Investigations**

The symptoms of vadha ubhakatham were more or less correlated with peripheral neuritis in allopathic view. So investigations meant for peripheral neuritis were done for vadha ubhakatham also

**Blood**

- Total count
- Difference Count
- ESR
- Hb
- Peripheral smear
- Cytology
- Complete haemogram
- Sugar (fasting, PP)
- Urea
- Creatinine
- Cholesterol
- Liver function test
- Serum protein
- Thyroid profile
Skin Clipping

- AFB

Urine

- Albumin
- Sugar
- Deposit

Motion

- Ova
- Cyst
- Occult Blood

Others

Nerve conduction study

On the basis of these investigations modern diagnosis and a parallel siddha diagnosis was made with the help of the following criteria the mukkurta nilaigal, envagai thervugal, 7 udal kattugal, nilam, kalam, vayathu etc.,

Investigations were found to be useful in assessing the progress of the disease and prognosis of the patient.

Selection of the trial drug

According to tridhosa theory laxatives were first given to normalize the vitiated vadham. For this Nilavagai chooranam 10gms with hot water at bed time was recommended before starting the specific treatment.

The drugs (Pathiriver chooranam and Medicated vennai) selected for the dissertation were subjected to pharmacological and biochemical analysis of the respected Department of Government Siddha Medical College, Palayamkottai.

Individual case sheet and proforma for each patient was maintained in the inpatient ward.

All the inpatient is advised to come to the out patient ward at regular intervals for further follow up after the discharge.
RESULTS AND OBSERVATION

The results were observed with respect of the following criteria by clinical study on 20 – Inpatients and 20 – out patients

1. Sex distribution
2. Age distribution
3. Kaalam distribution
4. Constitution of the body (Dhehi)
5. Gunam distribution
6. Religion distribution
7. Occupational status
8. Socio economic status
9. Habits distribution
10. Dietary pattern
11. Paruvakaalam distribution
12. Thinai distribution
13. Aetiological Factors
14. Clinical manifestations
15. Associated diseases
16. Kosam distribution
17. Mode of Onset
18. Duration of Illness
19. Derangement of mukkurtram
   A. Derangement of vadham
   B. Derangement of pitham
   C. Derangement of kabham
20. Ezhu udal kattugal
21. Envagai Thervugal
22. Duration of Treatment
23. Sensory system and Reflexes
24. Grading of results
25. Investigation tables
1. SEX DISTRIBUTION:

Table 1 illustrates the distribution of Sex

<table>
<thead>
<tr>
<th>S. No</th>
<th>Sex</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Male</td>
<td>9</td>
<td>45%</td>
</tr>
<tr>
<td>2.</td>
<td>Female</td>
<td>11</td>
<td>55%</td>
</tr>
</tbody>
</table>

For this dissertation study 20 In-patients and 20 Out-patients were selected.

Out of this In-patients 9 patients (45%) were males and 11 patients (65%) were females.

In Out-patients 13 patients (65%) were males and 7 (35%) were females.

2. AGE DISTRIBUTION:

Table 2 illustrates the distribution of Age

<table>
<thead>
<tr>
<th>S. No</th>
<th>Age in years</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>21-30</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>31-40</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>41-50</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>4.</td>
<td>51-60</td>
<td>9</td>
<td>45%</td>
</tr>
<tr>
<td>5.</td>
<td>61-70</td>
<td>6</td>
<td>30%</td>
</tr>
<tr>
<td>6.</td>
<td>71-80</td>
<td>3</td>
<td>15%</td>
</tr>
</tbody>
</table>

10% of the In-patients were observed in the age group between 41-50 years. 45% of the In-patients were observed in the age group between 51-60 years. 30% of the In-patients were
observed in the age group between 61-70 years. 15% of the In-patients were observed in the age group between 71-80 years.

5% of the Out-patients were observed in the age group between 21-30 years. 10% of the Out-patients were observed in the age group between 31-40 years. 25% of the Out-patients were observed in the age group between 41-50 years. 15% of the Out-patients were observed in the age group between 51-60 years. 45% of the Out-patients were observed in the age group between 61-70 years.

3. KAALAM DISTRIBUTION:
Table 3 illustrates the distribution of Kaalam

<table>
<thead>
<tr>
<th>S. No</th>
<th>Kaalam</th>
<th>In-Patients</th>
<th></th>
<th>Out-Patients</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Vadha Kaalam (1-33 Years)</td>
<td>1</td>
<td>5%</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Pitha Kaalam (34-66 years)</td>
<td>14</td>
<td>70%</td>
<td>15</td>
<td>75%</td>
</tr>
<tr>
<td>3.</td>
<td>Kaba Kaalam (67-100 years)</td>
<td>5</td>
<td>25%</td>
<td>5</td>
<td>25%</td>
</tr>
</tbody>
</table>

Among the In-Patients belonging to Pitha Kaalam are chiefly affected at the rate of 70% and Kabha Kalam are affected at 25% remaining 5% are affected at Vadha Kalaam

Among the Out-Patients belonging to Pitha Kalam are chiefly affected at the rate of 75% and Kabha Kalam are affected at 25%.
4. CONSTITUTION OF THE BODY (DHEHI):  
Table 4 illustrates the distribution of Dhehi

<table>
<thead>
<tr>
<th>S. No</th>
<th>Dhehi</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Vadha Dhehi</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>2.</td>
<td>Pitha Dhehi</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>3.</td>
<td>Kaba Dhehi</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td>4.</td>
<td>Thontha Dhehi</td>
<td>10</td>
<td>50%</td>
</tr>
</tbody>
</table>

Among the In-Patients Thontha dhehi are at the rate of 50% and 25% of patients are Vadha dhehi, and 15% are kabha dhehi remaining patients are Pitha dhehi.

Among the Out-Patients Thotha dhehi are at the rate of 50% and 20% of patients are Vadha dhehi, and 20% of Pitha dhehi, remaining patients are Kabha dhehi.

5. GUNAM DISTRIBUTION:
Table 5 illustrates the distribution of Gunam

<table>
<thead>
<tr>
<th>S. No</th>
<th>Gunam</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Sathuva Gunam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Rajo Gunam</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Thamo Gunam</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

In both In-Patients and Out-Patients cent percentage of the cases had Rajo Gunam.
6. RELIGION DISTRIBUTION:
Table 6: illustrates the distribution of Religion

<table>
<thead>
<tr>
<th>S. No</th>
<th>Religion</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Hindus</td>
<td>16</td>
<td>80%</td>
</tr>
<tr>
<td>2.</td>
<td>Muslims</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Christians</td>
<td>4</td>
<td>20%</td>
</tr>
</tbody>
</table>

Among the In-Patients majority of the patients are Hindus (80%) and the Remaining percentage are Christians.

Among the Out-Patients majority of the patients are Hindus (75%) and 15% are Christians, 10% are Muslims.

7. OCCUPATION:
Table 7: illustrates the distribution of Occupation among the patients

<table>
<thead>
<tr>
<th>S. No</th>
<th>Occupation</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Formers</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>2.</td>
<td>Coolies</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>3.</td>
<td>Retired Man</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>watch Man</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>5.</td>
<td>Drivers</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>6.</td>
<td>House Wifes</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>7.</td>
<td>Servant Maid</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td>8.</td>
<td>Merchants</td>
<td>2</td>
<td>10%</td>
</tr>
</tbody>
</table>
**In In-Patients:**

Formers, coolies, house wifes are affected at the rate of 20%, servant maid are affected at the rate of 15%, merchants; drivers are affected at the rate of 10%.

**In Out-Patients:**

House wife, Retired Man are affected at the rate of 25%, merchants are affected at the rate of 20%, coolies are affect at the rate of 10% watchman, driver, servant maid are affected at the rate of 5%.

**8. SOCIO – ECONOMIC STATUS:**

Table 8: illustrates the Socio – Economic Status of the patients

<table>
<thead>
<tr>
<th>S. No</th>
<th>Socio – Economic Status</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Rich</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Middle Class</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>3.</td>
<td>Poor</td>
<td>16</td>
<td>80%</td>
</tr>
</tbody>
</table>

Among, In-Patients 80% Patients are in poor status and the remaining patients are in middle class society.

Among, Out-Patients 50% Patients are in middle class and poor status.
9. HABITS:

Table 9 illustrates the distribution of habits

<table>
<thead>
<tr>
<th>S. No</th>
<th>Habits</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
<td>No.of.Cases</td>
</tr>
<tr>
<td>1.</td>
<td>Smoker</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>2.</td>
<td>Alcoholic</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>3.</td>
<td>Tobacco</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td></td>
<td>chewer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Tobacco</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td></td>
<td>Inhaler</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Among the In-Patients 20% of the patients are smokers, 15% are tobacco chewers, 5% are Tobacco Inhaler, 10% are alcoholics.

Among the Out-Patients 30% of the patients are smokers, 5% are alcoholics.

10. DIET:

Table 10 illustrates the distribution of diet among the patients

<table>
<thead>
<tr>
<th>S. No</th>
<th>Diet</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
<td>No.of.Cases</td>
</tr>
<tr>
<td>1.</td>
<td>Vegetarian</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>2.</td>
<td>Mixed diet</td>
<td>19</td>
<td>95%</td>
</tr>
</tbody>
</table>

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

Among the Out-Patients, majority of (75%) of the patients had mixed diet and remaining had vegetarian diet.

The table showed the highest incidence of the disease for the patients with mixed diet.
11. PARUVAKAALAM DISTRIBUTION:

Table 11: illustrates the distribution of the disease among the Paruva Kaalam

<table>
<thead>
<tr>
<th>S. No</th>
<th>Paruva Kaalam</th>
<th>Month</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>No.of. Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Kaar Kaalam</td>
<td>Aavani &amp; Puratasi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Koothir Kaalam</td>
<td>Iyppasi &amp; Karthigai</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>3.</td>
<td>Munpani Kaalam</td>
<td>Markazhi &amp; Thai</td>
<td>12</td>
<td>60%</td>
</tr>
<tr>
<td>4.</td>
<td>Pinpani Kaalam</td>
<td>Masi &amp; Panguni</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td>5.</td>
<td>Elavenil Kaalam</td>
<td>Chitirai &amp; Vaikasi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Muthuvenil Kaalam</td>
<td>Aani &amp; Aadi</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

The paruva kaalam changes play major role in the development of disease. In this study, In-Patients had developed symptoms during Munpani Kaalam (60%) and Koothir Kaalam (25%) Pinpani Kaalam (15%).

Out-Patients 100% cases are affected in Koothir Kaalam.
12. THINAI DISTRIBUTION

Table 12: illustrates the distribution of Thinai

<table>
<thead>
<tr>
<th>S. No</th>
<th>Thinai (Nilam)</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
<td>No. of Cases</td>
</tr>
<tr>
<td>1.</td>
<td>Kurinji</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>2.</td>
<td>Mullai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Marutham</td>
<td>17</td>
<td>85%</td>
</tr>
<tr>
<td>4.</td>
<td>Neithal</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>5.</td>
<td>Paalai</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Among the In-Patients people living in Marutham are affected at the rate of 85% and Neithal at the rate of 10% and Kurinji at the rate 5%.

Out-Patients people living in Marutham are affected at the rate of 80% and Kurinji at the rate 15% and Neithal at the rate of 5%.

13. AETIOLOGICAL FACTOR:

Table 13 illustrates the aetiological factor for the disease.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Precipitating factors</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
<td>No. of Cases</td>
</tr>
<tr>
<td>1.</td>
<td>Diabetes mellitus</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>2.</td>
<td>Anaemia</td>
<td>8</td>
<td>40%</td>
</tr>
<tr>
<td>3.</td>
<td>Leprosy</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Alcoholics</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>5.</td>
<td>Nutritional deficiency</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>6.</td>
<td>Drugs</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
The above table shows Among the In-Patients 50% of patients had diabetes melitus. 40% of patients had anaemia, 10% had nutritional disorder, Remaining 10% of the patients are alcoholics.

Among the Out-Patients 60% of patients had diabetes melitus, 35% had anaemia, 20% had Nutritional disorder, remaining patients are alcoholics.

14. CLINICAL MANIFESTATION:

Table 14 illustrates the distribution of Clinical Features.

Table 14

<table>
<thead>
<tr>
<th>S. No</th>
<th>Clinical Symptoms</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of. Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Gnawing sensation over legs and hands</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Numbness</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Burning sensation all over the body</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Polyphagia</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>5.</td>
<td>Restricted Movements (Gait disturbance)</td>
<td>6</td>
<td>30%</td>
</tr>
<tr>
<td>6.</td>
<td>Giddiness</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>7.</td>
<td>Paresthesia</td>
<td>18</td>
<td>90%</td>
</tr>
<tr>
<td>8.</td>
<td>Pricking pain with pin and needle sensation</td>
<td>16</td>
<td>80%</td>
</tr>
<tr>
<td>9.</td>
<td>Dung coated sensation</td>
<td>16</td>
<td>80%</td>
</tr>
<tr>
<td>10.</td>
<td>Tingling sensation</td>
<td>16</td>
<td>80%</td>
</tr>
<tr>
<td></td>
<td>Difficulties to hold chappels</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>--------------------------------</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td>12.</td>
<td>Trophic Ulcers</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>13.</td>
<td>Sleepless ness</td>
<td>6</td>
<td>30%</td>
</tr>
<tr>
<td>14.</td>
<td>Constipation</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>15.</td>
<td>Muscle wasting</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>16.</td>
<td>Peri arthritis</td>
<td>7</td>
<td>35%</td>
</tr>
</tbody>
</table>

Among the 40 cases (20 in-pateints & 20 out-patients) 100% of the patients had Gnawing sensation over legs and hands, numbness, burning sensation all over the body 90% patients had parasthesia, 80% patients had dung coated sensation, pricking pain with pin and needle sensation, Tingling sensation. Of the remaining, in-patients 50% had polyphagia, constipation. 30% had restricted movements, sleepless ness, 35% had peri arthritis, 25% had giddiness, and 15% had difficults to hold chapels wasting and tropic ulcers.

Of the remaining out-patients 60% had Polyphagia, 40% had sleepless ness, 30% had giddiness and constipation, 25% had Peri arthritis, 20% had restricted movements, 5% had muscle wasting and difficulties to hold chapels.
15. ASSOCIATED DISEASES

Table 15 illustrates the distribution of associated diseases

<table>
<thead>
<tr>
<th>S. No</th>
<th>Associated diseases</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Diabetes mellitus</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>2.</td>
<td>Hyper tension</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>3.</td>
<td>Osteo arthritis</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>4.</td>
<td>lumbar Spondylosis</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>5.</td>
<td>Trophic Ulcers</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>6.</td>
<td>Piles</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Anaemia</td>
<td>8</td>
<td>40%</td>
</tr>
</tbody>
</table>

Among the In-Patients 50% of patients are affected by the disease of Diabetes mellitus, 20% of patients are affected by hyper tension, 25% of patients are affected by Osteo arthritis, 10% of patients are affected by lumbar spondylosis and tropic ulcer. 40% of patients are affected by anaemia.

Among the Out-Patients 60% of patients are affected by the disease of Diabetes mellitus, 20% of patients are affected by hyper tension, 10% of patients are affected by Osteo arthritis, 5% patients affected by piles, 35% of patients are affected by anaemia.
16. MODE OF ONSET:

Table 16 illustrates the mode of onset of the disease

Table 16

<table>
<thead>
<tr>
<th>S. No</th>
<th>Mode of Onset</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Acute</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Sub acute</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td>3.</td>
<td>Chronic</td>
<td>17</td>
<td>85%</td>
</tr>
</tbody>
</table>

Among the In-Patients 85% of cases were affected in Chronic condition, 15% of cases were affected in sub acute condition.

Among the Out-Patients 50% of cases were affected in Chronic condition, 40% of cases were affected in sub acute condition, remaining 10% were affected in acute condition.

17. DURATION OF ILLNESS:

Table 16: illustrates the distribution of the Duration of Illness

Table 17

<table>
<thead>
<tr>
<th>S. No</th>
<th>Duration of Illness</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of. Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Upto 1 month</td>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>2.</td>
<td>1 month – 6 months</td>
<td>11</td>
<td>55%</td>
</tr>
<tr>
<td>3.</td>
<td>6 month – 1 year</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>4.</td>
<td>Above 1 year</td>
<td>4</td>
<td>20%</td>
</tr>
</tbody>
</table>
Among the In-Patients 55% of patients had the duration of 1 month to 6 months. 20% of patients had the duration of 6 month to 1 year and above 1 year. 5% of patients had the duration of up to 1 month.

Among the Out-Patients 50% of patients had the duration of up to 1 month. 40% of patients had the duration of 1 month to 6 months, 10% of patients had the duration of 6 month to 1 year.

18. KOSAM:

Table 18 illustrates the distribution of Kosam

<table>
<thead>
<tr>
<th>S. No</th>
<th>Kosam</th>
<th>In-Patients</th>
<th></th>
<th>Out-Patients</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of. Cases</td>
<td>Percentage</td>
<td>No.of. Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Annamaya kosam</td>
<td>20</td>
<td>100%</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Pranamaya kosam</td>
<td>20</td>
<td>100%</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Manomaya kosam</td>
<td>20</td>
<td>100%</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Gnanamaya kosam</td>
<td>20</td>
<td>100%</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>5.</td>
<td>Anandhamaya kosam</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Among the 40 Patients 100% patients are affected by Annamaya kosam, Pranamaya kosam, Manomaya kosam, Gnanamaya kosam.
19. Thridhosa Theory
The derangement undergone by the various types of Vadham Pitham and Kabham

Table 19.a Derangement of Vadham

<table>
<thead>
<tr>
<th>S. No</th>
<th>Classification of Vadham</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>Piranan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Abanan</td>
<td>15</td>
<td>75%</td>
</tr>
<tr>
<td>3.</td>
<td>Udhanan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Viyanan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>5.</td>
<td>Samanan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>6.</td>
<td>Nagan</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>7.</td>
<td>Koorman</td>
<td>12</td>
<td>60%</td>
</tr>
<tr>
<td>8.</td>
<td>Kirukaran</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>9.</td>
<td>Devathathan</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>10.</td>
<td>Dhananjeyyan</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Among the 40 cases (both in-Patient and Out-Patient) cent percentage of the patients had altered viyanan, samanan, devathathan.10% of the patients had altered Nagan 75% of the patients had altered abanan.

Of the remaining In-Pateints 60% of the patients had altered Koorman, 50% of the patients had altered Kirukaran.

Remaining Out-Patients 70% had altered Koorman, 60% had altered Kirukaran.
Table 19.b Derangement of Pitham

<table>
<thead>
<tr>
<th>S. No</th>
<th>Classification of Pitham</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Anarpitham</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>2.</td>
<td>Ranjahapitham</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>3.</td>
<td>Prasaha Pitham</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Aalosagapitham</td>
<td>12</td>
<td>60%</td>
</tr>
<tr>
<td>5.</td>
<td>Sadhaha Pitham</td>
<td>20</td>
<td>100%</td>
</tr>
</tbody>
</table>

In 40 pateints cent percentage had altered Prasha Pitham and sathaga Pitham.

Of the remaining In-Patients 50% had altered Anarpitham and altered Ranjaha Pitham, 60% had altered alosahaha pitham.

Of the remaining Out-Patients 70% had altered Alosaha Pitham 60% had altered Anarppitham 40% had altered Ranjaha Pitham.

Table 19.c Derangement of Kabham

<table>
<thead>
<tr>
<th>S. No</th>
<th>Classification of Kabham</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Avalambagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Kilethagam</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>3.</td>
<td>Pothagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Tharpagam</td>
<td>12</td>
<td>60%</td>
</tr>
<tr>
<td>5.</td>
<td>Santhigam</td>
<td>6</td>
<td>30%</td>
</tr>
</tbody>
</table>
Among the in-Patients 50% of the patients had altered Kilethagam, 60% of patients had altered tharpagam and 30% had sandhigam.

Among the out-Patients 60% had altered Kilethagam, 70% had altered Tharpagam, and 20% had altered Sandhigam.

20. EZHU UDAL KATTUGAL:

Table 20 illustrates the distribution of derangement of Udal Kattugal in the disease.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Udal Kattugal</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
<td>No.of.Cases</td>
</tr>
<tr>
<td>1.</td>
<td>Saaram</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Senneer</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>3.</td>
<td>Oon</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Kozhuppu</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>5.</td>
<td>Enbu</td>
<td>5</td>
<td>25%</td>
</tr>
<tr>
<td>6.</td>
<td>Moolai</td>
<td>12</td>
<td>60%</td>
</tr>
<tr>
<td>7.</td>
<td>Sukkilam / Suronitham</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

**In-Patients**

100% of the patients had altered Saaram, Senneer and Oon, 25% of the patients had altered Kozhuppu and Enbu and 60% of the patients had altered Moolai.

**Out-Patients**

100% of the patients had altered Saaram, Senneer and OOn, 10% of the patients had altered Kozhuppu and Enbu and 700% of the patients had altered Moolai.
21. ENVAGAI THERVUGAL:

Table 21 illustrates the distribution of Envagai Thervugal in the disease.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Envagai Thervugal</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of. Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Naa</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>2.</td>
<td>Niram</td>
<td>8</td>
<td>40%</td>
</tr>
<tr>
<td>3.</td>
<td>Mozhi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Valhi</td>
<td>12</td>
<td>60%</td>
</tr>
<tr>
<td>5.</td>
<td>Sparisam</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>6.</td>
<td>Malam</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>7.</td>
<td>mothiram</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td>8.</td>
<td>Naadi</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>a. Vadha Pitham</td>
<td>10</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td>b. Vadha Kabam</td>
<td>3</td>
<td>15%</td>
</tr>
<tr>
<td></td>
<td>c. Pitha Vadham</td>
<td>7</td>
<td>35%</td>
</tr>
</tbody>
</table>

Among the In-Patients

100% of patients had altered Naa and Sparisam, 50% of the patients had altered Malam and Moothiram, 60% of the patients had altered Vizhi. 50% of the patients had Vadha Pitha Naadi, 35% of the patients had Pitha Vadha Naadi, 15% of the patients had Vadha Kabha Naadi

Among the Out-Patients

100% of patients had altered Naa and Sparisam, 70% of the patients had altered vizhi, 60% of the patients had altered moothiram, 30% of the patients had altered malam, 60% of the patients had Vadha Pitha Naadi, 30% of the patients had Pitha Vadha Naadi, 10% of the patients had Vadha Kabha Naadi.
22. Duration of Treatment

Table 22 illustrates the distribution of duration of Treatment

<table>
<thead>
<tr>
<th>Table 22</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. No.</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>1.</td>
</tr>
<tr>
<td>2.</td>
</tr>
<tr>
<td>3.</td>
</tr>
</tbody>
</table>

The duration of the treatment of the in-patients as follows.
41-50 days 19 patients (95%), 51-60 days 1 patient (5%)

Among the out-Patients
30-40 days 7 patients (35%), 41 – 50 days 11 patients (55%),
51-60 days 1 patient (5%)

23. SENSORY SYSTEM

Table 23.a: illustrates the distribution of the Sensory System

<table>
<thead>
<tr>
<th>Table 23. a</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. No.</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>1.</td>
</tr>
<tr>
<td>2.</td>
</tr>
<tr>
<td>3.</td>
</tr>
<tr>
<td>4.</td>
</tr>
<tr>
<td>5.</td>
</tr>
<tr>
<td>7.</td>
</tr>
<tr>
<td>8.</td>
</tr>
</tbody>
</table>
Among the In-Patients, 100% of the Patients had diminished touch sensation. 10% diminished pain sensation, 20% had hypothermia. 30% had affected vibration sense.

Among the Out-Patients, 100% had diminished touch sensation. 30% had affected vibration sense.

**REFLEX DISTRIBUTION:**

*Table 23.b: illustrates the distribution of the Deep Tendon Reflex*

<table>
<thead>
<tr>
<th>S. No</th>
<th>Deep tendon reflex</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No of Cases</td>
<td>Percentage</td>
<td>No.of.Cases</td>
</tr>
<tr>
<td>1</td>
<td>Jaw jerk</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Biceps</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Triceps</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Supinator</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>Knee jerk</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>Ankle jerk</td>
<td>Diminished</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td></td>
<td>20</td>
</tr>
</tbody>
</table>

40 patients had diminished Ankle jerk.
25. Clinical Improvement:

Table 25 illustrates the distribution of clinical Improvement

<table>
<thead>
<tr>
<th>S. No</th>
<th>Result</th>
<th>In-Patients</th>
<th>Out-Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.of.Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Good</td>
<td>17</td>
<td>85%</td>
</tr>
<tr>
<td>2.</td>
<td>Fair</td>
<td>2</td>
<td>10%</td>
</tr>
<tr>
<td>3.</td>
<td>Poor</td>
<td>1</td>
<td>5%</td>
</tr>
</tbody>
</table>

The results were graded as

Among the In-Patients 85% had showed good results, 10% had showed fair results, 5% had showed poor results.

Among the out-Patients 80% had showed good results, 15% had showed fair results, 5% had showed poor results.
DISCUSSION

The clinical study of the disease “Vadha Ubhakatham” was carried out in post Graduate Pothu Maruthuvam Department, in-patient ward with administration of trial drug “Pathiriver Chooranam” and “Medicated vennai” was carried out under the guidance of professor, reader, lecturer and asst.lecturer of concerned department. The trial drugs administered to all the patients regularly.

The strategic use of the history and other clinical profiles is discussed here to enhance the clinical evaluation of the disease. All the patients are examined systematically and thoroughly investiagted.

Sex Distribution:

For this dissertation study 20 in-patients and 20 Out-Patients were selected. Out of these 20 in-patients 9 patients (45%) were males, and 11 patients (55%) were females.

Among Out-patients 13 patients (65%) were males and 7 patients (35%) were females. So Vadha Ubhakatham can affect both sexes, more or less equally.

Age Distribution:

Regarding age incidence in in-patients 10% of the cases belongs to the age group of 41-50 years, 45% are between 51-60 years, 30% are in between 61-70 years, 15% in between 71-80 years.

Among the Out-Patients 5% of the cases belongs to the age group 21-30 years, 10% are in between 31-40 years, 25% are in between 41-50 year, 15% are in between 51-60 years, 45% are in between 61-70 years.

So in Vadha Ubhakatham majority of cases affected in between 40-70 years.
Kaalam:
Among the 20 in-patients 70% of cases were in Pitha Kaalam and 25% in Kabha Kaalam and remaining 5% in Vadha Kaalam.
Among 20 Out Patients 75% of cases were in Pitha Kaalam and 25% in Kabha Kaalam.
Though “Vadha ubhakatham” can acquire in all Kaalam, but majority of cases are affected in Pitha Kaalam.

Constitution of the Body:
Among the 20 in-patients 50% of the cases were thondha thegi, 25% of the patients were Vadha thegi, 10% of the patients were pitha thegi, 15% were kabha thegi.
Among 20 out-patients 50% of the cases were thondha thegi, 20% of the patients were Vadha thegi and Pitha thegi, and remaining 10% of the patients were kabha thegi.
So Vadha Ubhakatham can affect all thegis.

Gunam:
Among the 20 in-patients and 20 out-patients cent percent of the cases had Rajo gunam.
So Vadha Ubhakatham is predominant in Rajo gunam patients.

Religious Status:
Out of 20 in-patients majority of the patients are Hindus (80%), Christians are 15%.
Out of 20 out-patients majority of the patients are Hindus (75%) Christians are 15%. Muslims are 10%.

Occupational Status:
Regarding occupational history among the in-patients House wives, Formers, Coolies are affected at the rate of 20%. Servant maid are affected at the rate of 15%, Merchants & drivers are
affected at the rate of 10%. Remaining 5% of the affected patients are watchman.

Among the out-patients House Wifes, retired man are affected at the rate of 25% merchants are affected at the rate of 10% watchman, drivers & servant maid are affected at the rate of 5%.

So Vadha Ubhakatham can affect all categories of people.

**Socio Economic Status:**

Among the 20 in-patients 20% of the cases were in middle class and 80% are poor people.

Among the 20 out-patients 50% of the cases were middle class and poor status.

**Habits:**

Among the 20 in-patients 20% of the patients were smokers, 15% of the patients were tobacco chewers, 10% of the patients were alcoholic 5% of the patients were tobacco inhaler.

Among the 20 out-patients, 30% of the patients were smoker, 5% of the patients were alcoholic.

So Vadha Ubhakatham may affect all bad habits patients.

Alcoholic abuse is one of the causes for neuritis.

**Dietary Changes:**

Among the 20 in-patients 95% of the patients were mixed diet and the remaining are vegetarians.

Among the 20 out-patients 75% of the patients were mixed diet and the remaining are vegetarians.

So “Vadha Ubhakatham” may affect both categories of people but predominant among the mixed diet.

**Paruvakaalam:**

The paruvakaalam changes play major role in the developement of the disease. In this study among the in-patients
25% of cases had developed symptoms during Koothir kaalam (25%), Munpani Kaalam (60%), Pinpani Kaalam (15%).

Among the Out patients 100% of patients had developed symptoms during Koothir Kaalam.

**Thinai Reference:**

Among 20 in-patients most of the people living in Marutham are affected at the rate of 85%, 10% of the patients in Neidhal, 5% of the patients are in Kurinji.

Among 20 out-patients most of the people living in Marutham are affected at the rate of 80%, 15% of the patients in Kurinji, 5% of the patients are in Neidhal.

Marutham is the area where the severity of the disease is less, but these incidences may be due to the alterations in the food, habit and other activities.

**Aetiological Factors:**

Among 20 in patients 50% of patients were affected by diabetes mellitus. 40% of patients were affected by anaemia; remaining 10% of patients were affected by alcohol intake.

Among 20 out patients 60% of patients were affected by diabetes mellitus. 35% of patients affected by anaemia, remaining 5% of patients were affected by alcoholic intake.

Literary evidence indicates that the disease Vadha Ubhakatham is common in diabetes mellitus.

**Associated diseases:**

In this study among the in-patients 50% of the cases were affected by diabetes mellitus, 20% had hyper tension, 25% had Osteo arthritis, and 10% had lumbar spondylosis and trophic ulcers.
Among the out-patients 60% of the patients were affected by diabetes mellitus, 20% had hyper tension, 10% had Osteo arthritis, 5% had piles.

**Mode of Onset:**

Among the In-Patients majority of cases (85%) were presented in chronic condition, remaining 15% were in sub acute condition.

Among the Out-patients 50% of cases were presented in chronic condition. 40% of cases were presented in the subacute condition remaining 10% were in acute stage.

So majority of chronic condition needs hospitalization other condition can cure in out-patient ward.

In Vadha ubhakatham majority of the cases were reported in the chronic condition.

**Duration of Illness:**

In In-Patients 20% were affected by the duration of above 6 month to 1 year and above 1 year. 55% were above 1 month to 6 months, 55 were upto 1 month duration affected.

In out-patients 50% were affected by the duration upto 1 month and 40% were above 1 month to 6 months. Remaining 10% were above 1 month – 1 year.

In Vadha Ubhakatham majority of the cases were reported in above 1month – 6 months.

In long duration the patients needs hospitalization.

**Observations of Clinical features of Vadha Ubhakatham are as follows:**

**Clinical Manifestation:**

Among the 40 cases (20 in-pateints & 20 out-patients) 100% of the patients had Gnawing sensation over legs and hands, numbness, burning sensation all over the body 90% patients had parasthesia, 80% patients had dung coated sensation, pricking
pain with pin and needle sensation, Tingling sensation. Of the remaining, in-patients 50% had polyphagia, constipation. 30% had restricted movements, sleeplessness, 35% had peri arthritis, 25% had giddiness, 15% had difficulties to hold chapels wasting and trophic ulcers.

Of the remaining out-patients 60% had Polyphagia, 40% had sleeplessness, 30% had giddiness and constipation, 25% had Peri arthritis, 20% had restricted movements, 5% had muscle wasting and difficulties to hold chapels.

Thridhosa Theory

A. Disturbances in Vadham:

Among the 40 cases (both in-Patient and Out-Patient) cent percentage of the patients had altered viyanan, samanan, devathathan. 10% of the patients had altered Nagan 75% of the patients had altered abanan.

Of the remaining In-Patients 5% had altered Piranan & altered Udhanan, 60% of the patients had altered Koorman, 50% of the patients had altered Kirukaran.

Remaining Out-Patients 70% had altered Koorman, 60% had altered Kirukaran.

Affected Abannan produced Poly urea and constipation.

Affected Viyanan produced numbness, tingling sensation, loss of sensation, burning sensation, body pain, muscle wasting are formed.

Affected samanan produces derangement of other vayus.

Affected naagan was found to the derangement in who were mentally depression and sluggishness.

Affected Koorman produced diminished vision.

Affected Kirukaran produce increase the appetite.
Affected Devathathan produces fatigue and tiredness and disturbances of sleep rhythm.

**B. Disturbances in Pitham**

Among the in-Patients 50% of the patients had altered Anar pitham, 50% of the patients had altered Ranchaga pitham, 60% of patients had altered Alosaham, 100% had altered Prasaham and Sathaham.

Among the out-Patients 60% had altered Anar pitham, 40% had altered Ranchaga pitham, 70% had altered Alosaham, 100% had altered Prasaham and Sathaham.

Affected Anarpitham produces increase the appetite.

Affected Ranchaga Pitham produces dyspnoea and reduced heamoglobin.

Affected Prasakapitham produces dryness and darkness of the skin and burning sensation of the body.

Affected Sathaha Pitham produces difficulty in attaining to their regular duties.

Affected Alosaha pitham diminished vision.

**C. Disturbance in Kabam:**

Among the in-Patients 50% of the patients had altered Kilethagam, 60% of patients had altered tharpagam and 25% had sandhigam.

Among the out-Patients 60% had altered Kilethagam, 70% had altered Tharpagam, and 10% had altered Sandhigam.

Affected Kelethagam produce increase appetite.

Affected pothaham produces tastelessness

Affected tharpagam produces burning sensation in the eyes.

Affected Sandhiham produces Pain and difficult to flex and extend joints.
Ezhu Udal Kattugal:

In- Patients,

100% of the patients had altered Saaram, Senneer and Oon.
60% of the patients had altered Moolai.
25% of the patients had altered Kozhuppu and Enbu

Out-Patients,

100% of the patients had altered Saaram, Senneer and Oon.
70% of the patients had altered Moolai.
10% of the patients had altered Kozhuppu and Enbu


Envagai Thervugal:

Among the In-Patients,

100% of patients had altered Naa and Sparisam, 50% of the patients had altered Malam and Moothiram, 60% of the patients had altered Vizhi, 50% of the patients had Vadha Pitha Naadi, 35% of the patients had Pitha Vadha Naadi and 15% of the patients had Vadha Kabha Naadi.

Among the Out-Patients,

100% of patients had altered Naa and Sparisam, 70% of the patients had altered vizhi, 60% of the patients had altered moothiram, 30% of the patients had altered malam, 60% of the patients had Vadha Pitha Naadi, 30% of the patients had Pitha Vadha Naadi and 10% of the patients had Vadha Kabha Naadi.
Affected Naa denotes dryness of tongue and pallor. Affected Niram denotes pallor of eye skin, nailas and all over body. Affected Vizhi denotes burning sensation of eye, pallor of eye diminished vision. Affected Sparisam denotes burning sensation and numbness of body. Affected Malam denotes constipation. Neikuri was found to be diabetic patient’s urine denotes odur of honey. Edai was increased, Manam ant flies were found to the attracted urine Nurai-frothy. Neikuri had showed in inpatients 5% vadha neer, 45% pitha neer and 50% kabha neer. In outpatients 10% vadha neer, 30% pitha neer and 60% kabha neer.

**Duration of Treatment**

The patients were treated in the hospital for maximum 40 days. The prognosis and result of the treatment was assessed on the basis of subjective feeling and objective parameters.

**Sensory system**

Among the In-Patients, 100% of the Patients had diminished touch sensation. 10% diminished pain sensation, 20% had hypothermia. 30% had affected vibration sense.

Among the Out-Patients, 100% had diminished touch sensation. 30% had affected vibration sense.

**Reflexes**

40 patients had diminished Ankle jerk.

**Investigation**

Routine haematological and urological investigation was done, during the time of admission and discharge for all cases.

Haematological investigation had showed raised Blood sugar and reduction in haemoglobin.

After the end of this regimen, raised haemoglobin and reduction of blood sugar recorded.
Special Investigation

Nerve conduction study was done for 2 patients it showed sensory disturbance the symptoms are reduced after treatment.

Results

All the 20 inpatients and 20 outpatients were treated with trial medicine. The results were assessed on the basis of subjective and objective improvement of symptoms and a sense of well being at the end of the treatment the results were categorized as follows.

**Good** : Complete subsitence of gnawing sensation, numbness, pricking pain, normal appetite and free movements.

**Fair** : Relief of gnawing sensation, numbness, pricking pain, normal appetite and some time restricted movements.

**Poor** : No improvement

Among the in-patients 85% of the patients showed good results 10% patients showed fair results remaining 5% of patients showed poor results.

Among the out patients 80% of patients showed good results 15% of patients shows fair results remaining 5% of patients shows poor results.
SUMMARY

The author feels privileged for carrying out the clinical study on the disease “Vadha Ubakatham” perhaps the incidence of Vadha Ubhakatham is increasing now a day. The disease is correlated with “Peripheral Neuritis” in allopathic view. It is now common in occurrence due to prevalence of Madhu megam and their complications, and dietary irregularities.

So the author had worked on this dissertation study with deep devotion and keen observation.

For the disease Vadha Ubhakatham internal medicine pathiriver choornam is given. Medicated vennai applied externally.

The medicines for the treatment of this disease have been chosen from the traditional siddha literatures, after extensively going through various siddha literatures. The medicines are prepared according to the norms strictly confirmed to siddhar’s ethics.

The aetiology, pathology, classifications, clinical features, diagnosis complications differential diagnosis, treatment were collected from a number of literatures both in Siddha system as well as in modern system of medicine.

In this study 20 patients of either sex and of varying age groups as in-patients and 20 patients as out-patient were selected and treated with the trial drugs for this study.

The selection of cases and management of cases during admission and after treatment was carried out under the supervision of Professor, Reader, Lecturer, and Asst. Lecturer of Post graduate Pothu Maruthuvam Department.

In order to normalize the provoked Vadha humour, laxative drug was given to all patients on the first day. The next day the trial
drugs were given to all the patients. From the observations and results, the author had en-lights that vadha ubhakatham was common in the following aspects.

The disease was more common in females.

Maximum incidence of age between 50 – 70 years i.e in pitha kaalam the incidence of the disease was higher in koothirkaalam (25% ip cases and 100% op cases) & Munpani Kaalam (60% ip cases)

The disease due to vitated vadham which subsequently leads to alterations in other dhosas.

Signs and symptoms along with various other factors mentioned in the case sheets were elaborately discussed in the previous chapter.

The siddha diagnosis were made with the help of Envagai thervugal especially naadi and neikuri. Naddi Nadai of the patients was found to be vadha pitham, vatha kabham and pitha vadham.

Majority of the patients had this disease due to nutritional disorder another one is diabetes mellitus. In poor status the patients having lake of nutritional elements in their diet. The author has find out the cause from dietary habits majority of patients are in poor status.

Regarding modern aspects Vadha ubhakatham is differentiated from other pathologies by the use of routine blood examination, blood sugar, blood urea, serum cholesterol, urine, motion examination. Nerve conduction study was done in 2 patients for diagnosis and to follow the progress of the patients.

So the diagnosis, of the vadha ubhakatham was made out by analyzing clinical signs, symptoms and differential diagnosis.

The efficacies of the drugs “Pathiriver chooranam, Medicated vennai” were studied and observed during the period of this
research. The diabetic patients are also benefited by consuming this internal medicine.

Daily monitoring of diabetic status was made by Urine analysis. In addition to that drugs every patient was advised to Yoga therapy (Mayurasanam) for better results.

Clinically there were marked reduction of pain, numbness, Gnawing sensation, and tingling sensation burning sensation along and reduced blood sugar, blood urea, and increased Hb% that were noted.

Pharmacological evaluation of the drug pathiriver chooranam has showed analgesic, anti inflammatory (acute and chronic) and Hypoglycemic action. Medicated vennai has anti – inflammatory action.

Biochemical analysis showed the drugs have the presence of ferrous iron, unsaturated compound, reducing sugar and amino acid. The clinical improvement is graded as good, fair, poor on the basis of symptoms relieved and the results observed during this study.

The in-patients are discharged after satisfactory, clinical improvement and advised to continue the treatment in out-patient department.

No side effects, adverse effects and complications were observed during these studies.
CONCLUSION

 вз In this clinical trial results were found to be satisfactory in 80% of cases.
 вз The trial drugs have excellent pharmacological action.
 вз The trial drugs were free from side effects and adverse effects.
 вз Further follow up of all these patients showed excellent reduction of their symptoms and a sense of well being.
 вз The trial medicine Pathiri ver chooranam has the taste Thuvarppu.

 Suvai - Thuvarppu
 Thanmai - Thatpam
 Pirivu - Inippu

 The taste Thuvarppu has skin soothing effect and blood purifying effect, as per siddha maruthuvanga surukkam.

 And the trial medicine has it thanmai as thatpam all the thatpam naturated drug has cooling effect to body so it acts well against burning sensation.

 After ingestion while the trial medicine reacts the gastric guice it changes to Inippu, at this stage Inippu cures the vadha disease and it gives strength to 5 sensory organs. From this study Pathiri ver Chooranam acts as antivadha drug on the basis of Suvai, Thanmai, Pirivu and Edhir urai.

 So Vadha Ubhakatham is well controllable with Pathiriver Chooranam and Medicated vennai.
PREPARATION OF PATHIRI VER CHOORANAM

Ingredients
Pathiriver - 1 k.g

Method
Pathiriver is dried and purified then nicely powdered and filter through cloth.

Dose
5 gms twice daily.

Adjuvant
Hot water

Indication
Madhumegam, Burning sensation in eye, ear, leg and hands, Eczema, pitham.

Expiry
6 months from the date of preparation.

Reference
Gunapadam - Mooligai Vaguppu Page No 665

Properties of the Pathiri

Pathiri
Common Tamil Name : Pathiri
Synonyms : Kanni, Paadalimaram, Paadalam, Punkali.
Botanical Name : Stereospermum suaveolens
Family : Bignoniaceae
Parts used : Leaf, flower Bark, root
Suvai : Thuvarppu (Astringent)
Thanmai : Thatpam (Cold)
Pirivu : Inippu (Sweat)
Actions : Diuretic, febrifuge, anti inflammatory, antibacterial, tonic
Chemical Constituents: glycoside, albuminous, saccharine and 6-0 glucosyl cutellariein, dinatin – 7-glucuronide

Uses:

“பாதிரி வேறு மாதுமதுக்கண்டம் பாதிக்கும் பாதுகாகம் தொடர்கூறு பாதுகாக்க தொடர்கூறு - பாதிக்கும் காற்றுகளின் காற்றுகள் தொடர்கூறு காற்றுகளும் மிக்கம் மிக்கம்”

Pathiri ver used for madhumegam, burning sensation in eye, ear, hand and leg, eczema, pitham. Decoction of roots used in intermittent and puperperal fevers and affections of the brain.

Preparation of Medicated vennai

Ingredients

- Illandai ilai - 100gms
- Cheek-kai ilai - 100gms
- Musumusukkai ilai - 100gms
- Karuva ilai - 100gms
- Nelli ilai - 100gms
- cow’s milk - 500ml

Method

Take the juice of the above leaves. Add cow’s milk to the leaf juice. Boil this mixture and cool. Keep this solution overnight to facilitate fermentation. Next morning the fermented solution is stirred well and a buttery mass will rise up then, that is collected in a clean bowel. This butter is used for external application preserves in a water.

Dose

Butter 5gms external application for 3 days.
**Indications**

Mehathinal undana erivu, pithathinal undana erivu.

**Expiry**

3 months

**Reference**

Sarabendhirar pitha roha sihitchai P.No 199

**Properties of the Individual component**

**Illandai**

<table>
<thead>
<tr>
<th>Common Tamil Name :</th>
<th>Illandai</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synonyms :</td>
<td>Illandai, kulladhi, kulvali, kole, korkodi, vadhari</td>
</tr>
<tr>
<td>Botanical Name :</td>
<td>Ziziphus manuritiana var, Maurtina</td>
</tr>
<tr>
<td>Family :</td>
<td>Rhamnaceae</td>
</tr>
<tr>
<td>Parts used :</td>
<td>Leaf, fruit, bark, root, root bark, whole plant</td>
</tr>
</tbody>
</table>

Suvi : Thuvarppu, Inippu

Thanmai : Thatpam (Cold)

Pirivu : Inippu (Sweat)

Therapeutic Actions : Emollient, cooling and refrigerent

**Chemical Constituents:**

Leafs contain, tannin and a crystallizable principle, ziziphic acid and sugar

**Uses:**

Leaves are given cooling and refrigerent, effect. Burning sensation present in body, sacbies, pitham, Megam, also cured.
Cheek-kai

<table>
<thead>
<tr>
<th>Common Tamil Name</th>
<th>Cheek-kai</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synonyms</td>
<td>Cheek-kai, cheyakai</td>
</tr>
<tr>
<td>Botanical Name</td>
<td>Acacia sinuata</td>
</tr>
<tr>
<td>Family</td>
<td>Mimosaceae</td>
</tr>
<tr>
<td>Parts used</td>
<td>Leaf, and pod</td>
</tr>
<tr>
<td>Suvai</td>
<td>Pulippu</td>
</tr>
<tr>
<td>Thanmai</td>
<td>Thatpam (Cold)</td>
</tr>
<tr>
<td>Pirivu</td>
<td>Karppu</td>
</tr>
<tr>
<td>Therapeutic Actions</td>
<td>Externally detergent and astringent</td>
</tr>
</tbody>
</table>

**Chemical Constituents:**

<table>
<thead>
<tr>
<th>Alkaloid saponins</th>
<th>11.2%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malic acid</td>
<td>12.75%</td>
</tr>
<tr>
<td>Resin</td>
<td>1%</td>
</tr>
<tr>
<td>Glucose</td>
<td>13.9%</td>
</tr>
<tr>
<td>Gum and colouring matter</td>
<td>21.5%</td>
</tr>
<tr>
<td>Grude fibre</td>
<td>22%</td>
</tr>
<tr>
<td>Ash</td>
<td>3.75%</td>
</tr>
</tbody>
</table>

**Uses:**

This plant has cleaning properties and protects the skin against micro organism.

Musumuskaai

<table>
<thead>
<tr>
<th>Common Tamil Name</th>
<th>Musumuskkai</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synonyms</td>
<td>Iyleyam, erukurangin kai, mosumuskkai</td>
</tr>
<tr>
<td>Botanical Name</td>
<td>Melothira moderaspatana</td>
</tr>
<tr>
<td>Family</td>
<td>Cucurbitaceae</td>
</tr>
<tr>
<td>Parts used</td>
<td>Leaves and Root</td>
</tr>
</tbody>
</table>
Suvai : Thuvarppu
Thanmai : Veppam
Pirivu : Karppu
Therapeutic Actions : Expectorant, astringent
Chemical Constituents : Columbin

Uses:
Leaves used in vitiligo and biliousness, kabham, cough.

Karuva Ilai
Common Tamil Name : Karuva Ilai
Synonyms : Lavanga pathiri
Botanical Name : Cinnamomum verum
Family : Lauracea
Suvai : Karppu
Thanmai : Veppam
Pirivu : Karppu
Therapeutic Actions : Astringent, Stimulant, carminative
Chemical Constituents : Eugenol, cinnamaledhyde, benzaldehyde, pinene, phellandrene, diterpene, safrole, methyleugenol borneol, geraniol, caryophyllene.

Uses:
Leaves used for anti inflammatory effect. It if also useful in mehasuram, kabha suram, asthma, vomiting.
**Nelli**

Common Tamil Name : Nelli  
Synonyms : Aamalaham, Aalaham, Aambal, amariham, thatthhari, thatththiri, koranham, merudhu pala, meethundhu.  
Botanical Name : Phyllanthus emblica  
Family : Euphorbiaceae  
Parts used : Leaves, flowers, bark, root, seeds and pod.  
Suvai : Pulippu, Thuvarppu and Inippu  
Thanmai : Thatpam  
Pirivu : Inippu  
Therapeutic Actions : Astringent, cooling diuretic  
Chemical Constituents : Trigalloyglucose, terchebin, covilagin, ellagic acid, phyllembic acid in the chief constituent, 3-0 gallated prodelphlinidin and procyanidin  

**Uses:**  
Leaves are useful in the inflammation useful in sinusities, vomiting, giddiness.

**Cow’s milk**

**Synonyms**

Sanskrit : Dughaksheera  
English : Milk  
Arab : Habit  
Persian : Sher  
Hindu  
Marathi : Dudh  
Kanada
Characters

Cow milk is an opaque white, (or) yellowish, white, emulsion, alkaline fluid, a little, more viscous, than water, taste is sweet, odour, faint and peculiar kept for a long time.

Specific gravity is between 1027 to 1037

under the microscope numerous minutes fat globules are seen floating in the form an emulsion.

The milk settles out in to 3 layers. The layer at the bottom of the vessel contains bacteria, cells and dirt. That at middle contains milk, plasma and small amount of fat (or) cream and a considerable number of bacteria and fat globules.

Composition of milk per 100ml

<table>
<thead>
<tr>
<th>Components</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salts</td>
<td>0.5%</td>
</tr>
<tr>
<td>Fat</td>
<td>3.5%</td>
</tr>
<tr>
<td>Non fat</td>
<td>8.0%</td>
</tr>
<tr>
<td>Casain</td>
<td>2.8%</td>
</tr>
<tr>
<td>Lactalumina</td>
<td>0.7%</td>
</tr>
<tr>
<td>Sugar</td>
<td>4.5%</td>
</tr>
<tr>
<td>Total solids</td>
<td>12.0%</td>
</tr>
<tr>
<td>Water</td>
<td>88.0%</td>
</tr>
</tbody>
</table>

Action

Milk is generally considered cooling, nutritive, strengthening and vitalizing also demulcent and emollient.
**BIO-CHEMICAL ANALYSIS**

**Preparation of the extract**

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

<table>
<thead>
<tr>
<th>S.no</th>
<th>Experiment</th>
<th>Observation</th>
<th>Inference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td><strong>Test for calcium</strong></td>
<td>2ml of the above prepared extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxalate solution is added to it.</td>
<td>No white precipitate is formed</td>
</tr>
<tr>
<td>2.</td>
<td><strong>Test for sulphate:</strong></td>
<td>2ml of the extract is added to 5% barium chloride solution.</td>
<td>No white precipitate is formed</td>
</tr>
<tr>
<td>3.</td>
<td><strong>Test for chloride</strong></td>
<td>The extract is treated with silver nitrate solution.</td>
<td>No white precipitate is formed</td>
</tr>
<tr>
<td>4.</td>
<td><strong>Test for carbonate</strong></td>
<td>The substance is treated with concentrated HCl.</td>
<td>No brisk effervescence is formed</td>
</tr>
<tr>
<td>5.</td>
<td><strong>Test for Starch</strong></td>
<td>The extract is added with weak iodine solution</td>
<td>No blue colour is formed</td>
</tr>
<tr>
<td></td>
<td>Test for iron</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>----------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td><strong>Ferric</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>The extract is treated with concentrated glacial acetic acid and potassium ferrocyanide.</td>
<td>No blue colour is formed</td>
<td>Absence of ferric iron</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Test of iron:</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.</td>
<td><strong>Ferrous:</strong></td>
</tr>
<tr>
<td></td>
<td>The extract is treated with concentrated Nitric acid and ammonium thio cyanate.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Test for phosphate</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.</td>
<td>The extract is treated with ammonium molybdate and concentrated nitric acid.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Test for albumin</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.</td>
<td>The extract is treated with Esbach’s reagent.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Test for Tannic acid</th>
</tr>
</thead>
<tbody>
<tr>
<td>10.</td>
<td>The extract is treated with ferric chloride.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Test for unsaturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.</td>
<td>Potassium permanganate solution is added to the extract.</td>
</tr>
<tr>
<td></td>
<td>Test for the reducing sugar</td>
</tr>
<tr>
<td>---</td>
<td>----------------------------</td>
</tr>
<tr>
<td>12.</td>
<td>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.</td>
</tr>
<tr>
<td></td>
<td>Yellow colour precipitate is formed</td>
</tr>
<tr>
<td></td>
<td>Indicates the presence of reducing sugar</td>
</tr>
</tbody>
</table>

The Bio chemical analysis showed that the pathiriver chooranam had ferrous Iron, unsaturated compound, Reducing sugar and amino acid.
AIM

To study the acute Anti-inflammatory effect of PATHIRIVER Chooranam.

PREPARATION OF THE TEST DRUG

1 gm of PATHIRIVER Chooranam was dissolved in 10 ml of water. Dose of 2 ml was given to each rat. This 2 ml contains 10mg of the test drug.

PROCEDURE

Nine healthy albino rats weighing 100 - 150 gm were taken and divided into three groups, each consisting of 3 rats.

First group was kept as control by giving distilled water of 2ml / 100mg of body weight. The second group was given Ibuprofen as dose of 20mg / 100g of body weight. The third group received the test drug PATHIRIVER Chooranam of 200 mg / kg body weight.

Before administration of test drug, the hind-paw volume of all rats was measured. This was done by dipping the hind-paw up to tibio - tarsal junction, in to a mercury plethysmography. While dipping the hind-paw, by pulling the syringe piston, the level of
mercury in the center small tube was made to coincide with red marking and reading was noted from the plethysmograph.

Soon after the measurement, the drugs were administrated orally. One hour later, a subcutaneous injection of 0.1ml of 1% w/v carrageen in water was made into planter surface of both hind-paw of each rat.

Three hours after carrageenin injection, the hind-paw volume was measured once again. The difference between the initial and final volume was calculated and compared.
The method is more suitable for studying the anti-inflammatory activity in acute inflammation.

### EFFECT OF PATHIRIVER CHOORANAM

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Group</th>
<th>Dose per 100gm of body weight</th>
<th>Initial Reading Value</th>
<th>Final Reading Value</th>
<th>Mean Difference</th>
<th>% of Inflammation</th>
<th>% of Inhibition</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Drug</td>
<td>100mg/1ml</td>
<td>0.80</td>
<td>1.0</td>
<td>0.20</td>
<td>25.0</td>
<td>75.0</td>
<td>SIGNIFICANT</td>
</tr>
<tr>
<td>2.</td>
<td>Standard Ibu Brufen</td>
<td>20 mg /1ml</td>
<td>0.80</td>
<td>0.85</td>
<td>0.05</td>
<td>6.25</td>
<td>93.75</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Control Water</td>
<td>1ml</td>
<td>0.65</td>
<td>1.5</td>
<td>0.85</td>
<td>100.0</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE**

The test drug has got *significant action* when compared with that of the standard drug.
AIM

To study the chronic anti-inflammatory activity of the drug, PATHIRIVER CHOORANAM in the rats by cotton pellets implantation (granuloma) methods.

PREPARATION OF THE TEST DRUG

1 gm of PATHIRIVER Chooranam was dissolved in 10 ml of water. A dose 1ml was given to each rat. This 1ml contains 10 mg of test drug.

PROCEDURE

Cotton pellets each weighing 10mg were prepared and sterilized in the autoclave for about one hour under 15 pounds atmospheric pressure.9 albino rats weighing between 150-200gms were selected and divided into 3 groups each containing 3 rats. Each rat was anaesthetized with ether and cotton pellets were implanted subcutaneously in the groin two on each side.

From the day of implantation first group was kept as control by giving distilled water 2 ml/100mg and second group of animals received PATHIRIVER Chooranam in a dose of 100mg / kg of body weight. The standard group of animals received paracetamol in a dose of 20mg / 100gm of body weight.

On the eighth- day the rats were sacrificed and the pellets were removed, dried to concordant weight and weighed. They were put in an incubator at 60 - 80°C and then the weight of the granulation tissue was determined separately.
## EFFECT OF PATHIRIVER CHOORANAM

<table>
<thead>
<tr>
<th>Serial No.</th>
<th>Name of Drugs / Group</th>
<th>Dose Per 100gm body weight</th>
<th>Pallet weight</th>
<th>Pallet weight of the Granuloma of drugs</th>
<th>Mean Differr</th>
<th>% of inflammation</th>
<th>% of inhibiton</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Pathiriver Chooranam</td>
<td>100mg/ml</td>
<td>10mg</td>
<td>95mg</td>
<td>-</td>
<td>38</td>
<td>62</td>
<td>Significant</td>
</tr>
<tr>
<td>2.</td>
<td>Ibu Brufen</td>
<td>20mg/1ml</td>
<td>10mg</td>
<td>56mg</td>
<td>-</td>
<td>22.4</td>
<td>77.6</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Water</td>
<td>1ml</td>
<td>10mg</td>
<td>250mg</td>
<td>-</td>
<td>100.00</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE**

The test drug has got **significant action** when compared with that of standard drug.
ANALGESIC ACTIVITY OF PATHIRIVER
CHOORANAM

AIM

To study the analgesic effect of PATHIRIVER Chooranam on albino rat by hot water bath method.

DRUG PREPARATION

1gm of PATHIRIVER Chooranam was dissolved in 10ml of distilled water and 1ml was given to each rat. This 1 ml contains 100 mg of PATHIRIVER Chooranam.

PROCEDURE

Three groups of healthy albino rats of both sexes were selected, each group having three rats. Each rat is weighing between 80-100 gm. The hot water bath is maintained at the temperature of 55°C.

The tail was dip into the bath, and time taken for each rat to remove its tail from the hot water bath was noted. The rat which takes more than 5 seconds for removal of its tail from hot water bath was excluded from the experiment.

First group was kept as control by giving distilled water of 1ml/100mg of body weight.

The second group was given paracetamol 20mg/100gm of body weight and kept as standard. Third group was given test drug PATHIRIVER Chooranam 200mg/kg of body weight.
Half-an-hour after drug administration the tail of each rat dipped in hot water bath, one by one and the time taken for each rat to remove its tail was noted. The whole experiment is repeated after another half-an-hour.

The results of control group, standard group and test drug group were tabulated and compared.

<table>
<thead>
<tr>
<th>Serial no.</th>
<th>Name of Drugs/groups</th>
<th>Dose/1 OOgm</th>
<th>Initial reading</th>
<th>After drug administration</th>
<th>Mean difference</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>PATHIRIVER Chooranam +</td>
<td>100mg/1ml</td>
<td>3.0</td>
<td>3.5 4.5 4.5</td>
<td>-</td>
<td>Moderate</td>
</tr>
<tr>
<td>2.</td>
<td>Paracetamol</td>
<td>20mg/1ml</td>
<td>2.5</td>
<td>4.0 5.0 6.5</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Control Water</td>
<td>1ml</td>
<td>2.5</td>
<td>2.5 2.5 3.0</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE**

The test drug has got Moderate action when compared with that of the standard drug.
HYPOGLYCEMIC EFFECT OF PATHIRIVER CHOORANAM IN RABBITS

AIM

To study the hypoglycemic effect of the test drug Pathiriver Chooranam.

PREPARATION OF THE TEST DRUG

1 gm of PATHIRIVER Chooranam was dissolved in 10 ml of distille water and 1 ml was given to each rabbit.

CONDITON OBSERVES DURING ADMINISTRATION

1) Three rabbits weighing around 500gm were selected.
2) Rabbits were made to fast more than 18hrs before and during Administration.
3) Rabbits were kept in clean condition.

EXPERIMENT

In the morning of the experiment before drug administrated blood samples were collected from the 3 rabbits at 0 hrs, for blood sugar analysis. The blood samples were collected from the marginal ear vein of the rabbit. Blood sugar analysis was done according to Folin-Wu method. There immediately the rabbits were treated with the drug as follows, one rabbit Glinbenclamide at a dose of 1mg/rabbit - The second rabbit received the test drug 2ml / rabbit 4- The third rabbit received distilled water 5ml / rabbit

Blood samples are collected at 1 Vz hour administration and the amount of blood sugar was estimated by Folin -Wu method. During the experiment period rabbits were not allowed to drink even water.
FOLIN-WU METHOD

0.2 ml of blood was taken in the test tube. In this add 3ml of distilled water, 0.4ml of 10% sodium tungstate and 0.4ml of 2/3 NH₂SO₄. Then the test tube was shaking well. Chocolate brown precipitate was formed. After centrifuging for 3 minutes protein free filtrate was obtained. 2ml of alkaline copper reagent was added. The mixture was taken in test tube and to this 2ml of alkaline copper reagent was added. The mixture was heated for 8 minutes and cooled. The 2ml of phosphomolybdic acid is added to this (blue colour formed.) and then the mixture was diluted to 25ml using distilled water. Then the mixture taken in a test tube. And the reading was taken using photoelectric calorimeter at 420n.

Blood sugar levels before and after the administration of the drug was estimated and percentage reduction in blood sugar level was calculated and tabulated. The different levels of 3 blood sugar and the reduction are shown in the table:

<table>
<thead>
<tr>
<th>s. No.</th>
<th>Groups / Drug</th>
<th>Dose/ 100 gm of body weight</th>
<th>Blood sugar</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Initial Value</td>
</tr>
<tr>
<td>1.</td>
<td>PATHIRIVER Chooranam</td>
<td>400mg / 10 ml</td>
<td>70</td>
</tr>
<tr>
<td>2.</td>
<td>Standard Dianil</td>
<td>1.25mg</td>
<td>75</td>
</tr>
<tr>
<td>3.</td>
<td>Control Water</td>
<td>10 ml</td>
<td>71</td>
</tr>
</tbody>
</table>

NOTE

The test drug PATHIRIVER CHOORANAM has got Moderate hypoglycemic activity.
ACUTE ANTI INFLAMMATORY STUDY OF MEDICATED VENNAI (EXTERNALLY)

AIM
To study the anti inflammatory (acute) activity of MEDICATED VENNAI.

PROCEDURE
Acute anti-inflammatory activity of Medicated Vennai was studied in healthy albino rats, weighing between 100-150 gms. For studying acute-inflammation rat hind-paw technique was used.

Six albino rats were selected and divided into two groups. Each containing three rats. To the first group distilled water was given and kept as control. Before the application of the drug, the hind-paw volumes of all rats were measured. This was done by dipping the hind-paw up to the tibio-tarsal junction in mercury plethysmography.

Subcutaneous injection of 0.1 ml of 1% carragenin (w/v) in water was made into plantar surface of both the hind-paw of each rat. To the test group Medicated Vennai was topically applied frequently over the inflamed surface in a thin layer. To the control group, no drug was applied over the inflamed surface. One-and-half the injection the hind-paw volume was measured once again. The difference between the initial and final volumes would show the amount of inflammation.

Taking the volume in the control group as 100% of inflammation or anti-inflammatory effect of the group is calculated. Tabulation of the results observed.
<table>
<thead>
<tr>
<th>S NO.</th>
<th>Name of Drug/Groups</th>
<th>Dose /100gm body weight</th>
<th>Initial reading average</th>
<th>Final reading average</th>
<th>Mean difference</th>
<th>Percentage inflammation</th>
<th>Percentage inhibition</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>MEDICATED VENNAI</td>
<td><strong>External</strong></td>
<td>0.7</td>
<td>1.3</td>
<td>0.65</td>
<td>81.25</td>
<td>18.25</td>
<td>Mild</td>
</tr>
<tr>
<td>2.</td>
<td>Ibuprofen</td>
<td><strong>20mg/1ml</strong></td>
<td>0.80</td>
<td>0.85</td>
<td>0.05</td>
<td>6.25</td>
<td>93.75</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Water</td>
<td><strong>1ml</strong></td>
<td>0.65</td>
<td>1.5</td>
<td>0.85</td>
<td>100.0</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE**

The test drug has got Mild anti-inflammatory activity.
Govt. Siddha Medical College & Hospital
Palayamkottai
Department of Post Graduate Maruthuvam
Branch – I
IP Case Sheet

Ward : Nationality :
I.p.no. : Religion :
Bed no. : Date of Admission :
Name : Date of Discharge :
Age /sex : Diagnosis :
Occupation : Medical officer :
Income :
Permanent address :
Local address :

Complaints & Duration :

History of present illness :

History of Past illness :

Family History :

Personal History :

Habits :
Veg / Non-veg/ smoker / Alcoholic / Tobacco chewer
History of exposure to extra marital contacts
General Examination
1) General Attitude 10) Venous pulse
2) Decubitus position 11) Pedal Oedema
3) Built 12) Nerve thickening
4) Temperature 13) Jaundice
5) Pulse rate 14) Anaemia
6) Heart rate 15) Cyanosis
7) Blood Pressure 16) Clubbing
8) J.V.P. 17) Engorged veins
9) Lymph adenopathy

SIDDHA SYSTEM EXAMINATIONS

NILAM
Kurinji
Mullai
Marutham
Neithal
Palai

PARUVA KALAM
Kar
Koothir
Munpani
Pinpani
Elavenil
Muthuvenil

GNANAINTHIRIAM
Mei
Vai
Kan
Mukku
Sevi

KANMAINTHIRAM
Kai
Kal
Vai
Eruvai Karuvai

KOSAM
Annamayakosam
Pranamayakosam
Manomayakosam
Ganamayakosam
Anandhamayakosam
MUKKUTRA NILAI

VATHAM
Pirananan
Abanan
Viyanan
Udhanan
Samanan
Naagan
Koorman
Kirugaran
Dhevathamthan
Thananjeyan

THEGI
Vatham
Pitham
Kabha
Thontham

MUKKUNAM
Sathuvam
Rajoatham
Thamogunam

PITHAM
Anar pitham
Ranjaga pitham
Saadhaga pitham
Alosaga pitham
Pirasaga pitham

KABAM
Avalambagam
Kilethagam
Pothagam
Tharpagam
Sandhigam

UDALTHATHUKKAL
Saaram
Senneer
Oon
Kozhuppu
Enbu
Moolai
Sukkilam / Suronidham

ENVAGAI THERVUGAL
Naadi
Sparisam
Naa
Niram
Mozhi
Vizhi
Malam
Moothiram
MODERN ASPECT

Clinical examination

1) Handedness.

2) Conscious level, orientation and degree of co-operation.

3) Meningeal sign

4) Speech Defect - general description Detailed analysis at the end of examination.

5) Cranial Nerves

6) The Motor System (arms, trunk, legs)
   Position of limbs and deformities
   Muscle bulk and presence of wasting
   Presence or absence of fasciculation
   Involuntary movements
   Muscle tone
   Muscle power with detailed analysis of any weakness.

7) Sensory system
   Pain sense
   Temperature sense
   Sense of light touch
   Position sense and sense of passive movement
   Vibration sense
   Sterognosis and graphaesthesia
   Two - point Discrimination.

8) The Reflexes

a) Superficial
   1) Corneal
   2) Conjunctiva!
   3) Abdominal
   4) Cremastric
   5) Plantar
b) **Deep tendon reflexes**
   1) Jaw jerk
   2) Biceps
   3) Triceps
   4) Supinator
   5) Knee jerk
   6) Ankle jerk

9) **Co-ordination**
   1) Finger nose test
   2) Finger to finger test
   3) Heel Knee test
   4) Romberg's sign
   5) Dis diadokokinesia

10) **Gait**

11) **Test for Autonomic function**
   1) Heart rate
   2) Pulse rate
   3) Standing test for orthostatic Hypostension
   4) Deep breath test
   5) Hand grip test

12) **Spine**
   1) Inspection
   2) Palpation
   3) Mobility

13) **Lower limb** - Straight leg raising test
## Investigation

### Blood
- Total Count
- Differential Count
- ESR
- Hb
- Complete haemogram
- Peripheral smear for cytology
- Haemoparasite
- Sugar
- Urea
- Creatinine
- Cholesterol
- Liver function test
- Serum protein

### Skin Clipping
- AFB

### Motion
- Ova
- Cyst
- Occult Blood
- Albumin
- Sugar
- Deposit

### Nerve Conduction Study

### Differential Diagnosis

### Case Summary

### Diagnosis

### Treatment

### Prognosis

### Diet

### Advices
Govt. Siddha Medical College & Hospital  
Palayamkottai  
Department of Post Graduate Maruthuvam  
Branch – I  

Discharge Summary

<table>
<thead>
<tr>
<th>S.No</th>
<th>Clinical Features</th>
<th>During Admission</th>
<th>During Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Gnawing sensation over legs and hands</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Numbness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Burning Sensation all over the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Polypagia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Giddiness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Paresthesia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Pricking pain with pin and needle sensation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Restricted movements</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Clinical features if any</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Place :
Date :
Govt. Siddha Medical College & Hospital
Palayamkottai
Department of Post Graduate Maruthuvam
Branch – I

OP Case Sheet

OP No : Treatment starting date :
Name : End of Treatment date :
Age / Sex : No. of days treated :
Occupation : Diagnosis :
Income : Results :
Address : Medical Officer :

Complaints and Duration:

General Examination

Temperature : Anaemia :
Blood Pressure : Jaundice :
Heart rate : Cyanosis :
Pulse rate : Clubbing :
Respiratory rate : Others if any :

In siddha aspects

Nilam : Dhehi :
Mukkunam : Ezhu Udal Kattugal :
Imporikal : Mukkutram :
Kanmendhriyam : Envagai Thervugal :
Kosam : Neerkuri :
Paruvakalam : Neikuri :
Mukkunam :
**Examination of Sensory system**

- Pain sense
- Temperature sense
- Sense of light touch
- Position sense and sense of passive movement
- Vibration sense
- Sterognosis and graphaesthesia
- Two - point Discrimination

**Ankle joint Reflex**

**Investigation**

**Blood**

- Total Count : Sugar : 
- Differential Count : Urea : 
- ESR : Creatinine : 
- Hb : Cholesterol : 
- Complete haemogram : Liver function test : 

**Motion**

**Urine**

- Peripheral smear for cytology
- Haemoparasite

**Skin Clipping**

- Serum protein
- AFB

- Ova
- Cyst
- Occult

- Albumin
- Sugar
- Deposit

**Nerve Conduction Study**
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