A STUDY ON

NARITHALAIVADHAM

Dissertation Submitted To

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DOCTOR OF MEDICINE (SIDDHA)

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DEPARTMENT OF NOI NAADAL

Government Siddha Medical College

Palayamkottai – 627 002

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INTRODUCTION

The word ‘Siddha’ comes from the word ‘siddhi’ which means ‘an object to be attained’ or ‘perfection’ or heavenly bliss’. Siddhi generally refers to Astama siddhi (ஏழு வாயில்) i.e., the eight great supernatural powers which are enumerated as Anima (ஆணிமை) etc.. Those who attained or achieved the above said powers are known as siddhars.

The siddhars were further the greatest scientists in ancient times. They were men of highly cultured intellectual and spiritual faculties combined with supernatural powers. Their works in tamil are supposed to be more valuable than many that have been written in Sanskrit. They contain a large number of valuable formulae and exhibit further minute enumerations of morbid symptoms.

As per siddha text, man is said to be the microcosm and the world the macrocosm; because what exists in the world exists in man; so man must be looked upon as an integral part of nature and not as anything separate from the latter. Further, the forces in the microcosm or man are identical with the forces of macrocosm or the world the natural forces acting in and through the various organs of the human body are intimately related is the similar or corresponding force acting in and through the organisms of the world.
In the organisms of man, these forces may act in an abnormal manner and cause diseases thereby.

Humoural pathology explains that man is mixture of the three cardinal humours viz Vali, Azhal, Iyam and that the relative proportion of these humours are responsible for a person’s physical and mental qualities and dispositions.

Disease, According to modern science is only a departure from a state of health and more frequently a kind of disturbance of the healthiness of the body to which any particular case of sickness is assigned. According to Siddhar’s philosophy, diseases in man do not originate himself, but influences which act upon him. As already stated, man is compared to the world, so any change in the elementary condition of external world has its corresponding change in the human organism. There is the feeling of oneness between the external and the internal world of man; and its is upon this oneness that the doctrine of humoural pathology i.e. the theory of Tridoshas (திருதில்லிய) is based. This may occur through different causes viz.
1. Derangement of the three humours
2. Astral influences
3. Poisonous substances
4. Psychological causes
5. Spiritual causes
6. Diseases originating from the soul

In Yugi vaithya chindamani, Yugi described 80 types of vadha diseases “Narithalaivadham” is one of the entity of the vadha diseases.
Man is not merely made up of muscle, bones and nerves as we think. According to siddhar’s thought,

Man (Microcosm) is having himself all the things within the universe (Macrocosm) like muscle, bones and nerves; both mind and soul are the part of the body.

Human body as made up of 2 kinds of bodies.

i. Sthula Sariram (visible body)

ii. Sukkuma Sariram (invisible body)

**Sthula Sariram includes,**

Bones, Muscles, Blood vessels, Nerves and all functional system of human body. It is known as functional units of body.

**Sukkuma Sariram,**

This is the basic for the Suthala Sariram. It makes the Sthula Sariram to be active.

The universe is made up of five basic elements called.

- **Earth (Prthivi)** - பரத்தி
- **Water (Appu)** - புதி
- **Fire (theyu)** - திய
- **Air (vayu)** - வாயு
- **Space (Aagayam)** - வெளியதா

4
As we said before the human body is also made up of these five basic elements.

The basic elements exists in two forms

i. Sthula form (பொன்னூரியல்) - Recognized by our sense

ii. Sukkuma form (செயூரியல்) - Not recognized by our senses

**Physiology [→] Basic process underlying the functioning of the species.**

A basic thing for functioning of human beings explained by siddhars includes.

- 96 Thathuvangal
- 7 Udalkattukal
- 6 Suvaigal

The factors which influence in functioning of human body are

- Udal vanmai
- Udal thee

Siddhars explained physiology on the bases of 96 thathuvangal (or) structural units. This explains the physical and chemical factors that are responsible for the origin, development and progression of life. They are as,
Pancha poothas

Earth - மழை

Water - கிளை

Fire - மனித்

Air - காற்று

Space – வெப்பம்

These five elements contribute our whole body structure and function.
Human Body [Built by Panchapoothas]  
[96 Thathuvagal]

External Thathuvas  
[Sthula Sariram]

(i) Gnanaenthiryam[5]  
[Five Sense Organs]

(ii) Pori 5  
[Functions of five Sense organs]

(iii) Kanmaenthiriyam-5  
[Functional Organs]

(iv) Kanmavidayam  
[Functions of Kanma enthriyam]

Internal Thathuvas  
[Sukkuma Sariram]

i) Anthakaranam – 4
ii) Arivu – 1
iii) Naadi – 10
iv) Vayu – 10
v) Aasayam – 5
vi) Kosam – 5
vii) Aatharam – 6
viii) Mandalam – 3
ix) Thodam – 3
x) Malam – 3
xi) Edanai – 3
xii) Gunam – 3
xiii) Vinai – 2
xiv) Raagam – 8
xv) Avathai -5
7 Udalkattukal

1. Saaram - It enriches the functions of body and mind
   (Energy through food stuffs)

2. Senneer (Blood) - It makes the basic functions of body perfectly

3. Oon (muscles) - It gives structure to our body and gives
   supports to joints

4. Kozhuppu (Fat) - Gives lubrication to our body organs to move.

5. Enbu (Bone) - It gives skeletal structure to body and protection

6. Moolai(Bone marrow)- It gives stability to bone

7. Venneer (Sexual full) - It helps to produce the new generation

Aarusuvaigal - We get from foods

   It has linked to uyirthathu, panchapootham, and body functions

- கைதுப்பு (Sweet) - Mann + Neer       Vali - Vayu +Mann
- முத்திப்பு (Sour) - Mann +thee       Azhal - Thee
- உப்பு (Salt) - Neer + thee         Iyam - Neer
- தக்து (Bitter) - Vayu + Aagayam
- காத்து (Pungent) - Vayu + thee
- கைலையுப்பு (Astringent)- Mann + Vayu
Any alteration takes place in suvaigal. It affects the uyirthathu and body functions.

Aurusvai (Any alterations)

Uyirthathukkal (Alterations)

Diseases (Noi)

**Udalthee**

It is our body’s core temperature or BMR (Basal metabolic rate)

Udal thee (Core temperature)

Increases      Normal         Decreases

Thin body built     Healthy body  indigestion
Increased appetite      and related diseases

**Udal vanmai (Innate immunity)**

It is genetically transmitted from parents and also get throw our environment.

When any alteration takes place in immunity diseases occur.

In Narithalaivadham physiological functions of body (sthula, sukkuma) is altered.
This poem indicates the importance of diagnosing pathology of the diseases.

The Greek word pathology means.

Pathos (or) Noi - “Disease”

Logos (or) Naadal - “To know the cause”

According to siddhar’s thought Noi (disease) is caused by alteration takes place in Uyirthathu by factors,
An alteration in Uyirthathu is caused by

i) Dietary factors

ii) Seasonal and environmental changes

iii) Genetic factors

iv) Immoral activities

v) Suppression of reflexes

vi) Microorganism

Above these factors will cause the disease (changes in Uyirthathu.)

These changes reflected in 7 Udalkattukkl and display the symptoms of disease.
Dietary factors. 
Seasonal, environment changes 
Suppression of reflexes  
 Genetic factors  
 Immoral activities  
 Germs

**Causes for disease**  
(Noi muthanadal)

Produces changes in Uyiruthukkal

Affected Uyiruthu  
(Vali, Azhal. Iyam)

Alteres 7 Udal thathukkal

Diseases occur

Envagai thervu (Stools of diagnosis)

**Diagnosis**  
Noi naddal

**Siddha system of Pathology**  
(Noi Naadal)

Diagnosis
First, we have to rule out the causes for diseases and pathogenesis

**Causes for diseases**

(i) **Dietary Factors**

We take the food in the form of Aru suvaigal.

```
Altered Aru suvaigal (food) →
Altered Uyirthathu →
Diseases
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(ii) **Seasonal variations (Paruvakaalam)**

1. **kaarkaalam (கார்காலம்)** - இணையினி, புள்ளியினி

   All three Uyirthathus are affected
   - Vali: Aggravated
   - Azhal: Accumulated
   - Iyam: Slightly change
2. Kootheir kaalam (கூத்தைக் காலம்) - வட்டமி, கரக்கிரீக

Vali - Normal
Azhal - Aggravated
Iyam - Normal

3. Munpanikaalam (முந்பாப்பாக்கிய காலம்) - பாரக்கி, காக்கி

Vali
Azhal - Neutralized
Iyam

4. Pinpanikaalam (பிந்தப்பாக்கிய காலம்) - பாரக்கி, பார்க்கி

Iyam - Accumulated

5. Elavenieerkaalam (ஏளவேணிக்கிய காலம்) - கிரீக்கிய, காக்கி

Iyam - Aggravated

6. Muthuveneerkaalam (முதுவேணிக்கிய காலம்) - அுளிக்கி, அுந்து

Iyam - Normal
Vali - Accumulated

Environmental Changes

There are 5 types of places in siddhar’s aspect

kurunchi (குருங்கி) - Iyam accumulated, Liver, Abdominal mass will develop

Mullai (முல்லை) - Vali, Azhal diseases may develop

Marutham (மாருதம்) - Place for human beings
Neithal (เนียล์) - Vali diseases, liver enlarged, Flatulence may develop

Paalai (เคช่า) - Not suitable for human living all diseases may develop

(iii) Genetic factors

"รามปุสิ่งษีสัมพันธ์มิตร บ่มนิADERD ภัยกิจ"

According this poem genetic factor are determine the human’s life and diseases.

In siddha aspects these diseases are called Kanma noikal (กันม่านิโคือล)

In siddha system many disease are said to be precipitated by kanmam which the deeds committed by an individual in his previous and the present births. The genetic dispositions of certain disease are probably the result of Kanmam. Vatha disease according “Agasthihar kanma kaandan 300” verse- 56 may also be precipitate by kanmam.
Cutting or denuding of green young living trees, breaking the legs of living beings, etc lead to vadha disease. These deeds are determined to the fellow beings and such psychosocial aspect of an individual implies psychogenesis of Vadha disease.

(iv) Immoral activities

In siddha aspect it is the don’ts and does of humans.

Eyamam (தினம) - Self restricts don’ts

Niyamam (நியமம) - Do’s

When who skip the dos and don’ts they will suffer by diseases.

(v) Suppression of reflexes

In Siddha aspect they are 14 reflexes. Suppression of these reflexes causes diseases.
These reflexes are mentioned below,

<table>
<thead>
<tr>
<th></th>
<th>Tamil Word</th>
<th>English Translation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Abana vayu (அபனவயு)</td>
<td>Flatus</td>
</tr>
<tr>
<td>2.</td>
<td>Thummal (தும்மல்)</td>
<td>Sneezing</td>
</tr>
<tr>
<td>3.</td>
<td>Siruneer (சிருநீர்)</td>
<td>Urine</td>
</tr>
<tr>
<td>4.</td>
<td>Malam (மலம்)</td>
<td>Stools</td>
</tr>
<tr>
<td>5.</td>
<td>Kottaavi (கோட்டாவி)</td>
<td>Owning</td>
</tr>
<tr>
<td>6.</td>
<td>Pasi (பசி)</td>
<td>Hunger</td>
</tr>
<tr>
<td>7.</td>
<td>Thagam (தகாம்)</td>
<td>Thirst</td>
</tr>
<tr>
<td>8.</td>
<td>Erumal (ஏருமால்)</td>
<td>Cough</td>
</tr>
<tr>
<td>9.</td>
<td>Elaippu (எலாய்பு)</td>
<td>Rest</td>
</tr>
<tr>
<td>10.</td>
<td>Thookkam (துக்காம்)</td>
<td>Sleep</td>
</tr>
<tr>
<td>11.</td>
<td>Vaanthi (வாங்டிக்)</td>
<td>Vomiting</td>
</tr>
<tr>
<td>12.</td>
<td>Kanneer (காஞ்சிக்)</td>
<td>Tears</td>
</tr>
<tr>
<td>13.</td>
<td>Sukkilam (சுக்கிலாம்)</td>
<td>Semen</td>
</tr>
<tr>
<td>14.</td>
<td>Uyirppu (உயிர்ப்பு)</td>
<td>Breathing</td>
</tr>
</tbody>
</table>
(vi) Micro organism

Some diseases are caused by micro organisms (Kirumi) According to siddhar’s thought

“குறிச்சை வண்டிகதும் நுயர்கொன்றி
மூலையும் விரைந்துகூரும் குருப்புக்
மற்றும் மூலையும் வண்டிகவும் குறிச்சை
புத்துயிர்ப்பு காட்டுத்து குறிச்சை
நூற்றூற்றும் புந்தரிக்கும் குறிச்சை
நூற்றூற்றும் காட்டுத்து குறிச்சை
புத்துயிர்ப்பு காட்டுத்து குறிச்சை
குறிச்சை வண்டிகதும் நுயர்கொன்றி விரைந்து.”

- குறிச்சை வண்டிகது

Above this poem explains

Anaemia, skin diseases, venereal diseases, urticarial rashes and fistula they are caused by micro organism (Kirumi)

Disease(Altration in Uyir,Udalthathu)

Altertations in Uyirthathu

Vali Thodam

- Darkness of motion
- Body pain
- Pricking pain
- Constipation
- Paralyzed limbs
- Mental disorders
Difficult in work

- Decreased
- Impairment of intelligence
- Giddiness

**Azhal thodam**

- Increased kapha symptoms
- Yellowish discoloration skin, urine
- Increased appetite
- Increased Thirst
- Burning Sensation
- Decreased sleep.

**Iyathodam**

- Loss of appetite
- Indigestion
- Cold

- Chills with Rigor
- Pallor
- Cough
- Fullness of stomach
- Excessive sleep
- Dysphonic
Destruction of joint

Giddiness

Decreased

Decreased Iyam in all body fluids

Increased sweating

Palpitation

7 Udalthathukkal

When food enters the body, it enriches Udalthathukkal one by one.

Food

1st Day (Rasa) - Plasma

2nd Day (Raktha) - Blood

3rd Day (Mamsa) - Muscles

4th Day (Kozhppu) - Fat, Lymph

5th Day (Asthi) - Bones, Cartilages

6th Day (majja) - Bone marrow

7th Day (Sukkila) - Reproductive fluids

Gives energy to body

If any causes affect this process, diseases will occur.
1. Saaram (Rasa)

It is the essence of digested food, circulating all over the body and gives energy to body.

**Increased State**

* Excessive Salivation
* Sound intolerance
* Excessive sleep etc..

**Decreased state**

* Wasting of muscles
* Weakness etc..

2. Senneer (Raktha)

It is produced from rasa. It is responsible for substance of life and provides colour and complexion to the skin.

**Increased state**

* Haemangiomas
* Spleenomegaly
* Leprosy
* Jaundice
* Nervous weakens
* Mental disorders
* Blood dyscariasis
* Hyper pigmentation
Decreased state

* Anaemia
* Dry skin
* Nervous weakness
* Desire to intake of sour food

3. Oon` (Mamsa)

Muscular tissues are produced from Rakata

Increased state

* Tumors
* Carcinoma
* Goiter
* Cyst

Decreased state

* Wasting
* Dryness
* Crackling sound in movement of joints etc.

4. kozhppu (medas)

Increased state

* Associated with mamsa diseases
* Obesity
* Hyper cholesteremia
**Decreased state**

* Wasting of muscles
* Decreased stability of joints
* Lethargy

5. **Enbu (Asthi)**

**Increased state**

* Hyper calcinosis
* Extra tooth formation
* Hypertrophy of bone tissues

**Decreased state**

* Osteoporosis
* Rickets

6. **Moolai (majja)**

**Increased state**

* Bone and joint disorder
* Ulcers
* Heaviness of eye and body

**Decreased state**

* Demyelination
* Dellusion
* Giddiness
7. Sukklathathu

**Increased state**

* Calculi formation (Urethra)
* Sexual perversion

**Decreased state**

* Impotency
* Infertility
* Weakness

**Diagnosis**

According to siddha aspect they are 8 stools of diagnosis.

- குணம் - Pulse
- பிரித்தை - Touch or palpation
- சுயந்த - Examination of tongue
- காசிபாரம் - Examination of complexion
- வைண்டி - Examination of speech
- வொய்சி - Examination of eye
- சுயக்கிழியம் - Examination of Urine
- பொசம் - Examination of stool

“எச்சைணி சிக்குள நி஗ப்பி நீர்த்தலമயம் தர்ந்து…”

- சுருக்கம்
Other Diagnostic stools in siddha

- Examination of urine it includes quantitative, Qualitative analysis

- Examination of urine by dropping gingelly oil

In our siddha system of pathology includes.

Noi muthal naadal

(Find out the causes for diseases)

Siddha system of Pathology
(Noi Naadal)

Noiarithal
(Find out the nature of diseases)

Noi kannaippu
Karuvigal
(Diagnostic Stools)
Envagai Thervu
Manikkadainool
Symptoms

Maruthuvam
(Treatment)
AIM AND OBJECTIVES

The main aim of this study is to evaluate the pathology of the disease “Narithalaivadham” with the help of Siddha and modern parameters.

Nowadays, there is so many treatments are done in other systems, but no satisfactory remedy in this disease Narithalaivadham. We have to find out the exert treatment by knowing correct pathology of Narithalaivadham.

Objectives of this study

1. Varied clinical presentation of the disease
2. Pathology of the disease
3. Changes in the mukkutram
4. Find out the relation to sex, climate, Habit occupations and genetic relation
5. To pave way for further studies and researchers in this field

6. To bring forth the high lights of Siddha system of diagnosis, Envagithervu

7. To propagate the views of siddhars in terms of Scientific proof to needy world.
ELUCIDATION ABOUT NARITHALAIVADHAM

According to the Literature Yugi Vaithya Chinthamani has been described “Narithalaivadham” under vadha roga nithnam.

The meaning of the words in this poem,

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<td>கர்ப்புகள்</td>
<td>Knee cap</td>
</tr>
<tr>
<td>அரசையாளம்</td>
<td>Force</td>
</tr>
<tr>
<td>கர்ப்புகள்</td>
<td>knee cap</td>
</tr>
<tr>
<td>பசுளை</td>
<td>Swelling</td>
</tr>
<tr>
<td>எஃபையாளம்</td>
<td>Firm in nature</td>
</tr>
<tr>
<td>கர்ப்புகள்</td>
<td>Accumulation of blood</td>
</tr>
<tr>
<td>கர்ப்புகள்</td>
<td>Not able to stand</td>
</tr>
<tr>
<td>கர்ப்புகள்</td>
<td>Joint</td>
</tr>
</tbody>
</table>
Difficulty to fold -  பலகக்கூற்றம்
Hacking -  கீழக்கண்ட
Narrowing of Joint cavity -  கிருட்டமாக்கல்
Like jackal’s head -  தட்டிப்போர்க்
Massive Swelling -  பெரும் மூழ்கு
Rapid pulsation -  திட்டா தெரியும் தட்டிப்போர்க்

(Swelling in the knee cap)

Firm Swelling due to Accumulation blood in the knee joint cavity

Difficulty in standing upright Position

Difficulty to fold the Joint

Damaging of articular cartilage, narrowing of joint cavity

Massive Swelling like jackal’s head
The poem’s lines are summarized as follows,

The stanza clearly depicted from the sudden onset of swelling in the knee joint. Swelling is due to accumulation of blood in the knee joint cavity. The nature of swelling is firm massive, painful and tender damaging of articular cartilage, narrowing of joint cavity. Because of swelling patient is not able to stand in upright position, difficulty in folding and rapid pulsation.

- Accumulation of Blood in the knee joint
- Damaging the articular cartilage
- Narrowing of joint cavity
- Massive Swelling like Jackal’s head
- Not able to stand in upright position and difficulty in folding
- Rapid pulsation
DETAILED PATHOLOGICAL VIEW OF DISSERTATION TOPIC

Siddha Aspect

In this paper, we shall focus on the aspect of Siddha. The Siddha aspect is an integral part of the traditional Indian medicine system. It deals with the principles of disease causation and treatment. The Siddha system is based on the concept of prakriti (constitutions), vata, pitta, and kapha (humors), and the dynamic balance between them.

The Siddha system recognizes the importance of maintaining this balance to prevent diseases. The treatment involves the use of herbal medicines, dietary management, and lifestyle changes. The Siddha system is known for its holistic approach to health and well-being.

In conclusion, the Siddha system provides a unique perspective on disease prevention and treatment. Its principles are widely practiced in traditional medicine systems across India.
வாதிகள் வலசம்:

"நான் விளக்கும் மற்றையதே எனக்கு என்று தான் துருக்கம் கருதும் காலம் தான் போர்க்காலம்

'நான் விளக்கும் மற்றையதே எனக்கு என்று தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான் தான்

- பதினெட்டு மலார்

பக்திப்பக்க வருகாயகால நாளை, முதல், பின்னர், குறுக்கு, முன்னான அல்லாமல் இரண்டாம் ஆண்டு ஆண்டுகள் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலவின் நிலava

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முடிய மாட்டோ செய்தோ

“நிற்போரின்மையில் மாட்டோ பார்த்தினை முடிய

தொழில்நுட்பவாசர் விளக்கம்

தொழில்நுட்பப்படா பார்த்தினை முடிய

சுருக்காம்பியலையும் வெளிப்பகுதியில் தொழில்நுட்பப்படா

பார்த்தினை பார்த்தினை முடிய

சுருக்காம்பியலையும் பார்த்தினை முடிய

சுருக்காம்பியலையும் கைப்பொருளாக்கிய
t

- இதுவடையில் குச்சம்

தொழில்நுட்பப்படா (தொழில்நுட்ப - செய்தியுள் செய்தியுள்)

தொடர்றுத் தொழில்நுட்பப்படா பார்த்தினை செய்தியுள்

பார்த்தினை 

சுருக்காம்பியலையும் பார்த்தினை முடிய

சுருக்காம்பியலையும் பார்த்தினை 

t

- இதுவடையில் குச்சம்

மாலிகராமரத்து விளக்கம் காட்சியில் விளக்கம் அதனால்

சுருக்காம்பியலையும் காட்சியில் விளக்கம் காட்சியில். விளக்கம் பார்த்தினை செய்தியுள் செய்தியுள்

சுருக்காம்பியலையும் பார்த்தினை முடிய

சுருக்காம்பியலையும்

“மாலிகராமரத்து விளக்கம் காட்சியில்

சுருக்காம்பியலையும் விளக்கம்”

அதாவது விளக்கம் பார்த்தினையும் மாலிகராமரத்து விளக்கம்

சுருக்காம்பியலையும் விளக்கம் காட்சியில்

- Blood accumulate in to joint cavity

- Swelling of knee Joint.
“Damaging of articular cartilage

Narrowing of joint cavity.

Massive swelling

Rapid pulsation due to hemorrhage”.

"It makes the situation worse, making it worse, increasing the pain, and increasing the swelling. The rapid pulsation due to hemorrhage makes it even worse."

The narrowing of the joint cavity and the swelling make it worse. The rapid pulsation due to hemorrhage increases the pain and swelling. The narrowing of the joint cavity makes it worse. The rapid pulsation due to hemorrhage makes it even worse."

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Narrowing of joint cavity.

Massive swelling

Rapid pulsation due to hemorrhage".

The narrowing of the joint cavity and the swelling make it worse. The rapid pulsation due to hemorrhage increases the pain and swelling. The narrowing of the joint cavity makes it worse. The rapid pulsation due to hemorrhage makes it even worse."

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In this Disease Narthalivatham following alterations are takes place.

II. Alterations in Mukkutra Nilaikkal

Vali

1. Pranan – Rapid pulsation
2. Abanan – Constipation
3. Udanan – Palloriness
4. Viyanan – Difficulty in movements
5. Samanan – Its balancing function is affected
6. Devathathan – Irritability

Azhal

1. Ranjaga pitham - Palloriness of tongue and conjunctiva.
2. Sadhaga pitham - Difficulty in doing routine works.
3. Prasagapitham - Colour changes in the skin at the site of swelling.

Iyam

1. Avalampagam - Its balancing function disturbed.
2. Santhigam - Difficulty in movements of joints.

Inference

Vali - Pranan, Abanan, Udhanan, Viyanan, Samanan, devathathan are affected.

Azhal - Ranjaga pitham, Sathaga pitham, Prasaga pitham are affected.

Iyam - Avalambagam and Santhigam are affected.
III. Alterations in Udal thathukkal

1. Saaram - Pain and discomfort.

2. Senner - Stagnation of blood in the site of swelling

3. Oon

   Initial stage - Some inflammatory changes (Swelling)

   Later stage - Disuse atrophy

4. Kozhuppu - Restricted joint movements

5. Enbu

   Difficulty to move, standing upright position, inflammatory changes and intra articular adhesions in the knee joint.

6. Moolai - Osteoporotic changes in later stage.

**Inference**

All 6 Udal Thathukkal are affected except Sukkilam/ Sronitham.
MODERN ASPECT

"புரேக்கல் பெருநீரும் கருத்துக்காட்டு விளங்கும்
பதிக்கத் துறையில் பெருநீரும் பயன்படுத்தும்".

Swelling of Knee joint is due to accumulation of blood in to Knee joint cavity.

**Swelling with accumulation of blood:**

Generally the blood flows inside the closed circuits of the blood vessels which are arteries, arterioles, capillaries, vein and venules. Physiologically, blood is in constant motion inside the circuits. The escape of blood from the blood vessel is called haemorrhage and these extravasations of blood in to the tissues. When the blood accumulates in one of the body cavities it is referred to as

<table>
<thead>
<tr>
<th>Cavity</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic cavity</td>
<td>Haemothorax.</td>
</tr>
<tr>
<td>Pericardial cavity</td>
<td>Haemopericardium.</td>
</tr>
<tr>
<td>Peritoneal cavity</td>
<td>Haemoperitoneum.</td>
</tr>
<tr>
<td>Joint cavity</td>
<td>Haemarthrosis.</td>
</tr>
</tbody>
</table>

**Bleeding in to knee joint cavity is caused by**

1. Ligament injuries.
2. Fracture of patella.
3. Fractures of tibial condyles.
4. Trauma.
5. Deficiency of VIII factor and other clotting factors.
   - Fall injury
   - Direct injury
   - Sports injury

6. Any defect in coagulative mechanism.

7. Increased fragility of vessels.

8. Platelet deficiency or dysfunction.

Ligament injuries and fractures of patella and tibial condyles are caused by sports injury and traumatic cause. Sometimes pathological cause.

Increased fragility of vessels and platelet deficiency do not cause serious bleeding problem. Most often they induce small haemorrhages, petechiae and purpura.

**Derangement in coagulation**

The deficiency of one of knowing clotting factors results in bleeding disorder. More often bleeding manifest as the development of large haematomas followed by injuries.

The activated factor IX, acting in concern with activated factor VIII and with platelet, phospholipids and platelet factor III from traumatized platelets, activates factor X.
In person with deficient factor VIII and IX the process of

1. Vaso constriction.

2. Platelets plug formation.

It occurs, but they lack in the process of conversion of fibrinogen to fibrin, resulting in bleeding which in turn leads to the formation of haematoma.

Extravasations of blood in to the joint cavity with resultant swelling known as haemarthrosis.

➢ The immediate vascular response is of “Transient vasoconstriction” of arterioles.

➢ Next follows “persistent progressive vasodilatation” which involve mainly the arterioles and to the extent venules and capillaries. Vasodilatation results in increased blood volume.

➢ Progressive vasodilatation, in turn, elevates the local hydrostatic pressure resulting in transudation of fluid into the extra cellular space thus forming the swelling

“துறுத்தையும் திகையும் காண்டு தீர்ந்தது

திருமுகையும் சுருளையும் பாதிக்கிறது”.

Due to the swelling, patient is not able to stand in upright position and flex the knee.

“திருமுகையும் சுருளையும் பாதிக்கிறது

காண்டத்தை வல்லுவே விளையாடும்”.
Damaging of articular cartilage further increases the swelling (like jackal’s head)

Repeated haemorrhages into joints produce synovial hyperplasia, haemosiderin deposition and fibrous scarring increased intra articular pressure, limitation of movements causes articular cartilage damage.

"தெரிகியும் கலந்துடையது"

Intera-articular adhesions will result in reduction of the knee joint cavity.

"செஞ்சியும் கருத்து"

Chronic inflammatory process occurs due to blood in cavity. Inflammation and swelling of the joint, accumulation of morbid fluid which are followed by over growth of articular cartilage and synovial membrane with destruction of those parts of the cartilages subjected to intra articular pressure and progressive deformity by assuring the shape of jackal’s head

"நான்கு கலந்துபட்டு மையவில் எழுந்து"

There by rendering the patient unable to walk or move about

"செஞ்சியும் போதுமையானது செய்தியே"

The knee becomes usually large and makes him difficulty to fold or stretch his leg

"தற்போது காட்டுவிதே குழுந்தரை ப்ரெச்சையை அழகையானே"
(This is caused by excited vali uniting with the vitiated blood produces the inflammation of knee joint.)

"நோக்கத்திலும் ரோஜூரோ பாலஒற்று"  

If any haemorrhage in body causes rapid pulsation. It is due to increased cardiac activity to compensate the circulatory blood loss. So the heart rate and pulse rate is increased in haemorrhagic conditions.
Schematic representation of Narithalivadham

Any injury (or) trauma in knee joint

→ Haemorrhage  → Rapid pulsation

→ Accumulation of blood in knee joint cavity

→ Swelling occur (Stasis of blood in joint cavity)

→ Haemosiderin deposition, intra-articular adhesions

→ Articular cartilage damage
  and
  Narrowing of joint cavity
  Limitation of movements

→ Further damaging to articular cartilage

→ Massive swelling (like Jackal’s head)
REVIEW OF LITERATURE

Narithalaivadham is also explained in Yugimuni Vaithya Kaviyam and Paravasasekaram.

**In Yugimuni Vaithya Kaviyam**

“The author explains Narithalaivadham is characterized by

- Pain and discomfort in knee joint.
- Massive swelling (like jackal’s head)
- Pain during walk

**In Pararasa Sekaram,**

“The author explains Narithalaivadham is characterized by

- Swelling in knee joint
- Accumulation of blood in knee joint cavity.
- Patient not able to stand and flex the knee.
- Swelling is tender and reddish like jackal’s head.
Above two authors explained Narithalaivadham is disease of knee joint.

But, Pararasekaram specify, the swelling is due to accumulation of blood. Same clinical features are explained by Yugi in Yugi Vaithya Cinthamani and Yugi Vaithya Kaviyam. According literatures we concluded Narithalaivadham is inflamatroy joint disease affecting knee joint due to accumulation of blood in knee joint cavity.
ANATOMY OF THE KNEE JOINT

➢ The Knee is the largest and most complex joint of the body. The complexity is the result of fusion of three joints in one. It is formed by fusion of the lateral femorotibial, medial femorotibial and femoropatellar joints.

Type

➢ It is compound synovial joint incorporating two condylar joints between the condyles of the femur and tibia and one saddle joint between the femur and the patella.

Articular Surfaces

➢ The articular surfaces are the large curved condyles of femur, flattened condyles of tibia and facets of patella.

➢ Lateral femoral condyle accommodates the large lateral facet of the patella and prevents subluxation and dislocation.

➢ The medial femoral condyles are longer in dimension in anteroposterior direction which allows greater rolling in this condyle.

➢ Medial tibial plateau is an oval disc concave in both sagittal and femoral planes. This accommodates larger and medial femoral condyle.
- Lateral tibial plateau is circular in shape. This facilitates rotation of the femoral condyle during movement.
- Patellar facets are medial and lateral facet which articulates with femur.

**Bursae**

- Bursae are a little fluid sac that helps muscle and tendons to slide freely as they knee moves. There are about 13 bursae in the knee joint in the front and in the sides.

**Anterior**

- Subcutaneous pre patellar bursa
- Subcutaneous infra patellar bursa
- Deep infra patellar bursa
- Supra patellar bursa

**Lateral**

- Deep to lateral head of gastronemius
- Between fibular collateral ligament and biceps femoris.
- Between fibular collateral ligament and popliteal tendon.
- Between popliteal tendon and lateral condyle of tibia.

**Medial**

- Deep to Medial head of gastronemius
- Ansarine bursa
- Deep to tibial collateral ligament
Deep to semimembranosus

Between Semimembranosus and Semi tendinosus.

**Ligaments**

- Ligaments are the connective tissue structures that connect or bind one bone to another either at or near a joint.
- The roles of the various ligaments of the knee have their importance to knee joint stability.
  - Fibrous capsule
  - Ligamentum Patellae.
    - This is the central portion of the common tendon of insertion of the Quadriceps femoris, the remaining portions of the tendon form the medial and lateral patellar retinacula.
    - The ligamentum patella is about 3 inches long and one inch broad.
    - It is attached above to the margins and rough posterior surface of the apex of the patella and below to the smooth, upper part of the tibial tuberosity.
  - Anterior cruciate ligament
  - Posterior cruciate ligament
  - Medial (or) tibial collateral ligament
  - Lateral (or) fibular collateral ligament
Medial Meniscus

Lateral Meniscus

Oblique popliteal ligament

Arcuate popliteal ligament

Transverse ligament.

**Blood supply**

- Five genicular branches of the popliteal artery.
- The descending genicular branch of the femoral artery.
- The descending branch of the lateral circumflex femoral artery
- Two recurrent branches of the anterior tibial artery.
- The circumflex fibular branch of posterior tibial artery.

**Nerve Supply**

- Femoral nerve, through its branches to the Vasti especially the Vastus Medialis.
- Sciatic Nerve through the genicular branches of the tibial and common peroneal Nerve.
- Obturator Nerve, through its posterior division
**Movements of the knee joint**

Active movements at the knee are flexion, extension, medial rotation and Lateral rotation.

**Table 1 : Muscles Producing Movements at the knee joint**

<table>
<thead>
<tr>
<th>Movement</th>
<th>Principle Muscles</th>
<th>Accessory Muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>i) Biceps Femoris</td>
<td>i) Gracilis</td>
</tr>
<tr>
<td></td>
<td>ii) Semitendinosus</td>
<td>ii) Sartorius</td>
</tr>
<tr>
<td></td>
<td>iii) Semimembranosus</td>
<td>iii) Popliteus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>iv) Gasstronemius</td>
</tr>
<tr>
<td>Extension</td>
<td>Quadriceps Femoris</td>
<td>Tensor Fasciae Latae</td>
</tr>
<tr>
<td>Medial rotation of</td>
<td>i) Popliteus</td>
<td>i) Sartorius</td>
</tr>
<tr>
<td>flexed leg</td>
<td>ii) Semi membranosus</td>
<td>ii) Gracilis</td>
</tr>
<tr>
<td></td>
<td>iii) Semitendinosus</td>
<td></td>
</tr>
<tr>
<td>Lateral rotation of</td>
<td>Biceps femoris</td>
<td></td>
</tr>
<tr>
<td>Flexed leg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**QUADRICEPS FEMORIS**

- Quadriceps is the combination of Rectus femoris, Vastus medialis, Vastus lateralis, and Vastus intermedius. Quadriceps found in the anterior part of the thigh and the function is knee extension.
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
</tr>
</thead>
</table>
| Rectus Femoris         | a. Straight head: from the upper half of the anterior inferior iliac spine  
                        | b. Reflected head: from the groove above the margin of the acetabulum and the capsule of the hip joint | Base of patella                                                  |
| Vastus Lateralis       | a. Upper part of intertrochanteric line.  
                        | b. Anterior and inferior borders of greater trochanter  
                        | c. Lateral lip of gluteal tuberosity  
                        | d. Upper ½ of lateral lip of linea aspera | a. Later part of the base of patella.  
                        | b. Upper 1/3 of the lateral border of patella expansion to the capsule of knee joint, tibia and iliotibial tract. |
| Vastus Medialis        | a. Lower part of intertrochanteric line,  
                        | b. Spiral line  
                        | c. Medial lip of linea aspera  
                        | d. Upper ¼ of medial supracondylar line | Medial 1/3 of the base and upper 2/3 of the medial border of the patella. |
Nerve Supply
- Femoral Nerve (L2,3,4)

Action
- Quadriceps is a strong extensor of the leg. This action is very important in standing, walking and running.
- In addition the Rectus femoris flexes the hip joint along with iliopsoas and helps to maintain the erect attitude.
- The Vastus medialis prevents lateral displacement of the patella
BIOMECHANICS OF KNEE JOINT

1. Stabilization

- Capsule and ligaments are the static stabilizers of knee joint, muscles are the dynamic stabilizers. Structures located in the medial and lateral sides of the knee joint provide medial and lateral stability.
- Static and dynamic stabilizers, medial and lateral compartment structures provide anteroposterior stability of the knee.
- Extensor retinacular provide anteromedial and anterolateral support.
- Popliteus is posterior lateral stabilizers.
- Anterior cruciate ligament and Hamstring work in a complementary manner to resist the forces that displace tibia anteriorly or shear the femur posteriorly.

Static Stabilizers

- Medial collateral ligament
- Lateral collateral ligament
- Anterior cruciate ligament
- Posterior cruciate ligament
- Oblique popliteal ligament
- Arcuate popliteal ligament
- Transverse ligament
**Dynamic Stabilizers**

- Quadriceps femoris
- Extensor Retinaculam
- Pesanserinus (Semitendinosus, Sartorius, Gracilis Muscles)

2. Q Angle

   The Q angle is the angle formed between a line connecting to the axis to mid point of the patella and a line connecting the tibial tubercle and the mid point of patella.

   An angle of 10° - 15° measured with knee either in full extension or slight flexion is considered to be normal.

3. Locking and Unlocking of Knee Joint

   Locking is a mechanism that allows the knee to remain in the position of full extension. Locking occurs as a result of medial rotation of femur during last stage of extension. Locking aided by the oblique pull of the ligament during the last stage of extension. Locking is produced by the quadriceps femoris muscles.

   In initial flexion the knee must unlock. A flexion force will automatically result in lateral rotation of the femur. Unlocking is brought about by the action of the popliteus muscle.
**PHYSIOLOGY OF BLOOD**

- Blood is a connective tissue in fluid form. It is considered as a fluid of life. Because it carries oxygen from lungs to all parts of the body and the carbon dioxide from all parts of the body to the lungs.

- Blood is red in colour. Arterial blood is scarlet red because it contains more oxygen and venous blood is purple red.

- The volume of blood in a normal adult is about 5 litres

- pH in normal conditions is 7.4

- Blood is five times more viscous than water. It is mainly due to red blood cells and plasma

**Composition of Blood**

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

**Blood Cells**

Three types of cells are present in the blood

- Red blood cells or Erythrocytes
- White blood cells or Leukocytes
- Platelets or Thrombocytes
Haemoglobin

- Haemoglobin (Hb) is the red pigment of blood present in RBC.
- Haemoglobin consists of 2 parts like 96% is a simple protein called globin and 4% is iron containing pigment called heme.
- Average haemoglobin (Hb) content in blood is 14-16 gm% in adult males 15 gm%, in adult females 14.5 gm%
- The function of haemoglobin is to carry the respiratory gases, oxygen and carbon dioxide.

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.
- The organic substances are proteins, carbohydrates, fat, amino acids, non protein nitrogenous substances, internal secretions, enzymes and antibodies.
- The inorganic substances are sodium, calcium, potassium, magnesium, chloride, iodide, iron, phosphates and copper.
COAGULATION OF BLOOD

- When blood is shed out or collected in a container, it loses its fluidity and becomes a jelly-like mass after a few minutes. This process is called coagulation or clotting of blood.

- The clot is a mesh of thin fibrils entangling the blood cells. These fibrils consist of fibrin. The fibrin is formed from fibrinogen.

**Mechanism of Coagulation Basic**

1. Damaged tissues of blood vessels
2. Damaged platelets
3. Thromboplastin
4. Ca$^{2+}$
5. Prothrombin activation
6. Ca$^{2+}$
7. Prothrombin (Inactive) → Thrombin (Active)
8. Fibrinogen (Soluble) → Fibrin threads (Clot) (Insoluble)
Actors involved in blood clotting

Coagulation of blood occurs through a series of reactions due to the activation of a group of substances. Those substances necessary for clotting are called clotting factors. Thirteen clotting factors are identified.

Blood clotting factors

- Factor I - Fibrinogen
- Factor II - Prothrombin
- Factor III - Thromboplastin
- Factor IV - Calcium
- Factor V - Quick’s Labile Factor (Proaccelerin (or) Accelerator globulin)
- Factor VI - Presence has not been proved
- Factor VII - Stable factor
- Factor VIII - Anti haemophilic factor
  *(Antihaemophilic globulin) AHF*
- Factor IX - Christmas factor (of) Plasma Thromboplastin Component (PTC)
- Factor X - Stuart – Prower factor
- Factor XI - Plasma thromboplastin antecedent (PTA)
- Factor XII - Hegman factor (contact factor)
- Factor XIII - Fibrin Stabilizing factor (Fibrinase)
CLOTTING MECHANISM

- Clotting of blood occurs in three stages
  - Formation of prothrombin activator
  - Conversion of prothrombin to thrombin
  - Conversion of fibrinogen to fibrin

- During the process of blood clotting, the clotting factors, which are in inactive forms, are converted into active forms and their enzymatic actions produce the successive reactions one after another in a cascading manner.

- Thus the various reactions involved in blood clotting are explained by enzyme cascade theory.

**Stage 1: Formation of Prothrombin Activator**

Prothrombin activator is formed in two ways.

a) Extrinsic pathway

  - In this, the formation of prothrombin activator is initiated by the tissue thromboplastin.

b) Intrinsic pathway

  - In this, the formation of prothrombin activator is initiated by platelets which are within the blood itself.
**Extrinsic pathway for the formation of prothrombin Activator**

- It occurs in the following sequence
  - Tissue thromboplastin (factor III) initiates this pathway. After injury, the damaged tissues release tissue thromboplastin. The thromboplastin contains proteins, phospholipids and glycoprotein which act as proteolytic enzymes.
  - The glycoprotein and phospholipids components of thromboplastin convert factor X into activated factor Xa, in the presence of factor VII.
  - The activated factor X reacts with factor V and phospholipids component of tissue thromboplastin to form prothrombin activator. This reaction requires the presence of calcium ions.
  - Factor V is activated by thrombin, which is formed from prothrombin. This factor V now accelerates formation of prothrombin activator and the other processes of blood clot. This effect of thrombin is called positive feed back effect of thrombin.

**Intrinsic Pathway for the formation of prothrombin Activator**

- Following is the sequence of events in intrinsic pathway.
  - During the injury, the blood vessel is ruptured. The endothelium is damaged and collagen beneath the endothelium is exposed.
• When factor XII (Hegman factor) comes in contact with collagen, it is converted into activated factor XII.

• The activated factor XII converts factor XI into activated factor XI in the presence of kinogen and prekallikrein.

• The activated factor XI activates factor IX in the presence of factor IV (Calcium)

• The activated factor IX activates factor X in the presence of factor VIII and calcium.

• When the platelet comes in contact with collagen of damaged blood vessel, it releases phospholipids.

• Now the activated factor X (as in the case of extrinsic pathway) reacts with platelet phospholipids and factor V to form prothrombin activator. This needs presence of calcium ions.

• Factor V is also activated by positive feedback effect of thrombin.

Stage 2: Conversion of Prothrombin into thrombin

➢ Prothrombin activator converts prothrombin into thrombin in the presence of calcium. Thrombin itself can accelerate this reaction by positive feed back mechanism. That is, the initial thrombin activates factor V. This is turn accelerates the formation of both extrinsic and intrinsic prothrombin activator.
Stage 3: Conversion of fibrinogen into fibrin

- During this, the soluble fibrinogen is converted into insoluble fibrin by thrombin. Initially, the fibrinogen is converted into activated fibrinogen due to loss of 2 pairs of polypeptides from each fibrinogen molecule.

- The activated fibrinogen is called fibrin monomer. This polymerizes with other monomer molecules to form fibrin.

- The first formed fibrin contains loosely arranged strands. This is modified later into a dense tight aggregate by fibrin stabilizing factor (XIII) and this reaction requires the presence of calcium ions.
**Stage of Coagulation of Blood**

**Intrinsic pathway**

Stage 1

- Endothelial damage + collagen exposure

- XII → XII a
  - Kinogen
  - Prekallikrein

- Xi → XI a
  - Calcium

- IX → IX a
  - VIII
  - Calcium

- X → X a

**Extrinsic pathway**

Stage 1

- Tissue trauma + Tissue thromboplastin

- Glycoprotein
  - Phospholipid

- VIII

**Stage 2**

- Prothrombin activator

**Stage 3**

- Prothrombin → Thrombin

- Fibrinogen a → Fibrinogen

- Fibrinogen

- Polymerization

- Loose strands of fibrin

- Xii
  - Calcium

**Tight blood clot**

\[ a = \text{activated} \]
Blood Clot

- The fibrin threads run in all directions. The red blood cells, white blood cells and the platelets get entrapped within the meshwork of fibrin. The entire mass of fibrin meshwork and the blood cells entrapped within this is called blood clot. The external blood clot is also called scab. This blood clot adheres to the opening of damaged blood vessel and prevents blood loss.

- Red blood cells and white blood cells are not necessary for clotting process. However, when clot is formed, these cells are trapped in it. The trapped red blood cells are responsible for the red colour of the clot.

Bleeding Time

- This is the time interval from oozing of blood after a cut or injury till arrest of bleeding

- The normal duration of bleeding time is 3 to 6 minutes. It is prolonged in purpura.

Clotting Time

- The time interval from oozing of blood after a cut or injury till the formation of clot is called clotting time.

- The normal duration of the clotting time is 3 to 8 minutes and it is prolonged in Heamophilia.
**Prothrombin time**

- Blood is collected and oxalated so that, the calcium is precipitated and prothrombin is not converted into thrombin. Thus, the blood clotting is prevented.
- Then a large quantity of tissue thromboplastin with calcium is added to this blood. Calcium nullifies the effect of oxalate.
- And, the tissue thromboplastin activates prothrombin and blood clotting occurs. During this procedure the time taken by blood to clot after adding tissue thromboplastin is called prothrombin time. Prothrombin time indicates the total quantity of prothrombin present in the blood.
- The normal duration of prothrombin time is about 12 seconds. The prothrombin time is prolonged in deficiency of prothrombin and other factors like factors I, V, VII, and X, However, it is normal in haemophilia.
HAEMOPHILIA

Definition

Haemophilia is a genetic bleeding disorder. Persons suffering from it have prolonged bleeding, mostly in their joints. It is due to absence of certain clotting factors in the blood. Prolonged bleeding may be after injury or can happen on its own.

Incidence

The incidence of haemophilia as per WHO findings is 1 person in a population of 10,000. Approximately 1,00,000 persons suffering from haemophilia in India. May be just 50% of them survive today.

Sex

Male – Commonly affected
Female – Carriers

Causes of Haemophilia

Haemophilia occurs due to lack of formation of prothrombin activator. That is why the coagulation time is prolonged. Bleeding time and prothrombin time are normal.

Types of Haemophilia

The formation of prothrombin activator is affected due to the deficiency of VIII or IX factor depending upon the deficiency of the factor involvement, haemophilia is classified into two types.
Haemophilia A

- It is also called the classic haemophilia, due to the deficiency of factor VII. 85% of people with haemophilia are affected by haemophilia A.

Haemophilia B

- It is also called Christmas disease, due to deficiency of factor IX. 15% of people with haemophilia are affected by haemophilia B.

Von Willebrand Disease

- Von willebrand disease is characterized by excessive bleeding even with a mild injury.
- It is due to inherited deficiency of Von willebrand factor.
- Von willebrand factor is a protein secreted by endothelium of damaged blood vessels and platelets. This protein is responsible for adherence of platelets to endothelium of blood vessels during hemostasis after an injury. It is also responsible for the survival and maintenance of factor VIII in plasma.
- The deficiency of Von Willebrand factor leads to reduction in platelet adhesion and secondary deficiency of factor VIII. This results in excessive bleeding which resembles the bleeding that occurs during platelet dysfunction or haemophilia.
- The gene for factors VIII and IX both are located on the X-Chromosome.
Table 3 Heamophilia

| If there is no Family history, The chance is 1 in 5000 male birth. About a 1/3 of all affected people with hemophilia belong to this category, which is due to new mutations. | If the mother is a carrier, 50% of her sons will be affected by the disease and 50% of her daughters will be carriers. | If the father has hemophilia, then all daughters will be carriers and all sons will be normal. |

A women is a indefinite carrier if

- Her father has haemophilia
- She has one son with haemophilia and first degree relatives (brother, uncle or other male relatives) are affected.
- She has two or more sons with haemophilia.

A Women is a possible carrier if

- She has one or more maternal relatives with haemophilia
- She has one son with haemophilia and no other affected relatives.

Chronic Musculo – Skeletal Manifestations

- Chronic haemophilia arthropathy
  - Chronic synovitis
  - Deforming arthropathy

- Contractures
- Fractures

Clinical Manifestations of Haemophilia

The clinical manifestations of haemophilia depend upon the factor levels.
- Mild – factor VIII or IX level is 5-30%
- Moderate – factor VIII or IX level is 2-5%
- Severe – factor VIII or IX level is <1%

**Table 4: Sites of bleeding**

<table>
<thead>
<tr>
<th>SI. No</th>
<th>Minor</th>
<th>Major</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Joints</td>
<td>Central nervous system</td>
</tr>
<tr>
<td>2.</td>
<td>Muscle/soft tissue</td>
<td>Gastro intestinal</td>
</tr>
<tr>
<td>3.</td>
<td>Mouth/ Gums</td>
<td>Neck/Throat</td>
</tr>
<tr>
<td>4.</td>
<td>Epitasis</td>
<td>Severe trauma</td>
</tr>
</tbody>
</table>

**Major** – causes serious problem  
**Minor** - manageable
### Table 5: Prevalence of bleeding in haemophilia

<table>
<thead>
<tr>
<th>SI. No.</th>
<th>Type of Haemorrhage</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Haemarthrosis</td>
<td>70-80</td>
</tr>
<tr>
<td>2.</td>
<td>Muscle</td>
<td>10-20</td>
</tr>
<tr>
<td>3.</td>
<td>Other major bleeds</td>
<td>5-10</td>
</tr>
<tr>
<td>4.</td>
<td>CNS bleeds</td>
<td>&lt; 5</td>
</tr>
</tbody>
</table>

### Table 6: Prevalence of bleeding in to joints

<table>
<thead>
<tr>
<th>SI. No.</th>
<th>Site</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Knee</td>
<td>45</td>
</tr>
<tr>
<td>2.</td>
<td>Elbow</td>
<td>30</td>
</tr>
<tr>
<td>3.</td>
<td>Ankle</td>
<td>15</td>
</tr>
<tr>
<td>4.</td>
<td>Shoulder</td>
<td>3</td>
</tr>
<tr>
<td>5.</td>
<td>Wrist</td>
<td>3</td>
</tr>
<tr>
<td>6.</td>
<td>Hip</td>
<td>2</td>
</tr>
<tr>
<td>7.</td>
<td>Others</td>
<td>2</td>
</tr>
</tbody>
</table>
PATHOLOGY

Haemarthrosis knee

Haemarthrosis means collection of blood in the joint cavity due to any injury or trauma in case of haemophilia and caused by ligament injury in normal person.

Haemarthrosis is the most common, most physically, economically and psychologically debilitating manifestation occurs in 90% of severe haemophilias.

Causes

Primary cause : Deficiency of VIII factor
Secondary cause : Traumatic injury

I.e. Fall injury, Direct injury, Sports injury

Haemarthrotic knee becomes, swollen warm, and markedly tender. Discoloration may be noted, indicating bleeding. There may be a related fever with leukocytosis, if the bleeding is mild, the joint may return to normal with in a few days with no sequelae or residual. In severe haemarthrosis the knee may remain inflamed for weeks or months.

Repeated haemarthrosis leads to hemosiderosis (a deposit of hemosiderin, granules consisting of ill-defined complexes of ferrichydroxides, polysaccharides, and proteins) of the synovium, gradual
degeneration of the capsule and other periarticular soft tissues repetitive and persistent haemarthrosis thins the cartilage.

**Haemarthrosis caused by trauma**

The following conditions included under this heading

1. Fractures of the tibial condyle
2. Fracture of patella
3. Ligament injuries of knee joints
   
   **Collateral ligaments**
   
   a. Medial collateral ligament
   
   b. Lateral collateral ligament

   **Cruciate ligaments**
   
   a. Anterior cruciate ligament (ACL)
   
   b. Posterior cruciate ligament (PCL)

4. Injuries to semilunar cartilage
   
   a. Medial semilunar cartilage
   
   b. Lateral Semilunar cartilage

5. Loose body in the knee

**Ligament injuries of the knee**

The collateral and cruciate ligaments are commonly injured in athletes, football and kabadi players.
Medial ligament injuries

The medial collateral ligament is more commonly injured than the lateral.

A sudden valgus or abduction strain at the knee associated with external rotation of tibia causes stretching of the medial ligament producing

a. Sprain of the ligament

b. Partial rupture of ligament

c. Complete rupture of ligament.

In case of sprain, there is pain in the medial aspect of the knee and tenderness over the upper and lower attachment of the ligament. In haemarthrosis the swelling occurs within an hour or two of the injury.

In case of partial rapture, the ligament is partially torn resulting in haemarthrosis.

Complete rupture of the medial ligament is caused by a very severe valgus strain to the knee. Marked swelling occur due to haemarthrosis.

Lateral collateral ligament

This is much less common than the injury to the medial ligament. The same types of injuries, sprain, partial rupture and complete rupture of the ligament occur due to a hit on the inner aspect of the weight bearing of knee joint, which forces the knee into a sudden adduction strain, causes haemarthrosis.
**Cruciate ligament injuries**

The cruciate ligaments play an important role in maintaining the stability of the knee joint. They may be injured alone or in combination with collateral ligament. The anterior cruciate ligament is more commonly injured. The common mechanism of injury is a forced full flexion of the knee joint with the tibia in internal rotation. It occurs injuries in gymnastic exercises, basketball and football. Clinically, the patient having acute pain, swelling and haemarthrosis of the knee joint.

**Injuries of the semilunar cartilages.**

Injuries to the meniscus are common in young adults and are often sustained by the football and kabadi players.

Violent abduction and external rotation, on flexed weight bearing knee causes a tear in the medial meniscus. The lateral meniscus is damaged by the opposite violence i.e. internal rotation and adduction violence of the tibia or semi flexed weight bearing knee joint.

**Clinical features**

- Acute pain
- Knee gets locked in flexion
- Swelling due to haemarthrosis

**Fractures**

Fractures of tibial condyles and patella also causes haemarthrosis
**Acute Haemarthrosis**

After minor injury, or even spontaneously bleeding is thought to arise from numerous vessels of stratum synoviale. This produces a polymorphonuclear leucocytosis as well as a lymphocytic and histiocytic cellular invasion leading to a synovitis with hyperplasia. Large amounts of the pigment haemosiderin accumulate throughout the synovial tissues. As the swelling rapidly increases, pain becomes a prominent feature with a stiffness of the joint usually in a flexed position as well as inhibition of muscle function.

**Chronic haemophilic arthropathy**

Haemophilic patients are now living longer with frequent bleeding and the total number is increasing so that chronic arthropathy remains a problem. Combined pathological and experimental investigations demonstrated that repeated haemorrhages into joints produce synovial hyperplasia, haemosiderin depositions and fibrous scaring.

Azarin (1985) have produced experimentally an on going haemarthrosis into the knee of dogs by creating a poplitial arteriovenous aneurysm bleeding into the joint. They studied not only the effect of blood on the tissues, but an increased intra articular pressure of (120-140mmHg). This is also very important in pathogenesis intra articular adhesions will result in reduction of the joint cavity and limitation of movements. The restrictions of movement will reduce the circulation of
nutrients in articular cartilage and leads to progressive articular cartilage breakdown. The bony changes are attributed partly to disuse osteoarthritis following cartilage breakdown. It appears that the initiation of cartilage breakdown is due to the combined effect of immobilization with poor nutrition and the release of enzymes from both the blood within the joint and inflamed synovial membrane. This causes breakdown of the glycosamioglycans of the matrix. Haemophilic synovium in culture produces more prostaglandin E at much higher levels than normal but less than found in rheumatoid synovitis, with significant loss of chondroitin sulphates from human cartilage.

The late radiographic appearance of the involved joints result from the effects of haemarthrosis, locate immobilization osteoporosis and mechanical changes in alignment the most common features seen in knee joint are,

- Epiphyseal over growth
- Patellar squaring
- Widening of inter condylar notch
- Diminished joint cartilage space
- Subchondral bone collapse
- Cyst formation
- Marginal osteopytic formation
- Deposition of haemosiderine in synovial membrane.
Schematic representation of Haemarthrosis

Repeated haemarthrosis

- Subchondral haemorrhage
- Activated Plasmin
- Bleeds easily
- Chronic Synovitis
  - Epiphyseal over growth
  - Due to Hyperemia

- Destruction of Biological shock Absorber
- Accumulation of intra articular iron deposits
- Hyper tropic Synovial Membrane

- Synovial Fibrosis
- Librations of lysosomal enzymes (Cathepsin-D)
  - Further Inflammatory Response
  - Cartilage break down products

- Articular cartilage Damage

Due to Hyperemia
Clinical features

1. Loss of movement is the most frequent finding
2. Fixed flexion contracture
3. Valgus and external rotation deformities
4. Wasting of quadriceps muscle.

Differential diagnosis

1. Acute suppurative arthritis

   Acute suppurative arthritis closely resembles haemarthrosis. The history is usually conclusive, but aspiration will resolve any doubt.

2. Rheumatic fever

   Typically the pain flits from joint to joint, but the onset of one joint may be misleadingly inflamed. However, there are no signs of bleeding.

3. Gout and pseudo Gout

   In adults acute crystal induced synovitis may closely resembles haemarthrosis. On aspiration the joint fluid is often turbid, with high white cell counts however microscopic examination by polarized light will show the characteristic crystals.

Diagnosis

Diagnosis is clinically obvious but X-Ray, ultra sonogram /CT scan may be occasionally needed to confirm the presence of a bleed.
I. Detailed history with particular emphasis on

- Age
- Onset of bleeding
- Whether bleeding spontaneous or trauma related
- Other affected members in the family
- History of trauma.

II. Complete physical Examination

Complete physical examination with particular care to record range of motion, deformities and muscle strength at knee.

Clinical features

- Swelling
- Pain
- Warmth
- Redness
- Stiffness
- Limitation of movements i.e…. extensor lag
- Rapid Pulsation
EVALUATION OF DISSERTATION TOPIC

Materials and methods

The clinical study on Narithalaividham was carried out at the Post Graduate Department of Noi Naadal branch in Govt Siddha Medical College, palayamkottai. Case selection and supervision

The following 20 cases selection and supervision was done in Peace Health Center, Tirunelveli and Tirunelveli Medical College. According to the finding of Ennvagithervu on Narithalivadham (Haemarthrosis) as mentioned by Yugi vaithiya chinthamani.

The patients are carefully examined systematically under the supervision of the professor and other staffs of post graduate Noi Naadal department.

The detailed history of the present and past illness and family history were observed. Typical picture of 20 cases was evaluated under siddha and modern parameters.

Evaluation of clinical parameters

- The detailed history and clinical features of the patients were taken carefully
- The clinical history
- Detail history of present and past illness
- Personal and family history
Clinical features for Narithalaivadham are

- Swelling in the knee joint
- Accumulation of blood in the knee joint cavity
- Difficulty in standing
- Difficulty to flex the knee
- Rapid pulsation
- Pain and tenderness

Study of siddha clinical diagnosed modes of investigations are

1. Poriyal arithal
2. Pulanal arithal
3. Vinathal
4. Mukkutra nilaigal
5. Udal kattukal
6. Envagai thervugal (Including Neerkuri, Neikuri)
7. Manikkadai nool

The clinical investigations

For further detailed study about the disease the following laboratory investigation was done in all cases.

1. Haematology
2. Bleeding time
3. Clotting time
4. Prothrombin time
5. Partial prothrombin time
6. Factor assay
7. Total count-WBC, RBC
8. Differential count
9. Haemoglobin
10. Erythrocyte Sedimentation Rate

**Bio chemical**
- Blood Urea
- Blood Sugar

**Urine**
- Albumin
- Sugar
- Deposits

**Motion**
- Ova
- Cyst

**Other test**
- X-Ray
- Ultra sonogram
- CT scan – if necessary
OBSERVATION AND RESULTS

Results are observed with respect to the following aspects

1. Age and Sex reference.

2. Family history.

3. Traumatical history

4. Mukkutranilai

5. Udal Thathukkal

6. Envagai Thervugal

7. Clinical Features.

8. Laboratory Findings.

1. Age and Sex reference.

Table 7: Age and Sex reference

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Up to 10 years</td>
<td>4</td>
<td>-</td>
</tr>
<tr>
<td>10 – 20 years</td>
<td>7</td>
<td>-</td>
</tr>
<tr>
<td>20 – 50 years</td>
<td>9</td>
<td>-</td>
</tr>
</tbody>
</table>

2. Family History

Table 8: Family History

<table>
<thead>
<tr>
<th>Family history</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive family history</td>
<td>11</td>
</tr>
<tr>
<td>Negative family history</td>
<td>9</td>
</tr>
</tbody>
</table>
3. Traumatic history

Table 9: Traumatic history

<table>
<thead>
<tr>
<th>Traumatic History</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>16</td>
</tr>
<tr>
<td>Negative</td>
<td>4</td>
</tr>
</tbody>
</table>

Mukkutranilai

A. Derangement of Vali

Table 10: Derangement of Vali

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types of Vadham</th>
<th>No of cases affected</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pranan</td>
<td>13</td>
<td>Rapid pulsation</td>
</tr>
<tr>
<td>2</td>
<td>Abanan</td>
<td>3</td>
<td>Constipation</td>
</tr>
<tr>
<td>3</td>
<td>Viyanan</td>
<td>20</td>
<td>Difficulty in movements of affected area</td>
</tr>
<tr>
<td>4</td>
<td>Uthanam</td>
<td>20</td>
<td>Colour changes</td>
</tr>
<tr>
<td>5</td>
<td>Samanan</td>
<td>20</td>
<td>Its balancing function is disturbed.</td>
</tr>
<tr>
<td>6</td>
<td>Nagan</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>Koorman</td>
<td>2</td>
<td>Diminished vision</td>
</tr>
<tr>
<td>8</td>
<td>Kirukaran</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>Devathathan</td>
<td>5</td>
<td>Irritablity</td>
</tr>
<tr>
<td>10</td>
<td>Dananjeyan</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
### B. Derangement of Azhal

**Table 11: Derangement of Azhal**

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types of Azhal</th>
<th>No of cases affected</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anar pitham</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Ranjaga pitham</td>
<td>16</td>
<td>Paleness in conjunctiva and tongue.</td>
</tr>
<tr>
<td>3</td>
<td>Sadhaga pitham</td>
<td>20</td>
<td>Difficulty in doing routine works.</td>
</tr>
<tr>
<td>4</td>
<td>Aalosaga pitham</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>Prasaga pitham</td>
<td>20</td>
<td>Colour changes in the skin at the site of injury.</td>
</tr>
</tbody>
</table>

### C. Derangement in Iyam

**Table 12: Derangement in Iyam**

<table>
<thead>
<tr>
<th>S. No</th>
<th>Types of Iyam</th>
<th>No of cases affected</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Avalampagam</td>
<td>20</td>
<td>It’s balancing function disturbed.</td>
</tr>
<tr>
<td>2</td>
<td>Kilethagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Bothagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Tharpagam</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>Santhigam</td>
<td>20</td>
<td>Difficulty in movements of joints.</td>
</tr>
</tbody>
</table>
3. Udal Thathukkal

**Table 13: Udal Thathukkal**

<table>
<thead>
<tr>
<th>S. No</th>
<th>Udal Thathukkal</th>
<th>No of cases</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Saaram</td>
<td>20</td>
<td>Pain, discomfort</td>
</tr>
<tr>
<td>2</td>
<td>Senneer</td>
<td>20</td>
<td>Stagnation of blood in the site of injury.</td>
</tr>
<tr>
<td>3</td>
<td>Oon</td>
<td>20</td>
<td>Swelling</td>
</tr>
<tr>
<td>4</td>
<td>Kozhuppu</td>
<td>20</td>
<td>Restricted it movement</td>
</tr>
<tr>
<td>5</td>
<td>Enbu</td>
<td>20</td>
<td>Inflammatory changes</td>
</tr>
<tr>
<td>6</td>
<td>Moolai</td>
<td>3</td>
<td># (fracture)</td>
</tr>
<tr>
<td>7</td>
<td>Sukilam / Sronitham</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

4. Manikadai Nool (Viral kadai Alavu)

**Table 14: Manikadai Nool**

<table>
<thead>
<tr>
<th>Viral kadai Alavu</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>10 ¼</td>
<td>10</td>
</tr>
<tr>
<td>10 ½</td>
<td>8</td>
</tr>
</tbody>
</table>
### 5. The Picture of Envagai Thervugal

**Table 16: Envagai Thervugal**

<table>
<thead>
<tr>
<th>Cases No</th>
<th>Naadi</th>
<th>Sparisam</th>
<th>Naa</th>
<th>Niram</th>
<th>Mozhi</th>
<th>Vizhi</th>
<th>Molam</th>
<th>Moothiram</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>VK,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>2</td>
<td>VK,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>3</td>
<td>VP,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
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<td>N</td>
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</tr>
<tr>
<td>4</td>
<td>VP</td>
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<td>A</td>
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<td>A</td>
<td>N</td>
<td>N</td>
</tr>
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<td>5</td>
<td>VP</td>
<td>A</td>
<td>A</td>
<td>A</td>
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<tr>
<td>6</td>
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<td>NA</td>
<td>A</td>
<td>A</td>
<td>N</td>
</tr>
<tr>
<td>7</td>
<td>VK</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
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<td>N</td>
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<tr>
<td>8</td>
<td>VK</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>9</td>
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<td>VK,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
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<td>N</td>
</tr>
<tr>
<td>11</td>
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<td>A</td>
<td>NA</td>
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<tr>
<td>12</td>
<td>VK</td>
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<td>A</td>
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<tr>
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<td>VP,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>A</td>
<td>N</td>
</tr>
<tr>
<td>14</td>
<td>VK,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
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<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>16</td>
<td>VP,RP</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>NA</td>
<td>A</td>
<td>A</td>
<td>N</td>
</tr>
<tr>
<td>17</td>
<td>VK,RP</td>
<td>A</td>
<td>N</td>
<td>A</td>
<td>NA</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>18</td>
<td>VP,RP</td>
<td>A</td>
<td>N</td>
<td>A</td>
<td>NA</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>19</td>
<td>PV</td>
<td>A</td>
<td>N</td>
<td>A</td>
<td>NA</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>20</td>
<td>VK</td>
<td>A</td>
<td>N</td>
<td>A</td>
<td>NA</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

N  - Normal  
NA - Not Affected  
A  - Affected  
VP - Vadha Pitham  
PV - Pitha Vadham  
KV - Kabha Vadham  
RP - Raid Pulsation
## Clinical features

### Table 17: Clinical features

<table>
<thead>
<tr>
<th>S No.</th>
<th>Clinical Features</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Knee joint swelling, Pain</td>
<td>20</td>
</tr>
<tr>
<td>2.</td>
<td>Accumulation of blood in knee joint cavity</td>
<td>20</td>
</tr>
<tr>
<td>3.</td>
<td>Pain, tenderness</td>
<td>20</td>
</tr>
<tr>
<td>4.</td>
<td>Difficulty in movements (standing, walking)</td>
<td>15</td>
</tr>
<tr>
<td>5.</td>
<td>Rapid pulsation</td>
<td>13</td>
</tr>
</tbody>
</table>
## Factor assay-Report

### Table 18: Factor assay-Report

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age</th>
<th>Factor-Report</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>09</td>
<td>VIII- factor activity decrease (35%)</td>
</tr>
<tr>
<td>2</td>
<td>09</td>
<td>VIII- factor activity decrease (30%)</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
<td>VIII- factor activity decrease (40%)</td>
</tr>
<tr>
<td>4</td>
<td>12</td>
<td>VIII- factor activity decrease (18%)</td>
</tr>
<tr>
<td>5</td>
<td>11</td>
<td>VIII- factor activity decrease (20%)</td>
</tr>
<tr>
<td>6</td>
<td>10</td>
<td>VIII- factor activity decrease (25%)</td>
</tr>
<tr>
<td>7</td>
<td>17</td>
<td>VIII- factor activity decrease (20%)</td>
</tr>
<tr>
<td>8</td>
<td>46</td>
<td>VIII- factor activity decrease (22%)</td>
</tr>
<tr>
<td>9</td>
<td>09</td>
<td>VIII- factor activity decrease (30%)</td>
</tr>
<tr>
<td>10</td>
<td>23</td>
<td>VIII- factor activity decrease (20%)</td>
</tr>
<tr>
<td>11</td>
<td>16</td>
<td>VIII- factor activity decrease (25%)</td>
</tr>
<tr>
<td>12</td>
<td>30</td>
<td>VIII- factor activity decrease (20%)</td>
</tr>
<tr>
<td>13</td>
<td>32</td>
<td>VIII- factor activity decrease (10%)</td>
</tr>
<tr>
<td>14</td>
<td>18</td>
<td>VIII- factor activity decrease (16%)</td>
</tr>
<tr>
<td>15</td>
<td>24</td>
<td>VIII- factor activity decrease (19%)</td>
</tr>
<tr>
<td>16</td>
<td>58</td>
<td>No deficient of clotting factor (100%)</td>
</tr>
<tr>
<td>17</td>
<td>30</td>
<td>No deficient of clotting factor (100%)</td>
</tr>
<tr>
<td>18</td>
<td>14</td>
<td>No deficient of clotting factor (110%)</td>
</tr>
<tr>
<td>19</td>
<td>50</td>
<td>No deficient of clotting factor (100%)</td>
</tr>
<tr>
<td>20</td>
<td>56</td>
<td>No deficient of clotting factor (110%)</td>
</tr>
</tbody>
</table>

**Normal Value:** Factor activity = (70-120%)
Table 19: Laboratory Investigation

<table>
<thead>
<tr>
<th>Case No</th>
<th>BT</th>
<th>CT</th>
<th>PT</th>
<th>PTT</th>
<th>PT count</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2 min 30sec</td>
<td>12 min</td>
<td>12.5 sec</td>
<td>32 sec</td>
<td>1.2 lakes</td>
</tr>
<tr>
<td>2</td>
<td>2 min 30sec</td>
<td>14 min</td>
<td>12 sec</td>
<td>30 sec</td>
<td>1.6</td>
</tr>
<tr>
<td>3</td>
<td>3 min 30sec</td>
<td>11 min</td>
<td>15 sec</td>
<td>34 sec</td>
<td>1.4</td>
</tr>
<tr>
<td>4</td>
<td>2 min 30sec</td>
<td>8 min</td>
<td>14 sec</td>
<td>28 sec</td>
<td>1.2</td>
</tr>
<tr>
<td>5</td>
<td>2 min</td>
<td>14 min 30sec</td>
<td>12 sec</td>
<td>37 sec</td>
<td>1.5</td>
</tr>
<tr>
<td>6</td>
<td>2 min 60sec</td>
<td>13 min</td>
<td>15 sec</td>
<td>28 sec</td>
<td>1.8</td>
</tr>
<tr>
<td>7</td>
<td>6 min</td>
<td>14 min 30sec</td>
<td>12.3 sec</td>
<td>27 sec</td>
<td>1.8</td>
</tr>
<tr>
<td>8</td>
<td>3 min</td>
<td>14 min</td>
<td>11.4 sec</td>
<td>34 sec</td>
<td>1.8</td>
</tr>
<tr>
<td>9</td>
<td>5 min</td>
<td>13 min</td>
<td>12 sec</td>
<td>40 sec</td>
<td>1.6</td>
</tr>
<tr>
<td>10</td>
<td>5 min</td>
<td>13 min</td>
<td>11.3 sec</td>
<td>35 sec</td>
<td>1.5 lakes</td>
</tr>
<tr>
<td>11</td>
<td>2 min 30 sec</td>
<td>12 min</td>
<td>12 sec</td>
<td>32 sec</td>
<td>3.7</td>
</tr>
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<tr>
<td>13</td>
<td>2 min</td>
<td>14 min</td>
<td>15 sec</td>
<td>32 sec</td>
<td>1.6</td>
</tr>
<tr>
<td>14</td>
<td>2 min</td>
<td>12 min</td>
<td>12 sec</td>
<td>34 sec</td>
<td>2.1</td>
</tr>
<tr>
<td>15</td>
<td>3 min</td>
<td>12 min</td>
<td>14 sec</td>
<td>28 sec</td>
<td>1.8</td>
</tr>
<tr>
<td>16</td>
<td>2 min</td>
<td>4 min</td>
<td>12.5 sec</td>
<td>12 sec</td>
<td>1.8</td>
</tr>
<tr>
<td>17</td>
<td>3 min</td>
<td>4 min</td>
<td>13 sec</td>
<td>15 sec</td>
<td>1.4</td>
</tr>
<tr>
<td>18</td>
<td>2 min</td>
<td>4 min</td>
<td>12 sec</td>
<td>3413 sec</td>
<td>1.2</td>
</tr>
<tr>
<td>19</td>
<td>2 min</td>
<td>4 min</td>
<td>14 sec</td>
<td>14 sec</td>
<td>1.6</td>
</tr>
<tr>
<td>20</td>
<td>2 min</td>
<td>4 min 30sec</td>
<td>15 sec</td>
<td>12 sec</td>
<td>1.4</td>
</tr>
</tbody>
</table>

Normal Values

- Bleeding time: 2 - 4 min
- Clotting time: 3 - 6 min
- Prothrombin time: 0 - 27 sec
- Partial thermoplastic time: 0 - 25 sec
- Platelet count: 2 lakes
### Table 20: Lab report

<table>
<thead>
<tr>
<th>Case No</th>
<th>Blood</th>
<th>ERS</th>
<th>Bio Chemical</th>
<th>Urine</th>
<th>Motion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tc cells cumm</td>
<td>DC Cells</td>
<td>1/2 hr mm</td>
<td>1 hr mm</td>
<td>Hb</td>
</tr>
<tr>
<td>1</td>
<td>10000</td>
<td>60 39 1</td>
<td>6</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>9000</td>
<td>67 29 4</td>
<td>6</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>3</td>
<td>18000</td>
<td>47 50 3</td>
<td>20</td>
<td>42</td>
<td>10</td>
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<tr>
<td>4</td>
<td>18600</td>
<td>74 21 5</td>
<td>14</td>
<td>30</td>
<td>8.4</td>
</tr>
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<td>11000</td>
<td>79 23 2</td>
<td>20</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>11000</td>
<td>50 49 2</td>
<td>14</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>7</td>
<td>12000</td>
<td>49 48 4</td>
<td>18</td>
<td>20</td>
<td>9</td>
</tr>
<tr>
<td>8</td>
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<td>49 48 3</td>
<td>16</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>9</td>
<td>10000</td>
<td>50 48 2</td>
<td>14</td>
<td>16</td>
<td>9.6</td>
</tr>
<tr>
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<td>12000</td>
<td>50 48 2</td>
<td>16</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
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<tr>
<td>12</td>
<td>11000</td>
<td>50 48 2</td>
<td>16</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>13</td>
<td>10000</td>
<td>49 48 3</td>
<td>12</td>
<td>14</td>
<td>10</td>
</tr>
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<td>10800</td>
<td>49 48 3</td>
<td>16</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>15</td>
<td>13000</td>
<td>49 48 4</td>
<td>16</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>16</td>
<td>10000</td>
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<td>14</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>17</td>
<td>10000</td>
<td>50 48 2</td>
<td>4</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>18</td>
<td>1000</td>
<td>40 50 5</td>
<td>18</td>
<td>25</td>
<td>8.5</td>
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<td>19</td>
<td>11000</td>
<td>52 44 3</td>
<td>10</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>20</td>
<td>10000</td>
<td>42 47 4</td>
<td>12</td>
<td>13</td>
<td>13</td>
</tr>
</tbody>
</table>

**Normal Value:**
- Total count of WBC: -4000 - 11,000
- P %: 40 - 65%
- L %: 30 - 50%
- E %: 2 - 6%

**Hb-**
- Men: 13-17 gms%
- Women: 12.0 - 15.5 gms%

**ESR**
- ½ hour: 5-15 mm
- 1 hour: 5 – 15mm

**Blood Sugar**
- 80 - 140mg/dL

**Urea**
- 10-40mg/dL
STATISTICAL ANALYSIS OF NARITHALAIVADHAM

The Narithalaivatham study subjects were analyzed with statistics namely mean, D; and percentages. The inferences are made by the statistical test of student’s t’ test and the results are analysed by Relative Risk (RR), Attributable Risk (AR) and odds Ratio (OR).

Discussion and result

Age and etiology

The study subjects of Narithalaivadham are classified mainly into two categories namely

1. Hemophilia with Trauma
2. Trauma

The two groups were classified based on their age and analysed with following table.
Table 21: Age and etiology

<table>
<thead>
<tr>
<th>Age group</th>
<th>Haemophilia with trauma</th>
<th>Trauma</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-9</td>
<td>3</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>10-19</td>
<td>7</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>20-29</td>
<td>2</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>30-39</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>40-49</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>50-59</td>
<td>Nil</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>5</td>
<td>20</td>
</tr>
<tr>
<td>Mean</td>
<td>18.7</td>
<td>41.6</td>
<td>24.5</td>
</tr>
<tr>
<td>S.P</td>
<td>10.6</td>
<td>19.0</td>
<td>16.2</td>
</tr>
<tr>
<td>‘t’</td>
<td>3.415</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Significant</td>
<td>P&lt;0.01</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The mean age of haemophilia with trauma cases are 18.7 ± 10.6 and trauma cases are 41.6±19 the difference of mean age is statistically significant (t =3.415 and p <0.01d) That means, the incidence of Narithalaivadham among the haemophilia with trauma cases are quite increased than the trauma cases.
Family history

The family history ratio of haemophilia was analyzed and interpreted as follows.

Table 22: Family history

<table>
<thead>
<tr>
<th>Family history of haemophilia</th>
<th>Heamophilia</th>
<th>Trauma</th>
<th>Total</th>
<th>R.R</th>
<th>A.R</th>
<th>O.R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>11</td>
<td>0</td>
<td>11</td>
<td>2.1</td>
<td>52.4%</td>
<td>31.6</td>
</tr>
<tr>
<td>Negative</td>
<td>4</td>
<td>5</td>
<td>9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>5</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Relative Risk of family history in hemophilia cases are without family history is 2.1 times greater than the without family history. Attributable risk (AR) of the positive family history is 52.4%. Among the hemophilia cases the Odd Ratio of family history is 31.6 times greater than the Negative family history.
History of Trauma

The history of trauma risk ratio is analysed and interpreted in the following table.

Table 23: Traumatic history

<table>
<thead>
<tr>
<th>Traumatic History</th>
<th>Haemophilia</th>
<th>Trauma</th>
<th>Total</th>
<th>RR</th>
<th>A.R</th>
<th>O.R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>15</td>
<td>1</td>
<td>16</td>
<td>8.7</td>
<td>88.5%</td>
<td>93.0</td>
</tr>
<tr>
<td>Absent</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>8.7</td>
<td>88.5%</td>
<td>93.0</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>5</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

From the above table it may increase the risk of trauma presentation among the Haemophilia cases is 8.7 times greater than traumatic Narithalivadham cases. The presence of traumatic history is an odd among the Haemophilic Narithalaivadhan cases is 93 times greater than the traumatic Narithalivadham.
Mukkutra Nilai

The Narithalaivadham. Subjects were diagnosed through the mukkutranilai and its components are tabulated

**Table 24: Presentage distributes of mukkutranilai**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Component</th>
<th>Number</th>
<th>Types</th>
<th>Alerted cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>1.</td>
<td>Vali</td>
<td>20</td>
<td>Viyanan</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Samanan</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pranan</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Abanan</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Udanan</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Koorman</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Devathathan</td>
<td>5</td>
</tr>
<tr>
<td>2.</td>
<td>Azhal</td>
<td>20</td>
<td>Ranjagapitham</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sadhaga pitham</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Prasaga pitham</td>
<td>20</td>
</tr>
<tr>
<td>3.</td>
<td>Iyam</td>
<td>20</td>
<td>Avalambagam</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Santhigam</td>
<td>20</td>
</tr>
</tbody>
</table>

The above table shows that the viyanan, samanan, sadhaga pitham, prasaga pitham, Avalam bayam and santhigam and ant persent absorbed in the study subjecte. Koorman is absorbed only 10% of cases. Ranjaga pitham is absorbed in 80% of the study subjects. Pranan is absorbed in 65% of subjects. Abanan and Udanan are absorbed in 15% of subjects. Devathathan is absorbed in 25% of cases.
**Udalthathukkal**

The study subjects were analysed based on the observation of udal thethukkal and results are presented in the table.

**Table 25: Observation of udal thathukkal**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Udal thathukkal</th>
<th>N</th>
<th>Affected cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>1.</td>
<td>Saaram</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>2.</td>
<td>Senneer</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>3.</td>
<td>Oon</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>4.</td>
<td>Kozhuppu</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>5.</td>
<td>Enbu</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>6.</td>
<td>Moolai</td>
<td>20</td>
<td>3</td>
</tr>
</tbody>
</table>

From the above table the Udalthathukkal namely Saaram, Senneer, Oon, Kozhuppu and Enbu are observed in cent present of the study subjects. Only 15% of the cases and exposed to moolai.

**Mani kudai Nool**

The cases were measured based on the viral kadaialavu and they were analyzed and presented in the table.

**Table 26: Mani kudai Nool**

<table>
<thead>
<tr>
<th>Viral kadaialavu</th>
<th>10</th>
<th>10 ⅓</th>
<th>10 ⅔</th>
<th>Total</th>
<th>Mean</th>
<th>S.P</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of cas</td>
<td>2</td>
<td>10</td>
<td>8</td>
<td>20</td>
<td>10.325</td>
<td>10.25</td>
<td>9.9 To 10.7</td>
</tr>
</tbody>
</table>
From the above analysis it may be listed that the mean viralkadaialavu of the Narithalai vadham cases in the population. 10.248 to 10.402. That means the meaned median will be 10.25 of viral kadaialavu.

**Envagai thervugal**

The rule of eight is siddha medicine selecting and diagnosing the diseased persons are tabulated and the results are analysed in the below mentioned table.

**Table 27: Envagai thervugal**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Envagai thervuga</th>
<th>N</th>
<th>Type</th>
<th>Cases affected</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>1</td>
<td>Naadi</td>
<td>20</td>
<td>Vali Iyam</td>
<td>10, 50</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Azhal Vali</td>
<td>1, 5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Vali Azhal</td>
<td>9, 45</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Rapid pulsation</td>
<td>13, 65</td>
</tr>
<tr>
<td>2</td>
<td>Sparisam</td>
<td>20</td>
<td>-</td>
<td>20, 100</td>
</tr>
<tr>
<td>3</td>
<td>Naa</td>
<td>20</td>
<td>-</td>
<td>16, 86</td>
</tr>
<tr>
<td>4</td>
<td>Niram</td>
<td>20</td>
<td>-</td>
<td>20, 100</td>
</tr>
<tr>
<td>5</td>
<td>Mozhi</td>
<td>20</td>
<td>-</td>
<td>0, 0</td>
</tr>
<tr>
<td>6</td>
<td>Vizhi</td>
<td>20</td>
<td>-</td>
<td>16, 80</td>
</tr>
<tr>
<td>7</td>
<td>Malam</td>
<td>20</td>
<td>-</td>
<td>3, 15</td>
</tr>
<tr>
<td>8</td>
<td>Moothiram</td>
<td>20</td>
<td>-</td>
<td>0, 0</td>
</tr>
</tbody>
</table>
Clinical Features

Table 28: Clinical Features

<table>
<thead>
<tr>
<th>S.No</th>
<th>Clinical features</th>
<th>Cases</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>1</td>
<td>Knee joint swelling, pain</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>2</td>
<td>Accumulates of blood in knee joint cavity</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>3</td>
<td>Pain, tenderness</td>
<td>20</td>
<td>100</td>
</tr>
<tr>
<td>4</td>
<td>Difficulty in movements</td>
<td>15</td>
<td>75</td>
</tr>
<tr>
<td>5</td>
<td>Rapid pulsation</td>
<td>13</td>
<td>65</td>
</tr>
</tbody>
</table>

Cent percent of subjects is having swelling, pain and blood accumulation. 75% of subjects having difficulty in movements. 65% of subjects having Rapid pulsation.
Laboratory Investigations

The lab investigations are compared to Narithalaivadham cases of Haemophilia and Trauma cases.

Table 29: Lab investigations results.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name are Investigations</th>
<th>Haemophilia</th>
<th>‘t’</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>N</td>
<td>Mean</td>
<td>S.p</td>
</tr>
<tr>
<td>1</td>
<td>Clotting time</td>
<td>15</td>
<td>12.47</td>
<td>1.63</td>
</tr>
<tr>
<td>2</td>
<td>Partial thromboplastin time (PPT)</td>
<td>15</td>
<td>32.1</td>
<td>3.69</td>
</tr>
<tr>
<td>3</td>
<td>D.Cout (P %)</td>
<td>15</td>
<td>54.73</td>
<td>10.29</td>
</tr>
<tr>
<td>4</td>
<td>E.S.R. 1 hour (mm)</td>
<td>15</td>
<td>20.47</td>
<td>11.68</td>
</tr>
<tr>
<td>5</td>
<td>Hb /gm</td>
<td>15</td>
<td>9.353</td>
<td>.752</td>
</tr>
</tbody>
</table>

The above table evaluate the data reports interpret of the study subject’s physiological measures.

The clotting time of Haemophilia with trauma cases are recorded the mean clotting time of Haemophilia trauma cases is longest than the trauma cases. The deferens statistically signifient (pl 0.00001) the other measures like PPT and Hb are statistically highly significant (pl 0.001). there is no significant measure is are observed in Dc count (P%) and E.S.R/ 1 hr (P>.05).
DISCUSSION

Saint Yugi has classified diseases into two types they are,

- Functional disorder
- Organic invasions.

The functional units of our body are the three vital forces, which are Vali, Azhal and Iyam. Any disturbance in the vital Humour will affect the function of the organ. In chronic condition, it may lead to pathological changes in the affected organ.

Vali is the initiator of all activities of our body. It is important in the connecting network of the body from sense organ to brain and tissue to tissue and even cell to cell.

The clinical studies on all selected cases were undergone investigation by both Siddha as well as modern allied parameters.

INTERPRETATION OF CLINICAL HISTORY

1. Family history

Most of the cases explained a positive family history of the disease.

2. Age group

No specific age group is mentioned in this disease.
3. Sex

Mostly male is affected because of this disease is also related with X-linked recessive disorder.

3. Occupation

Players are majority affected due to sports injury.

4. Clinical features

All the patients depicted the clinical features mentioned in the poem “Narithalaivadham” in the text book of “Yugi Vaithya Chinthamani”.

5. History of previous illness

11 out of 20 Narithalaivadham patients had past history of same symptoms.

INTERPRETATION OF SIDDHA PARAMETERS

3. Interpretation of Envagai thervugal

1. Naadi

In naadi diagnosis of the all patients, the observed naadi is ValiYam, Vali Azhal, and Azhal Vali.

2. Sparisam

Swelling tenderness and warmth is present in knee joint.

3. Naa

Many patients having palorness of tongue in examination
4. Niram

On observations the body colour of all the patients are normal. But the affected area (knee joint) is swollen and reddish black is colour.

5. Mozhi

All patients were having normal speech. No alteration in mozhi.

6. Vizhi

Most of the patients having paleness of conjunctiva in examination

7. Moothiram

<table>
<thead>
<tr>
<th>Niram</th>
<th>Normal straw colour in all patients.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manam</td>
<td>No abnormal odor.</td>
</tr>
<tr>
<td>Enjal</td>
<td>No deposition.</td>
</tr>
</tbody>
</table>

Neikuri

Most of the patients Neikuri exhibited as the oil spreads medium speed and look like round shape with some sieve hole [சிவப்பு கூர்கு]. In some patients the oil spreads with low speed and look like round shape only.
II. Interpretation of Mukkutra Nilaikkal

Vali

1. Pranan
   Most of the patients are having rapid pulsations.

2. Abanan
   Some of the patients are having constipation.

3. Udanan
   Most of the patients are having colour changes

4. Viyanan
   All the patients were having difficulty in movements (Flexion and extension) due to swelling.

5. Samanan
   Its balancing function is affected.

6. Koorman
   Some of the patients were having visual disturbances.

7. Devathathan
   Some of the patients are irritable
Azhal

1. Ranjaga pitham

Some of the patients were affected. They are having paleness of tongue and conjunctiva.

2. Sadhaga pitham

All the patients were having difficulty in doing routine works.

3. Prasagapitham

All the patients were having colour changes (Reddish) in the skin at the site of swelling (Knee joint)

Iyam

1. Avalampagam

All the patients were affected, it’s balancing function disturbed.

2. Santhigam

All the patients were having difficulty in movements of joints.

Inference

Vali - Pranan, Abanan, Udanan, Koorman, Viyanan, Samanan and Devathan are affected.

Azhal - Ranjaga pitham, Sathaga pitham, Prasaga pitham are affected.

Iyam - Avalambagam and Santhigam are affected.
III. Interpretation in Udal thathukkal

1. Saaram
   
   All patients were having pain and discomfort.

2. Senner
   
   All patients were having stagnation of blood in the site of swelling (Knee Joint)

3. Oon
   
   All patients were having
   
   Initial stage - Some inflammatory changes (Swelling)
   Later stage - Disused atrophy

4. Kozhuppu
   
   All patients were having restricted joint movements

5. Enbu
   
   All patients were having difficulty to move, standing upright position, inflammatory changes and intra articular adhesions in the knee joint.

6. Moolai
   
   Some of the patients were having osteoporotic changes in later stage.

Inference

All 6 Udal Thathukkal are affected except Sukkila/ Sronitham.
Interpretation of allied parameters

Suspected cases were subjected to screening test of Haematology.

Total Count WBC - Normal.
Total Count RBC - Normal.
Differential Count for WBC - Normal, some times lymphocytes

Monocytes were increased.

Hb - Most of patients become anemic

due to chronic haemorrhage.

Bleeding time - Only 2 patients were affected.
Clotting time - 15 patients out of 20 cases are

affected.

Prothrombin time - Normal
Partial thromboplastin time - 15 out of 20 patients were affected.

Inference

Most of the patients have prolonged thromboplastin time, clotting
time and anemia.

Factor assay

Factor VIII decreased in 15 cases out of 20.

Inference

Most of the patients were having decreased activity of factor VIII

X - Ray

5 out of 20 patients having ligament injury, patella fracture and
tibial condyle fracture.
HIGH LIGHTS OF THE DISSERTATION TOPIC

The disease is characterized by the swelling in the knee joint is due to prolonged accumulation of blood in the knee joint cavity.

Patients were having complaints of not able to stand in upright position and difficulty in movements of joint. It is due to damaging of articular cartilage and narrowing of joint cavity. Articular cartilage damage is caused by inflammatory response of blood, in knee joint cavity.

Further inflammatory process causes massive swelling like (Jackal’s head like) and deformity.

Few of them, shows the symptom like Rapid pulsation.

All of these correlate with “Narithalaivadham” explained by our great siddhar Yugimuni.
Identification of disease and its pathogenesis are pre requisite for medical practice. A detailed history taking, clinical examinations as per siddha guidelines are necessary to arrive at precise diagnosis.

The study on Narithalivadham was carried out in the dissertation, giving importance to the characteristics of the disease like accumulation blood in knee joint cavity, hacking massive tender swelling, difficulty in movements, standing and rapid pulsation.

Diagnosis can be carried out by detailed history taking, classical clinical examination of siddha system, Via, Envagithervugal Including Neerkuri, Neikuri, Manikkadai nool and changes in seven physical constituents and three Humours.

This study on Narithalaivadham may be correlates with haemarthrosis, which has given relevance to modern clinical entity.
A Study to Diagnose “Narithalaivadham” through Siddha

Diagnostic Methodology

SELECTION PROFORMA


6. Name: ________________ 7. Age (Years): [ ] 8. Sex: [M] [F]


11. Address: …………………………………………………………

…………………………………………………………

………………………………………………………………

12. Complaints and duration:

………………………………………………………………………………

………………………………………………………………………………

………………………………………………………………………………

13. History of present illness:

………………………………………………………………………………

………………………………………………………………………………

………………………………………………………………………………

14. Past history:

………………………………………………………………………………

………………………………………………………………………………

………………………………………………………………………………

15. Family History:

………………………………………………………………………………

………………………………………………………………………………

………………………………………………………………………………
16. **Habits**

1. Betelnut chewer: [ ] Yes [ ] No
2. Tea: [ ] Yes [ ] No
3. Coffee: [ ] Yes [ ] No
4. Alcohol: [ ] Yes [ ] No
5. Exercise: [ ] Yes [ ] No
6. Yoga: [ ] Yes [ ] No

7. Food habits: V □ NV □ M □

---

17. **GENERAL ETIOLOGY FOR NARITHALAI VATHAM**

1. Haemophilia: [ ] Yes [ ] No
2. Trauma: [ ] Yes [ ] No
3. Rheumatoid arthritis: [ ] Yes [ ] No
4. In square playing: [ ] Yes [ ] No
5. Heavy weight lifting: [ ] Yes [ ] No
6. Improper yoga and exercise: [ ] Yes [ ] No
7. Obesity: [ ] Yes [ ] No
8. Ligament injury: [ ] Yes [ ] No
9. Starvation: [ ] Yes [ ] No
10. Sleeping in daytime: [ ] Yes [ ] No
11. Awakening in night time: [ ] Yes [ ] No

---

18. **GENERAL EXAMINATION**

1. Weight (kg): [ ]
2. Temperature (°F): [ ]
3. Pulse rate/minute: [ ]
4. Heart rate/minute: [ ]
<table>
<thead>
<tr>
<th></th>
<th>1. Yes</th>
<th>2. No</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Pallor</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>8. Jaundice</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>9. Cyanosis</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>10. Lymphadenopathy</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>11. Pedal edema</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>12. Clubbing</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>13. Jugular venous pulsation</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

19. VITAL ORGANS EXAMINATION

<table>
<thead>
<tr>
<th></th>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Heart</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>2. Lungs</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>3. Brain</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>4. Liver</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>5. Kidney</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>6. Spleen</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>7. Stomach</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>
20. NAA
1. Maa Padinthiruthal
   a. Present  
   b. Absent  
2. Niram
   a. Karuppu  
   b. Manjal  
   c. Velluppu  
3. Suvai
   a. Pulippu  
   b. Kaippu  
   c. Inippu  
4. Vedippu
   a. Absent  
   b. Present  
5. Vai neer ooral
   a. Normal  
   b. Increased  
   c. Reduced  

21. NIRAM
   a. Karuppu  
   b. Manjal  
   c. Velluppu  

22. MOZHI
   a. Sama oli  
   b. Urattha oli  
   c. Thazhlntha oli  

23. VIZHI
1. Niram
   a. Karuppu  
   b. Manjal  
   c. Sivappu  
   d. Velluppu  
2. Kanneer
   a. Present  
   b. Absent  
3. Erichchal
   a. Present  
   b. Absent  
4. Peelai seruthal
   a. Present  
   b. Absent  

112
24. MEI KURI

1. Veppam
   a. Mitham   b. Migu   c. Thatpam

2. Viyarvai
   a. Normal   b. Increased   c. Reduced

3. Thodu vali
   a. Absent   b. Present

25. MALAM

1. Niram
   a. Karuppu   b. Manjal
   c. Sivappu   d. Velluppu

2. Sikkal
   a. Present   b. Absent

3. Sirutthal
   a. Present   b. Absent

4. Kalichchal
   a. Present   b. Absent

5. Seetham
   a. Present   b. Absent

6. Vemmai
   a. Present   b. Absent
26. MOOTHIRAM

A. NEER KURI

1. Niram
   a. Venmai  □  b. Manjal  □  c. Crystal clear  □

2. Manam
   a. Present  □  b. Absent  □

3. Nurai
   a. Nill  □  b. Increased  □  c. Reduced  □

4. Edai (Ganam)
   a. Normal  □  b. Increased  □  c. Reduced  □

5. Enjal (Alavu)
   a. Normal  □  b. Increased  □  c. Reduced  □

B. NEI KURI

1. Aravam  □  2. Mothiram  □

3. Muthu  □  4. Aravil Mothiram  □

5. Aravil Muthu  □  6. Mothirathil Aravam  □

7. Mothirathil Muthu  □  8. Muthil Aravam  □

9. Muthil Mothiram  □  10. Asathiyam  □

11. Mellena paraval  □

27. NAADI (KAI KURI)

A. Naadi Nithanam
a. Kalam

1. Kaarkaalam  □  2. Koothirkaalam  □

3. Munpanikaalam  □  4. Pinpanikaalam  □

5. Ilavenirkaalam  □  6. Muthuvenirkaalam  □
b. Desam

1. Kulir
2. Veppam
3. Vayathu

1. 1-33yrs
2. 34-66yrs
3. 67-100yrs

D. Udal Vanmai

1. Iyyalbu
2. Valivu
3. Melivu

e. Vanmai

1. Vanmai
2. Menmai

f. Panbu

1. Thannadai
2. Puranadai
3. Illaittha
4. Kathithal
5. Kuthithal
6. Thullal
7. Azhutthal
8. Padutthal
9. Kalatthal
10. Munnookku
11. Pinnokku
12. Suzhalal
13. Pakkanokku

B. Naadi nadai

1. Vali
2. Azhal
3. Iyyam
4. Vali Azhal
5. Vali Iyyam
6. Azhal Vali
7. Azhal Iyyam
8. Iyyavali
9. Iyya Azhal

28. MANIKADAI NOOL (Viral Kadai Alavu)

29. IYMPORIGAL / IYMPULANGAL

1. Normal
2. Affected

1. Mei
2. Vaai
3. Kan
4. Mookku
5. Sevi
### 30. KANMENTHIRIYANGAL / KANMAVIDAYANGAL

<table>
<thead>
<tr>
<th></th>
<th>1. Normal</th>
<th>2. Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Kai</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Kaal</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Vaai</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Eruvai</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Karuvaai</td>
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</table>

### 31. YAAKAI

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>2. Azhal</th>
<th>3. Iyam</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Vali</td>
<td></td>
<td>Azhal</td>
<td>Iyam</td>
</tr>
<tr>
<td>4. Vali Azhal</td>
<td></td>
<td>Vali Iyam</td>
<td>Azhal Vali</td>
</tr>
<tr>
<td>7 Azhal. Iyam</td>
<td></td>
<td>Iyavali</td>
<td>Iya Azhal</td>
</tr>
</tbody>
</table>

### 32. GUNAM

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>2. Rajso Gunam</th>
<th>3. Thamo Gunam</th>
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</thead>
<tbody>
<tr>
<td>1. Sathuva Gunam</td>
<td></td>
<td>Rajso Gunam</td>
<td>Thamo Gunam</td>
</tr>
</tbody>
</table>

### 33. UYIR THATHUKKAL

#### A. Vali

<table>
<thead>
<tr>
<th></th>
<th>1. Normal</th>
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</thead>
<tbody>
<tr>
<td>1.</td>
<td>Uyirkkaal (Praanan)</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Kelnokkukaal (.Abaanan)</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Nadukkaal (Samaanan)</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Melnakukkal (Udhaanan)</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Paravukkaal(Viyaanan)</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Vaanthikkal (Naahan)</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Vizhikkaal (.Koorman)</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Thummikaal (.Kirukaran)</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Kottavikkaal (Devathathan)</td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>Veeingukkaal (.Dhananjeyan)</td>
<td></td>
</tr>
<tr>
<td>B. Azhal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------</td>
<td>------------------</td>
<td>------------------</td>
</tr>
<tr>
<td>1. Normal</td>
<td>1Aakkannal (.Anala pitham)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 Olloliththee (Prasaka pitham)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 Vannayeri (Ranjaka pitham)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4 Nokku Azhal (Aalosaka pitham)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 Aatralangi (Saathaka pitham)</td>
<td></td>
</tr>
<tr>
<td>2. Affected</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C. Iyam</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Normal</td>
<td>1. Alilyam (Avalambagam)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2. Neerppi Iyam (Kilethagam)</td>
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</tr>
<tr>
<td></td>
<td>3. Suvaikaan Iyam (Pothagam)</td>
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</tr>
<tr>
<td></td>
<td>4. Niraivu Iyam (Tharpagam)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5. Ondri Iyam (Santhigam)</td>
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<td>2. Affected</td>
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<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>34. Udal Thathukkal</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>1. Normal</td>
<td>1. Saaram</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2. Shenneer</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3. Oon</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4. Kozhuppu</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5. Enbu</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6. Moolai</td>
<td></td>
</tr>
<tr>
<td></td>
<td>7. Suronitham / Sukkilam</td>
<td></td>
</tr>
<tr>
<td>2. Affected</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 35. Mukkutra Migu Gunam

#### I. Vali Migu Gunam

<table>
<thead>
<tr>
<th>Condition</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emaciation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Blackish discolouration of body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Desire to take hot food</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Shivering of body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Abdominal distension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Constipation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Weakness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Weakness of sense organs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Giddiness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Sluggishness</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### II. Azhal Migu Gunam

<table>
<thead>
<tr>
<th>Condition</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Yellowish discolouration of the skin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Yellowish discolouration of the eye</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Yellowish discolouration of urine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Yellowish discolouration of faeces</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Increased appetite</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Burning sensation in the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Insomnia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### III. Iyyam Migu Gunam

<table>
<thead>
<tr>
<th>Condition</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Excessive salivation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Reduced activeness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Heaviness of the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Whitish discoloration of the body</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
5. Chillness of the body
6. Reduced appetite
7. Cough
8. Increased sleep
9. Sluggishness

36. NOI UTRA KAALAM
   1. Kaarkaalam
   2. Koothirkaalam
   3. Munpanikaalam
   4. Pinpanikaalam
   5. Ilavenirkaalam
   6. Muthuvenirkaalam

37. NOI UTRA NILAM
   1. Kurinji
   2. Mullai
   3. Marutham
   4. Neithal
   5. Palai

38. Date of Birth
39. Time of Birth
40. Place of Birth

41. NATCHATHIRAM
   1. Aswini
   2. Barani
   3. Karthikai
   4. Rohini
   5. Mirugaseeridam
   6. Thiruvathirai
   7. Punarpoosam
   8. Poosam
   9. Aayilyam
   10. Makam
   11. Pooram
   12. Uttiram
   13. Astham
   14. Chithirai
   15. Swathi
   16. Visakam
   17. Anusam
   18. Kettai
   19. Moolam
   20. Pooradam
   21. Utthiradam
   22. Thiruvonam
   23. Avittam
   24. Sadayam
   25. Poorattathi
   26. Utthirattathi
   27. Revathi
   00. Not known
### 42. RASI

1. Mesam  
2. Rishabam  
3. Midhunam  
4. Kadakam  
5. Simmam  
6. Kanni  
7. Thulam  
8. Viruchiham  
9. Dhanusu  
10. Maharam  
11. Kumbam  
12. Meenam  
00. Not known

### 43. JOINT EXAMINATION

1. **Size**
   
<table>
<thead>
<tr>
<th></th>
<th>Rt.</th>
<th>Lt.</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Upper</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Middle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c. Lower</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. **Shape**
   
<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Rt</td>
<td>....................</td>
</tr>
<tr>
<td>Lt</td>
<td>....................</td>
</tr>
</tbody>
</table>

3. **Movements**
   
<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Flexion</td>
<td>.....................</td>
</tr>
<tr>
<td>b. Extension</td>
<td>.....................</td>
</tr>
<tr>
<td>c. Medial rotation</td>
<td>.....................</td>
</tr>
<tr>
<td>d. Lateral rotation</td>
<td>.....................</td>
</tr>
</tbody>
</table>
### 44. INVESTIGATION

#### A. BLOOD

1. **TC (Cells/cumm)**:  

2. **DC (%):**
   - 1.P
   - 2.L
   - 3.E
   - 4.B
   - 5.M

3. **Hb (gms %)**:  

4. **E.S.R. (mm/hr):**  
   - 1.1/2h
   - 2.1hr
   - 3.Blood

5. **Sugar (R) (mgs%)**:  

6. **Bleeding time**:  

7. **Cloting time**:  

8. **Prothrombin time**:  

9. **Clotting factors**:  

10. **RA factor**:  

#### B. URINE

1. **Albumin**:  
   - 0.Nil
   - 1.Trace
   - 2.+
   - 3.++
   - 4.+++  

2. **Sugar**:  
   - 0.Nil
   - 1.Trace
   - 2.+
   - 3.++
   - 4.+++  

3. **Deposits**:  
   - 1. Yes
   - 2. No
     - A. Pus cells
     - B. Epithelial cells
     - C. RBCs
     - D. Crystals  

#### C. MOTION TEST

1. **Ova**

2. **Cyst**

3. **Occult Blood**
45. X-Ray-knee joint

46. Doppler study

47. CT scan-knee joint

48. CLINICAL SYMPTOMS OF NARITHALAI VATHAM.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Massive swelling in knee joint (like Jackal’s head)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Accumulation of blood in knee joint cavity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Not able to stand in upright position</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Difficulty in movements of joints</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Rapid pulsation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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- Theerayar Vaagadam
- Madurai Tamil Agarathi.
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