A STUDY ON

VATHA PANDU

Dissertation submitted to

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DEPARTMENT OF POTHU MARUTHUVAM

GOVERNMENT SIDDHA MEDICAL COLLEGE

PALAYAMKOTTAI – 627 002.

APRIL – 2013
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ACKNOWLEDGEMENT
ACKNOWLEDGEMENT

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INTRODUCTION

The Siddha Medicine is one of the oldest medical systems known to mankind. Contemporary Tamizh literature mentions the origin of the medical system from Southern India in the state Tamil Nadu, as part of the trio Indian medicines - ayurveda, siddha and unani. Reported to have surfaced more than 2500 years ago, the Siddha system of medicine is considered one of the most antiquated traditional medical systems.

"Siddhargal” or Siddhas are the great scientists of ancient days mainly from Southern India who laid foundation for this system of medication. Siddhars are spiritual adepts who possessed the ashta siddhis, or the eight supernatural powers. Sage Agathiyar is considered the guru of all Sidhars, and the Siddha system is believed to have been handed over to him by Lord Muruga, son of the Hindu God - Lord Shiva and Goddess Parvathi. So, are the siddhars the followers of Lord Shiva (Shaivaites). Agathiyar is the first Siddhar, and his disciples and other siddhars of other schools contributed thousands of texts on Siddha, including medicine, and form the propounders of the system in this world.

This disease Vathapandu correlate with Anaemia in allopathic entity. The word anaemia actually means “Lack of blood” in old Greek. This disease affects one of five people in the developed world and affects
almost everyone who suffers from malnutrition, Chronic bleeding disease. It is a common disorder in women of reproductive age and those who are pregnant as well as infants.

This disease according to Siddha concept is caused due to disturbances in blood which is one of the seven dhatus in our body. Great Siddhar Yugi muni quotes about the seven humours as follows.

\[
\text{The seven humours are formed from panchaboothas by pancheekaranam. Thirumoolar says this as}
\]

- 2086
This blood is subjected to various researches since ancient days. It was also believed that the man’s life is decided by blood in those days. Blood acts as mirror that reflects any pathological changes in the body. According to pattinathar there are crores of uyir anukkal in blood. He says

Recent researches also justifies this statement. In our body, blood represents “Appu” Kooru. Sathaga nadi says this as follows.
Vatha pandu, one of the type of pandu which is caused by disturbances in blood affects many crores of people in a developing country like India. Environmental pollution, lack of a well balanced diet, infections and poisonous substances etc. are all promoters of this disease.

Though this disease can be diagnosed and treated easily if left untreated in its advanced stage it may affect important internal vital organs like heart, kidney digestive system etc.

For the above mentioned reasons I have selected to study this disease with Thiratcha Choornam. (Agasthiar Pari Pooranam 400).
ANEMIA

Introduction

Anemia is defined as a hemoglobin concentration in blood below the lower limit of the normal range for the age and sex of the individual. In adults, the lower extreme of the normal hemoglobin is taken as 13.0 g / dl for males and 11.5 g / dl for females. Newborn infants, have higher hemoglobin level and, therefore, 15g / dl is taken as the lower limit at birth, whereas at 3 months the lower level is 9.5 g / dl. Although hemoglobin value is employed as the major parameter for determining whether or not anemia is present, the red cell counts, hematocrit (PCV) and absolute values (MCV, MCH and MCHC) provide alternate means of assessing anemia.

Generally, anemia occurs because of:

1. Decreased production of RBC,
2. Increased destruction of RBC or
3. Excess loss of blood from the body.

All these incidents are caused either by inherited disorders or environmental influences such as nutritional problem, infection and exposure to drugs or toxins.
Prevalence of Anemia

Anemia is one of the most common health problems in India. The problem is much more in rural than the urban areas. The high-risk groups for anemia are pregnant and lactating females and children. Prevalence in this subgroup has been found to vary from 50-90% in different parts of India. Almost all interventions at national and local level have focussed predominantly on these groups. Reliable data on the prevalence of anemia in adult population (non-pregnant females and adult males) is not available. During an epidemiological study on prevalence of hypertension in rural population, we studied prevalence of anemia among adult individuals.
AIM AND OBJECTIVES

The aim of the work is to evaluate the clinical efficacy of the trial drug (Thiratcha Choornam) in vatha pandu, one of the types of Pandu.

The Vatha Pandu to be correlated with clinical condition called “Anaemia”.

I has chosen Vatha Pandu for the dissertation study because It affects all age from children to old age.

The disease is diagnosed in (1) Clinical methods of Siddha Medicine and (2) Relevant modern diagnostic methods.

The selection of cases is from the out-patient department and in-patient department of Government Siddha Medical College.

This dynamic approach deals very much in detail, with the work done with devotion and dedication to bring out following objectives.

1. To collect through study of various literatures dealing with definition, classification, sign and symptoms, naadi, diagnosis, diets.

2. To expose the reliability of siddhar’s diagnostic principles.

3. To know the clinical efficacy of the trial drug.

4. Bio chemical investigations and pharmacological action of the medicine tried on this disease.
5. To have an idea of the incidence of the disease with Sex, age, habitat, occupation, income, social status, diet etc.
ABSTRACT

Since being the commonest disease in the society, number of suffers increasing day by day, I has chosen the disease “VATHA PANDU” for his dissertation work. The evidence of the disease VATHA PANDU is derived from “Yugi Vaidhya Chinthamani – 800”. The signs and symptoms mentioned in Yugi Vaidhya Chinthamani book closely resembles with that of Anemia in modern medicine.

20 Inpatients and 20 Out patients of both sexes were selected. They were administered with the trial medicine “Thiratcha Choornam” 2 gms BD with Ghee during the whole study period. Agathiyar Paripooranam (page no 57, 58) (Rathina Nayagar & Sons, Chennai – 79, 1995).

The trial medicine was subjected to biochemical and pharmacological analysis.

At the end of the trial study, the majority of the cases showed good results.
REVIEW OF SIDDHA LITERATURE

SIDDHA ASPECTS

Siddha system is said to have originated from LORD SIVA AGASTHIYAR is the first disciple of Siva, nobody deny the fact that Agasthiyar was Guru and pioneer to propagate this siddha system of medicine.

The siddha system of medicine is based on the Tridhosha theory according to which the human system is medicated by three vitiating elements (or) Thatus. The functional units of living beings namely vatham pitham and kabam. Diseases are produced by disequilibrium of these three vitiating elements, which may be due to various causes like dietetic regimen, life style patterns and the ecological imbalance etc.
The disease pandu has its historical importance. The word pandu has been derived from HINDU EPIC “MAHABHARATHAM” where the father of five heroes “Pancha Pandavar” is pandu. It is said that this man, when born was very pale and anaemic and hence this condition was named after him as pandu.

**Synonyms of Pandu**

Venmai noi, Velluppu noi, Ven pandam.

**Iyal (Definition)**

Pandu is the disease of Raktha thathu characterized by pallor of skin mail beds and conjunctivae.

The great siddhar Agathiar define pandu in the following verses.

அபிளகுரு வேகலாம காசுகும்பங்கணம

நூற்றிலிரு மாசாமல் நீனின்

புனிதார் புணிதில் புணிதம் விமானம் மாசாமலேண்ம

வேளகுருகு குளம்

- அவர்லிங்கம் கொள்ளி கருப்பம்

சாக்கிஃ சித்த குருகிஞ்

சுந்தரா விறு நீன் காசுகும்பங்க

- அவர்லிங்கம் கொள்ளகல்
Formation of Raktham according to Siddhars

As the digestion takes place in the body “Rasa thathu” is formed on the very first day on the second day “Raktham” is formed from the Rasa thathu. And from Raktham masmisam is formed, from this kozhuppu is formed, from kozhuppu enbu is formed, from enbu moolai is formed, from moolai sukilam is formed on the 3\textsuperscript{rd}, 4\textsuperscript{th}, 5\textsuperscript{th}, 6\textsuperscript{th} and 7\textsuperscript{th} day respectively.

It is to be noted that the nutrients absorbed after digestion are responsible for the metabolic functions and the formation of blood.

Noi Varum Vazhi (Aetiology)

a) According to YUGIMUNI, AETIOLOGY in defined as follows.

Noi Varum Vazhi (Aetiology)

- அதிகம் குறுகிறது ரசாக் வேலா
- அதிகம் உயிரிலிருந்து மண்டுறை நெடுந்து பிரித்தலை பாதுப்படுத்தலை
- பிரித்தலை மூக்கவில் விளக்கப்படும்
- பிரித்தலை குறுகிறும் வீடு அசெக்கடுமை
- பிரித்தலை முக்கள்வில் வீடு பிரித்தலை
- பிரித்தலை குறுகிறும் வீடு பிரித்தலை
- பிரித்தலை வெடும் வகையையும் வெடுமை
- பிரித்தலை வெடும்மாறு மாறுபடும்
- பிரித்தலை வெடும் மாறு மாறிகளிடம் மாறில் மனைவி.

பாரா பக்கத்தில் விளக்கம்

பாரா பக்கத்தில் விளக்கம் முதுமையாகும்

12
Yugi says that the faulty way of living brings about this pandu condition while listing out the wrong life styles, he says about excessive in take of food stuffs with salt and sour items, staying in hot conditions excessive in take of pan and nap etc.

b) **Cause of Pandu from Thanvanthiri Vaidyam**

- மாட்டை மக்களும் பாதுகாப்பு பின்பற்றியனவும் காட்டுக்கொண்ட தென்மாண்
- மாட்டை மக்களும் பின்பற்றியது நிகழ்வையும் பாதுகாப்பு
- பிறந்துள்ள தன்மாண்டு உடன்பிடிக்கை பாதுகாப்பு
- மாட்டை மக்களும் அணுவமும் பாதுகாப்பு
- மாட்டை மக்களும் பின்பற்றியது மாட்டை மக்களும்
- பிறந்துள்ள தன்மாண்டு உடன்பிடிக்கை 
- மாட்டை மக்களும் அணுவமும் பாதுகாப்பு
- மாட்டை மக்களும் பின்பற்றியது மாட்டை மக்களும்

- சுற்றுக்கோள் கூறினாக்கியது
Imbalance between the three thathus perversion of appetite in the form of geophagia excessive heat accumulation due to altered abana, sorrow, psycho social factors and also closely accompanied with several transmitted disease.

**Pandu – Kanma Noi**

c) IN AGASTHIAR PARI POORNAM – 400 pandu is considered to be the one among the kanma noi.

The same thought was again confirmed in Agasthiar Vaidyam Mun – 80.
d) AGASTHIAR GUNAVAGADAM says

"wrong food habits and injudicious diet, menorrhagia in females, chronic diarrhea, blood loss due to various aetiology, stress and strain are some of the causes of pandu."

e) Due to worm infestations

The above stanza from Gurunadi illustrate the worm infestation.
Pathological loss of blood may occur due to various factors. One among them is worm infestation leading to chronic blood loss from the intestinal region. Hence heavy infestations are bound to cause anaemia.

f) Another concept from THERAYAR VAGADAM denotes the causative factor of pandu noi.

Therayar Vagadam says, thorn of fish, paddy ban, bone, stones, old rice, hairs in food are the dietetic causes.

Secondly severe constipation, drinking of polluted water sleeping in an abnormal posture then over indulgence with ladies are all bring out veluppu noi.
g) In AGATHIAR VAGADAM the causes of pandu noi is mentioned as follows.

By which the irresponsible and disobedience parents, unhospitality to people, speaking lie, gets anger on others stimulates pithadhosha which results in pandu noi.

h) In THERAYAR MAHA KARISAL Aetiology of pandu is defined as follows.
NOI ENN (CLASSIFICATION)

YUGI discuss this disease under five pandu noi

THANVANTHIRI classified into five types of pandu noi

AGASTHIYAR classified into five types of pandu noi
Our Siddhars classified this pandu noi under three dhoshos and have given names according to their concept. The signs and symptoms of classified disorders are almost identical in the description of the disease.

**Classification of pandu noi**

<table>
<thead>
<tr>
<th>Yugi Chinthamani</th>
<th>Thanvanthiri Vaithiyam</th>
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<tbody>
<tr>
<td>Vatha pandu</td>
<td>Vatha pandu</td>
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<td>Pitha pandu</td>
<td>Pitha pandu</td>
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<tr>
<td>Kapha pandu</td>
<td>Kapha pandu</td>
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<tr>
<td>Mukkutra pandu</td>
<td>Mukkutra pandu</td>
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<tr>
<td>Visha pandu</td>
<td>Pitha Vatha pandu</td>
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<td></td>
<td>Pitha kappa pandu</td>
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<td>Sanni patha pandu</td>
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<thead>
<tr>
<th>Vaidyasara Sankirakam</th>
<th>Agasthiyar Gunavagadam</th>
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<tr>
<td>Vatha pandu</td>
<td>Vatha pandu</td>
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<td>Pitha pandu</td>
<td>Pitha pandu</td>
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<tr>
<td>Moola pandu</td>
<td>Moola pandu</td>
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<tr>
<td>Moola pitha pandu</td>
<td>Nanju pandu</td>
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<tr>
<td>Visha pandu</td>
<td>Miruthiya pandu</td>
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<td>T.V.Samba Sivam</td>
<td>Roga Nirnaya</td>
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<tr>
<td>Pillai Dictionary</td>
<td>Saram</td>
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<td>Vatha pandu</td>
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<td>Pitha pandu</td>
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<td>Kapha pandu</td>
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<td>Mukkutra pandu</td>
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<td>Oothu pandu</td>
<td>Visha pandu</td>
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<td>Neer pandu</td>
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<td>Eri pandu</td>
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<tr>
<td>Visha pandu</td>
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Various Siddha literature enumerate the types of pandu in different classification but all of these consists Vatha pandu as one of the variety.

Noi Kurikunangal (Clinical features)

a) Premonitory Symptoms

Pandu patients exhibit the following symptoms from their initial stage of development itself. The patient experience insidious onset of fatigability lassitude, difficult in breathing on exertion, diminished vision, faintness, palpitation, pallor-ness of the skin.

b) Signs and Symptoms of pandu

According to Gunavagadam

Pallorness of the face, eyes, lips, tongue, pallor of the skin, pallor of the nail, oedema of the feet due to CCF.
The features of Vatha pandu are parallel with that of Yugimuni’s features.

Vatha pandu as told in Yugimuni’s Vaidya Chinthamani’s

In Association with general signs and symptoms of pandu due to vatha vitiation the below mentioned signs and symptoms are also present. They are abdominal discomfort, loss of appetite, increased thirst, shivering of the body with generalised oedema.
**Roga Nirnaya Saram**

Pain all over the body, tremor, discolouration of nail, conjunctiva.

**Mukkutra Verupadugal (Pathology of vatha pandu)**

Our Siddha medicine is based upon thridoshic theory. Due to intrinsic and extrinsic factor, the pitha in the body gets altered so digestion affected by the affected pitha thathu. In the blood stream the ranjagam which is the branch of pitha which gives colour to the blood is unable to entertain these ill digested and improperly assimilated nutrients with Raktha thathu. So a defect manifest in the haemopoisis. So vatha pandu produces pallor of the tongue, nailbeds, conjunctiva, and skin etc.

The detoriation of thathu with pitha and vatha, produces oedema. These altered tridosha finally altered the normal structure and functions of the seven thathus.

Agasthiar in his vadha kaviyam say that for upsetting and deranging all the functions of vatham and pitham is mainly due to the dietetic factors.

**PINIYARI MURAIMEI (Diagnosis)**

Piniyari muraimai is the method of diagnosing the disease affecting the man. It is based upon three main principles.
They are
1. Poriyalarithal
2. Pulanalarithal
3. Vinathal

Pori is considered as the five senses of perception namely Nose, Tongue, Eye, Skin and Ear. While pulan are five objects of senses. They are Smell, Taste, Sight, Sensation and Sound. Vinathal is obtaining the information regarding the history of the disease its clinical features etc from the patient or his immediate relatives who are taking care of him, when the patient is not in a position to speak or if the patient is a child.

The above principles correspond to the methodology of inspection, palpation and interrogation of modern medicine
Alavai

Alavai is a parameter through which one can assess the real properties merits and demerits of the things using the five senses as the instruments.

Alavai is very much useful in diagnosing vatha pandu as follow.

1. **Kandal (Observation)**

   By observing the palloriness of skin, conjunctiva, nail beds, mucous membrane of lip and tongue.

2. **Karuthal (Inference)**

   Patient complaints of fatigue, palpitation, dyspnoea on exertion the physician can have a clue for the diagnosis of vath a pandu.

3. **Urai (Authority)**

   In addition to the above manifestation the kabha, kabha vatham, kabha pitham, vatha kabham felt in the vatha pandu patient further confirms the diagnosis.

   This has been quoted in

   காப்பறம் காப்பறும் காப்பவதம் காப்பத்தான்
   பாசுகற் காண்பரிசையம் பிள்ளைய காண்பர் வந்தும்
   குறிண்டு பந்தமையார் நிலாரஞ்சல் பலரும்
   காப்பறம் காப்பறும் காப்பவதம் காப்பத்தான்

   - அகரங்கக் காம்.
உணவுணர்வு செய்யும் போது தின்பகுதியில்
தேர்வு செய்ய முடியாது பின்னர்த்தவுல்லார்
வெளியிலே சின்னங்கள் உள்ளறை காண்பது
தேர்வு செய்ய முடியாது மாற்றால் காண்பது
உணவுணர்வு உருவாக்கவும் செய்யும் நிகழ்வு
தேர்வு தேர்வு மாற்றும் மற்றும் உறுப்பு செய்யும்

- சக்தியுடலில்

உணவுணர்வு உருவாக்கிய பேருந்துச் சேவை,

மணிக்கோட்டுகளில் முதல் மையம்

கல்லூற்று உருவாக்கும் நிகழ்வு

பிறங்காலத்தில் முழு நிலை நிகழ்வு

மராத்திய மாற்று தினம் நிலையில் நிகழ்வு

கல்லூற்று கடந்து வேறுபடும் மாற்றம்

துறவுப் பாதுகாப்பு நடைபெறும்

- சக்தியுடலில்

மணிக்கோட்டு உருவாக்கிய நிகழ்வு

செய்யலாம் முன்னெச்சரித்திய நிகழ்வை

மணிக்கோட்டு நிலையில் முதல் மையம்

செய்யலாம் செய்யலாம் மாற்றால் துறவு

மணிக்கோட்டு நிலையில் மாற்றம் நிகழ்வு

- சக்தியுடலில்

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Enn Vagai Thervugal

Diagnosis is confirmed by Enn vagai thervugal, which are named as follows.

Naa,
Niram,
Mozhi,
Vizhi,
Malam,
Moothiram,
Naadi

Sparism

This is evident from the version of Theran one of the great Siddhars.

In Agasthiyar vaidhya vallathi – 600 Ennvagai thervugal has been mentioned as Attavitha paritchai
அகஷியார் வாத்தா வால்தா 600

Agasthiyar Vaidhya

Vallathi – 600
Naa (Tongue)

The colour change according to vatha pitha and kabha, mukkutra disease, dyness or wet, coated or not, excessive salivation, redness, ulceration, pallor, yellowish discolouration of the tissues, neoplastic growth, conditions of teeth its colours, condition of the gums, taste, loss of appreciation, speech, deviation of the mouth angle, movements of the tongue can be made out.

In vatha pandu noi, the tongue was pallor moist, and glossy and the sense of taste would be altered.

Niram (Colour of the skin)

Colour indicating vatha, pitha, kabha and three dhoshas, yellow or pallor or redness of the skin, any bluish discolouration.

In vatha pandu noi, the colour of the skin, tongue, nailbeds and conjunctiva were very pale.

Mozhi (voice)

Clarity of speech or any disturbance, loud voice, slurring, crying, talk induced by hallucination, breathlessness can be made out.

In vatha pandu noi patients were spoken in diminished voice. Some were in breathlessness.
Vizhi (Eyes)

Any abnormal colour change indicating the three doshas derangements, pallor, excessive lacrimation, sub conjunctival bleeding, drooping of eye lids, visual disturbance, any specific diseases of the eyes.

In all vatha pandu noi patients pallor of the conjunctiva was observed.

Malam (Faeces)

Quantity, semisolid, colour, odour, froth, abnormal consistency indicating indigestion, frequency, constipation, sulphur smell, foul smell etc. are observed.

In vatha pandu noi, constipation is usually encountered.

Moothiram (Urine)

Quantity, colour, Odur, froth, frequency, deposits, presence of abnormal constituents such as sugar, protein etc.

Examination contains two phases Neerkuri (The colour indication Neikuri (The oil indication)
Neerkuri

The patient must take well cooked food on the previous day. The intake must be proportionate to the degree of his appetite. Food should be taken at appropriate time. He must have sound sleep on that night. The urine is collected on the dawn of the next day in a glass container and closed immediately to prevent evaporation. The specimen must be examined within 1½ hours. This procedure should be followed strictly in order to get accurate reading of Neerkuri and Neikuri.

In vatha pandu not straw yellow coloured urine was observed.

Neikuri
The specimen is kept open in a glass dish being exposed well to sunlight. But it should not be disturbed by the movement of the wind. Then add one drop of gingely oil by a glass rod. Observe keenly the position and spreading of the oil drop dropped on the surface of the urine.

In vatha pandu not snake like spreading was observed.

1) **Sparism (palpation)**

The following points are elicited by sparism, temperature of the skin any abnormal growth, hyper sensitiveness dryness of skin ulcers, oedema, etc.

In vatha pandu noi the sparism was warm.

**Naadi**

Naadi is the vitiating element of the body which are vatham, pitham and kabam. Naadi is the vital force. The examination of naadi has been recognised as one of the principal means of diagnosis and prognosis of the disease from time immemorial. The great Siddhar Thiruvaluvar says,

Any change in the three doshas is best diagnosed by feeling the Naadi.
At the lower end of radius, Naadi is felt as vatham pitham and kabam with the tips of index, middle and ring fingers respectively.

They are formed by the combination of

- **Edakali** + **Abanan** - vatham
- **Pinkalai** + **Piranann** - pitham
- **Suzhumunai** + **Samanan** - kabam

The ratio between vatham, pitham, kabam, is $1:½:¼$ respectively.
On the basis of the examination of the senses and on the basis of eight special examination and interrogation all the details of the disease factor are collected and their final diagnosis is confirmed with those finding made on naadi paritchai.

In vatha pandu noi the following types of naadi are seen commonly.

1. Kabam
2. Kaba vatham
3. Kaba pitham
4. Vatha kabam.

Besides Ennvagai thervugal, a disease can also be diagnosed by means of other methods namely Thinaigal, Paruvakalanga, Uyirthathukkal, udal thathukkal. Hence a through knowledge about the disease can be attained systematically and properly in siddha system of medicine. A combination of all these diagnostic criteria are very helpful to attain a proper
diagnosis with a full complete entity based on basic principles of Siddha science.

**Thinaigal**

Nilam is classified into five types, depending on the surrounding vegetation, landscape and ecological stage, study of five places is very much necessary as some diseases are common in particular land.

1. Kuringi : Mountain and its surroundings liver diseases and flurosis are common
4. Neithal : Sea and seashore liver disease occur in combination with other diseases.
5. Palai : Desert and its surroundings vatha, pitha and kaba diseases occur.

In all five types of lands “Vatha Pandu” is probable of occurrence
Paruvakalangal

Siddhars have classified a year into six seasons each consisting two months. They are

1. Kaar kalam : Avani and Purattasi
2. Koothir kalam : Iyppasi and Karthigai
3. Mun pani kalam : Margazhi and Thai
4. Pinpani kalam : Maasi and Panguni
5. Elavenir kalam : Chithirai and Vaikasi

Some of the diseases are commonly prevalent during a particular paruvakalam and a study of it will be of much use for diagnosis.

In all six seasons “vatha pandu” is probable of occurrence.

Udal Vanmai (Immunity)

Siddhars classified this udal vanmai into three types. They are

1. Iyarkai Vanmai
2. Seyarkai Vanmai
3. Kala Vanmai
Iyarkai Vanmai

Natural immunity of the body caused by mukkutram right from birth onwards.

Seyarkai Vanmai

Improving the health by intake of nutritious food materials, activities and medicines.

Kala Vanmai

Development of immunity according to age and environment.

When udal vanmai is affected, there may be possibility of “vatha pandu”.

Uyir thathukkal

Three initiating elements are the functional units of the body, namely vatham, pitham and kabam.

Vatham

Vatham is the kinetic energy, which influences all motions.

Vatham is located in the abanan, motion, edakalai, spermatic cord, pelvic bone skin, nerves, joints, hair follicles and muscles, bones air and thigh. It is classified into ten types. The are
1. **Piran Nan (Uyirkkal)**
   This control knowledge, mind and five objects of senses useful for breathing and digestion.

2. **Abanan (Keezhnokkukal)**
   It expels stools, urine, semen and foetus.

3. **Udhanan (Melnokkukal)**
   It responsible for all upward visceral movements such as vomiting, eructation, nausea.

4. **Viyanan (Paravukal)**
   It is used to feel all type of sensations. It carries nutrients to all over the body. Flexes and extends the movable joints.

5. **Samanan (Nadukkal)**
   It prevents and exhibits the over activity of other vayus.

6. **Nagan**
   It controls the blinking of eyes.

7. **Koorman**
   Responsible for vision, yawning and lacrimation.

8. **Kirukaran**
   Responsible for salivation, nasal secretion and appetite.
9. Thevathan

Sleep, fatigue, tiredness, anger are caused by Thevathan.

10. Thananjeyan

It produces oedema of the body and hyperacusis in the ears. It escapes on the 3\textsuperscript{rd} day after death by bursting the cranium.

In case of “VATHA PANDU”

1. Abanan : (expells stools)
2. Viyanan : (It carries nutrients all over the body)
3. Udhanam : (Vomiting)
4. Samanan : (Due to other vayus are affected)
5. Kirukaran : (Appetite affected)
6. Thevathan : (Tiredness and sleep affected)

Above the vauyus are mainly affected other vauyus are not affected commonly.

Pitham

Located in urinary bladder, heart, head, pingalai, umbilicus, abdomen, piranan, stomach, blood, sweat, skin and eye.

Pitham is classified into five types. They are
1. Anal Pitham : This gives appetite and helps for digestion.

2. Ranjaga Pitham : It gives colour to the blood.

3. Pirasaga Pitham : It gives complexion to the skin.

4. Alosaga Pitham : It is responsible for vision.

5. Sathaga Pitham : It is responsible for the action what we think.

In the case of VATHA PANDU

1. Anal Pitham : (Appetite and digestion)

2. Ranjagam : (Colour to the blood)

3. Pirasagam : (Complexion of the skin)

4. Sathaga Pitham : (Unable to carryout regular works properly)

These are mainly affected.

Kabam

Located in samanan, semen, head, tongue, fat, bone marrow, blood, nose, chest, nerves, bones, brain, large intestine, eye, stomach and pancreas.

Kabam is classified into five types. They are

1. Avalambagam

Heart is the center for avalambagam. It controls all the other kabams.
2. Kilethagam

Stomach is the center for kilethagam. It gives moisture and helps in the digestion.

3. Pothagam

Tongue is the center for pothagam. It is responsible for the sense of taste.

4. Tharpagam

Head is the center for Tharpagam. It gives cooling to the eyes.

5. Santhigam

It lies in the joints and is responsible for the action of joints. In the case of “vatha pandu”

<table>
<thead>
<tr>
<th>Kabam</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kilethagam</td>
<td>(Digestion)</td>
</tr>
<tr>
<td>Pothagam</td>
<td>(Taste sensation)</td>
</tr>
<tr>
<td>Avalambagam</td>
<td>(It controls all other kabam)</td>
</tr>
</tbody>
</table>

These are commonly affected.

Ezhu udal kattugal

These are seven basic principles, which constitute the entire body.

1. Saram

Strengthens the body and mind.
2. **Senneer**

   It is responsible for knowledge, strength, boldness and healthy complexion.

3. **Oon**

   It gives structure and shape of the body and is responsible for the movements of the body.

4. **Kozhuppu**

   It lubricates the joints and facilitates their functions.

5. **Enbu**

   It protects all the internal organs and gives the structure to the body.

6. **Moolai**

   Present inside the core of the bones and gives strength.

7. **Sukkilam / Suronitham**

   Meant for reproduction (male and female respectively)

In the case of “vatha pandu”

1. Saram - Tiredness.
2. Senneer - Reduced haemoglobin
3. Oon - Movements affected
4. Kozhuppu - Functions affected.

These are mainly affected other udal kattugal are not affected commonly.
Noi Nithanam

Prognosis of vatha pandu noi.

Yugimuni says vatha pandu noi is curable.

The prognosis of pandu becomes grave if diarrhea occurs. This is stated with the prognosis of other disease as follows.

The other condition stated are piramegam, vatha soolai, gunmam, kshayam, Janni, Sobai, mathumegam and kamalai. This is stated in sathaga nadi as
In pandu noi and in certain other disease if a combination of emaciation, dyspnoea and hiccough occur, the patient will die. In sathaga naadi it is stated as follows.

Kannusamiyam quoted that scanty micturition in pandu noi will cause the death,

Maruthuvam

- மருதுவம் 8000
So our Siddha treatment is not only for complete healing but also for the prevention and rejuvenation. This is said as follows:

Kappu (Prevention)

Neekkam (Treatment)

Niraivu (Restoration)

Siddha system has unequivocally stated that even during the time of conception. Some effects creep into the fertilized embryo. The defects form the basis for the manifestation of certain constitutional diseases later on during the existence of the individual.

Diseases are produced by the unequilibrium of three thathus which may be due to various causes like diet, lifestyle patterns, mental and physical activities.

When treating for cure of the diseases the following principles must be noted.
So it is essential to know the disease and the cause for the onset of disease the nature of the patient, the severity of the illness, the seasons and the time of occurrence of the disease must be observed.

**LINE OF TREATMENT FOR VATHA PANDU**

The aim is to normalize the vitiated mukkutram, vayus and the affected Raktha thathu. As this disease is caused by the detoriation in Ranjaga pitham, effective medicinal preparations have to be administered in the beginning itself to raise the Raktha thathus achieve normal function of it.

Before starting the actual treatment, the presence of absence of toxins in the body produced due to derangement of three thathus. This explained in siddha as follows.
Usually for pitha disease emetics are to be given to alter the deranged pitham. But there are some exceptions to this rule for instance in veluppu not since the patient is already week and drowsi. The administration of vomiting medicines is excluded form the line of treatment.

1. To bring out the Thridhosha to its normal physiological activites, laxative are to be administered.

2. Improving haemoglobin content of blood, iron preparations are used.


4. Pathiam ie diet and other restrictions to normalize the affected thathu and to maintain a longer drug action.

Patients were treated with Agathi keerai kudineer 100 to 200ml at bed time as laxative and Murukkanvithai mathirai (1) with hot water as deworm theraphy. Then they were treated with Ilagu Suyamakini Chenduram and Nellikai Legium.
Diet

Siddha system lays a great importance on the observation of rules regarding diet in everyday life because the siddha system has rightly realized that the basic factor of the body is food, that is Annamaya kosam is the first among the five kosams constituting our physical and mental existence to prevent the occurrence of the disease, elaborate inference regarding food item is out daily diet is given in the text books of siddha.

Thirumoolar says

Herbs, Metals and Minerals used for pandu noi

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கீழில் பின்னர் வரும் எட்டு விழாக்களுக்கு நீண்ட நேர்
விளையாட்டு விளையாட்டு புனைவுக்காக காதி

மிகுதி விளையாட்டு முன்னேற்றத் தீவில்

விளையாட்டு புனைவுப்பு அறிக்கை

விளையாட்டு காத்தை விளையாட்டு காத்தைலுக்கு

விளையாட்டு தொகுதியில் விளையாட்டு

விளையாட்டு பொருளாக தாக்குவதோடு விளையாட்டு

அளவேற்று விளையாட்டு விளையாட்டு பொருளாக தாக்குவதோடு

விளையாட்டு தொகுதியில் விளையாட்டு விளையாட்டு

- கைப்பிட்டிய தாக்குத்தோடு
**Diet Regimen for pandu noi**

Consuming unbalance and incompatible diet is considered to be the prime causative factor for upsetting the threedosha balance leading to manifestations of various ailments. Regarding diet Regimen in vatha pandu the following food items are recommended.

**Keerai vagaikal (Greens)**

Greens like karisalai, ponnanganni, Arukkeerai, Sirukeerai, Murungai keerai and Manithakkali keerai having haematinic property may be given daily.

**Kaikarikal (Vegetables)**

Kathiri pinju, Avarai pinju, Murungai pinju, Vazhai kachal may be given along with other food stuffs.

**Pazhavakaigal (Fruits)**

Pereechai, Orange, Kodimunthiri, Naral, Apple, Atthi are also rich sources for iron which are taken.

Easily digestible foods like Ganjee, Mutton soups, bone soups must be given in acute stages of vatha pandu noi. The goat liver soup is given with orange or apple or grape juice to these patients.
After the normal appetite is restored properly prepared flesh of kadai, kowthari and udumbu can also be given. They tone up the body in the debilitating condition and improve “Raktham” formation.

If oedema is present barley canjee prepared with vellarivithu, mulan vithu and poosunai vithu can be given. This will act as diuretic and reduce oedema.

When the symptoms were improved the patients were advised to practice Yoga and Pranayama according to their physical and mental conditions.
ANEMIA

BLOOD :

Introduction :

Blood is a connective tissue in fluid form.

It is considered as the.

- Fluid of life because it carries oxygen from the lungs to all parts of the body and carbon dioxide from all parts of the body to the lungs.
- Fluid of growth because it carries nutritive substances from the digestive system and hormones from endocrine glands to all the tissues.
- Fluid of health because it protects the body against the diseases and gets rid of the waste products and unwanted substances by transporting them to the excretory organs like kidneys.

Properties of blood :

1. Colour :

   Arterial blood – Scarlet red.
   Venous blood – Purple red.

2. Volume :

   Average volume of blood in a normal adult – 5 L.
   Volume of blood in a new born baby – 450 ml.
3. Reaction & pH :
   Slightly alkaline.
   pH → 7.4

4. Specific Gravity :
   Specific gravity of total blood – 1.052 to 1.061
   Specific gravity of blood cells – 1.092 to 1.101
   Specific gravity of plasma – 1.022 to 1.026.

5. Viscosity :
   Five times more viscous than water.

**Composition of blood :**

Blood Contains the blood cells which are called formed elements and the liquid portion known as plasma.

1. Blood cells or formed elements :
   Three types of cells are present in the blood. They are
   i) Red blood cells or Erythrocytes.
   ii) White blood cells or Leukocytes.
   iii) Platelets or Thrombocytes.
2. Plasma:

Plasma is a straw coloured clear liquid. It contains 91 to 92% of water and 8 to 9% of solids. The solids are the organic and the inorganic substances.

**Functions of blood:**

1. Nutrient function:

    Nutritive substances like glucose amino acids, lipids and vitamins derived from digested food are absorbed from gastrointestinal tract and carried by blood to different parts of the body for growth and production of energy.

2. Respiratory function:

    Blood carries oxygen from alveoli of lungs to different tissues and carbon dioxide from tissues to alveoli.

3. Excretory function:

    Waste products formed in the tissues during various metabolic activities are removed by blood and carried to the excretory organs like kidney, skin, liver etc, for excretion.
4. Transport of hormones & enzymes:

The hormones, secreted by ductless (endocrine) glands are released directly into the blood. The blood transports these hormones to their target organs/tissues. Blood also transports enzymes.

5. Regulation of water balance:

Water content of the blood is freely interchangeable with interstitial fluid. This helps in the regulation of water content of the body.

6. Regulation of acid base balance:

The plasma proteins and hemoglobin act as buffers and help in regulation of acid base balance.

7. Regulation of body temperature:

Because of the high specific heat of blood, it is responsible for maintaining the thermoregulatory mechanism in the body.

8. Storage function:

Water and some important substances like protein, glucose, sodium and potassium are constantly required by the tissues. Blood serves as a readymade source for these substances.

9. Defensive function:

White blood cells play an important role in the defense of the body.
RED BLOOD CELLS

Introduction:

Red blood cells are also known as erythrocytes (erythros – red).

The red colour of the red blood cell (RBC) is due to the presence of the colouring pigment called hemoglobin.

RBCs play a vital role in transport of respiratory gases.

RBC Count:

<table>
<thead>
<tr>
<th></th>
<th>SI units</th>
<th>Non – SI units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>4.5 – 6.5 x 10^12/L</td>
<td>4.5 – 6.5 x 10^6/mm^3</td>
</tr>
<tr>
<td>Female</td>
<td>3.8 – 5.8 x 10^12/L</td>
<td>3.8 – 5.8 x 10^6/mm^3</td>
</tr>
</tbody>
</table>

Morphology of Red blood cells:

Normal Shape:

Normally, the RBCs are disk shaped and biconcave (dumb bell shaped).

Normal Size:

Diameter : 7.2µ (6.9 – 7.4µ)

Thickness : At the periphery, it is thicker with 2.2µ and
at the centre, it is thinner with 1µ. The difference in thickness is because of the biconcave shape.

Surface area : 120 sq.m.
Volume : 85 – 90 cu.m.

**Normal Structure :**

RBCs are non–nucleated formed elements in the blood. So, DNA is absent. Mitochondria also are absent and the energy is produced from glycolytic process. Golgi apparatus is also absent in RBC. RBCs do not have insulin receptor and so the glucose uptake by this all is not controlled by insulin.

RBC has a special type of cytoskeleton which is made up of actin and spectrin. Both the proteins are anchored to transmembrane proteins by means of another protein called ankyrin.

**PROPERTIES OF RED BLOOD CELLS :**

1. Rouleaux formation :
When blood is taken out of the blood vessel, the RBCs pile up one above another like the pile of coins. This property of the RBC is called rouleaux (pleural = rouleau) formation.

2. Specific gravity:

Specific gravity of RBC = 1.092 to 1.101

3. Packed cell volume:

When the blood is collected in a centrifuge tube along with proper anticoagulant and centrifuged for a period of 30 minutes at a speed of 3000 rpm the RBCs settle at the bottom of the tube leaving the clear plasma at the top.

The RBCs form 45% of the total blood. This is called the packed cell volume or hematocrit. The volume of plasma is 55%.

4. Suspension stability:

During circulation, the RBCs remain suspended uniformly in the blood. This property of the RBCs is called the suspension stability.

**ERYTHROPOIESIS:**

Erythropoiesis is the process which involves the origin, development and maturation of erythrocytes.
**Site of Erythropoiesis:**

<table>
<thead>
<tr>
<th>I.</th>
<th>Fetal life</th>
<th>Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>First 2 months of intrauterine life.</td>
<td>Mesenchyme of yolk sac.</td>
</tr>
<tr>
<td>2.</td>
<td>From 3(^{rd}) month onwards.</td>
<td>Liver, Spleen and Lymphoid organs.</td>
</tr>
<tr>
<td>3.</td>
<td>During last 3 months of intrauterine life</td>
<td>Red bone marrow and Liver.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>II.</th>
<th>After birth:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Upto the age of 5 to 6 years</td>
<td>Red bone marrow of all bones.</td>
</tr>
<tr>
<td>2.</td>
<td>Age 6 yrs to 20 yrs.</td>
<td>Red bone marrow of long bones and all the membranous (flat) bones.</td>
</tr>
<tr>
<td>3.</td>
<td>After the age of 20 yrs.</td>
<td>From Membranous bones like vertebra, sternum, ribs, scapula, iliac bones and skull bones and from the ends of long bones.</td>
</tr>
</tbody>
</table>

During bone disorders, the RBCs are produced in spleen.
Stages of Erythropoiesis:

Uncommitted pluripotent hemopoietic stem cell.
↓
Committed pluripotent hemopoietic stem cell.
↓
Colony forming blastocyte
↓
Colony Forming unit – E.
↓
Proerythrocyte
↓
Early normoblast (Basophilic erythroblast)
↓
Intermediate normoblast (Hemoglobin starts appearing)
↓
Late normoblast (Nucleus disintegrates and disappears)
↓
Reticulocyte (Immature RBC)
↓
Matured Erythrocyte
It requires 7 days for the development and maturation of RBC from proerythroblast. It requires 5 days up to the stage reticulocyte. The reticulocyte takes 2 more days to become the matured RBC.

**Changes during Erythropoiesis:**

The stem cells of the colony forming unit – E (CFU – E) pass through different stages and finally become the matured RBCs. During these stages four important changes are noticed.

1. Reduction in size of the cell (from the diameter of 25µ to 7.2µ)
2. Disappearance of nucleoli and nucleus
3. Appearance of hemoglobin.
4. Change in the staining properties of the cytoplasm.

**Factors necessary for Erythropoiesis:**

Development and maturation of erythrocytes require variety of factors which are classified into 3 categories:

I) General factors.

II) Maturation factors.

III) Factors necessary for hemoglobin formation.
I. General Factors:

General factors necessary for erythropoiesis are:

1. Erythropoietin.
2. Thyroxine.
3. Hemopoietic growth factors
4. Vitamins.

1. Erythropoietin:

The most important general factor for erythropoiesis is the hormone erythropoietin. It is also called homopoietin or erythrocyte stimulating factor.

Erythropoietin is a glycoprotein secreted by peritubular capillaries of kidney.

Hypoxia is the stimulant for the secretion of erythropoietin.

Erythropoietin promotes the following processes:

i) Production of proerythroblasts from the stem cells in CFU – E of the bone marrow.

ii) Development of proerythroblasts into matured RBCs through the normoblastic stages.
iii) Release of matured erythrocytes into blood. Even some reticulocytes (immature erythrocytes) are released along with matured RBCs. After secretion, it takes 4 to 5 days to show the action.

2. **Thyroxine**:

Being a general metabolic hormone, thyroxine accelerates the process of erythropoiesis at many levels.

3. Hemopoietic growth factors:

Hemopoietic growth factors or growth inducers are the interleukins and stem cell factor (Steel factor). Generally, these factors induce the proliferation of pluripotent stem cells. Interleukins involved in erythropoiesis are IL – 3, IL -6 and IL -11.

4. Vitamins:

The vitamins, which are necessary for erythropoiesis are:

- Vitamin B, 
- Vitamin C, 
- Vitamin D and 
- Vitamin E.
II. Maturation factors:

Vitamin B12, intrinsic factor and folic acid are necessary for the maturation of RBCs.

1. Vitamin B$_{12}$ (Cyanocobalamin):

This is essential for maturation of erythrocytes. The deficiency of vitamin B$_{12}$ causes pernicious anaemia. So vitamin B$_{12}$ is called antipernicious factor.

Source of vit. B12:

Vit. B$_{12}$ is called extrinsic factor because it is obtained mostly from diet. Its absorption from the intestine requires the presence of intrinsic factor of castle. Vit. B$_{12}$ is stored mostly in the liver and in small quantity in muscle. When necessary, it is transported to the bone marrow to promote maturation of RBCs. It is also produced in the large intestine by the intestinal flora.

Action of vit. B12:

Vit. B$_{12}$ is essential for synthesis of DNA. Its deficiency leads to failure in maturation of the cell and reduction in the cell division. Also, the cells are larger with fragile and weak cell membrane.
2. Intrinsic Factor of castle:

   It is produced in gastric mucosa and it is essential for the absorption of Vit. B\textsubscript{12} from intestine into the blood. In the absence of intrinsic factor, Vit. B\textsubscript{12} is not absorbed. This happens in severe gastritis, ulcers and gastrectomy. The deficiency of intrinsic factor also causes pernicious anemia since the Vit. B\textsubscript{12} is not absorbed. The extrinsic and intrinsic factors are together called hematinic principle.

3. Folic acid:

   Folic acid is also essential for maturation. It is required for the synthesis of DNA. In the absence of folic acid, the synthesis of DNA decreases causing failure of maturation. This leads to anemia in which, the cells are larger and appear in megaloblastic (Proerythroblastic) stage. Anemia due to folic acid deficiency is called megaloblastic anemia.

**III. Factors necessary for hemoglobin formation:**

   Various materials are essential for the formation of hemoglobin in the RBCs. The deficiency of these substances decreases the production of hemoglobin leading to anemia. Such factors are:
1. First class proteins and amino acids:

   Proteins of high biological value are essential for the formation of hemoglobin. Amino acids derived from these proteins are required for the synthesis of protein part of hemoglobin, the globin.

2. Iron:

   It is necessary for the formation of heme part of the hemoglobin.

3. Copper:

   It is necessary for the absorption of iron from the gastrointestinal tract.

4. Cobalt and nickel:

   Cobalt and nickel are essential for the utilization of iron during hemoglobin formation.

5. Vitamins:

   Vit. C, Riboflavin, Nicotinic acid and pyridoxine are also essential for the formation of Hemoglobin.

**VARIATIONS IN SIZE OF RED BLOOD CELLS:**

   Under physiological conditions, the size of RBC in venous blood is slightly larger than those in arterial blood.

   In pathological conditions, the variations in size of RBCs are:

   1. Microcytes – decrease in size.

   2. Macrocytes – increase in size.
3. Anisocytes – cells without uniform size.

Microcytes:

Microcytes are present in:

i) Iron deficiency anaemia.
ii) Prolonged forced breathing.
iii) Increased osmotic pressure in blood.

Macrocytes:

Macrocytes are present in:

i) Megaloblastic anemia.
ii) Muscular excercise.
iii) Decreased osmotic pressure in blood.

Anisocytes:

Anisocytes occurs in pernicious anemia.

VARIATIONS IN SHAPE OF RBCs:

The shape of RBCs is altered in many conditions including different types of anemia.

1. Genation:

   Shrinkage as in hypertonic solution.

2. Spherocytosis:

   Globular form as in hypotonic solution.
3. **Elliptocytosis**:  
   Elliptical shape as in certain types of anemia.

4. **Sicke cell**:  
   Crescentic shape as in sickle cell anemia.

5. **Poikilocytosis**:  
   Unequal shapes due to deformed cell membrane. The shape will be of flask hammer or any other unusual shape.

**VARIATIONS IN STRUCTURE OF RBCs:**

1. **Punctate Basophilism**:  
   The striated appearance of RBCs by the presence of dots of basophilic materials (porphyrin) is called punctate basophilism. It occurs in conditions like lead poisoning.

2. **Ring**:  
   Ring or twisted strands of basophilic material appear in the periphery of the RBCs. This is also called the goblet ring. This appears in the RBCs in certain types of anemia.

3. **Howell – Jolly Bodies**:  
   In certain types of anemia, some nuclear fragments are present in the ectoplasm of the RBCs. These nuclear fragments are called howell – Jolly bodies.
LIFESPAN AND FATE OF RBCs:

Average lifespan of RBC is about 120 days. After the lifetime the senile (old) RBCs are destroyed in reticuloendothelial system.

When the cells become older (120 days) the cell membrane becomes more fragile. The diameter of the capillaries is less or equal to that of RBC. The younger RBCs can pass through the capillaries easily. However, because of the fragile nature, the older cells are destroyed while trying to squeeze through the capillaries. The destruction occurs mainly in the capillaries of spleen, because the diameter of splenic capillaries is very small. So, the spleen is called graveyard of RBCs.

The destroyed RBCs are fragmented. From the fragmented parts, the hemoglobin is released and degraded into iron, globin and porphyrin. Iron combines with the protein apoferritin to form ferritin, which is stored in body. Globin enters the protein depot. The porphyrin is degraded and finally forms bilirubin which is excreted by liver through bile.

Daily 10% RBCs, which are senile, are destroyed in normal young healthy adults. It causes release of about 0.6g% of hemoglobin into the plasma. From this, 0.9 to 1.5 mg% bilirubin is formed.
FUNCTIONS OF RBCs :

1. Transport of oxygen from the lungs to the tissues :

   Hemoglobin in RBC combines with oxygen to form oxyhemoglobin. About 97% of oxygen is transported in blood in the form of oxyhemoglobin.

2. Transport of carbon dioxide from the tissues to the lungs :

   Hemoglobin combines with carbondioxide and form carboxyhaemoglobin. About 30% of carbon dioxide is transported in this form.

   RBCs contain a large amount of the carbonic anhydrase. This enzyme is necessary for the formation of bicarbonate from water and carbon dioxide. Thus it helps to transport carbon dioxide in the form of bicarbonate from tissues to lungs. About 63% of carbon dioxide is transported in this form.

3. Buffering action in blood :

   Hemoglobin functions as a good buffer. By this action, it regulates the hydrogen iron concentration and thereby plays a role in the maintenance of acid base balance.

4. In Blood group determination :

   RBCs carry the blood group antigens like a agglutinogen, B agglutinogen and Rh factor. This helps in determination of blood group and enables to prevent reactions due to incompatible blood transfusion.
HEMOGLOBIN (Hb)

Introduction:

Hemoglobin is the red blood pigment, exclusively found in erythrocytes. It is a chromoprotein forming 95% of dry weight of RBC and 30 to 34% of wet weight. The molecular weight of hemoglobin is 68,000. It performs two important biological functions concerned with respiration:

i) Delivery of \( \text{O}_2 \) from the lungs to the tissues.

ii) Transport of \( \text{CO}_2 \) and protons from tissues to lungs for excretion.

It also acts as a buffer.

NORMAL Hb LEVELS:

Average hemoglobin (Hb) content in blood is 14 to 16g%. However, the value varies depending upon the age and sex of the individual.

Age:

At birth : 25g%

After 3\(^{rd}\) month : 20g%

After 1 Year : 17g%

From puberty onwards : 14 to 16g%

At the time of birth, Hb content is very high because of increased RBC count.
### Hb level

<table>
<thead>
<tr>
<th>Sex</th>
<th>In ST unit</th>
<th>in non – ST unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>In adult males</td>
<td>130 – 180 g/l</td>
<td>13 – 18 g/dl</td>
</tr>
<tr>
<td>In adult females</td>
<td>115 – 165 g/l</td>
<td>11.5 – 16.5 g/dl</td>
</tr>
</tbody>
</table>

**STRUCTURE:**

Hb is a conjugated protein. It consists of a protein combined with an iron containing pigment. The protein part is globin and the iron containing pigment is heme. Hence also forms a part of structure of myoglobin (oxygen binding pigment in muscles)

**Iron:**

Normally, it is present in ferrous (Fe$^{2+}$) form. It is an unstable or loose form in some abnormal conditions, the iron is converted into ferric (Fe$^{3+}$) state, which is a stable form.

**Porphyrin:**

The pigment part is called porphyrin it is formed by four pyrrole rings (tetraphrrole) called I, II, III and IV. The pyrrole rings are attached to one another by methane (CH$_4$) bridges.

The iron is attached to N- of each pyrrole ring and N- of globin molecule.
Globin:

This contains four polypeptide chains. Among the four polypeptide chains, two are $\alpha$ – chains and two are $\beta$ Chains.

(i) $\alpha$ Chain:

Molecular weight – 15, 126.

No. of amino acids – 141.

(ii) $\beta$ Chain:

Molecular weight - 15, 866

No. of amino acids – 146.

NORMAL MAJOR TYPES OF HEMOGLOBINS:

<table>
<thead>
<tr>
<th>Type</th>
<th>Composition and symbol</th>
<th>Percentage of total Hb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb A1</td>
<td>$\alpha_2 \beta_2$</td>
<td>90%</td>
</tr>
<tr>
<td>Hb A2</td>
<td>$\alpha_2 \delta_2$</td>
<td>&lt; 5%</td>
</tr>
<tr>
<td>Hb F</td>
<td>$\alpha_2 r_2$</td>
<td>&lt; 2%</td>
</tr>
<tr>
<td>Hb A_{1c}</td>
<td>$\alpha_2 \beta_2$ glucose.</td>
<td>&lt; 5.4%</td>
</tr>
</tbody>
</table>

SYNTHESIS OF Hb:

Synthesis of Hb actually starts in proerythroblastic stage. However, Hb appears in the intermediate normoblastic stage only. The production of
the hemoglobin is continued until the stage of reticulocyte. The heme portion of hemoglobin is synthesized in mitochondria from acetic acid and the glycine. The sequence of events in the synthesis of hemoglobin:

1. During krebs cycle, the acetic acid is converted into succinyl CoA
2. Two molecules of succinyl CoA combine with two molecules of glycine to form pyrrole compound.
3. Four pyrrole compounds combine to form protoporphyrin.
4. Protoporphyrin is of many types. Only protoporphyrin IX is involved in the formation of heme molecule by combining with iron.
5. Each heme molecule combines with one globin molecule to form hemoglobin.

The protein part, globin is synthesized in ribosomes.

**DESTRUCTION OF Hb:**

After the lifespan of 120 days, the RBC is destroyed in the reticuloendothelial system particularly in spleen and the hemoglobin is released into plasma. Soon, the hemoglobin is degraded in the reticuloendothelial cells and split into globin, iron and porphyrin.

Globin is utilized for the resynthesis of Hb. Iron is stored in the body as ferritin and hemosiderin, which are reutilized for synthesis for new Hb.
Porphyrin is converted into a green pigment called biliverdin. In human being, most of the biliverdin is converted into a yellow pigment called bilirubin. Bilirubin and biliverdin are together called the bile pigments.

FUNCTIONS:

1. Transport of respiratory gases:

   The main function of hemoglobin is the transport of respiratory gases:

   (i) Oxygen from the lungs to tissues.

   (ii) Carbon dioxide from tissues to lungs

   (i) Transport of oxygen:

   When oxygen binds with Hb, a physical process called oxygenation occurs resulting in the formation of oxyhemoglobin. The iron remains in ferrous state in this compound. Oxyhemoglobin is an unstable compound and the combination is reversible, ie. The oxygen can be released from this compound.

   When oxygen is released from oxyhemoglobin, it is called reduced hemoglobin or ferrohemoglobin.
(ii) Transport of carbon dioxide:

When carbon dioxide binds with hemoglobin, carbhemoglobin is formed. It is also an unstable compound and the combination is reversible, i.e. the carbon dioxide can be released from this compound. The affinity of Hb for carbon dioxide is 20 times more than for oxygen.

2. Buffer action:

Hb acts as a buffer and plays an important role in acid base balance.

IRON

Introduction:

The total content of iron in an adult body is 3 – 5 g. About 70% of this occurs in the erythrocytes of blood as a constituent of Hb. Hence is the most predominant iron containing substance. It is a constituent of several proteins/enzymes (hemoproteins) – hemoglobin, myoglobin, cytochromes, xanthine oxidase, catalase, tryptophan pyrrolase peroxidase, certain other proteins contain non heme hence iron eg. Transferrin. Ferritin, hemosiderin.

Distribution of iron in the body:

The approximate distribution of iron in the body is as follows:

- In the hemoglobin: 65 to 68%
- In the muscle as myoglobin: 4%
As intracellular oxidative heme compound : 1%

In the plasma as transferrin : 0.1%

Stored in the reticuloendothelial system : 25 to 30%

Dietary requirements :

Adult man - 10 mg / day

Menstruating woman - 18 mg / day.

Pregnant and lactating women - 40 mg / day

Sources :

Dietary iron is available in two forms called heme and non-heme.

Heme iron is present in fish, meat and chicken. Non-heme iron is available in vegetables, grains and cereals.

Rich Sources - Organ meats (liver, heart, kidney)

Good Sources - Leafy vegetables, pulses,

Cereals, fish, apples,

Dried fruits, molasses.

Poor Sources - Milk, wheat, polished rice.
Absorption of iron:

Iron is mainly absorbed from the small intestine. It is absorbed through the intestinal cells by pinocytosis and transported into the plasma. Bile is essential for the absorption of iron.

Transport and storage of iron:

Immediately, after absorption into the blood, iron combines with a β globulin called apotransferrin to form transferrin and is transported in this form in the plasma. Iron combines loosely with the globin and can be released easily at any region of the body.

Iron is stored in large quantities in the reticuloendothelial cells and liver hepatocytes. In other cells also, it is stored in small quantities. In the cytoplasm of the cell, iron is stored as ferritin in large amount. Small quantity of iron is also stored as hemosiderin.

Factors affecting iron absorption:

1. Acidity, ascorbic acid and cysteine promote iron absorption.
2. In iron deficiency anemia, fe absorption is increased to 2-10 times that of normal.
3. Small peptides and amino acids favour iron uptake.
4. Phytate (found in cereals) and oxalate (found in leafy vegetables) interfere with iron absorption.

5. A diet with high phosphate content decreases iron absorption while low phosphate promotes.

6. Impaired absorption of iron is observed in malabsorption syndromes such as steatorrhea.

7. In patients with partial or total surgical removal of stomach and intestine, iron absorption is severely impaired.

**Daily loss of iron:**

In males, about 1 mg of iron is excreted everyday through feces. In females, the amount of iron loss is very much high. This is because of the menstruation.

1g of hemoglobin contains 3.34 mg of iron. Normally 100ml of blood contains 15g of hemoglobin and about 50mg of iron. So, if 100ml of blood is lost from the body, there is a loss of about 50mg of iron. In females, during every menstrual cycle, about 50 ml of blood is lost by which 25 mg of iron is lost. That is why, the iron content is always less in females than in males.

Iron is lost during hemorrhage and blood donation also. If 450ml of blood is donated, about 225mg of iron is lost.
**Regulation of total body iron:**

Absorption and excretion of iron are maintained almost equally under normal physiological conditions. When the iron storage is saturated in the body, it automatically reduces the further absorption of iron from the gastrointestinal tract by feedback mechanism.

The factors which reduce the absorption of iron are:

1. Stoppage of apotransferrin formation in the liver, so that, the iron could not be absorbed from the intestine.
2. Reduction in the release of iron from the transferrin so that, transferrin is completely saturated with iron and further absorption is prevented.

**Functions:**

1. Iron mainly exerts its functions through the compounds in which it is present.
   - Hemoglobin and myoglobin are required for the transport of O2 and CO2.
   - Cytochromes and certain non-heme proteins are necessary for electron transport chain and oxidative phosphorylation.
   - Peroxidase, the lysosomal enzyme, is required for phagocytosis and killing of bacteria by neutrophils.
2. Iron is associated with effective immunocompetence of the body.
PACKED CELL VOLUME AND BLOOD INDICES

Packed cell volume:

Packed cell volume (PCV) is the hematocrit value expressed as the percentage of cellular elements with that of whole blood. Or it is the measure or proportion of blood volume that is occupied by RBCs.

Normal values of PCV or Hematocrit:

<table>
<thead>
<tr>
<th></th>
<th>In SI units</th>
<th>In Non SI units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>0.40 – 0.54</td>
<td>40 – 54%</td>
</tr>
<tr>
<td>Females</td>
<td>0.37 – 0.47</td>
<td>37 – 47%</td>
</tr>
</tbody>
</table>

BLOOD INDICES:

The various blood indices are,

1. Mean corpuscular volume (MCV)
2. Mean corpuscular hemoglobin (MCH)
3. Mean corpuscular hemoglobin concentration (MCHC)
1. **Mean corpuscular volume (MCV)**:

MCV is the average volume of a single RBC and it is expressed in cubic micron (cu.µ)

$$MCV = \frac{Volume\ of\ packed\ cells\ in\ ml\ per\ 1000\ ml\ of\ blood}{RBCs\ in\ million\ per\ cu.\ mm.\ of\ blood}$$

Normal value:

<table>
<thead>
<tr>
<th>Normal value</th>
<th>In SI units</th>
<th>In non SI units</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCV</td>
<td>78 – 98 fl</td>
<td>78 – 98 cu.µ.</td>
</tr>
</tbody>
</table>

MCV is more in pernicious anemia and megaloblastic anemia in which the RBCs are macrocytic in nature. MCV is less in microcytic anemia.

2. **Mean corpuscular Hemoglobin (MCH)**:

It is the quantity or amount of Hb present in one RBC. It is expressed in microgram or picogram (pg).

$$MCH = \frac{Hb\ in\ grams\ per\ 1000\ ml\ of\ blood}{RBC\ count\ in\ million\ /\ cu.\ mm}$$
Normal value:

\[ MCH = 27 - 32 \text{ pg.} \]

It decreases or remains normal in pernicious anemia and megaloblastic anemia, in which RBCs are macrocytic and normochromic or hypochromic. It decreases in hypochromic anemia. When MCH is normal, it is called normochromic state.

3. **Mean corpuscular Hemoglobin concentration (MCHC):**

This is the concentration of Hb in one RBC. It is the amount of Hb expressed in relation to the volume of one RBC. So, its unit is percentage.

\[
\text{Hb in grams} / 100\text{ml of blood} \\
\text{MCHC} = \frac{\text{Hb in grams}}{\text{PCV in 100ml of blood}} \times 100 \\
\]

Normal Value:

\[ \text{MCHC} = 30 \text{ to } 38\% \]

This is the most important absolute value in the diagnosis of anemia. It decrease in iron deficiency anemia in which RBCs are microcytic and hypochromic.
4. **Color Index (CI):**

This is the ratio between the percentage of Hb and the percentage of RBCs in the blood. Actually, it is the average Hb content in one cell of a patient compared to average Hb content in one cell of a normal person.

\[
\text{Color index} = \frac{\text{Hemoglobin} \%}{\text{RBC} \%}
\]

Where 
\[
\text{Hb} \% = \frac{\text{Hb content in the subject}}{\text{Normal Hb content}} \times 100
\]

\[
\text{RBC} \% = \frac{\text{RBC count in the subject}}{\text{Normal RBC count}} \times 100
\]

Normal Value:

\[
\text{CI} = 0.8 \text{ to } 1.2.
\]

It raises in pernicious anemia and megaloblastic anemia. It is reduced in iron deficiency anemia. And, it is normal in normocytic normochromic anemia.
ANEMIA

Introduction:

Anemia is defined as a hemoglobin concentration in blood below the lower limit of the normal range for the age and sex of the individual. In adults, the lower extreme of the normal hemoglobin is taken as 13.0 g/dl for males and 11.5 g/dl for females. Newborn infants have higher hemoglobin level and, therefore, 15 g/dl is taken as the lower limit at birth, whereas at 3 months the lower level is 9.5 g/dl. Although hemoglobin value is employed as the major parameter for determining whether or not anemia is present, the red cell counts, hematocrit (PCV) and absolute values (MCV, MCH and MCHC) provide alternate means of assessing anemia.

Generally, anemia occurs because of:

1. Decreased production of RBC.
2. Increased destruction of RBC or
3. Excess loss of blood from the body.

All these incidents are caused either by inherited disorders or environmental influences such as nutritional problem, infection and exposure to drugs or toxins.
Pathophysiology of Anemia:

Subnormal level of Hb causes lowered oxygen – carrying capacity of the blood. This, in turn, initiates compensatory physiologic adaptations such as:

- Increased release of oxygen from Hb;
- Increased blood flow to the tissues;
- Maintenance of the blood volume; and
- Redistribution of blood flow to maintain the cerebral blood supply.

Eventually, however, tissue hypoxia develops causing impaired functions of the affected tissues. The degree of functional impairment of individual tissues is variable depending upon their oxygen requirements. Tissues with high oxygen requirement such as the heart, CNS and the skeletal muscle during exercise, bear the clinical effects of anemia.

Clinical features of Anemia:

The Hb level at which symptoms and signs of anemia develop depends upon 4 main factors.

1. The speed of onset of anemia:

   Rapidly progressive anemia causes more symptoms than anemia of slow onset as there is less time for physiologic adaptation.
2. The severity of anemia:

Mild anemia produces no symptoms or signs but a rapidly developing severe anemia (Hb below 6g/dl) may produce significant clinical features.

3. The age of the patient:

The young patients due to good cardiovascular compensation tolerate anemia quite well as compared to the elderly. The elderly patients develop cardiac and cerebral symptoms more prominently due to associated cardiovascular disease.

4. The hemoglobin dissociation curve:

In anemia, the affinity of Hb for oxygen is depressed as 2, 3 – BPG in the red cells increase. As a result, oxyhemoglobin is dissociated more readily to release free oxygen for cellular use, causing a shift of the oxyhemoglobin dissociation curve to the right.

In older patients, there may be symptoms of.

Cardiac failure.

Angina Pectoris,

Intermittent claudication,

Confusion and

Visual disturbances
Signs :

A few general signs common to all types of anemias are as under :

1. Pallor :

Pallor is the most common and characteristic sign which may be seen in the mucous membrane, conjunctivae and skin.

2. Cardiovascular system :

A hyperdynamic circulation may be present with tachycardia, collapsing pulse, cardiomegaly, midsystolic flow murmur, dyspnoea on exertion, and in the case of elderly, congestive heart failure.

3. Central nervous system :

The older patients may develop symptoms referable to the CNS such as attack of faintness, giddiness, headache tinnitus, drowsiness, numbness and tingling sensations of the hands and feet, dizziness or vertigo especially when standing and increased sensitivity to cold.

4. Respiratory system :

There is increase in rate and force of respiration. Sometime, it leads to breathlessness and dyspnoea. Oxygen hemoglobin dissociation curve is shifted to right.
5. Ocular manifestations:

Retinal hemorrhages may occur if there is associated vascular disease or bleeding diathesis.

6. Reproductive system:

Menstrual disturbances such as amenorrhoea and menorrhagia and loss of libido are some of the manifestations involving the reproductive system in anemic subjects.

7. Renal system:

Mild proteinuria and impaired concentrating capacity of the kidney may occur in severe anemia.

8. Gastrointestinal system:

Anorexia, flatulence, nausea, constipation and weight loss may occur.

9. Metabolism:

Basal metabolic rate increases in severe anemia.

10. Muscles:

Muscles become weak and the patient feels lack of energy and fatigued quite often and quite easily.

11. Nails & Hair:

Pallor can be seen in nail bed. The nails become brittle and easily breakable. Thinning, loss and early grayness of hair occur.
Classification of anemia:

Anemia is classified by 2 methods:

a. Morphological classification.
b. Etiological classification.

A. Morphological classification:

Based on the red cell size, hemoglobin content and red cell indices, anemias are classified into 4 types:

1. Normocytic normochromic anemia:

   The size and Hb content of RBC are normal. But the number of RBC is less. MCV, MCH, MCHC are all normal.

   eg. After acute blood loss.
   Hemolytic anemias,
   Bone marrow failure,
   Anemia of chronic disorders.

2. Macrocytic Normochromic Anemia:

   The RBCs are larger in size with normal Hb content. The RBC count decrease.
3. **Macrocytic hypochromic Anemia** :

   The RBCs are larger in size. The hemoglobin content in the cell (MCH) is less so the cells are pale.

   In macrocytic anemias, MCV is raised.

   eg: Megaloblastic anemia due to deficiency of Vit B12 or folic acid.

4. **Microcytic hypochromic Anemia** :

   The RBCs are smaller in size.

   MCV, MCH, MCHC are all reduced.

   eg: Iron deficiency anemia.

       Sideroblastic anemia

       Thalassemia.

       Anemia of chronic disorders.

**B. ETIOLOGICAL CLASSIFICATION** :

On the basis of the etiology (cause of the disease), the anemia is divided into 5 types:

1. Hemorrhagic anemia.
2. Hemolytic anemia.
4. Aplastic anemia.
5. Anemia of chronic disease.
(1) **Hemorrhagic anemia**:

Anemia due to hemorrhage is known as hemorrhagic anemia. It occurs both in acute and chronic hemorrhagic conditions. Hemorrhage occurs in conditions like accident. Ulcer, excessive uterine bleeding, purpura and hemophilia.

**Acute hemorrhage**:

It refers to sudden loss of a large quantity of blood as in the case of accident. Within about 24 hours after the hemorrhage, the plasma portion of blood is replaced. However, the replacement of RBCs does not occur quickly and it takes at least 4-6 weeks. So with less number of RBCs, hemodilution occurs.

However, morphologically the RBCs are normocytic, normochromic.

Decreased RBC count causes hypoxia which stimulates the bone marrow to produce more number of RBCs so, the condition is corrected within 4–6 weeks.

**Chronic hemorrhage**:

It refers to loss of blood by internal or external bleeding over a long period of time.

It occurs in conditions like peptic ulcer, purpura, hemophilia and menorrhagia.
Due to continuous loss of blood, lot of iron is lost from the body causing iron deficiency. This affects the synthesis of Hb resulting in less Hb content in the cells. The cells also become small. Hence, the RBCs are microcytic and hypochromic.

(2) **HEMOLYTIC ANEMIA** :

Hemolysis means destruction of RBCs hemolytic anemia occurs because of excess destruction of RBCs.

Causes of excess hemolysis are:

- Liver failure
- Renal disorder.
- Hypersplenism.
- Burns.
- Infections like malaria and septicemia.
- Poisoning by chemical substances like lead, coal and tar.
- Presence of isoagglutinins like anti Rh
- Congenital or acquired default in the shape of RBCs.

When the shape is abnormal, RBCs become more fragile and hemolysis occurs easily. It occurs in two inherited conditions called sickle cell anemia and thalassemia.
(i) Sickle cell anemia:

It is a congenital anomaly and found mostly in blacks. It is due to the abnormal hemoglobin called hemoglobin S (normal adult Hb is Hb A). In this, $\alpha$ chains are normal and $\beta$ chains are abnormal. The molecules of Hb S polymerize into long chains and precipitate inside the cells. Because of this, RBCs attain sickle (crescent) shape and become more fragile leading to hemolysis.

In children, hemolyzed sickle cells aggregate and block the blood vessels leading to infarction. The infarction is common in small bones. The infarcted small bones in hand and foot results in varying length in the digits. This condition is known as hand and foot syndrome. Jaundice also occurs in these children.

Clinical features:

The clinical manifestations of homozygous sickle cell disease are widespread. The symptoms begin to appear after 6th month of life when most of the HbF is replaced by HbS. Infection and folic acid deficiency result in more severe clinical manifestations. These features are as under:
1. **Anemia:**

There is usually severe chronic hemolytic anemia (primarily extravascular) with onset of aplastic crisis in between, the symptoms of anemia are generally mild since Hbs gives up oxygen more readily then HbA to the tissues.

2. **Vaso–occlusive phenomena:**

Patients of SS develop recurrent vaso–occlusive episodes throughout their lives due to destruction of capillary blood flow by sickled red cells upon deoxygenation or dehydration. Vasoobstruction affecting different organs and tissues results in infarcts which may be of 2 types:

(a) Microinfarcts affecting particularly the abdomen, chest, back and joints and are the cause of recurrent painful crises in SS.

(b) Macroinfarcts involving most commonly the spleen (the splenic sequestration, autosplenectomy), bone marrow (pains), bones (aseptic necrosis, osteomyelitis), lungs (pulmonary infections), kidneys (renalcortical necrosis), CNS (stroke), retina (damage) and skin (ulcers), and result in anatomic and functional damage to these organs.
3. Constitutional symptoms:

In addition to the features of anemia and infarction, patients with SS have impaired growth and development and increased susceptibility to infection due to markedly impaired splenic function.

(ii) THALASSEMIA:

The thalassemias are a diverse group of hereditary disorders in which there is reduced rate of synthesis of one or more of the globin polypeptide chains. Thalassemias are first described in people of mediterranean countries (North Africa, Southern Europe) from where it derives its name ‘mediterranean anemia’. The word ‘thalasse’ in Greek means ‘the sea’ since the condition was found commonly in regions around the Mediterranean basin. It also occurs in the middle east India, south – east Asia and in general, in blacks.

Normally, an individual inherits 2 $\beta$-globin genes located one each on two chromosomes 11, and 2 $\alpha$ - globin genes one each on 2 chromosomes 16, from each parent i.e. normal adult Hb is $\alpha_2 \beta_2$ (Hb A). Depending upon the whether the genetic defect or deletion lies in transmission of $\alpha$ - or $\beta$- globin chain genes, thalassemias are classified into $\alpha$ - and $\beta$ - thalassemias.
a) \( \alpha \) - Thalassemia:

\( \alpha \)- Thalassemias are disorders in which there is defective synthesis of globin chains resulting in depressed production of hemoglobins that contain chains (ie) HbA, HbA2 and HbF. The \( \alpha \)-thalassemias are most commonly due to deletion of one or more of the \( \alpha \)- chain genes. Depending upon the no. of genes deleted, \( \alpha \) thalassemias are classified into 4 types.

1. Four \( \alpha \)- gene deletion – Hb Bart’s Hydrops foetalis.
2. Three \( \alpha \)- gene deletion – HbH disease.
3. Two \( \alpha \)- gene deletion - \( \alpha \)- thalassemia trait.
4. One \( \alpha \)- gene deletion - \( \alpha \)- thalassemia trait (carrier).

Clinical features:

Hb Bart’s Hydrops foetalis is incompatible with life due to severe tissue hypoxia. The condition is either fatal in utero or the infant dies shortly after birth of born alive, the features of severe Rh hemolytic disease are present.

HbH Disease:

HbH Disease is generally present as a well – compensated hemolytic anemia. The features are intermediate between that of \( \beta \)-thalassemia minor and major. The severity of anemia fluctuates and may full to very low levels
during pregnancy or infections. Majority of patients have splenomegaly and may develop cholelithians.

\(\alpha\)-Thalassemia trait:

\(\alpha\)-thalassemia trait due to two \(\alpha\)-chain gene deletion is asymptomatic. It is suspected in a patient of refractory microcytic hypochromic anemia in whom iron deficiency and \(\beta\) - thalassemia minor have been excluded and the patient belongs to the high – risk ethnic group.

\(\alpha\)-Thalassemia trait (carrier):

One gene deletion \(\alpha\)-thalassemia trait is a silent carrier state.

6) \(\beta\) - Thalassemia:

The \(\beta\) - thalassemias are caused by decreased rate of \(\beta\) - chain synthesis resulting in reduced formation of HbA in the red cells. The molecular pathogenesis of the \(\beta\) - thalassemias is more complex. In contrast to \(\alpha\)-thalassemias, gene deletions rarely ever cause \(\beta\) - thalassemia and is only seen in an entity called Hereditary persistence of foetal Hemoglobin (HPFH). Instead, most of \(\beta\) - thalassemias arise from different types of mutations of \(\beta\) - globin gene resulting from single base changes. The symbol \(\beta^0\) is used to indicate the complete absence of synthesis while \(\beta^+\) denotes partial synthesis of the \(\beta\) - globin chains.
Depending upon the extent of reduction in $\beta$ – chain synthesis, there are 3 types of $\beta$-thalassemia:

1. **Homozygous form:**

   $\beta$-thalassemia majors Mediterranean or Cooley’s anemia. It is the most severe form of congenital hemolytic anemia. It is further of 2 types.
   
   a) $\beta^0$ thalassemia major characterised by complete absence of $\beta$-chain synthesis.
   
   b) $\beta^+$ thalassemia major having incomplete suppression of $\beta$-chain synthesis.

2. **$\beta$-thalassemia intermedia:**

   It is $\beta$-thalassemia of intermediate degree of severity that does not require regular blood transfusions. These cases are genetically heterozygous ($\beta^0/\beta$ or $\beta^+/\beta$).

3. **Heterozygous form:** $\beta$-thalassemia minor (trait):

   It is a mild asymptomatic condition in which there is moderate suppression of $\beta$-chain synthesis.
Clinical features:

1. β - thalassemia major:

   Clinical manifestations appear insidiously and are as under.
   
   a) Anemia starts appearing within the first 4 - 6 months of life when
   the switch over from r – chain to β – chain production occurs:
   
   b) Marked hepatosplenomegaly occurs due to excessive red cell destruction, extramedullary hematopoiesis and iron overload.
   
   c) Expansion of bones occurs due to marked erythroid hyperplasia leading to thalassemic facies and malocclusion of the jaw.
   
   d) Iron overload due to repeated blood transfusions causes damage to the endocrine organs resulting in slow rate of growth and development, delayed puberty, diabetes mellitus and damage to the liver and heart.

2. β - Thalassemia. Minor:

   Clinically, the condition is usually asymptomatic and the diagnosis is generally made when the patient is being investigated for a mild chronic anemia. The spleen may be palpable.

3. NUTRITION DEFICIENCY ANEMIA:

   Nutritive substances such as iron, proteins and vitamins like C, B_{12} and folic acid are necessary for erythropoiesis. The deficiency of these substances leads to nutrition (nutritional) deficiency anemia.
(i) IRON DEFICIENCY ANEMIA:

Iron deficiency anemia is the most common type of anemia. It develops due to inadequate availability of iron for Hb synthesis. The RBCs are microcytic and hypochromic. Causes of iron deficiency anemia are:

Loss of blood.
Decreased intake of iron.
Poor absorption of iron from intestine.
Increased demand for iron in conditions like growth and pregnancy.

Clinical features:

Iron deficiency anemia is much more common in women between the age of 20 and 45 yrs than in men; at periods of active growth in infancy, childhood and adolescence, and is also more frequent in premature infants. Initially, there are usually no clinical abnormalities. But subsequently, in addition to features of the underlying disorder causing the anemia, the clinical consequences of iron deficiency manifest in two ways – anemia itself & epithelial tissue changes.

a) Anemia:

The onset of iron deficiency anemia is generally slow. The usual symptoms are of weakness, fatigue, dyspnoea on exertion, palpitations and
pallor of the skin, mucous membranes and sclerae. Older patients may develop angina and congestive cardiac failure. Patients may have unusual dietary cravings such as PICA. Menorrhagia is a common symptom in iron deficient women.

b) Epithelial tissue changes:

Long – standing chronic iron deficiency anemia causes epithelial tissue changes in some patients. The changes occur in the nails (Koilonychia or spoon – shaped nails), tongue (atrophic glossitis), mouth (angular stomatitis), and oesophagus causing dysphagia from development of thin, membranous webs at the postcricoid area (plummer – wilson syndrome)

(ii) PROTEIN DEFICIENCY ANEMIA;

Due to deficiency of proteins, the synthesis of hemoglobin is reduced. The RBCs are macrocytic and hypochromic.

(iii) PERNICIOUS ANEMIA (or) ADDISON’S ANEMIA:

It is due to atrophy of the gastric mucosa because of autoimmune destruction of parietal cells. The gastric atrophy results in decreased production of intrinsic factor and poor absorption of vitamin B₁₂ which is the
maturation factor for RBC. The RBCs are larger and immature with almost normal hemoglobin content. So, cells are macrocytic and normochronic.

It was very difficult to treat the patients and the disease was considered to be fatal. So, it was called pernicious anemia. The synthesis of Hb is almost normal in this type of anemia.

Pernicious anemia is common in old age and it is more common in females than in males. It is associated with other autoimmune diseases like disorders of thyroid gland, Addison’s disease etc.,

Clinical Features:

The disease has insidious onset and progresses slowly. The clinical manifestations are mainly due to vitamin B_{12} deficiency. These include: lemon yellow colour of skin (due to anemic paleness and mild jaundice), anaemia, glossitis, neurological abnormalities (neuropathy, subacute combined degeneration of the spinal cord, retrobulbar neuritis), gastrointestinal manifestations (diarrhoea, anorexia, weight loss, dyspepsia), hepatosplenomegaly, congestive heart failure and hemorrhagic manifestations.

(iv) MEGALOBLASTIC ANEMIA:

The megaloblastic anemias are disorders caused by impaired DNA synthesis and are characterised by distinctive abnormality in the
hematopoietic precursors in the bone marrow in which the maturation of the nucleus is delayed relative to that of the cytoplasm. Since cell division is slow but cytoplasmic development progress normally, the nucleated red cell precursors tend to be larger which Ehrlich in 1880 termed megaloblasts. Megaloblasts are both morphologically and functionally abnormal with the result that the mature red cells formed from them and released into the peripheral blood are also abnormal in shape and size, the most prominent abnormality being macrocytosis.

The underlying defect for the asynchronous maturation of the nucleus is defective DNA synthesis due to deficiency of vitamin B\textsubscript{12} (cobalamin) and / or folic acid (folate)

Clinical features :

Deficiency of Vit B\textsubscript{12} and folate may cause the following clinical manifestations which may be present singly or in combination and in varying severity.

1. Anemia :

Macrocytic megaloblastic anemia is the cardinal feature of deficiency of Vit. B\textsubscript{12} and / or folate. The onset of anemia is usually insidious and gradually progressive.
2. Glossitis:

Typically, the patient has a smooth, beefy, red tongue.

3. Neurologic manifestations:

Vit. B₁₂ deficiency particularly in patients of pernicious anemia, is associated with significant neurological manifestations in the form of subacute combined, degeneration of the spinal cord and peripheral neuropathy. While folate deficiency may occasionally develop neuropathy only. The underlying pathologic process consists of demyelination of the peripheral nerves, the spinal cord and the cerebrum signs and symptoms include numbness, paraesthesia, weakness, ataxia, poor finger coordination and diminished reflexes.

4. Others:

Mild jaundice, angular stomatitis, purpura, melanin pigmentation, symptoms of malabsorption, weight loss and anorexia.

(4) APLASTIC Anemia:

Aplastic anemia is defined as pancytopenia (ie. simultaneous presence of anemia, leucopenia and thrombocytopenia) resulting from aplasia of the bone marrow. The red bone marrow is reduced and replaced by fatty tissues. The underlying defect in all cases appears to be sufficient reduction in the number of hematopoietic pluripotent stem cells which
makes them unable to divide and differentiate. But, the RBCs are normocytic and nonmochronic.

**Etiology and Classification:**

Based on the etiology, aplastic anemia is classified into 2 main types: primary and secondary.

A. Primary aplastic anemia:

1. Fanconis anemia (congenital).

   Autosomal recessive inheritance.

2. Immunologically mediated (acquired).

B. Secondary aplastic anemia:

1. Drugs:

   (i) Dose – related aplasia eg. with antimetabolites (methotrexate), mitotic inhibitors (daunorubicin), alkylating agents (busulfan), nitrose urea, anthracyclines.

   (ii) Idiosyncratic aplasia eg: with chloramphenol sulfa drugs, oxyphenbutazone, phenylbutazone, chlorpromazine, gold salts.

2. Toxic Chemicals eg : Benzene derivatives, insecticides, arsenicals

3. Infection eg : infectious hepatitis, EB virus infection, AIDS, other viral illness

4. Miscellaneous eg : associated with SLE and therapeutic X-rays
Clinical features:

The onset may occur at any age and is usually insidious. The clinical manifestations include:

1. Anemia and its symptoms like mild progressive weakness and fatigue.
2. Hemorrhage from various sites due to thrombocytopenia such as from the skin, nose, gums, vagina, bowel and occasionally in the CNS and retina.
3. Infections in the mouth and throat are commonly present.
4. The lymph nodes, liver and spleen are generally not enlarged.

ANEMIA OF CHRONIC DISEASES:

Anemia of chronic diseases is the second common type of anemia. It is characterized by short life span of RBC’s caused by disturbance in iron metabolism or resistance to erythropoietin action. Anemia develops after few months of sustained disease.

Anemia in chronic disorders is usually normocytic normochromic but can have mild degree of microcytosis and hypochromia unrelated to iron deficiency.

1. Anemia in chronic infections / inflammations
   a) Infections eg: tuberculosis, lung abscess, pneumonia, osteomyelitis, subacute bacterial endocarditis, pyelonephritis.
b) Non – infections inflammations eg : Rheumatoid arthritis, SLE, vasculitis, dermatomyositis, scleroderma, sarcoidosis, Crohn’s disease.

c) Disserminated Malignancies eg : Hodgkin’s disease, disseminated carcinomas and sarcomas.

2. Anemia of renal disease :

   Eg : Uraemia, renal failure

3. Anemia of hypometabolic state :

   Eg : Endocrinopathies (myxoedema, Addison’s disease, hyperthyroidism, hypopituitarism) protein malnutrition, scurvy, pregnancy, liver disease.

   The severity of anemia is usually directly related to the primary disease process. The anemia is corrected only if the primary disease is alleviated

Investigations :

   Hemoglobin estimation CBC

   Peripheral blood film examination.

   Red cell indices

   (MCV, MCH & MCHC)

   Leucocyte and platelet count

   Reticulocyte count
ESR

Biochemical investigations

Blood Sugar

Blood Urea

Serum cholesterol

Serum Protein

Serum iron

Total iron binding capacity (TIBC)
MATERIAL AND METHODS

MATERIAL:

A clinical trail on vatha pandu was carried out in Govt. Siddha. Medical College, Palayamkottai.

The 40 cases with clinical signs and symptoms of Vatha Pandu of both sexes of difference age (17-75) groups were selected and studied cases were selected in the department of maruthuvam (OP and IP) clinical parameters.

Cases were selected from out-patient and in-patient ward and the parameters of my care selection were

Symptoms

Looking pale, fatigue, swelling of the body and oedema, feeling of weakness, dry skin, white sclera, general malaise, numbness, glossitis, dyspnoea on exertion. Palpitations, heart murmur, angular stomatitis with soreness, angina, intermittent claudication of the legs, head ache, female abnormal menstruation, decreased energy.

Clinical Examination

Patients were subjected to physical examination on siddha methodology piniyari muraimai. It has 3 main principles which are Poriyalarithal pulanalarithal, vinathal and effected through “Envagai thervugal”.

Coming to the modern methodology a detailed clinical history was recorded and also physical examination was carried out.

All the cases were subjected to following investigation.

• Nadi paritchai, Neerkuri, Neikuri.
• Complete blood analysis TC, DC, Hb.

Total RBC, peripheral blood smear, E.S.R, PCV, MCV, MCH, MCHC.

• Routine urine analysis albumin, sugar, deposit.
• Stools for ova, cyst and occult blood.

Bio chemical analysis of serum cholesterol, blood urea and blood sugar was carried out.

**Method of Treatment**

Siddha system of medicine is based on mukkutra theory and hence the treatment is mainly aimed to bring down the three doshas to its equilibrium, thereby restoring the physiological condition of the three dhoshas.

It is necessary to treat the worm infestation, as is the prime Aetiology for Anaemia “M.V. pills was given (Dose -1- tablet early morning with hot water for deworming the patient.

**Trial Medicine :**

Treatment should be given to neutralise the vitiated mukkutram. The trial medicine used in the present study is

1. Thiratcha Choornam – 2gm திராத்தா் சோரோண்

This medicine was given orally after meals. This medicines was prepared in the postgraduate gunapadam pratical hall with the knowledge
and guidelines of staffs by P.G. Maruthuvam department of Govt. Siddha Medical College, Palayamkottai.

Biochemical analysis and pharmacological studies of the drug was done to know the active principles and actions of trial medicine.
1. **Sex Distribution**

Table – 1 : Illustrates the Sex distribution and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Sex</th>
<th>Out patients</th>
<th>In 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>6</td>
<td>30</td>
</tr>
<tr>
<td>2.</td>
<td>Female</td>
<td>14</td>
<td>70</td>
</tr>
</tbody>
</table>

2. **Age Distribution**

Table – 2 : Illustrates the Age distribution and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Age group in Years</th>
<th>Out patientes</th>
<th>In 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>3</td>
<td>15</td>
</tr>
<tr>
<td>3.</td>
<td>41-50</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>4.</td>
<td>51-60</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>5.</td>
<td>61-70</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>6.</td>
<td>Above 70</td>
<td>1</td>
<td>5</td>
</tr>
</tbody>
</table>

Above table Shows, the incidence is high in age between 61 – 70.
3. **Kaalam**

Table – 3 : Illustrates the Kaalam and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Kaalam</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Vatha Kaalam</td>
<td>4</td>
<td>20%</td>
</tr>
<tr>
<td>2.</td>
<td>Pitha Kaalam</td>
<td>14</td>
<td>70%</td>
</tr>
<tr>
<td>3.</td>
<td>Kapha Kaalam</td>
<td>2</td>
<td>10%</td>
</tr>
</tbody>
</table>

From the above study, the maximum number of cases were treated in Pitha Kaalam.

4. **Constitution of the body**

Table – 4 : Illustrates the Constitution of the body and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Constitution of the body</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Vatha Thegi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Pitha Thegi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Kapha Thegi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Thontha Thegi</td>
<td>20</td>
<td>100%</td>
</tr>
</tbody>
</table>

In all, the patients had Thontha Thegi.
5. **Gunam**

Table – 5 : Illustrates the Gunam and its relative percentage.

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Gunam</th>
<th>Out patientes</th>
<th>In 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 this present study cent percent cases belongs to Rajogunam.

6. **Religion**

Table – 6 : Illustrates the Religion and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Religion</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Hindu</td>
<td>16</td>
<td>80</td>
</tr>
<tr>
<td>2.</td>
<td>Christian</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>3.</td>
<td>Muslim</td>
<td>1</td>
<td>5</td>
</tr>
</tbody>
</table>

In this study, the maximum number of patients were Hindus.
7. **Type of work**

Table – 7 : Illustrates the Type of work and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Type of work</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Sedentary worker</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Moderate worker</td>
<td>17</td>
<td>85</td>
</tr>
<tr>
<td>3.</td>
<td>Hard worker</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>

8. **Socio Economic Status**

Table – 8 : Illustrates the Socio Economic and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Socio Economic Status</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>High Class</td>
<td>17</td>
<td>85</td>
</tr>
<tr>
<td>2.</td>
<td>Middle Class</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>3.</td>
<td>Low Class</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

From the above study, economically poor class peoples were prone to Anaemia.
9. Habits

Table – 9 : Illustrates the Habits and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Habits</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Alcoholic</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>2.</td>
<td>Smoker</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>3.</td>
<td>Tabacco Chewer</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4.</td>
<td>None</td>
<td>16</td>
<td>80</td>
</tr>
</tbody>
</table>

10. Dietary Pattern

Table – 10 : Illustrates the Dietary Pattern and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Dietary Pattern</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Mixed Diet</td>
<td>15</td>
<td>75</td>
</tr>
<tr>
<td>2.</td>
<td>Vegetarian</td>
<td>5</td>
<td>25</td>
</tr>
</tbody>
</table>

From the above study most of the patients were taken mixed diet.
Table – 11: Illustrates the Paruva Kaalam and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Paruva Kaalam</th>
<th>Months</th>
<th>Out patientes</th>
<th>In 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>Kaarkalam</td>
<td>Aavani, Purattasi</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>2.</td>
<td>Koothir Kaalam</td>
<td>Iyppasi, Karthigai</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>3.</td>
<td>Munpani Kaalam</td>
<td>Markazhi, Thai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Pinpani Kaalam</td>
<td>Masi, Panguni</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Elavenil Kaalam</td>
<td>Chitirai, Vaikasi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6.</td>
<td>Muthuvenil Kaalam</td>
<td>Aani, Aadi</td>
<td>9</td>
<td>45</td>
</tr>
</tbody>
</table>

The above table shows most of the patients were treated in Muthuvenil and Karkalam.
12. Thinai

Table – 12: Illustrates the Thinai and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Thinai</th>
<th>Out patients</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Kurinji</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Mullai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Marutham</td>
<td>20</td>
<td>100%</td>
</tr>
<tr>
<td>4.</td>
<td>Neithal</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Paalai</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Above the table shows most of the patients were from marutha nilam.

13. Weight Distribution

Table – 13: Illustrates the Weight and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Weight Distribution</th>
<th>Out patients</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Over Weight</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>2.</td>
<td>Ideal Weight</td>
<td>16</td>
<td>80</td>
</tr>
<tr>
<td>3.</td>
<td>Low Weight</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>
14. Clinical Manifestations features:

Table – 14: Illustrates the clinical manifestations and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Clinical Manifestations</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Pallorness of skin mucous membrane</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>2.</td>
<td>Fatigue / weakness</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>3.</td>
<td>Dyspnoea on Exertion</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>4.</td>
<td>Palpitation</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>5.</td>
<td>Constipation</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>6.</td>
<td>Head ache</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td>7.</td>
<td>Angular Stomatitis</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>
15. **Kosam:**

Table – 15: Illustrates the Kosam and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Kosam</th>
<th>Out patients</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Annamaya Kosam</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>2.</td>
<td>Pranamaya Kosam</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>3.</td>
<td>Manomaya Kosam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Vingnanamaya Kosam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Ananthamaya Kosam</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

In all the patients, Annamayakosam, Pranamaya Kosam affected.
16. **Disturbances in Vatha:**

Table – 16: Illustrates the Disturbances in Vatha and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Vatham</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Pranan</td>
<td>12</td>
<td>60</td>
</tr>
<tr>
<td>2.</td>
<td>Abanan</td>
<td>8</td>
<td>40</td>
</tr>
<tr>
<td>3.</td>
<td>Viyanan</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>4.</td>
<td>Udhanan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Samanann</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>6.</td>
<td>Nagan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Koorman</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8.</td>
<td>Kirukaran</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>9.</td>
<td>Devathathan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10.</td>
<td>Dhananjeyan</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Above table shows most of the cases had altered Abanan, pranan, viyanan, samanan & kirukaran.
17. Disturbances in Pitham:
Table – 17: Illustrates the Disturbances in Pitham and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Vatham</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Analapitham</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Ranjagapitham</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>3.</td>
<td>Prasakapitham</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>4.</td>
<td>Alosagapitham</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Sathagapitham</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Above the table shows most of the cases had altered Ranjagapitham, Prasakapitham.

18. Disturbances in Kapham:
Table – 18: Illustrates the Disturbances in Kapham and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Vatham</th>
<th>Out patientes</th>
<th>In 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>12</td>
<td>60</td>
</tr>
<tr>
<td>2.</td>
<td>Kilethagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Pothagam</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>4.</td>
<td>Tharpagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Santhigam</td>
<td>7</td>
<td>35</td>
</tr>
</tbody>
</table>

Above the table shows most of the cases had altered avalambagam, santhigam, & Pothagam.
19. **Involvement of Ezhu Udalkattugal:**

Table – 19: Illustrates the Involvement of ezhu Udalkattugal and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Vatham</th>
<th>Out patientes</th>
<th>In 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>Seneer</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>3.</td>
<td>Oon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Kozhuppu</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>Enbu</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>6.</td>
<td>Moolai</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Sukkilam / Suronitham</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

From the above study all the patients had altered Saaram, seneer.
20. **Envagai Thervugal:**

Table – 20: Illustrates the Condition of Envagai Thervugal and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Vatham</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Naadi</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>2.</td>
<td>Sparisam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Naa</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>Niram</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>5.</td>
<td>Mozhi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6.</td>
<td>Vizhi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7.</td>
<td>Malam</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>8.</td>
<td>Moothiram</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

All the patients showed Pitha Kalapu Naadi (Pitha Vatham and Vathapitham).
## 21. Neerkuri - Neikuri:

Table – 21: Illustrates the Neerkuri - Neikuri and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Vatham</th>
<th>Out patientes</th>
<th>In patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Cases</td>
<td>Percentage</td>
</tr>
<tr>
<td>1.</td>
<td>Neerkuri (Straw yellow in Colour &amp; Clear)</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>2.</td>
<td>Neikuri</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>a. Aravena neendathuvatham</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>b. Aazhi pol paraviyathupitham</td>
<td>17</td>
<td>85</td>
</tr>
<tr>
<td></td>
<td>c. Muthothu nindrathukabam</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>d. Thonthaneer</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In this present study of neerkuri showed straw yellow in colour and . Most the patients had Patha Neer.
22. Assessment of Result:

Table – 22: Illustrates the Assessment of Results and its relative percentage

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Grade</th>
<th>Out patientes</th>
<th>In 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>10</td>
<td>50</td>
</tr>
<tr>
<td>2.</td>
<td>Fair</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>3.</td>
<td>Poor</td>
<td>3</td>
<td>15</td>
</tr>
</tbody>
</table>

Most of the patients good results. At the end of the treatment the inpatients were discharged and they were advised to follow up treatment in P.G. Department of Maruthuvam O.P. for further management.
Kaalam

Hindu

Vatha Kaalam
Pitha Kaalam
Kapha Kaalam

Religion

Hindu
Christian
Muslim
**Socio Economic Status**

![Socio Economic Status Chart](chart1.png)

**Habits**

![Habits Chart](chart2.png)
Disturbances in Vatha

Assessment of Result
RESULTS

Anaemia is corrected by the administration of medicinal iron. The response is evident within 10 days as improved well being and return of appetite, reduction of various symptoms.

Results were observed on the basis of two main criteria one of the basis of clinical improvement and other results derived from the blood picture. For all practical purpose the beneficial response of Thiratcha Choornam (internally) correcting anaemia may be taken as a suggestive evidence of anemia retrospectively. Results were derived on the basis of the following investigate parameter.

1. Haemoglobin – in gram / dl
2. Packed all volume (percentage)
3. Hb% - in gram / dl
4. Mean corpuscular Volume – MCV (fl)
5. Mean corpuscular haemoglobin – MCH (piccogram)
6. Mean Corpuscular Haemoglobin Concentration (gm/dl) clinical improvement.
1. Breathlessness on exertion was present in all cases was not present in all cases after treatment likewise.

2. Feeling of weakness was present in all cases, before treatment and not present after treatment.

3. Palloriness of skin, mucous membrane was present in all cases, it was Moderately reduced in 16 cases after treatment.

4. Palpitation was present in nine patients moderately reduced in all cases after treatment.

5. Constipation was present in 22 patients was not present in all cases after treatment.

6. Head ache was present in 17 patients and was not present in 17 patient after treatment.

7. Angluar stomatitis was present in 10 patients and was not present in 10 patients after treatment.

**Hematological Report :**

1. At the time of admission haemoglobin percentage was decreased. RBC count was low in majority of the cases.

2. In minority of patient MCV is also reduced.
3. No of days treated was 48 days, the efficacy was established through this period.

4. The raise of haemoglobin was good. (it varied in range between 1gm – 3gm)
DISCUSSION

A small group of 40 patients with complaints of vatha pandu were selected. The diagnosis was again established and confirmed with the help of Envagai Thervugal, modern biochemical investigations like total red blood corpuscles count, haemoglobin percentage, MCHC, MCH, MCV, PCV, peripheral blood smear. These cases were given the trial drug.

During the course of treatment, the patient was subjected to routine investigation in urine, Albumin, sugar, deposits, sp. Gravity in blood urea, serum cholesterol, serum protein, in stools ova, cyst, in order to exclude other disorders. Eventhough anaemia is present in other systemic disorders, infectious disease (e.g. T.B) and with other connective tissue disorders they were excluded.

Age / Sex

The patients selected for my study were between 17-75 years, of both sexes. It depends upon various etiological factors. Regarding sex in this study, occurrence ratio is comparably more in Female than Male.
Occupation, Study and Habit

As shown in table No.2, 32 patients were from poor socio-economic status and 8 from middle class family. Diet with rich nutrient fruits, milk, egg is closely binds to earning of an individual’s income.

Etiology

It is observed that nutritional deficiency was noted in all the 40 cases. A well-balanced diet should include a sufficient amount of vegetables fruits for organic minerals and vitamins, together with other elements in a balanced proportion. Proper care of food and the manner and environment in which it is produced is of great importance in the preservation of health. In this nine patients who are strict vegetarians and due to their poor socio-economic status and ignorance of nutritious diet, they tends to take poor diet, which leads to vatahpandu noi. This is shown in table No 4. PANDU NOI occurs irrespective of religion.

Thinai & Kalam

According to table No. 11 the present study is contrary to what has been mentioned by many ancient siddhars. Marutham is a non-prevalent zone of any diseases, but various environmental changes and changes in the lifestyle in marutham, disease vathapandu noi, which occurs irrespective of
any season. Most of the cases subjected to this study were done in Muthu Vehil Karkalam. Aggravation of signs and symptoms were not observed in any season.
SUMMARY AND CONCLUSION

The research on “Vatha Pandu” was chosen with an intention to give solace to the patients who are suffering from this disease without any noxious effects.

Various literatures having relevant reference to the disease, vatha pandu were collected from both Siddha system as well as in modern system of medicine.

The efficacy of the trial drug “Thiratcha chooranam” was studied and observed during research.

Pandu an ancient clinical entity, with its historical importance correlates to the modern clinical entity Anaemia in every aspects. In “Vatha Pandu” noi chest discomfort, fatigue, dyspnoea on exertion, Palpitation headache palloriness of the skin, tongue, mucous membrane of the lips, palms and hands and conjunctiva, tachycardia are noted. The clinical features observed by various Siddhars perfectly match with clinical features discussed in western text books without any doubt.

The feasibility of considering vatha pandu as anaemia is observed after considering the siddha etiological factors which coincide to the modern aspects.
The diagnosis is established by siddha and modern methodology revealed that Nutritional Deficiency is common cause for America. The incidence is slightly greater in females than males due to their excessive physiological needs in menstrual cycle, lactation, pregnancy.

Regarding age incidence, this disease occurs at any age.

It’ll be seen that sound diet is essential for overcoming disease. Proper care of food and the manner and environment in which it is produced is of great importance in the preservation health.

The clinical trial conducted with 40 patients.

The trial drug is easily tolerable by patients without any untoward side effects.

Biochemistry and pharmacological analysis reveal that the test drugs have significant haematinic effect.
CONCLUSION

The dissertation medicine Thiratacha Choornam and is a potent haematinic drugs for the remedy of Vatha pandu (Anaemia).

Research findings shows that 75% of patients were completely cured and also proved clinically.

The trial drug was very effective to the patients and there was no recurrence of symptoms. Cost of the drug is very cheap and free from side effects. So they are useful for long term purpose.

If a man strictly adhere to the immutable laws derived by Siddhars. He weill lead a healthy and happy life.
ANNEXURE I

PHARMACOLOGICAL ANALYSIS OF TRIAL MEDICINES

THIRATCHA CHOORNAM

<table>
<thead>
<tr>
<th>No.</th>
<th>Medicinal Plant (Scientific Name)</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anacardium Occidentale</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>2</td>
<td>Phonex Dactilifera</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>3</td>
<td>Glycyrrhiza Labra</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>4</td>
<td>Cyperus Rotundus</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>5</td>
<td>Coriandrum Sativam</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>6</td>
<td>Nymphaea Nouchali</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>7</td>
<td>Mristica Fragrans</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>8</td>
<td>Syzygium Maromaticum</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>9</td>
<td>Zingiber Officinale</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>10</td>
<td>Plectranthus Vettiveroides</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>11</td>
<td>Mesua Nagassarium</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>12</td>
<td>Cinnamomum Verum</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>13</td>
<td>Santalum Album</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>14</td>
<td>Bambusa Arundinaceae</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>15</td>
<td>Abies Spectabilis</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>16</td>
<td>Hemidesmus Indicus</td>
<td>1 Balam (35 mgs)</td>
</tr>
<tr>
<td>17</td>
<td>Illicium Verum</td>
<td>1 Balam (35 mgs)</td>
</tr>
</tbody>
</table>
18  பலையைல்வி (SEEDS OF PHYLLANTHUS EMBLCA) - 1 Balam (35 mgs)
19  பென்னியரி (FRIED ORYZA SATIVA) - 1 Balam (35 mgs)
20  சின்னமொம் பலகு (CHINNAMOMUM TAMALA) - 1 Balam (35 mgs)
21  பிப்பர்லு (PIPER LONGUM) - 1 Balam (35 mgs)
22  சசகார் - 1 Balam (35 mgs)

சிளுவார் வகையில் வரும் சர்க்கரை:

சர்க்கரையில் காட்டு பட்டியல்:

1. பெருந்துப்பொி - மெரையாக்கத்தாக அதுகைத் தந்துகைத் தோகாலை

2. களிப்பாட்டுப்பொி - மெரையாக்கத்தாக அதுகைத் தோகாலை

3. அகர்மையான- துடுவாலாம் கேளித் பொில் கேளித் தோகாலை

4. செல்லுமாக்காட்டு - குப்பால் நேர்க்க அனு குப்பால் அனுக்கு

5. அர்ப்பிக்கும் கிழையும் - குப்பால் நேர்க்க குப்பால் அனுக்கு

6. காய்வாதுக குப்பால் - குப்பால் நேர்க்க குப்பால் அனுக்கு

7. குப்பாலாக்கும் - மெரையாக்கத்தாக அதுக்கைத் தோகாலை.

8. மெரையாக்காக்கும் - குப்பால் காலைகாலை குப்பால் அனுக்கு

தோகாலை.
9. குறுக்கு - நூற்றாண்டு தொடர்ந்து கோவைகள் எழுந்து வழிக்கோள் விளம்பம்.

10. நவநிதம் - நூற்றாண்டு தொடர்ந்து கோவைகள் எழுந்து வழிக்கோள் விளம்பம்.

11. தொண்டாங்கி - மதிப்பிறகு எழுந்து கோவைகள் அம்சபெறும் வழிக்கோள் விளம்பம்.

12. நான்காண்டம் - நூற்றாண்டு தொடர்ந்து கோவைகள் எழுந்து வழிக்கோள் விளம்பம்.

13. தொண்டாங்கி - நூற்றாண்டு தொடர்ந்து கோவைகள் அம்சபெறும் வழிக்கோள் விளம்பம்.

14. தோற்காண்டம் - 100 வருடம் நூற்றாண்டு நூற்றாண்டு விளம்பம்.

15. தொண்டாங்கி - நூற்றாண்டு தொடர்ந்து கோவைகள் எழுந்து வழிக்கோள் விளம்பம்.

16. தொண்டாங்கி - தொண்டாங்கி அம்சபெறும் வழிக்கோள் விளம்பம்.

17. குறுக்கு - நூற்றாண்டு தொடர்ந்து பொழுது வழிக்கோள் விளம்பம்.

18. குறுக்கு - தொண்டாங்கி எழுந்து எழுந்து வழிக்கோள் விளம்பம்.

19. குறுக்கு - தொண்டாங்கி கோவைகள் எழுந்து 1 வெளி எழுந்து வழிக்கோள் விளம்பம்.

20. குறுக்கு - தொண்டாங்கி வழிக்கோள் விளம்பம்.

21. குறுக்கு - தொண்டாங்கி எழுந்து வழிக்கோள் விளம்பம்.

22. குறுக்கு - தொண்டாங்கி எழுந்து வழிக்கோள் விளம்பம்.
ஒப்புபட்டால்:

அகத்து காப்பா சுருக்கக்கோளம் குழுவில் இருந்த ஆண்டு தள்ளுகின்றது. அனைத்துப் பொருள்கள் காப்பா குழுவுக்கு வருவது காலகாலியாக தொடர்ந்து வருகிறது. அவற்றுடன் தொடர்ந்து காண்புப் பகுதியில் அகாதத் காப்பா குழுவின் செயல்பாடு செய்யப்படும்.

அந்தால் : 2 கிளைம்

அம்மக்கள் : பூமி

முதல் வருந்தால் காலம் : காலம், காலம் 1 கிளைமல்

கிளைக் குறிப்பிட்டிட : மாசு, மாசு, முக்கியமான காலத்தில், தொகுதியாக, குறிப்பிட்டிட

- அகாதத் குழுவின் 400

1. முக்கியமான திட்டம் : B.N: Anacardium Occidentale

அம்மக்கள் : Anacardiaceae

போதுமத வருந்தால் : விளக்கு

கோட்டம் : இன்னும், கோட்டம் - கோட்டம், பிட்டிய - இன்னும்

போதுமத வருந்தால் : இறைச்சி

விளக்கு கோட்டம், கோட்டம் விளக்கு கோட்டம், விளக்கு கோட்டம்

- கோட்டம்.
2. **凤凰木** : B.N : Phoenix Dactylifera
   
   **性质** : 木
   
   **用途** : 木
   
   **成分** : 钙, 铁 - 7.3mg / 100 gm
   
   It contains Fe - 7.3mg / 100 gm and has found it to be a rich source of vitamin ‘C’ (3mg / 100gm)

   - Wealth of India

3. **桂皮** : B.N : Illicium Verum
   
   **性质** : Magnoliaceae
   
   **用途** : 桂, 老 - 木
   
   **成分** : 木
   
   **性质** : Antivatha, Astrigent, Febrifuge, Nutritive

   - Wealth of India
Alcohol Anethole is the chief constituent.

4. アルコールエーテール : Glycyrrhiza Glabra
    植物名 : Fabaceae
    本属名 : Glycyrrhiza
    属名 : Glabra
    科名 : Fabaceae
5. **கோவிக் கிளையாக்கு** : Cyperus rotundus

**குறிப்பிட்டு** : Cyperaceae

**கும்பக்கு** : கூற்றுப்பினி

- கோவிக் கிளையாக்கு
- கூற்றுப்பினி
- கோவிக்
- கூற்றுப்பினி
- கோவிக்
- கூற்றுப்பினி

பாசிக்கும் பொருள்

- கோவிக் கிளையாக்கு, கூற்றுப்பினி பொருள், கூற்று பிறக், கூற்றுப்பினி, கூற்றுப்பினி, கூற்றுப்பினி, பொருள் பிறக், கூற்றுப்பினி, கூற்றுப்பினி, கூற்றுப்பினி, பொருள் பிறக், கூற்றுப்பினி, பொருள்

fat sugar gum, carbohydarate, esnential oil, alpuminous matter, starch fibr and ash.

-Hand book on medicinal herp with uses.

6. **மார்க்கத்தார் கிளையாக்கு** : B.N : Coridandru Sativum

**குறிப்பிட்டு** : Apieaceae

**காலை** : காலை, காலை புது, காலை புது

**பிணியு** : காலை

**வெப்பைத்து** : பொருள் விளையாட்டு

7. Scientific Name: B.N: Myristica Fragrans

Common Name: Myristicaceae

Family: Myristicaceae

Description: B.N: Myristica Fragrans

moisture – 25.2, protein – 5.2, fat – 8.9, fibre-9.5, carbohydrate -46, and mineral matter – 5.2%, Calcium -740, Phosphorus -100, and iron – 4.9mg, iodine-50mg, vitamin – carotene thamine-0.08, and riboflavin-0.13mg, nicotinic acid is alsopresent.

-The Hand Book on medicinal herps with uses
9. **Nymphae Nouchali, Brum**

- **Family:** Nymphaeaceae
- **Active Constituents:** Air roots – Essential Oil P-Methoxy Salicylic aldehyde as the major constituents. Others B-Sitosterol, and amyrin, lupuol terracyclic trtpene alcohols, small amount of resin, fatty acids, tannins, scaponin, a glycoside
11. துளைநாய்க்கப் பூக்குளி : Cinnamomum Famala

அமர்சம் : Lauraceae

கலை : கரட்பு, நிலிம - நிலம்பு

பொருள் : கரட்பு

சதுவால் : நிலம்பு முண்பாக்கக்கலை

அதிசயம் வருளும்

பொருள்கிளைநுண்ணைப்

சிம்மகைசூரி

பருத்தல் : 

பெருநாய்க்க, குருநாய்க்க, நஞ்சனா, குருநாய்க்க, கிருந்தை, குருநாய்க்க, கோலாலம், கான்பு, கரட்பு, நிலம்பு நிலம்பு

சியமமான சிறந்தம்.

Cinnamic, acid, linaloy eugenol, Acitate, Benzaldehyde, Camphor, Cadinane, Terpineol, Geraniol, Pebzol acetate

12. ஆபில்ஸ்பெக்டேபிலிஸ் : Ables Spectabilis

அமர்சம் : Pinaceae

பசைந்தை என்பது : திறை

கலை : கரட்பு, நிலிம, நிலம்பு,

பொருள் : கரட்பு

சதுவால் : பருத்தல்

அதிசயம் வருளும்
The Leaves contain nitrogen 1.44, and Ash – 4.06, the also contain and alkaloid 0.16% and essential oil.

-The Hand book on medicinal herps with uses.

13.  பிபர் லங்கம் : Piper longum
     பிபரையை : Piperaceae

மரபிக்குச் சம்பவம் : காப்பு, ஆணிக்

கலை : தோலைக்காட்டு, காண்டை – காப்பு

பிரிவு : தனிப்பு

ஊர்மங்கள் : ஊர்மப்பழங்களாகிய

அரசன் அரசின்

மாதாணம் :

சலை, தூளம், தூரிப், தூரிப் முதல் கொண்டு மாதாணம் காண்கை நேரடியாக ஆரம்பம் பற்றிய விளக்கங்கள்.

N hexadecane n- heptadecane n-octa decane n- nanodeana n-eicosane terpinonane zingiperane symaney phenathlyalchol
14. **Name of the plant**: Bambusa arudinaceae  
**Family**: Bambusaceae

- **Uses**: It is used for making furniture, utensils, and building materials. It is also used in traditional medicine for various purposes.

- **Nutritional content**: It contains protin, fat, carbohydrates, minerals, calcium, phosphorus, iron, magnesium, sodium, copper, Chlorin, Vitamin, Riboflavin, niacin, choline.

15. **Name of the plant**: Oryza Sativa Linn  
**Family**: Poaceae

- **Uses**: It is used for making rice, which is a staple food in many parts of the world. It is also used in traditional medicine for various purposes.

- **Nutritional content**: It contains protin, fat, carbohydrates, minerals, calcium, phosphorus, iron, magnesium, sodium, copper, Chlorin, Vitamin, Riboflavin, niacin, choline.
16. மேலி பொருள் : Mesua naga Ssarium

நன்றி : Clusiaceae

பெயரும் தலைமு : மரக்க பு

கலை : பிறகுசம்பாயம்

தோல் : பும்

பிளிளு : காம்பு

வெப்பங்கள் : காம்பு, வெப்பங்கள்

அதைப்படி அறிந்து

பிள்ளைத்தொலை : 

பற்றிப் பெயர் அடையும், பல வெப்பங்கள், கரையாற்றம்

அதிகம் விளக்கம்.

17. கர்ணகம் : Santalum Album

நன்றி : Santalaceae

பெயரும் தலைமு : வரு, கர்நூல்

கை : காம்பு, பிறகுசம்பாயம்

சுமுகம் : பும், தொலை

பிளிளு : தொலை, மாம்பு

வெப்பங்கள் : பிறகுசம்பாயம்

பற்றிப் பெயர் அடையும்

அதைப்படி அறிந்து

கரையாற்றால்

அதிகம் விளக்கம்
18. கதை : Zingiber Officinale

கிரியம் : Zingiberaceae

மப்புறை ஒளி : கற்பேழ் (சணகாலம்கு)

கலை : கால்பு, குள்ளம் - ஓலம்

பிளியு : கால்பு

இயற்கையை : ஓலம்பொழுதல்கை

மரநியல்கை : கண்டலாணா கரியாராய, கர்த்திகியால், புதியோப்பால், ஓலம், கிரியம் ஓலம், கொளை, கொளம், அதிர்க்கள், மின்றும், கல்லால், குழ்த்தும்போனை, கண்டல் கொள்ள ஓலம், கொளம், இன்றும் குழம்பை, சூர்யகிங் ஓலம்.
19. **Cinnamomum Verum Presi**
   - Family: Myrtaceae
   - Common Name: Cinnamon

20. **Phyllanthus Emblica**
   - Family: Euphorbiaceae
   - Common Name: Amla
21. ஓலைப்பற்று : Plectranthus vettiveroides

கிரிப்பல் : Labiaceae

மலாமுரிய வகையான : நீலம்

கலை : கருப்பு, நீலம் - நீண்டு, பிற்பகு - வெளிப்பு

நேரம் : கனிமச்சுற்று மலைச்சாலை

பட்டியல் : 

கிரிப்பல், ஓலைப்பற்றல் பிரிவில், கலையிலானால், கனிமச்சுற்றுமுனையர், 

நேரம் வட்டமான, நீண்டு மச்சாலை, நேரம் மலைச்சாலை நீண்டு வெளியும் விலங்கு.

22. பாறைகள்
ANNEXURE II

BIO-CHEMICAL ANALYSIS OF THRATCHA CHOORANAM

PREPARATION OF THE EXTRACT

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

QUALITATIVE 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>TEST FOR CALCIUM</td>
<td>A white precipitate is formed</td>
<td>Indicates the presence of Calcium.</td>
</tr>
<tr>
<td></td>
<td>2ml of the above prepared extract is add 2ml of 4% ammonium oxalate taken in a clean test tube. To this Solution.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>TEST FOR SULPHATE</td>
<td>A white precipitate is formed</td>
<td>Indicates the presence of sulphate</td>
</tr>
<tr>
<td></td>
<td>2ml of the extract is added to 5% Barium chloride solution.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>TEST FOR CHLORIDE</td>
<td>A white precipitate is formed</td>
<td>Indicates the presence of chloride</td>
</tr>
<tr>
<td></td>
<td>A white precipitate is formed</td>
<td>The extract is treated with Silver nitrate Solution.</td>
<td></td>
</tr>
<tr>
<td>Test</td>
<td>Substance Tested</td>
<td>Description</td>
<td>Result</td>
</tr>
<tr>
<td>------</td>
<td>------------------</td>
<td>-------------</td>
<td>--------</td>
</tr>
<tr>
<td>4. TEST FOR CARBONATE</td>
<td>The substance is treated with Concentrated HCL</td>
<td>No Brisk effervescence is formed</td>
<td>Absence of Carbonate</td>
</tr>
<tr>
<td>5. TEST FOR STARCH</td>
<td>The extract is added with weak iodine solution.</td>
<td>Blue Colour is formed</td>
<td>Indicates the presence of starch</td>
</tr>
<tr>
<td>6. TEST FOR FERRIC IRON</td>
<td>The extract is acidified with Glacial acetic acid and add Potassium ferro cyanide</td>
<td>No Blue colour is formed</td>
<td>Absence of Ferric Iron</td>
</tr>
<tr>
<td>7. TEST FOR FERROUS IRON</td>
<td>The extract is treated with Concentrated nitric acid and Ammonium thic cyanide solution.</td>
<td>Blood red colour is formed</td>
<td>Indicates the presence of ferrous Iron</td>
</tr>
<tr>
<td>8. TEST FOR PHOSPHATE</td>
<td>The extract is treated with ammonium Molybdate and concentrated nitric acid</td>
<td>No yellow precipitate is formed</td>
<td>Absence of Phosphate</td>
</tr>
<tr>
<td>9. TEST FOR ALBUMIN</td>
<td>The extract is treated with Esbatch’s Reagent</td>
<td>No yellow precipitate is formed</td>
<td>Absence of Albumin</td>
</tr>
<tr>
<td>10. TEST FOR TANNIC ACID</td>
<td>The extract is treated with ferric Chloride</td>
<td>No blue black precipitate formed</td>
<td>Absence of Tannic acid</td>
</tr>
</tbody>
</table>
| 11. TEST FOR UNSATURATION | Pottassium permanganate | It gets decolourised | Indicates the presence of an
<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>TEST FOR THE REDUCING SUGAR</td>
<td>colour change occurs</td>
</tr>
<tr>
<td></td>
<td>5ml of Benedict’s qualitative Solution is taken in a test tube and allowed to boil for 2 mins and add 8-10 drops of the extract and again boil it for 2 mins</td>
<td>Indicates the presence of Reducing sugar</td>
</tr>
<tr>
<td>13</td>
<td>TEST FOR AMINO ACID</td>
<td>Violet Colour is formed</td>
</tr>
<tr>
<td></td>
<td>One or two drops of the extract is placed on a filter paper and dried it well. After drying 1% Ninhydrin is sprayed over the same and dried it well.</td>
<td>Indicates the presence of Amino Acid</td>
</tr>
<tr>
<td>14</td>
<td>TEST FOR ZINC</td>
<td>No white precipitate is formed</td>
</tr>
<tr>
<td></td>
<td>The extract is treated with Pottassium ferrocyanide</td>
<td>Absence of Zinc</td>
</tr>
<tr>
<td>15</td>
<td>TEST FOR THE COPPER :</td>
<td>A brown precipitate is formed</td>
</tr>
<tr>
<td></td>
<td>The extract is treated with acetic acid and potassium Ferrocyanide are added.</td>
<td>Copper is conformed.</td>
</tr>
</tbody>
</table>
ANNEXURE III

PHARMACOLOGICAL ANALYSIS OF TRIAL MEDICINES

Study on the Haematinic effect of

Thiratcha Choornam

Variety of preparations in siddha system of medicine are well known for its haematinic effects, of which Thiratcha Choornam is one of the best. To provide the efficacy of Thiratcha Choornam an attempt was made to study its effect using “Albino rats”. For this purpose, rats were made anaemic by the following procedure.

Artificially Induced Anaemia

The albino rats taken for this experiment, were kept in aluminium cages and provided with drinking water and milk. The administration of the Thiratcha Choornam under investigation was started, when the hemoglobin level fell to 6 to 6.5 gram / 100ml. At the beginning of the experiment, RBC count, Hb% were determined.

I. Study of Rates

The albino rats were first divided in two equal groups, with three rats in each group. The first group received water. The 2nd group received the drug at a dose of 100 mg / 100gm body weight. All the above procedures
were continued for six weeks in once a day dose. The Haemoglobin levels or rats were measured I, II, III, IV weeks. The results observed are tabulated in the following chart.

**STUDY OF HAEMATINIC ACTION**

**Thiratchai Choornam**

<table>
<thead>
<tr>
<th>Serial No</th>
<th>Drugs</th>
<th>Dose</th>
<th>Before drug Administration</th>
<th>After Drug Administration</th>
<th>Remarks</th>
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<tr>
<td></td>
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<td>Initial Reading</td>
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<tr>
<td>1</td>
<td>Control (Water) 2ml</td>
<td>2 ml</td>
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<td>6.2</td>
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<td></td>
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<td>6.0</td>
<td>6.0</td>
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<td>6.2</td>
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<td>6.2</td>
<td>6.1</td>
</tr>
<tr>
<td>2</td>
<td>Tiratchai Choornam 100 mg</td>
<td>100 mg</td>
<td>6</td>
<td>6.0</td>
<td>6.2</td>
</tr>
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<td></td>
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<td>6.4</td>
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<td>6.4</td>
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<td>6.6</td>
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**Result:**

The trial drug Tiratcha Choornam has got significant haematinic action.
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<th><strong>Nationality</strong></th>
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<td><strong>Name</strong></td>
<td><strong>Date of Admission</strong></td>
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<tr>
<td><strong>Age</strong></td>
<td><strong>Date of Discharge</strong></td>
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</tr>
<tr>
<td><strong>Sex</strong></td>
<td><strong>No. of days treated</strong></td>
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<tr>
<td><strong>Occupation</strong></td>
<td><strong>Diagnosis</strong></td>
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<tr>
<td><strong>Income</strong></td>
<td><strong>Results</strong></td>
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<td><strong>Address</strong></td>
<td><strong>Medical Officer</strong></td>
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<th><strong>Complaints &amp; Duration</strong></th>
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<th><strong>Family History</strong></th>
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CLINICAL EXAMINATION - SIDDHA SYSTEM

PORIPULANGAL:

Sensation:

Tate:

Sight:

Smell:

Touch:

KANMEDRIYAM:

Kai:

Kaal:

Vaai:

Eru Vaai:

Karu Vaai:

Gunam:

Sathuvam:

Rajo:

Thamo:

Nilam:

Kurinchi:

Mullai:
Marutham :
Neithal :
Palai :
YAKKAI :
Vatham :
Pitham :
Kabam :
Kalappu :
PARUVAKALAM:
Kar :
Koothir :
Munpani :
Pinpani :
Elavenil :
Mudhuvenil :
UTKAYAM, ADAHAKAYAM :
Puyam :
Sayam :
Kaal :
Patham :
Karuvaai  :
PIRA URUPPUKALIN NILLAI :
Eruthyam  :
Puppusam  :
Kalleeral  :
Manneeral  :
Kudal  :
Siruneeragam  :
Mummalam  :
Viyarvai  :
Malam  :
Moothiram  :
MUKKUTRANGAL  :
VATHAM  :
Pranan  :
Abanan  :
Viyanan  :
Uthanan  :
Samanan  :
Nagan  :
Koorman :  
Kirukaran :  
Devathathan :  
Dhananjeyan :  
PITHAM :  
Analam :  
Ranjagam :  
Pirasagam :  
Aalosagam :  
Sathagam :  
KABAM :  
Avalambagam :  
Kilaethagam :  
Pothagam :  
Tharpagam :  
Sandhigam :  

ENVAGAI THERVUGAL :  
Na :  
Niram :  
Mozhi :  
Vizhi
Sparisam
Malam
Niram
Edai
Ilagal
MOOTHIRAM
NEERKURI
Niram
Edai
Manam
Nurai
Enjal
NEIKURI
Vatham
Pitham
Kabam
NAADI
EZHU UDARKATTUGL
Saram
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<td>Kozhuppu</td>
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<td>Enbu</td>
<td></td>
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<td>Moolai</td>
<td></td>
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<td>Sronitham</td>
<td></td>
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<tr>
<td>Sukkilam</td>
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</tbody>
</table>
GENERAL EXAMINATION

Consciousness : General appearance :

Stature : Height :

Weight : Nourishment :

Skin Changes : Facies :

Anaemia : Jaundice :

Cyanosis : JVP :

Pedal Oedema : Engorged Venis :

Abdominal distension : Premature Cataract :

Congenital anomaly :

Temperature :

Pulse : Rate Rhythm Volume Character

Blood pressure : mm/Hg (R) (L)

U.L.

L.L.

Respiratory Rate :

Heart Rate :
SYSTEMIC EXAMINATION

Examination of Cardiovascular System : 
Examination of Respiratory System : 
Examination of Abdomen : 
Examination of Central Nervous System : 

Laboratory Investigations

Blood

TC
DC  P   L   E   B   M
Hb%
RBC Count
Platelet Count
Reticulocyte count
PCV
MCV
MCH
MCHC
Serum Protein
Serum Cholesterol
Blood Urea
Peripheral blood smear

ESR ½ hr

1 hr

Motion

Ova

Cyst

Occult Blood

URINE

Albumin

Sugar

Deposit

Bile Salt

Bile Pigment

SPECIAL INVESTIGATION

1. Bariummeal and endoscopy

2. Bone marrow examination

3. Skiagram

4. Sputum for AFB:

5. Radiological Investigation:
6. Ophthalmoscopic Examination:

7. E.C.G.

DIFFERENTIAL DIAGNOSIS:

CASE SUMMARY:

DIAGNOSIS:

PROGRESS NOTE:

<table>
<thead>
<tr>
<th>Date</th>
<th>Complaints</th>
<th>Hb%</th>
<th>Wt.Kgs</th>
<th>Drug</th>
<th>Complications</th>
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</tbody>
</table>
PROFORMA OF VATHA PANDU
(ANAEMIA)

Name of the medical unit:
I.P No:
Name: Date of Admission:
Age / Sex: Date of Discharge:
Sex:
No. of days treated:
Occupation:
Diagnosis:
Income:
Results:
Nationality / Religion:
Medical Officer:

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<th>Patient condition at the time of discharge</th>
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<tr>
<td>Signs :</td>
<td>Signs :</td>
</tr>
<tr>
<td>G/E :</td>
<td>G/E :</td>
</tr>
<tr>
<td>Weight :</td>
<td>Weight :</td>
</tr>
<tr>
<td>Blood Pressure :</td>
<td>Blood Pressure :</td>
</tr>
<tr>
<td>Blood : Hb%</td>
<td>Blood : Hb%</td>
</tr>
<tr>
<td>TRBC %</td>
<td>TRBC %</td>
</tr>
<tr>
<td>PCV</td>
<td>PCV</td>
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<tr>
<td>MCHC</td>
<td>MCHC</td>
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<tr>
<td>MCV</td>
<td>MCV</td>
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<td>Serum Cholesterol : Blood Smear</td>
<td>Serum Cholesterol : Blood Smear</td>
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<td>Serum Protein</td>
<td>Serum Protein</td>
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<tr>
<td>Occult Blood :</td>
<td>Occult Blood :</td>
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BIBLIOGRAPHY

SIDDHA TEXTS

- Yugi Vaidhya Chinthamani – 800
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- Jeevarakshmirtham
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- HUTCHISON’S CLINICAL METHODS
- DAVIDSON’S PRINCIPLES AND PRACTICE OF MEDICINE
- BAILEY AND LOVE’S SHORT PRACTICE OF SURGERY
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<th>Name</th>
<th>Age / Sex</th>
<th>No of days</th>
<th>Hb gm/d</th>
<th>RBC Million</th>
<th>PCV</th>
<th>MCV</th>
<th>MCH</th>
<th>MCHC</th>
<th>Peripher al blood</th>
<th>Hb gm/dl</th>
<th>RBC Million</th>
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<td>68</td>
<td>22</td>
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<td>4.88</td>
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<td>MH</td>
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<td>33</td>
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AOD - Date of Admission  
TC - Thiratchai Chooranam  
DOD - Date of discharge  
NODO- Number of days Treated
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